Canadian Sleep Society | Société Canadienne du Sommeil

The 4th Conference of the Canadian Sleep Society

April 26-28, 2009

Toronto, Marriott Downtown Eaton Centre Hotel

For more information please visit
www.css-meeting.ca

LIST OF PRESENTING AUTHORS

Ahmadi, Negar	021
Amini, Reza	038
Arnedt, Todd	010
Baltzan, Marc	071
Barakat, Marc	011
Bauermann, Tonya	078, 079
Beaulieu-Bonneau, Simon	007, 018
Belanger, Lynda	032, 033
Black, Josh	009
Boudreau, Philippe	040, 041
Brooks, Patti	053
Burgess, Christian	054
Capua, Maya	066
Caron, Aimee M.	005
Chapotot, Florian	077
Chaput, Geneviève	019
Currie/Mongrain, Valerie	049
Davidson, Fiona	044
Dostie, Valerie	008
Driver, Helen	069
DuBord, Marq-Andre	058, 059
Dunai, Andrea	067
Fogel, Stuart	051
Fordyce, Laree	075
Fortier-Brochu, Emilie	025
Fradette, Lorraine	030
Ghashghai, Arash	065
Grace, Kevin	056
Grundy, Anne	043
Huynh, Christophe	016
Jarrin, Denise	006
Kertesz, Rona	029
Lafortune, Marjolaine	003
LeBlanc, Melenie	023
Madjou, Thierno	052
Madore, Alex	013
Martin, Nicolas	001
Martin, Jeanne-Sophie	042
McInrue, Erin	080
Milner, Catherine	020
Mink, Joe	076
Mongrain, Valerie	050
Montandon, Gaspard	060
Moreau, Vincent	031
Nesbitt, Darren	012
Nielsen, Tore	036
Novak, Markta	068
Omisade, Antonina	004
Ouellet, Daniel	037
Perozzo, Cristina	026
Ray, Laura	081
Rizzo, Dorrie	073
Rochette, Annie-Claude	015
Saleh, Philip	022
Saletin, Jared	063
Samuels, Charles	039, 072
Sanhez, Rafael Ochoa	045
Sasseville, Alexandre	046
Schwarz, Peter	055
Shechter, Ari	047, 048
Shen, Jianhua	024
Solomonova, Elizaveta	035
Spurr, Kathy	070
Stenstrom, Philippe	034
St-Jean, Geneviève	027
Stremler, Robyn	002
Sullivan, Glendon E.	074
Tadjalli, Arash	061
Tessier, Sophie	014
Turcotte, Isabelle	028
Vecchio, Laura Marie	057
Viens, Isabelle	062
Wade, Terry	064
Wiseman-Hakes, Catherine	017

LIST OF POSTERS & AUTHORS

Poster Categories
Monday, April 27, 2009
Sleep Deprivation
Sleep & Performance
Neuropsychology & Psychology
Insomnia
Dreaming

Poster Categories
Tuesday, April 28, 2009
Chronobiology
Physiology & Neurophysiology
Respiratory – Movement-Related Sleep Disorders
Technology & Procedures

POSTER SESSION A: MONDAY, APRIL 27, 2009

SLEEP DEPRIVATION

P001 EFFECTS OF SLEEP DEPRIVATION AND DAYTIME RECOVERY SLEEP ON SELF-EVALUATION ACCURACY
Martin N.[1,2], Dostie V. [1,2] and Carrier J. [1,2]

P002 SLEEP DURING PEDIATRIC HOSPITALIZATION
Stremler, R[1, 2], Weston, J[1], Dhukai, Z[1], Lumb, A[1], Wong, L[1], Adams, S[2], Weiss, S[2], Parshuram, C[2]

P003 AGE-RELATED DIFFERENCES IN N-REM SLOW OSCILLATIONS: EFFECTS OF SLEEP DEPRIVATION
Lafortune, M.[1,2], Viens, I.[1,2], Jacob-Lessard, A.[1,2], Poirier, G.[1,2], Vandewalle, G.[1,2], Barakat, M.[1,2], Martin, N.[1,2], Filipini, D.[1], & Carrier J.[1,2]

P004 IMPACT OF ACUTE SLEEP RESTRICTION ON CORTISOL AND LEPTIN LEVELS IN YOUNG WOMEN.
Omisade A.[1], Buxton M. O.[2] and Rusak B.[1,3,4]

P005 SHORT DAILY SLEEP OPPORTUNITIES AMELIORATE THE EFFECTS OF SLEEP LOSS ON ENERGY EXPENDITURE IN RATS
Caron A.M. and Stephenson R.

SLEEP & PERFORMANCE

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

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

P008 DO DIFFERENT LEVELS OF SLEEPINESS MODULATE EFFECTS OF CAFFEINE ON VIGILANCE?
Dostie, V[123]., Robillard, R.[123], Filipini, D[3]., Selmaoui, B.[4] and Carrier, J.[123]

P009 BENEFITS OF A DAYTIME NAP FOR STUDENTS ON A UNIVERSITY CAMPUS
Black J., Ling E., and Cote K.

P010 SLEEP QUALITY AND NEXT-DAY PVT PERFORMANCE AFTER HEAVY ALCOHOL CONSUMPTION
Arnedt J.T.[1], Almeida A.[2], Hunt S.[2], Gokhale M.[3], Rohsenow D.[4], & Howland J.[2]

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

P012 POST-LEARNING REDUCTION IN SLOW-WAVE SLEEP AFTER MENTAL PRACTICE OF THE ROTARY PURSUIT TASK
Nesbitt D., Peters K.R., DeCicco T. and Smith C.T.

P013 IDENTIFICATION OF A RAPID EYE MOVEMENT SLEEP WINDOW FOR THE WIN-SHIFT RADIAL ARM MAZE TASK
Madore A.R.[1], DeLay S.[1], Legault M.G.[1]

NEUROPSYCHOLOGY & PSYCHIATRY

P014 SLEEP EEG IN AUTISM AND PERFORMANCE ON THE EMBEDDED FIGURE TEST
Sophie Tessier,[1,4] Christianne Bolduc,[1,4] Élyse Limoges,[1,4] Édith Ménard,[4] Laurent Mottron,[2,3,4] Roger Godbout.[1,2,3,4]

P015 DISSOCIATION BETWEEN STAGE 2 SIGMA EEG ACTIVITY AND SLEEP SPINDLE DENSITY IN HIGH-FUNCTIONING AUTISM
Annie-Claude Rochette,[1,4] Élyse Limoges[1,4], Élyse Chevrier[1,3,4], Laurent Mottron,[2,3,4] Roger Godbout.[1,2,3,4]

P016 SLEEP PATTERNS IN ADOLESCENTS WITH BIPOLAR DISORDER OR BORDERLINE PERSONALITY DISORDER
Huynh C.[3], Guilé J.M.[1,3], Breton J.J.[1,3], Cohen D.[2], Chevrier É.[4] and Godbout R.[1,4]

P017 SLEEP/WAKE DISTURBANCE FOLLOWING SEVERE TRAUMATIC BRAIN INJURY; IMPACT ON RECOVERY OF COGNITIVE-COMMUNICATION FUNCTION: A CASE STUDY
Wiseman-Hakes, C.[1,2] , Murray, BJ, [2,3,4], Victor, JC[5]

P018 DAYTIME SLEEPINESS AFTER MODERATE/SEVERE TRAUMATIC BRAIN INJURY: PRELIMINARY FINDINGS
Beaulieu-Bonneau S., Roy M.-A., and Morin C. M.

P019 RELATIONSHIP AMONG SUBJECTIVE SLEEP COMPLAINTS, HEADACHES, AND MOOD ALTERATIONS FOLLOWING A MILD TRAUMATIC BRAIN INJURY
Chaput, G.[1, 2], Giguère, J-F. [2], Chauny, J-M.[2] , Denis, R.[2] , Lavigne, G.[1, 2]

P020 SPONTANEOUS K-COMPLEXES IN STAGE 2 SLEEP ARE REDUCED FOLLOWING TRAUMATIC BRAIN INJURY (TBI)
Milner C.E. and Cote K.A.

P021 INVESTIGATION OF SLEEP IN CHRONIC TREATMENT-RESISTANT DEPRESSED PATIENTS
Ahmadi N, Shapiro CM

P022 A PRELIMINARY ATTEMPT AT DEFINING 'SLEEP MARKERS OF DEPRESSION' CATEGORICALLY AND EXAMINING THEIR ASSOCIATION WITH SUBJECTIVE LOW MOOD.
Saleh, P[1, 2, 3], Shahid, A[1, 2], Chung, F[4] and Shapiro, CM[1, 2, 3]

INSOMNIA

P023 EPIDEMIOLOGY OF INSOMNIA IN A CANADIAN POPULATION-BASED SAMPLE
LeBlanc, M.[1,2], Bélanger, L. [1,2], Mérette, C. [2,3], Savard J. [1,4], Morin, C.M. [1,2]

P024 SLEEP DISTURBANCES IN CHINESE EARTHQUAKE VICTIMS
Shen J.[1], Wang L.[2], Shi Z.[2], Zhang Y.[2], Xin Y.[3], Wang W.[2], Shan S.[4], Zheng S.[5], Shapiro C.M.[1]

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

P026 INSOMNIA AS A RISK FACTOR FOR HEALTH PROBLEMS: A LONGITUDINAL STUDY
Perozzo, C.[1, 2], Gagnon, C.[1, 2], LeBlanc, M.[1, 2], Savard, J.[1, 3], & Morin, C. M.[1, 2]

P027 CLASSIFICATION OF INSOMNIA SUFFERERS BASED ON LABORATORY PSG RECORDINGS AND SUBJECTIVE SLEEP REPORTS
St-Jean G.[1-2] and Bastien C.H. [1-2]

P028 ERP MEASURES DURING SLEEP IN PSYCHOPHYSIOLOGICAL AND PARADOXICAL INSOMNIA SUFFERERS
Turcotte I.[1], Adam A-M.[1], Lecarpentier M.[1] and Bastien C.[1]

P029 EVENT-RELATED POTENTIALS (ERPs) REVEAL FAILURE TO INHIBIT STIMULI DURING THE PRE-SLEEP WAKING PERIOD FOR PATIENTS WITH SLEEP-ONSET INSOMNIA
Kertesz R. S., Cote K. A.

P030 BAROREFLEX SENSITIVITY DURING SLEEP AND WAKEFULNESS IN PRIMARY INSOMNIA : PRELIMINARY RESULTS
Authors: Lorraine Fradette, BSc[1,2], Pennestri Marie-Hélène, BSc[1], Jacques Montplaisir, MD, PhD[2,3], Charles M. Morin, PhD[4,5], Roberto Colombo BEng[6], Paola A.Lanfranchi, MD, MSc[2,7]

P031 DOES CBT FOR INSOMNIA ALTER SLEEP MISPERCEPTIONS?
Moreau V., Gagnon C., Lamy M., Ivers H. and Morin C. M.

P032 IMPACT OF CBT FOR INSOMNIA AND CBT COMBINED WITH MEDICATIONS ON DAYTIME FUNCTIONING
Bélanger, L.[1], Sanchez-Ortuño, M.[1,2], Ivers, H[1]., Morin, C. M.[1]

P033 ARE THE EFFECTS OF INSOMNIA TREATMENT ON DAYTIME MEASURES CLINICALLY IMPORTANT?
Sanchez-Ortuño, M.[1,2], Bélanger, L.[1], Ivers, H.[1], Morin, C. M.[1]

DREAMING

P034 SCHIZOPHRENIA-LIKE COGNITION IN REM SLEEP MENTATION
Stenstrom P.[1,2], Nielsen T.[1,3], Solomonova E.[1,2], Lara-Carrasco J.[1,2]

P035 LUCID DREAMING IS ASSOCIATED WITH SLEEP PARALYSIS BUT NOT NIGHTMARES
Solomonova, E.[1,2], Nielsen T.[1,3], Stenstrom, P.[1,2]

P036 BEHAVIORAL ENACTMENT OF DREAMING IN A NORMAL POPULATION
Nielsen T.[1], Svob C.[2], Kuiken D.[2]

P037 EVOLUTION OF GENDER DIFFERENCES IN THE DREAMS OF UNIVERSITY STUDENTS
Ouellet D., Duchesne-Pérusse, A., Paquette-Biron, M., Sabourin C., De Koninck J.

P038 WORD ASSOCIATIONS IMPROVE AUTOMATIC ANALYSIS OF DREAM EMOTIONAL TONE
Amini R., Ouellet D., Sabourin C., De Koninck J.

POSTER SESSION B: TUESDAY, APRIL 28, 2009

CHRONOBIOLOGY

P039 SCREENING FOR SLEEP QUALITY AND CHRONOTYPE IN ELITE WINTER ATHLETES
Samuels C[1,2,], Fryer S[1]

P040 EFFECT OF CIRCADIAN AND SLEEP-WAKE STATE ON HEART RATE VARIABILITY IN HUMANS
Philippe Boudreau[1], Guy Dumont[2], Diane B. Boivin[1]

P041 EFFECT OF BRIGHT LIGHT ON PERFORMANCE OF POLICE OFFICERS ON ROTATING SHIFTS
Boudreau P.[1], Tremblay G.M.[1], Boivin D.B.[1]

P042 DAYTIME SLEEPINESS AND NATURAL LIGHT EXPOSURE IN STUDENTS WHO WORK DURING THE SCHOOL YEAR: PRELIMINARY RESULTS
Martin J.-S.[1], Hébert M.[2], Ledoux É.[3], Laberge L.[4]

P043 SLEEP DURATION, LIGHT EXPOSURE AND BIOMARKERS OF MELATONIN AMONG ROTATING SHIFT NURSES
Grundy A.[1], Sanchez M.[1], Richardson H.[1], Tranmer J.[1,2], Graham C.[3], Aronson K.[1].

P044 CHANGES IN CIRCADIAN RHYTHM EVIDENT DURING AN ACUTE RANDOMIZED PLACEBO-CONTROLLED TRIAL OF METHYLPHENIDATE
Davidson, F., Ironside, S., & Corkum, P.

P045 SELECTIVE INCREASE OF SLOW WAVES SLEEP (SWS) BY A NOVEL MELATONIN PARTIAL AGONIST
R.Ochoa-Sánchez [1], G. Spadoni [3], A. Bedini [3] , M. Mor [4], S. Rivara [4], F. Fraschini [5] , G.Tarzia [3] , G. Gobbi [1,2]

P046 SHORT EXPOSURE TO BLUE-ENRICHED WHITE LIGHT DOES NOT FURTHER IMPACT ALERTNESS LEVEL WHEN USED AT THE END OF THE NIGHT
Sasseville A[1], Houle J[1], Hebert M[1]

P047 POLYSOMNOGRAPHIC SLEEP ACROSS THE CIRCADIAN AND MENSTRUAL CYCLES IN HEALTHY FEMALES
Ari Shechter [1,2] and Diane B. Boivin [1,2]

P048 QUANTITATIVE SLEEP ELECTROENCEPHALOGRAM ACROSS THE MENSTRUAL CYCLE IN WOMEN WITH PREMENSTRUAL DYSPHORIC DISORDER
Ari Shechter [1,2], Paul Lesperance [3] and Diane B. Boivin [1,2]

PHYSIOLOGY & NEUROPHYSIOLOGY

P049 SLEEP-WAKE AND CIRCADIAN CONTRIBUTION TO CHANGES IN PER2 PROTEIN IN WHOLE LIVING MICE
Curie T.[1], Mongrain V.[1], Dorsaz S.[1], Maret S.[1], Emmenegger Y.[1], Franken P[1].

P050 DECREASING THE STRESS RESPONSE DOES NOT RESCUE THE IMPAIRED HOMEOSTATIC SLEEP REBOUND IN DBA/2J MICE
Mongrain V.[1], Curie T.[1], Gip P.[2], Heller H.C.[2], Franken P.[1,2]

P051 INCREASED GABA-ERGIC ACTIVITY IN THE PEDUNCULOPONTINE NUCLEUS REDUCES REM SLEEP AND IMPAIRS LEARNING IN RATS
Fogel S.[1], Smith C.[2] and Beninger R.[1]

P052 REM SLEEP INSOMNIA AND DECREASED PPT CHOLINERGIC NEURONS FOLLOWING MYOCARDIAL INFARCTION IN THE RAT
Bah T.M.[1], Laplante F.[4], Kaloustian S.[1], Sullivan R.[2,4], Rousseau G.[1,3], Godbout R.[1,2]

P053 $GABA_B$-MEDIATED INHIBITION PLAYS A CRITICAL ROLE IN MEDIATING REM SLEEP ATONIA
Brooks, P.L. and Peever, J.H.

P054 NORADRENERGIC MODULATION OF MUSCLE TONE DURING CATAPLEXY IN HYPOCRETIN/OREXIN KNOCKOUT MICE
Burgess C.R. and Peever J.H.

P055 NORADRENERGIC TRANSMISSION TRIGGERS MUSCLE TONE BY AMPLIFYING GLUTAMATERGIC DRIVE ONTO SOMATIC MOTONEURONS
Schwarz P.B., Yee N., Mir S. and Peever J.H.

P056 ENHANCED CHOLINERGIC ACTIVITY AT THE HYPOGLOSSAL MOTOR NUCLEUS SUPPRESSES GENIOGLOSSUS MUSCLE ACTIVITY
Grace K.[1], Liu H.[1], Nolan P.[2], Horner R.[1]

P057 EFFECT OF ETHANOL ON MEDULLARY RESPIRATORY ACTIVITY AND SLEEP
LM Vecchio[1], H Liu[2], S Harding[3], A Le[3], RL Horner[1, 2]

P058 PKA-MEDIATED MODULATION OF RESPIRATORY-DRIVE TRANSMISSION TO HYPOGLOSSAL MOTONEURONES *IN VIVO*
DuBord M.-A., Liu H., Horner R. L.

P059 NOVEL MECHANISM UNDERLYING OPIOID-INDUCED RESPIRATORY DEPRESSION: SUPPRESSION OF MOTOR DRIVE FROM THE MEDULLA TO UPPER-AIRWAY MUSCLES
Hajiha M., DuBord M.-A., Liu H., Horner R. L.

P060 OPIOID-SENSITIVE SITE IN THE MEDULLA UNDERLYING SUPPRESSION OF BREATHING IN THE SLEEPING ADULT RAT
Gaspard Montandon and Richard L. Horner

P061 SLEEP LOSS REDUCES APNEA-INDUCED RESPIRATORY NEUROPLASTICITY
Tadjalli A.[1], Duffin J[2]. and Peever J.H.[1,2]

P062 N-REM SLEEP SLOW OSCILLATIONS AMPLITUDE AND DENSITY IN THE YOUNG AND MIDDLE-AGED MEN AND WOMEN
Viens, I[1,3], Lafortune, M[1,3], Poirier, G[1], Paquet, J[1], Barakat, M[1,3], Vandewalle, G[1,3], Martin, N[1,3], Robillard, R[1,2,3] and Carrier, J[1,2,3]

P063 ETHNIC DIFFERENCES IN SLEEP ARCHITECTURE IN HEALTHY, NORMOTENSIVE YOUNG ADULTS ARE ASSCOCIATED WITH NOCTURNAL HEART RATE VARIABILITY
Saletin J.[1], Klick B.[1], Smith M.[1]

RESPIRATORY & MOVEMENT-RELATED SLEEP DISORDERS

P064 SLEEP-RELATED BREATHING PROBLEMS AFFECT BLOOD PRESSURE REGULATION IN CHILDREN
Wade TJ[1], Reid GJ[2], Fitzgibbon LK[1], Coverdale NS[1], Cairney J[3], O'Leary DD[1]

P065 THE IMPACT OF SLOW WAVE SLEEP (SWS) AND SLEEP FRAGMENTATION ON FATIGUE VERUS DAYTIME SLEEPINESS IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA (OSA)
Ghashghai A., Chung SA, and Shapiro CM

P066 FREQUENCY OF OBSTRUCTIVE SLEEP APNEA IN PAEDIATRIC PATIENTS WITH ADENOTONSILLAR HYPERTROPHY
CAPUA C, CHUNG SA, MARCU S, JOVANOVIC D, AND SHAPIRO CM.

P067 OBSTRUCTIVE SLEEP APNEA IS HIGHLY PREVALENT AMONG KIDNEY TRANSPLANTED PATIENTS: RESULTS OF THE SLEPT STUDY
Dunai A.[1], Molnar M. Zs.[2,3], Novak M.[3,4], Czira M. E.[3], Lindner A.[3], Lazar A. S.[3], Fornadi K.[3,5], Zoller R.[3], Szentkiralyi A.[3], Mucsi I.[1,3]

P068 PERIODIC LIMB MOVEMENTS IN SLEEP AND CARDIOVASCULAR RISK IN KIDNEY TRANSPLANTED PATIENTS
Novak M.[1,2,3], Mucsi I.[1,3], Czira E.M.[1], Lindner A.[1,] Fornadi K.[1,4], Lazar A.S.[1], Dunai A.[3], Zoller R.[1,3], Szentkiralyi A.[1], Madarasz Cs.[1], Kiss Z.[5],Molnar M. ZS.[1,6]

P069 EVALUATION OF A PORTABLE MONITOR COMPARED WITH POLYSOMNOGRAPHY FOR THE DIAGNOSIS OF OBSTRUCTIVE SLEEP APNEA
Driver HS.[1,2], Bjerring K.A.[2], Toop F.[2], Pereira E.[2], Stewart S.C., Munt P.[1], Fitzpatrick M.F.[1]

P070 ANALYSIS OF HOSPITAL DISCHARGE DATA TO DETERMINE THE USE OF CONTINUOUS POSITIVE AIRWAY PRESSURE IN CANADIAN HOSPITALIZED PATIENTS WITH SLEEP APNEA
Kathy Spurr, RRT, RPSGT, MHI (c)[1], Adam Webber, MSc.[2], Debra Morrison, MD, FRCPC[3], Robert W Gilbert, PhD[1]

P071 THE MOUTH LEAK SYNDROME IN PATIENTS INITIATING NCPAP FOR OSAS
Baltzan M.A.,[1,2,3] Garcia-Asensi A.,[3] Parenteau M,[3] Dabrusin R.,[1] Tanzimat G,[3] Kassissia I.,[3] Wolkove N.[1]

TECHNOLOGY & PROCEDURES

P072 WHAT IS THE LEVEL OF SLEEP KNOWLEDGE IN FAMILY MEDICINE RESIDENTS?
Samuels C.H[1,2,] Cohen R[1], Fryer S[1]

P073 WHAT MAKES SOME PATIENTS WANT TO INVESTIGATE THEIR SLEEP?
Rizzo, D.[1], Bailes, S.[1], Baltzan, M.[2,7,8], Grad, R.[3,5], Kassissia, I.[4,8], Fichten, C.[1,5,6], Creti, L.[1], Libman, E[1,7].

P074 COMPARISON OF SYNCHRONIZED LEVEL 1 AND LEVEL 3 SLEEP STUDIES
Sullivan, G. E.; Morehouse, R.; Savoy, A.

P075 A RETROSPECTIVE, OBSERVATIONAL STUDY SHOWING PATIENTS WITH A NORMAL LEVEL III SLEEP STUDY AND NORMAL OSA PRETEST PROBABILITY FACTORS MAY STILL REQUIRE ADDITIONAL INVESTIGATIONS
Fordyce L[1], Samuels C.H[1,2], Oram C[1], Wallins B[1]

P076 SPECIALIZED NEW PROGRAMMING TO FACILITATE RESEARCH ORIENTED ANALYSIS OF POLYSOMOGRAPHIC STUDIES
Mink J, Skomro R. and Reid J.

P077 ARTIFICIAL NEURAL NETWORK SCORING OF HUMAN SLEEP-WAKE STAGES COMBINING SHORT-EPOCH FEATURE EXTRACTION AND POST-PROCESSING INFERENCE RULES
Florian Chapotot Ph.D.[1], Lukas Zoubek B.M.E.[2] and Guillaume Becq Ph.D.[3]

P078 THE INFLUENCE OF SLEEP QUALITY ON MENTAL AND PHYSICAL HEALTH IN THE CANADIAN COMMUNITY HEALTH SURVEY
Bauermann, T.M.[1] & Najbor, R.M. [2]

P079 THE DISCRIMINANT VALIDITY OF THE SLEEP PROBLEMS INVENTORY
Bauermann, T.M.[1], Parker, J.D.A. [2], Wood, L.M. [2]

P080 THERE MAY BE NO FIRST NIGHT EFFECT FOR AMBULATORY POLYSOMNOGRAPHY IN OLDER ADULTS
McInrue, E.[1], Hoehn, J. [1], Cosenza, S. [1], Buenaver, L.[1], Smith M.T.[1]

P081 SYSTEMATIC OPTIMIZATION OF AUTOMATED SLEEP SPINDLE DETECTION
Ray LB.[1], Peters KR.[1], Fogel SM.[2], Smith CT. [1]

POSTER PRESENTATIONS

SLEEP DEPRIVATION

P001

EFFECTS OF SLEEP DEPRIVATION AND DAYTIME RECOVERY SLEEP ON SELF-EVALUATION ACCURACY

Martin N.[1,2], Dostie V. [1,2] and Carrier J. [1,2]
[1]Centre d'étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Cœur de Montréal, Montréal, QC, Canada; [2]Département de psychologie, Université de Montréal, Montréal, QC, Canada

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

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

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

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

Support: This research was supported by scholarships from the Canadian Institutes of Health Research (CIHR), and grants from CIHR, the Fonds de la Recherche en Santé du Québec (FRSQ) and the Natural Sciences and Engineering Research Council of Canada (NSERC).

P002

SLEEP DURING PEDIATRIC HOSPITALIZATION

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

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

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

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

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

Support: Dr. Stremler received the J. Christian Gillin, MD Research Award from the Sleep Research Society Foundation in support of this project. Dr. Stremler's work is also supported by a New Investigator Award from the Canadian Institutes of Health Research. Dr. Parshuram is recipient of a Career Scientist Award from the Ontario Ministry of Health and Long Term Care.

P003

AGE-RELATED DIFFERENCES IN N-REM SLOW OSCILLATIONS: EFFECTS OF SLEEP DEPRIVATION

Lafortune, M.[1,2], Viens, I.[1,2], Jacob-Lessard, A.[1,2], Poirier, G.[1,2], Vandewalle, G.[1,2], Barakat, M.[1,2], Martin, N.[1,2], Filipini, D.[1], & Carrier J.[1,2]

[1]Centre d'étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Cœur de Montréal, QC, Canada; [2] Département de psychologie, Université de Montréal, Montréal, QC, Canada

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

Methods: We used an automatic algorithm to assess the effects of age, sleep loss and topography on N-REM sleep slow oscillations (SO; >75 μm). Twenty-four healthy volunteers with no sleep disorders were separated in two groups: Young (6W, 6M; 24.2y ± 3.3), and Middle-aged (6W, 6M; 53.8y ± 3.7). Each subject participated in a baseline nocturnal sleep and a daytime recovery

sleep (after 25-hours of wakefulness). SO detection was performed on artefact free sections of NREM sleep for Fp1, F3, C3, P3, and O1 (linked-ears), with an automatic algorithm using published criteria (Massimini et al. 2004).

Results: Three-way ANOVAs (Age group*Sleep condition*Derivation) were performed on SO amplitude and density (nb/min). In both age groups, SO density was higher during daytime recovery sleep compared to baseline sleep and this effect was stronger in Fp1 and F3. Compared to baseline sleep, SO amplitude increased during daytime recovery sleep and this effect was more prominent in young compared to older subjects in FP1.
Conclusion: Age-related difference in the effects of the sleep deprivation was observed on SO amplitude only, and not on SO density, particularly in anterior derivations. This may be explained by age-related decline in the capacity to synchronize larger neuronal populations after sleep deprivation, especially in frontal areas.

References: Massimini, M., Huber, R., Ferrarelli, F., Hill, S., & Tononi, G. (2004). The sleep slow oscillation as a traveling wave. *Journal of Neuroscience, 24*(31), 6862-6870.

Support: This research was supported by scholarships from the Canadian Institutes of Health Research (CIHR), and grants from CIHR, the Fonds de la Recherche en Santé du Québec (FRSQ) and the Natural Sciences and Engineering Research Council of Canada (NSERC).

P004

IMPACT OF ACUTE SLEEP RESTRICTION ON CORTISOL AND LEPTIN LEVELS IN YOUNG WOMEN.

Omisade A.[1], Buxton M. O.[2] and Rusak B.[1,3,4]
Departments of [1]Psychology, [3]Psychiatry, and [4]Pharmacology, Dalhousie University, Halifax, NS, Canada; [2]Department of Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA.

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

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

Results: After sleep restriction, participants showed reduced morning cortisol levels ($p = 0.02$), elevated morning leptin levels ($p = 0.04$), elevated afternoon/evening cortisol AUC values ($p = 0.008$), and a slower rate of decline in cortisol ($p = 0.04$). Women in the follicular phase showed greater changes in morning cortisol ($p = 0.05$), afternoon/evening AUC values ($p = 0.03$), and

cortisol slope of decline ($p = 0.03$) compared to women in the luteal phase. Craving scores were unaffected by sleep loss and unrelated to hormone measures.

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

Support: Financial support was provided by the Capital District Health Authority Research Fund and the Department of Psychiatry, Dalhousie University.

P005

SHORT DAILY SLEEP OPPORTUNITIES AMELIORATE THE EFFECTS OF SLEEP LOSS ON ENERGY EXPENDITURE IN RATS

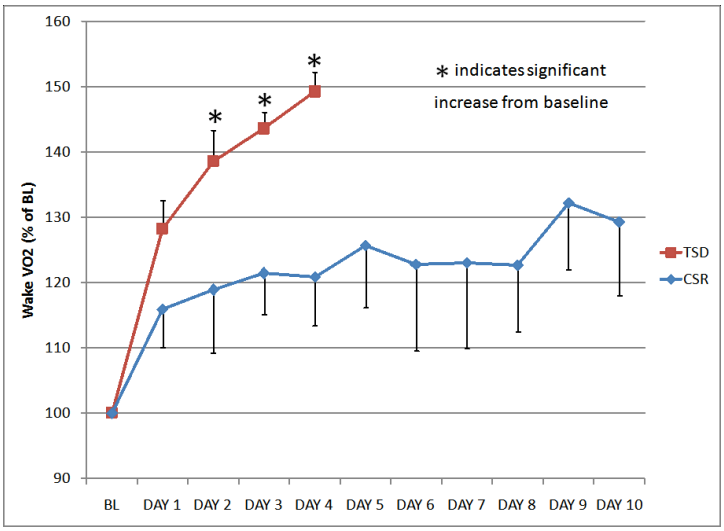
Caron A.M. and Stephenson R.
Department of Cell & Systems Biology, University of Toronto, Toronto, ON, Canada M5S 3G5

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

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

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

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



References: 1. Everson C.A., Bergmann B.M., and Rechtschaffen A. (1989). *Sleep* 12(1): 13-21
Support: Natural Sciences and Engineering Research Council of Canada (NSERC)

SLEEP & PERFORMANCE

P006

LOWER SUBJECTIVE SOCIOECONOMIC STATUS IS ASSOCIATED WITH SHORTER SLEEP DURATION, POORER SLEEP QUALITY, AND MORE DAYTIME SLEEPINESS IN CHILDREN AND ADOLESCENTS

Jarrin, D.C.[1], Silverstein, J.E. [1], & McGrath, J.J.[1]
[1]Department of Psychology, Concordia University, Montreal, QC, Canada

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

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

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

Conclusions: Lower subjective socioeconomic is associated with greater risk for sleep problems in children and adolescents, suggesting sleep may be one pathway underlying the socioeconomic

gradient in health. Future researchers should aim to elucidate how specific sleep constructs (e.g., sleep architecture) may explain how socioeconomic status "gets under the skin" to affect health.

References:

Drake, C., Nickel, C., Burduvali, E., Roth, T., Jefferson, C., & Badia, P. (2003). The Pediatric Daytime Sleepiness Scale (PDSS): Sleep habits and school outcomes in middle school children. *Sleep, 26,* 455-458.

Goodman, E., Adler, N.E., Kawachi, I., Frazier, A.L., Huang, B., & Colditz, G.A. (2001). Adolescents' perceptions of social status: development and evaluation of a new indicator. *Pediatrics, 108,* e31.

P007

SUBJECTIVE EXCESSIVE DAYTIME SLEEPINESS IN A COMMUNITY-BASED SAMPLE: FREQUENCY AND ASSOCIATED FACTORS

Beaulieu-Bonneau S., Fortier-Brochu E., LeBlanc M., Vallières A., and Morin C. M.
École de psychologie, Université Laval, Québec, Canada

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

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

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

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

Support: Supported by the Canadian Institutes of Health Research

P008

DO DIFFERENT LEVELS OF SLEEPINESS MODULATE EFFECTS OF CAFFEINE ON VIGILANCE?

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

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

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

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

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

P009

BENEFITS OF A DAYTIME NAP FOR STUDENTS ON A UNIVERSITY CAMPUS

Black J., Ling E., and Cote K.
Department of Psychology, Brock University, St. Catharines, ON, Canada

Introduction: Sleepiness is a problem for students at all levels of education, and naps are known to alleviate some negative effects of sleep deprivation[1]. We hypothesized that a 20-minute nap opportunity while on campus would lead to improvements in well-being and performance that would continue throughout the day during classes and on the commute home.

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

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

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

References: [1] Milner, C. E. and Cote, K. A. (2008). Benefits of napping in healthy adults: impact of nap length, time of day, age, and experience with napping. *Journal of Sleep Research, in press.*

P010

SLEEP QUALITY AND NEXT-DAY PVT PERFORMANCE AFTER HEAVY ALCOHOL CONSUMPTION

Arnedt J.T.[1], Almeida A.[2], Hunt S.[2], Gokhale M.[3], Rohsenow D.[4], & Howland J.[2]
[1]Sleep and Chronophysiology Lab, Department of Psychiatry, University of Michigan; [2]Department of Social and Behavioral Sciences, Boston University School of Public Health; [3]Data Coordinating Center, Boston University School of Public Health; [4]Center for Alcohol and Addiction Studies, Brown University

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

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

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

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

Support: R01 AA12087 (J Howland)

P011

EFFECTS OF CONSOLIDATION OF PROCEDURAL MOTOR MEMORY TRACES ON SLOW AND FAST SPINDLES

Barakat M.[1,3], Doyon J.[1,3], Debas K.[1,3], Morin A.[1,3], Poirier G.[2], Viens I.[2,3], Lafortune M.[2,3], Vandewalle G.[1,2], Carrier J. [1,2,3]

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

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

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

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

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

Support: This research was supported by a grant from the Canadian Institutes of Health Research (CIHR) to JD, JC and al.

P012

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

Nesbitt D., Peters K.R., DeCicco T. and Smith C.T.
Department of Psychology, Trent University, Peterborough, Ontario, Canada

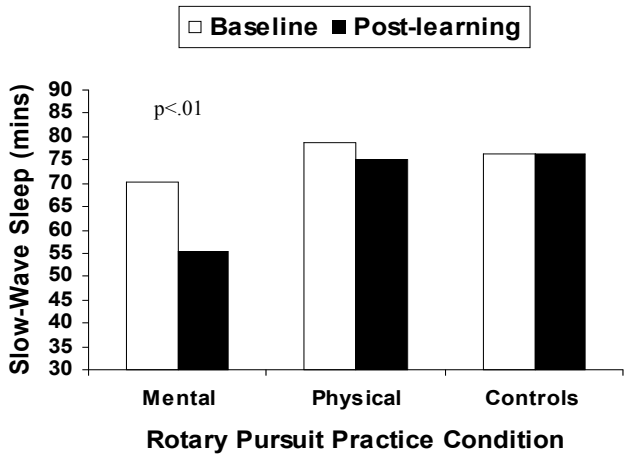
Introduction: Previous studies have reported learning related sleep changes after physical practice on a motor task. The present study assessed sleep changes occurring after acquisition of a motor task (rotory pursuit task) using mental practice. Mental practice refers to the cognitive rehearsal of a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

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

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

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

A



P013

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

Madore A.R.[1], DeLay S.[1], Legault M.G.[1]
[1]Department of Psychology, Laurentian University, Sudbury, ON, Canada

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

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

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

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

P014

SLEEP EEG IN AUTISM AND PERFORMANCE ON THE EMBEDDED FIGURE TEST

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

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

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

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

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

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

Supported by the Canadian Institutes of Health Research

P015

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

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

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

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

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

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

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

Supported by the Canadian Institutes of Health Research and the Fonds de la recherche en santé du Québec.

P016

SLEEP PATTERNS IN ADOLESCENTS WITH BIPOLAR DISORDER OR BORDERLINE PERSONALITY DISORDER

Huynh C.[3], Guilé J.M.[1,3], Breton J.J.[1,3], Cohen D.[2], Chevrier É.[4] and Godbout R.[1,4]
[1]Department of Psychiatry, Université de Montréal (Québec) Canada; [2] Université Pierre et Marie Curie, Hôpital Pitié-Salpétrière, Psychiatrie de l'enfant et de l'adolescent, Paris, France; [3]Pedopsychiatry Program, Mood disorders Clinic, Hôpital Rivière-des-Prairies; [4]Neurodevelopmental Disorders Program, Sleep Laboratory & Clinic, Hôpital Rivière-des-Prairies; [5]Centre de recherche Fernand-Seguin, Hôpital Rivière-des-Prairies, Montréal (Québec) Canada.

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

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

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

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

Supported by the Canadian Institutes of Health Research

P017

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

Wiseman-Hakes, C.[1,2] , Murray, BJ, [2,3,4], Victor, JC[5]
[1] Graduate Dept. of Rehabilitation Science, [2] Toronto Rehab Institute, [3] University of Toronto, Dept. of Neurology, [4] Sunnybrook Hospital, [5] University of Toronto Dalla Lana School of Public Health

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

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

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

Conclusions: Clinical and research evidence suggests that timely and effective diagnosis and management of sleep/wake disturbances post TBI may facilitate recovery of maximal cognitive-

communication potential. Based on results of this study, we conclude that for certain persons, pharmacological management of sleep and alertness post traumatic brain injury can result in functional subjective improvements in sustained attention, language processing and verbal memory. Further research in this area is warranted.

P018

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

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

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

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

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

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

Support: Supported by the Canadian Institutes of Health Research

P019

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

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

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

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

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

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

P020

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

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

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

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

complexes were visually counted in Stage 2 sleep by two raters. K-complex density (# events/minute of Stage 2) was compared between groups on both nights.

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

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

Support: Ontario Neurotrauma Foundation (ONF) Graduate Fellowship

References:

1. Ouellet MC, et al. (2006). Insomnia in patients with traumatic brain injury: frequency, characteristics, and risk factors. J Head Trauma Rehab, 21:199-212.
2. Colrain, IM. (2005). The K-Complex: a 7-Decade History. Sleep, 28:255-73.
3. Bastien, CH, et al. Spontaneous K-Complexes in Chronic Psychophysiological Insomnia, in submission.

P021

INVESTIGATION OF SLEEP IN CHRONIC TREATMENT-RESISTANT DEPRESSED PATIENTS

Ahmadi N, Shapiro CM
Psychiatry and Sleep Research Unit, University Health Network, Toronto, ON, Canada

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

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

Results: Seven of the participants were females and the average age for the whole group was 50±7 years. The average HAM-D score (± SD) was 24± 4. The average sleep onset latency and the average REM latency after sleep onset were 22±14 min and 253±119 min respectively (all patients were on antidepressants). The average sleep efficiency was 81±4%. The average slow wave was sleep percentage was 8±9% and the average REM sleep percentage was 13±8%. The average arousal index was 27±15/hr. Three of the nine patients had an undiagnosed moderate to

severe sleep apnea. Three of the patients had a moderate to severe periodic leg movement disorder and five of the patients had "fragmented" sleep. The average mean MSLT was 10±4 min and two of the patients had severe daytime sleepiness.

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

P022

A PRELIMINARY ATTEMPT AT DEFINING 'SLEEP MARKERS OF DEPRESSION' CATEGORICALLY AND EXAMINING THEIR ASSOCIATION WITH SUBJECTIVE LOW MOOD.

Saleh, P[1, 2, 3], Shahid, A[1, 2], Chung, F[4] and Shapiro, CM[1, 2, 3]
[1]Department of Psychiatry, University of Toronto; [2]Youthdale Child and Adolescent Sleep Centre, Toronto, Ontario; [3]Department of Cell and Systems Biology, University of Toronto; [4]Department of Anesthesia, Toronto Western Hospital

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

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

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

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

INSOMNIA

P023

EPIDEMIOLOGY OF INSOMNIA IN A CANADIAN POPULATION-BASED SAMPLE

LeBlanc, M.[1,2], Bélanger, L. [1,2], Mérette, C. [2,3], Savard J. [1,4], Morin, C.M. [1,2]
[1] École de psychologie, Université Laval, Québec, Canada. [2] Centre de recherche Université Laval-Robert-Giffard, Québec, Canada. [3] Département de psychiatrie, Université Laval, Québec, Canada. [4] Centre de recherche en cancérologie de l'Université Laval, l'Hôtel-Dieu de Québec, Québec, Canada.

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

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

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

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

Research supported by Canadian Institutes of Health Research grant (#42504)

P024

SLEEP DISTURBANCES IN CHINESE EARTHQUAKE VICTIMS

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

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

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

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

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

P025

ARE INSOMNIA SYMPTOMS STABLE OVER TIME? A 5-YEAR PROSPECTIVE STUDY IN THE GENERAL POPULATION

Fortier-Brochu, É., Ivers, H., Beaulieu-Bonneau, S., LeBlanc, M. & Morin, C. M.
Université Laval, Québec, Canada

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

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

Results: Overall, 52.7% of participants remained classified within the same insomnia subtype over the five-year period, with 1.7% having sleep onset insomnia, 46.2% having maintenance insomnia and 4.8% having mixed insomnia. Within the remaining 47.3% of participants for whom the nature of symptoms changed over time, 36.9% did not experience a predominant insomnia subtype, 7.4% had predominant sleep onset insomnia, 38.7% had predominant maintenance insomnia and 17.1% had predominant mixed insomnia. For individuals classified as

having sleep onset insomnia at a given assessment period (time X), the probability of having sleep onset insomnia the next time they had insomnia symptoms (time X + 1) was 32.3% (see figure). For those classified as having maintenance insomnia, the probability of remaining with maintenance insomnia the next time they experienced insomnia symptoms was 82.1%. Finally, for individuals classified as having mixed insomnia, the probability of having mixed insomnia the next time they experienced insomnia symptoms was 49.8%.

Conclusion: The stability of insomnia subtypes varies among individuals with insomnia and seems to differ depending on the nature of the subtype. While maintenance insomnia appears relatively stable across time, sleep onset and mixed insomnia seem more volatile.

P026

INSOMNIA AS A RISK FACTOR FOR HEALTH PROBLEMS: A LONGITUDINAL STUDY

Perozzo, C.[1, 2], Gagnon, C.[1, 2], LeBlanc, M.[1, 2], Savard, J.[1, 3], & Morin, C. M.[1, 2]
[1]École de psychologie, Université Laval, Québec, QC, Canada; [2]Centre d'étude des troubles du sommeil, Centre de recherche Université Laval-Robert-Giffard, Québec, QC, Canada; [3]Centre de recherche en cancérologie de l'Université Laval, Hôtel-Dieu de Québec, Québec, QC, Canada

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

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

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

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

Support: This research was supported by a grant from the Canadian Institutes of Health Research (# 42504).

P027

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

St-Jean G.[1-2] and Bastien C.H. [1-2]
[1]École de psychologie, Université Laval, Québec, Canada; [2]Laboratoire de neurophysiologie humaine, Centre de recherche Université Laval Robert-Giffard, Québec, Canada

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

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

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

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

P028

ERP MEASURES DURING SLEEP IN PSYCHOPHYSIOLOGICAL AND PARADOXICAL INSOMNIA SUFFERERS

Turcotte I.[1], Adam A-M.[1], Lecarpentier M.[1] and Bastien C.[1]
[1]Department of Psychology, Laval University, Quebec, Quebec, Canada.

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

Methods: Eight Psy-I (mean age = 40.4y), 9 Para-I (mean age = 43.1y) and 10 GS (mean age =

43.9y) underwent four consecutive nights of PSG (N1 to N4). ERPs N1 and P2 were recorded during the fourth night in stages 2, 3-4 and REM. Auditory stimuli consisted of 'standard' frequent (70 dB, 2000 Hz, .85 probability) and 'deviant' rare stimuli (90 dB, 1500 Hz, .15 probability).

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

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

Support: Canadian Institutes of Health Research (# 49500 and # 86571).

P029

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

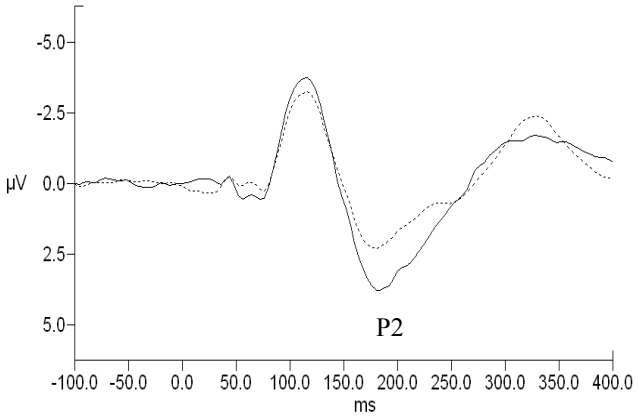
Kertesz R. S., Cote K. A.
Department of Psychology, Brock University, St. Catharines, ON, Canada

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

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

.

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



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

P030

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

Authors: Lorraine Fradette, BSc[1,2], Pennestri Marie-Hélène, BSc[1], Jacques Montplaisir, MD, PhD[2,3] , Charles M. Morin, PhD[4,5], Roberto Colombo BEng[6], Paola A.Lanfranchi, MD, MSc[2,7]
[1] Sciences Biomédicales, Faculté de Médecine, Université de Montréal, Montréal, Québec, Canada
[2] Sleep Disorders Center, Hôpital du Sacré-Coeur de Montréal, Montréal, Québec, Canada
[3] Department of Psychiatry, Université de Montreal, Montréal, Québec, Canada.
[4] École de Psychologie, Université Laval, Québec, Québec, Canada
[5] Centre d'étude des troubles du sommeil, Centre de recherche Université Laval-Robert-Giffard, Québec, Québec, Canada
[6] Bioengineering Department, Salvatore Maugeri Foundation, Veruno, Italy
[7] Department of Medicine, Division of Cardiology, Hôpital du Sacré-Coeur de Montréal and Université de Montréal, Québec, Canada

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

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

Results: Total slope, αLF and αlumped were highly similar between insomniacs and good sleepers during wakefulness, stage 2 and REM sleep. However, these measures of BRS during stage 2 tended to be lower in insomniacs with impaired sleep efficiency (SE) at PSG (SE<85%, N=6 subjects) versus those with preserved SE (≥85%, N=5 subjects). Values were respectively: total slope 10.3±5.7 vs 19.5±9 ms/mmHg, p=0.1; αLF 7.2±3.9 vs 12.6±4.4 ms/mmHg; p=0.06; and αlumped 10.2±6.3 vs 18.8±8.6 ms/mmHg; p=0.1.

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

P031

DOES CBT FOR INSOMNIA ALTER SLEEP MISPERCEPTIONS?

Moreau V., Gagnon C., Lamy M., Ivers H. and Morin C. M.
École de psychologie, Université Laval, Québec, QC

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

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

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

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

Support: National Institute of Mental Health (MH060413)

P032

IMPACT OF CBT FOR INSOMNIA AND CBT COMBINED WITH MEDICATIONS ON DAYTIME FUNCTIONING

Bélanger, L.[1], Sanchez-Ortuño, M.[1,2], Ivers, H[1]., Morin, C. M.[1]
[1] Université Laval, Québec, Canada; [2] Universidad de Murcìa, Murcìa, Spain

Introduction: This study examined the effects of CBT for insomnia delivered alone to CBT combined with hypnotic medications (CBT+Med) on daytime functioning variables. A second objective was to compare the effects of different extended treatment strategies on daytime functioning. **Methods.** One hundred and sixty individuals (61% women; mean age: 50.3 years) with persistent insomnia were randomized either to a CBT alone or CBT+Med condition. After

initial treatment, patients treated with CBT alone were then randomized either to extended CBT (6 months) or to "no additional treatment". Those treated with CBT+Med were randomized to CBT combined with intermittent medication use or CBT with medication taper. Daytime functioning variables included fatigue (Multidimensional Fatigue Inventory; MFI), anxiety (Beck Anxiety Inventory; BAI), depression (Beck Depression Inventory; BDI), health perception (SF-36) and beliefs about sleep (Dysfunctional Beliefs about Sleep, DBAS-16).

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

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

Support: National Institute of Mental Health (MH60413)

Keywords: Insomnia, treatment, CBT

P033

ARE THE EFFECTS OF INSOMNIA TREATMENT ON DAYTIME MEASURES CLINICALLY IMPORTANT?

Sanchez-Ortuño, M.[1,2], Bélanger, L.[1], Ivers, H.[1], Morin, C. M.[1]
[1] Université Laval, Québec, Canada; [2] Universidad de Murcìa, Murcìa, Spain

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

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

impairment reduction, i.e. recovered or improved, in each treatment condition were then compared.

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

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

Support: National Institute of Mental Health (MH60413)

Keywords: Insomnia, treatment, CBT

DREAMING

P034

SCHIZOPHRENIA-LIKE COGNITION IN REM SLEEP MENTATION

Stenstrom P.[1,2], Nielsen T.[1,3], Solomonova E.[1,2], Lara-Carrasco J.[1,2]
[1]Sleep Research Center, Hôpital du Sacré-Coeur de Montreal, Québec
[2]Psychology Department, Université de Montréal, Québec
[3]Psychiatry Department, Université de Montréal, Québec

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

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

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

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

References.

Gottesmann, C. (2006). The dreaming sleep stage: a new neurobiological model of schizophrenia? *Neuroscience, 140*, 1105-1115.

Hobson, J.A., (2004). A model for madness? *Nature, 430*, 21.

Support. Natural Sciences and Engineering Research Council.

P035

LUCID DREAMING IS ASSOCIATED WITH SLEEP PARALYSIS BUT NOT NIGHTMARES

Solomonova, E.[1,2], Nielsen T.[1,3], Stenstrom, P.[1,2]
[1]Sleep Research Center, Hôpital du Sacré-Coeur de Montreal
[2]Psychology Department, Université de Montréal
[3]Psychiatry Department, Université de Montréal

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

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

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

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

Acknowledgments: The study was funded by 'Fonds de recherche en santé du Québec'

P036

BEHAVIORAL ENACTMENT OF DREAMING IN A NORMAL POPULATION

Nielsen T.[1], Svob C.[2], Kuiken D.[2]
[1]Department of Psychiatry, Université de Montreal, Montreal, QC, Canada; [2]Department of Psychology, University of Alberta, Edmonton, AB, Canada.

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

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

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

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

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

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

REFERENCES

1. Nielsen T and Paquette T: Dream-associated behaviors affecting pregnant and postpartum women, Sleep. 2007;30:1162-1169.

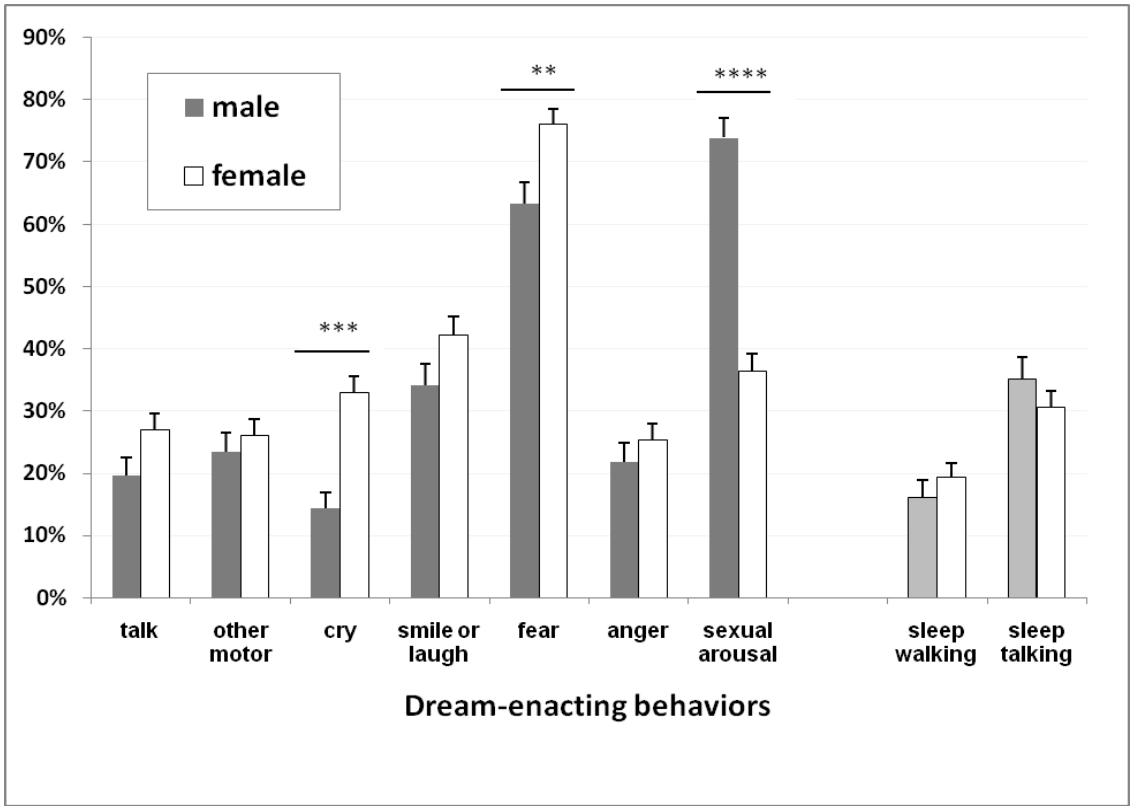


Figure 1. Percent of subjects reporting dream-enacting speaking, motor activity, emotions and sexual arousal 'sometimes' or 'often'. Females reported more frequent behaviors on all items except sexual arousal, which was characteristic of males (unpaired t-tests for gender: **p<.005; ***p<.000001; ****p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

P037

EVOLUTION OF GENDER DIFFERENCES IN THE DREAMS OF UNIVERSITY STUDENTS

Ouellet D., Duchesne-Pérusse, A., Paquette-Biron, M., Sabourin C., De Koninck J.
School of Psychology, University of Ottawa, Ottawa, ON, Canada

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

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

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

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

References:

[1]Hall, C. S., & Van De Castle, R. L. (1966). *The content analysis of dreams*. East Norwalk, CT: Appleton-Century-Crofts.

[2]Hall, C. S., Domhoff, G. W., Blick, K. A., & Weesner, K. E. (1982). The dreams of college men and women in 1950 and 1980: A comparison of dream contents and sex differences. *Sleep*. 5(2), 188-194.

Support: Social Sciences and Humanities Research Council of Canada

P038

WORD ASSOCIATIONS IMPROVE AUTOMATIC ANALYSIS OF DREAM EMOTIONAL TONE

Amini R., Ouellet D., Sabourin C., De Koninck J.
School of Psychology, University of Ottawa, Ottawa, ON, Canada

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

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

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

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

Reference: Razavi A. H., **Amini R.,** Sabourin C., Sayyad Shirabad, J., Nadeau, D., Matwin S. & De Koninck J. (2008). Classification of emotional tone of dreams using machine learning and text analyses. Sleep, 31, A380-381.

CHRONOBIOLOGY

P039

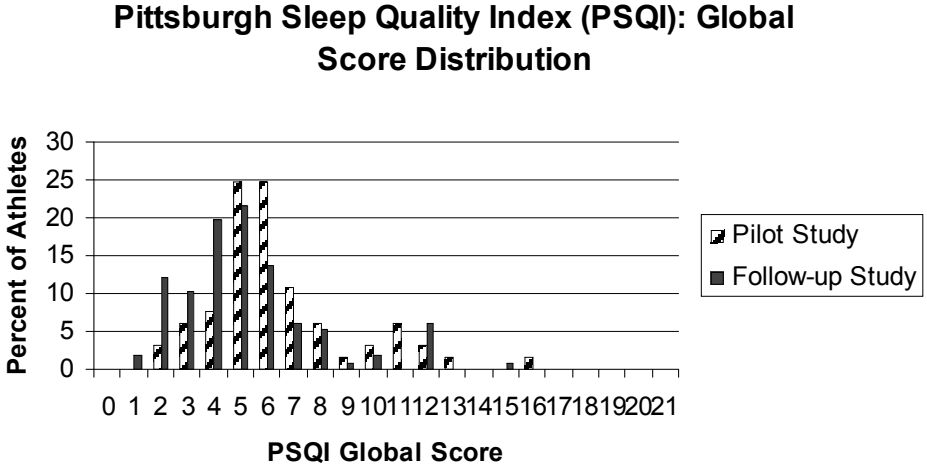
SCREENING FOR SLEEP QUALITY AND CHRONOTYPE IN ELITE WINTER ATHLETES

Samuels C[1,2,], Fryer S[1]
Centre for Sleep and Human Performance, Calgary, AB, Canada[1]
Faculty of Medicine, University of Calgary, Calgary, AB, Canada[2]

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

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

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



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

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

P040

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

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

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

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

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

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

Support: This work was supported by the Canadian Institutes of Health Research (CIHR) and the National Sciences and Engineering Research Council of Canada (NSERC). P. Boudreau and D. B. Boivin are supported by IRSST and FRSQ, respectively.

P041

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

Boudreau P.[1], Tremblay G.M.[1], Boivin D.B.[1]
[1]Centre for Study and Treatment of Circadian Rhythms, Douglas Mental Health University Institute, McGill University, Montréal, Québec, Canada

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

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

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

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

Reference: 1) Boivin DB, Tremblay GM, and James FO. Working on atypical schedules. Sleep Med 8: 578-89, 2007.
2) Dinges DF, et al. Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4-5 hours per night. Sleep 20: 267-77, 1997.

Support: This work was supported by the Institut de Recherche Robert-Sauvé en Santé et en Sécurité du Travail (IRSST). P. Boudreau and D. B. Boivin are supported by IRSST and Fond de la Recherche en Santé du Québec (FRSQ), respectively.

P042

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

Martin J.-S.[1], Hébert M.[2], Ledoux É.[3], Laberge L.[4]
1 ÉCOBES, Cégep de Jonquière, Saguenay, Québec; 2 Centre de recherche Université Laval Robert-Giffard, Québec; 3 Institut de recherche en santé et en sécurité du travail, Montréal; 4 Département de psychologie, Université du Québec à Chicoutimi, Saguenay, Québec, Canada

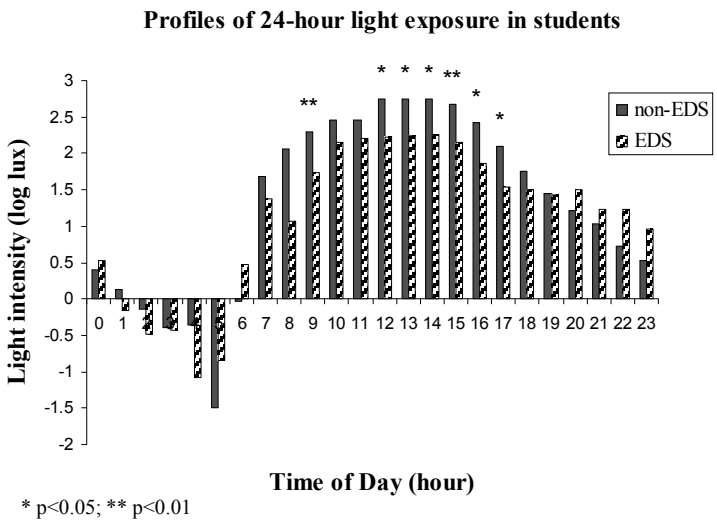
Introduction: Adolescents and young adults were acknowledged as a population at increased risk of daytime sleepiness. Recent studies showed that bright light exposure decrease subjective sleepiness. The objective was to assess whether excessive daytime sleepiness (EDS) in students who work during the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

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

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

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

Figure 1.



P043

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

Grundy A.[1], Sanchez M.[1], Richardson H.[1], Tranmer J.[1,2], Graham C.[3], Aronson K.[1].
[1]Department of Community Health and Epidemiology, Queen's University, Kingston, ON, Canada [2]School of Nursing, Queen's University, Kingston, ON, Canada [3]Department of Anatomy and Cell Biology, Kingston, ON, Canada

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

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

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

Discussion: Since peak melatonin production occurred at night among all workers, to avoid false conclusions regarding the associations of sleep duration and light intensity with melatonin

levels, confounding by circadian rhythm must be taken into account in future studies. In this study sleep duration was not correlated with urinary melatonin levels, suggesting it may not be a good proxy for melatonin production.

Support: CIHR Transdisciplinary Training Program in Cancer Research; Breast Cancer Action Kingston; Programme of Research in Environmental Etiology of Cancer, NCIC.

P044

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

Davidson, F., Ironside, S., & Corkum, P.
Department of Psychology, Dalhousie University, Halifax, Nova Scotia, Canada

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

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

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

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

P045

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

R.Ochoa-Sánchez [1], G. Spadoni [3], A. Bedini [3] , M. Mor [4], S. Rivara [4], F. Fraschini [5] , G.Tarzia [3] , G. Gobbi [1,2]
[1]Neurobiological Psychiatry Unit, Dept. of Psychiatry, McGill Univ. [2]Centre de Recherche Fernand-Seguin, Univ. of Montreal, Canada. [3] Inst. of Pharmaceutical Chemistry and Toxicology, Univ of Urbino, Italy. [4] Pharmaceutical Dept. University of Parma, Italy. [5] Dept. of Pharmacology, Chemiotherapy and Medical Toxicology, Univ.of Milan, Italy.

Introduction: Melatonin is a neurohormone implicated in the regulation of sleep and circadian rhythms and binds two G-protein coupled brain receptors, MT_1 and MT_2. The differential role of these two receptors in sleep function is completely unknown. Here we propose a novel drug acting as an MT_2 receptor partial agonist, called UCM765 (*N*-{2-[(3-Methoxyphenyl)-phenylamino]ethyl}acetamide), with a 100-fold higher affinity for the human recombinant receptor MT_2 ($pK_i = 10.18$) than for MT_1 ($pK_i = 8.38$) .

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

Results: Rats treated with UCM765 exhibited a shorter latency (min) of onset of the first slow wave sleep (SWS) episode compared to control ($p<0.05$) similar to diazepam. The UCM 765 also increased the total duration (min) of the SWS ($p<0.05$). Then, we administered the selective MT_2 antagonist 4-PPDOT (10mg/kg) prior to UCM765. The antagonist 4-PPDOT blocked the effects on the latency of SWS ($p=0.02$) as well on the duration of SWS ($p=0.006$). Results for paradoxical sleep (PS, or REM sleep) indicate that UCM765, prolonged the latency of the first episode of PS ($p<0.05$), similar to diazepam, but did not change the duration or quantity of PS episodes. Next, we assessed the properties of UCM765 (40 mg/kg, six times per day, every 4 hours) across 24 hours. Remarkably, the 24-hour EEG and EMG showed that UCM765 increases SWS only during the inactive or light phase ($p=0.006$) and not during the active or dark phase.

Conclusion: These results suggest that the MT_2 partial agonist UCM765 may be a novel and selective hypnotic drug that increases SWS (restorative sleep) during the sleeping period with no effect during active time.

Support: CIHR

P046

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

Sasseville A[1], Houle J[1], Hebert M[1]
[1]CRULRG, Université Laval, Quebec City, QC, Canada

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

Methods: Ten participants (5 M, 5 F) were tested on two consecutive nights, during which the VAS and the Conners' continuous performance test 2 (CPT II) were performed at 23h30, 1h30, 3h30 and 5h30. First night served as baseline where subjects were maintained awake and tested under less than 5 lux. On the second night, they were exposed to two 500 $\mu W/cm^2/s$ 30 minutes pulses of light at 3h00 and 5h00, produced by a blue-enriched white LED source (Litebook^{TM}).

Since participants wore blue blockers for the first pulse, the light source was set so that 500 $\mu W/cm^2/s$ of light was received by the eyes behind the glasses.

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

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

P047

POLYSOMNOGRAPHIC SLEEP ACROSS THE CIRCADIAN AND MENSTRUAL CYCLES IN HEALTHY FEMALES

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

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

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

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

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

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

References:

1. Dijk DJ and Czeisler CA. Contribution of the Circadian Pacemaker and the Sleep Homeostat to Sleep Propensity, Sleep Structure, Electroencephalographic Slow, and Sleep Spindle Activity in Humans. J Neuroscience 1995; 15(5):3526-3538.

Support: Research supported by the Canadian Institute of Health Research (CIHR)

P048

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

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

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

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

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

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

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

References:

2. Driver HS, Dijk DJ, Werth E, Biedermann K, Borbély AA. Sleep and the Sleep Electroencephalogram across the Menstrual Cycle in Young Healthy Women. J Clin Endocrinol Metab 1996; 81(2):728-735.
3. Baker FC, Kahan TL, Trinder J and Colrain. Sleep Quality and the Sleep Electroencephalogram in Women with Severe Premenstrual Syndrome. Sleep 2007; 30(10):1283-1291.

Support**:** Research supported by the Canadian Institute of Health Research (CIHR).

PHYSIOLOGY & NEUROPHYSIOLOGY

P049

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

Curie T.[1], Mongrain V.[1], Dorsaz S.[1], Maret S.[1], Emmenegger Y.[1], Franken P[1].
[1]Center for Integrative Genomics, University of Lausanne, Lausanne, Switzerland

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

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

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

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

This work was supported by the Swiss National Foundation (3100A0-111974), the Novartis Consumer Health Foundation, and the Marie Curie Intra-European Fellowship (PIEF-GA-2008-221254). *Per2Luc* mice were generously provided by Dr. J.S. Takahashi.

P050

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

Mongrain V.[1], Curie T.[1], Gip P.[2], Heller H.C.[2], Franken P.[1,2]
[1]Center for Integrative Genomics, University of Lausanne, Lausanne, Switzerland; [2]Stanford University, Stanford, CA, USA.

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

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

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

Discussion: While the larger SD-mediated increase in corticosterone observed in D2 mice could partly explain the larger increase in *mPer* expression in this strain, a higher stress response seems not to underlie the lower EEG delta power rebound after SD in this strain. The findings also indicate that stress does not importantly contribute to the homeostatic regulation of EEG delta power.

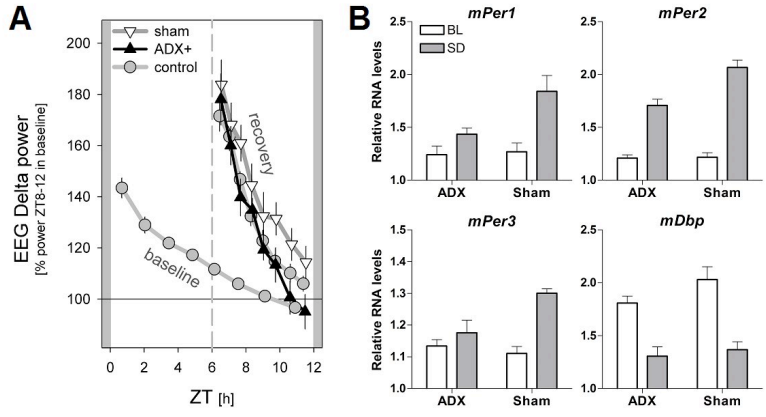


Figure 1: A) Dynamics of EEG delta power during the Light periods of baseline and recovery after 6h SD before and after ADX or sham surgery. **B**) Forebrain expression of *mPer1-3* and *mDbp* at ZT6 after ADX or sham surgery for baseline conditions (BL) or after 6h SD.

References: Franken et al., *J Neurosci*, 21, 2610-21, 2001; *BMC Neurosci*, 8, 87; 2007; Hairston et al., *Neurosci Lett* 315, 29-32, 2001; Tobler et al., *Neurosci Lett* 35, 297-300, 1983.
Research supported by the Univeristy of Lausanne, the NIH (MH67752), FNS (3100A0-111974), and an NSERC fellowship.

P051

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

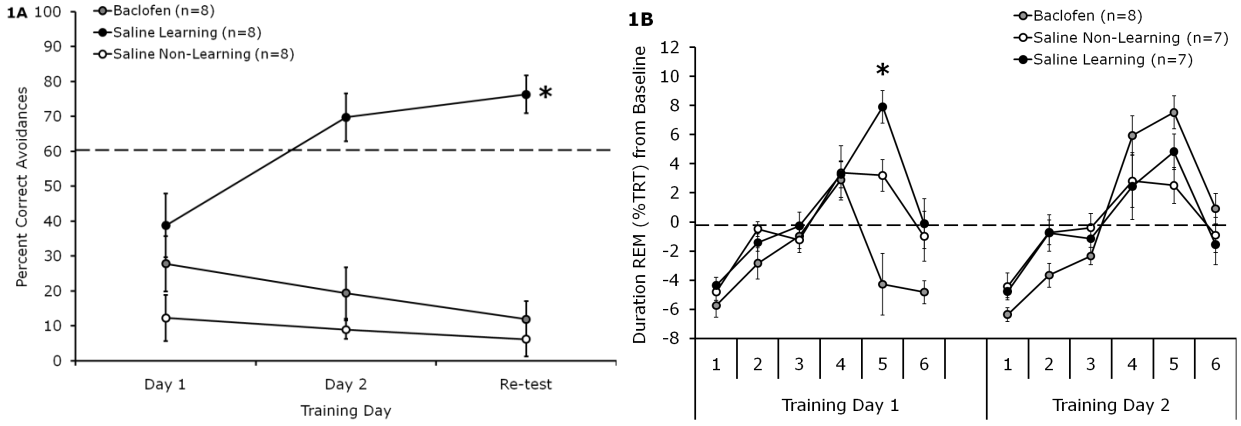
Fogel S.[1], Smith C.[2] and Beninger R.[1]
[1]Centre for Neuroscience Studies, Queen's University, Kingston, ON, Canada; [2]Department of Psychology, Trent University, Peterborough, ON, Canada

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

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

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

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



Support. Natural Sciences and Engineering Research Council of Canada to CS and RB.

P052

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

Bah T.M.[1], Laplante F.[4], Kaloustian S.[1], Sullivan R.[2,4], Rousseau G.[1,3], Godbout R.[1,2]
[1]Centre de Biomédecine, Hôpital du Sacré-Cœur de Montréal ; [2]Département de Psychiatrie, Université de Montréal (Qc), Canada ; [3]Département de Pharmacologie, Université de Montréal (Qc), Canada ; [4]Centre de recherche Fernand-Seguin, Hôpital Louis-H Lafontaine, Montréal (Qc), Canada.

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

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

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

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

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

Supported by: the Canadian Institutes of Health Research and the Natural Science and Engineering Research Council of Canada.

P053

$GABA_B$-MEDIATED INHIBITION PLAYS A CRITICAL ROLE IN MEDIATING REM SLEEP ATONIA

Brooks, P.L. and Peever, J.H.
Dept. Cell & Systems Biology and Physiology, University of Toronto, Toronto, ON, Canada

Introduction: Skeletal muscle tone is potently suppressed during REM sleep. We recently found that REM sleep atonia could not be prevented in masseter muscles by blockade of glycine and $GABA_A$ receptors at the trigeminal motor pool. REM atonia persisted even when glycine- and $GABA_A$-mediated inhibition was blocked and high doses of AMPA were simultaneously applied. Accordingly, we concluded that REM atonia is triggered by an unidentified inhibitory

mechanism(s). Although $GABA_B$ receptors are present on somatic motoneurons and postsynaptically inhibit them, the role of these receptors in mediating REM atonia is unknown. The aim of this study was to determine if $GABA_B$ receptors play a role in suppressing muscle tone during REM sleep.

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

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

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

P054

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

Burgess C.R. and Peever J.H.
Department of Cell and Systems Biology, University of Toronto, Toronto, ON, Canada

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

Methods: We used reverse microdialysis to apply phenylephrine (an α_1-noradrenergic receptor agonist) onto trigeminal motoneurons during cataplectic episodes in freely-behaving hypocretin KO mice. We quantified levels of masseter muscle tone during cataplexy (and across the sleep-wake cycle) before and after noradrenergic excitation of trigeminal motoneurons. Cataplexy and sleep-wake state were determined using EEG, EMG (neck and left/right masseters) and videography.

Results: We quantified 14 episodes of cataplexy while applying either aCSF (n=7) or 1mM phenylephrine (n=7) at the trigeminal motor pool in 4 hypocretin KO mice. We showed that masseter muscle tone was potently suppressed below waking levels during individual cataplectic attacks ($p=0.003$). Importantly, we found that increasing noradrenergic drive by activating α_1-noradrenergic receptors at the trigeminal motor pool significantly increased masseter tone during cataplexy ($p=0.004$); however, such activation did not restore masseter tone to waking levels ($p=0.008$).

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

P055

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

Schwarz P.B., Yee N., Mir S. and Peever J.H.
Departments of Physiology & Cell and Systems Biology, University of Toronto, Toronto, ON, Canada

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

Methods: To test this hypothesis, we used reverse-microdialysis to apply an α_1-adrenoceptor agonist (phenylephrine) and glutamate-receptor agonist (AMPA) and antagonist (CNQX) onto trigeminal motoneurons in both anaesthetized and freely-behaving rats. This allowed us to determine whether noradrenergic neurotransmission onto trigeminal motoneurons affects masseter muscle tone by increasing spontaneous motoneuron activity or whether it acts to amplify prevailing glutamate-driven excitation.

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

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

P056

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

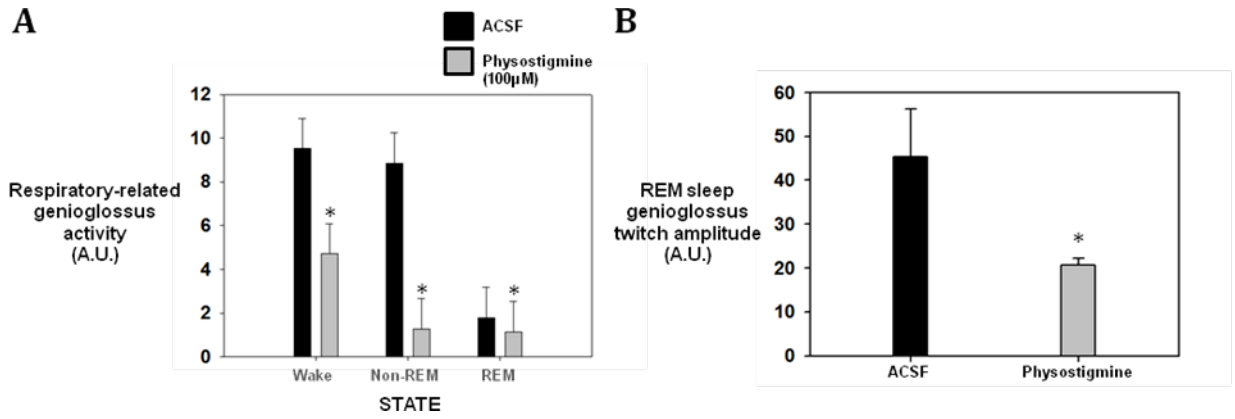
Grace K.[1], Liu H.[1], Nolan P.[2], Horner R.[1]
[1]Departments of Medicine and Physiology, University of Toronto, Toronto, ON, Canada; [2] The Conway Institute of Biomolecular and Biomedical Research, University College Dublin, Dublin, Ireland

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

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

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

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



Supported by: CIHR

P057

EFFECT OF ETHANOL ON MEDULLARY RESPIRATORY ACTIVITY AND SLEEP

LM Vecchio[1], H Liu[2], S Harding[3], A Le[3], RL Horner[1, 2]
Departments of Physiology[1] and Medicine[2], University of Toronto, and Centre for Addiction and Mental Health[3]

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

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

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

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

Supported by: CIHR

P058

PKA-MEDIATED MODULATION OF RESPIRATORY-DRIVE TRANSMISSION TO HYPOGLOSSAL MOTONEURONES *IN VIVO*

DuBord M.-A., Liu H., Horner R. L.
Departments of Medicine and Physiology, University of Toronto, Toronto, Ontario, Canada

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

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

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

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

Supported by: Canadian Institutes of Health Research

P059

NOVEL MECHANISM UNDERLYING OPIOID-INDUCED RESPIRATORY DEPRESSION: SUPPRESSION OF MOTOR DRIVE FROM THE MEDULLA TO UPPER-AIRWAY MUSCLES

Hajiha M., DuBord M.-A., Liu H., Horner R. L.
Departments of Medicine and Physiology, University of Toronto, Toronto, ON, Canada.

Introduction: Upper-airway obstruction and respiratory depression are serious clinical hazards associated with the administration of opioid analgesics. However, it has not been determined whether opioids directly suppress the activity of the central respiratory motoneurones that ultimately constitute the source of motor drive to the respiratory muscles. Furthermore, it has also recently been demonstrated that opioids induce acetylcholine release at the medullary

hypoglossal motor nucleus, the source of motor outflow to the genioglossus, an important lingual respiratory muscle that helps maintain the upper airway open for effective breathing. Acetylcholine at the hypoglossal motor nucleus has an inhibitory effect on motoneuronal activity via muscarinic-receptor stimulation, but the physiological relevance of this opioid-induced acetylcholine release has not been established. We hypothesized that ***(1)*** local delivery of the μ-opioid-receptor agonist fentanyl into the hypoglossal motor nucleus will suppress genioglossal muscle activity *in vivo*, and that ***(2)*** a component of this suppression is mediated by acetylcholine.

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

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

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

Supported by: Canadian Institutes for Health Research, Ontario Thoracic Society

P060

OPIOID-SENSITIVE SITE IN THE MEDULLA UNDERLYING SUPPRESSION OF BREATHING IN THE SLEEPING ADULT RAT

Gaspard Montandon and Richard L. Horner
Departments of Medicine and Physiology, Faculty of Medicine, University of Toronto. ON

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

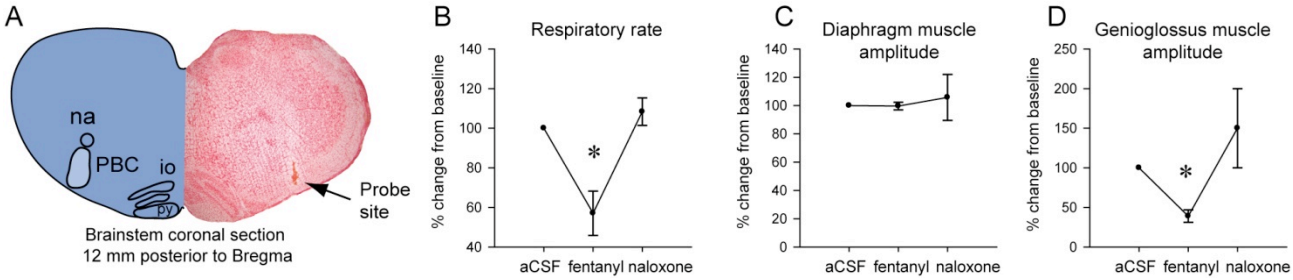
Methods. We recorded activities of the diaphragm muscle and the genioglossus muscle of the tongue - an important muscle involved in upper airway patency - in anaesthetized or freely-behaving adult rats with exogenous drugs applied to the PBC by microdialysis probes.

Results. In anaesthetized rats (n=7), application of the μ-opioid receptor agonist fentanyl (100 μM) to the PBC (panel A) decreased respiratory rate ($p=0.004$, panel B), without affecting diaphragm muscle amplitude ($p=0.81$, panel C), and suppressed genioglossus muscle activity ($p=0.007$, panel D), with this depression reversed by the antagonist naloxone (100 μM).

Preliminary data in 2 freely behaving rats showed that fentanyl (150 μM) at the PBC also reduced respiratory rate in non-REM sleep (~30%) but not wakefulness (~3%).

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

Support. GM was the recipient of the Canadian Lung Association - CIHR Postdoctoral Fellowship Award. Research was supported by the CIHR.



P061

SLEEP LOSS REDUCES APNEA-INDUCED RESPIRATORY NEUROPLASTICITY

Tadjalli A.[1], Duffin J[2]. and Peever J.H.[1,2]
Departments of Physiology[2] and Cell & Systems Biology[1], University of Toronto, Canada.

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

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

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

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

P062

N-REM SLEEP SLOW OSCILLATIONS AMPLITUDE AND DENSITY IN THE YOUNG AND MIDDLE-AGED MEN AND WOMEN

Viens, I[1,3], Lafortune, M[1,3], Poirier, G[1], Paquet, J[1], Barakat, M[1,3], Vandewalle, G[1,3], Martin, N[1,3], Robillard, R[1,2,3] and Carrier, J[1,2,3]

[1]Centre d'étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Cœur de Montréal, Montréal, Qc, Canada, [2]Centre de recherche de l'Institut Universitaire de Gériatrie de Montréal, Montréal, Qc, Canada, [3]Département de psychologie, Université de Montréal, Montréal, Qc. Canada

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

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

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

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

Support: This research was supported by scholarships and grants from the Canadian Institutes of Health Research (CIHR), the Fonds de Recherche en Santé du Québec (FRSQ) and the Natural Sciences and Engineering Research Council of Canada (NSERC).

P063

ETHNIC DIFFERENCES IN SLEEP ARCHITECTURE IN HEALTHY, NORMOTENSIVE YOUNG ADULTS ARE ASSCOCIATED WITH NOCTURNAL HEART RATE VARIABILITY

Saletin J.[1], Klick B.[1], Smith M.[1]
[1]Behavioral Sleep Medicine Program, Department of Psychiatry and Behavioral Sciences, Johns Hopkins School of Medicine, Baltimore, MD, USA.

Introduction: Recent preliminary work from our group[1] has found ethnic disparities in sleep architecture, notably, healthy African-Americans demonstrated decreased slow-wave sleep relative to Caucasian-Americans. These disparities exist even in young, healthy sleepers, self-reported as healthy sleepers. A common hypothesis points to the increased global stress accompanying minority status. While subjective stress reports and hypothalamic-pituitary-adrenal axis measures have failed to explain the effect, the use of heart rate variability (HRV) analyses, known to have marked ethnic disparities, offers new insight into both differences in sleep architecture and how sleep may relate to greater cardiac health and outcome.

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

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

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

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

Support (optional): This work was supported by NINDS R21NS051771 (MTS).

P064

SLEEP-RELATED BREATHING PROBLEMS AFFECT BLOOD PRESSURE REGULATION IN CHILDREN

Wade TJ[1], Reid GJ[2], Fitzgibbon LK[1], Coverdale NS[1], Cairney J[3], O'Leary DD[1]
[1] Department of Community Health Sciences, Brock University, St Catharines, Ontario
[2] Departments of Psychology, Family Medicine, and Pediatrics, The University of Western Ontario, London, Ontario
[3] Departments of Family Medicine and Psychiatry, McMaster University, Hamilton, Ontario

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

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

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

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

P065

THE IMPACT OF SLOW WAVE SLEEP (SWS) AND SLEEP FRAGMENTATION ON FATIGUE VERUS DAYTIME SLEEPINESS IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA (OSA)

Ghashghai A., Chung SA, and Shapiro CM
Sleep Research Laboratory, Department of Psychiatry, University Health Network; University of Toronto and International Sleep Clinic, West Parry Sound Health Centre, Ontario, Canada

Introduction: Fatigue and daytime sleepiness are common complaints in OSA patients. Although fatigue can be a result of many pathophysiological conditions, it has been documented

that more patients report fatigue, as compared to sleepiness, in this population. The aim of this study was to determine if fatigue changes after CPAP treatment and whether or not changes in fatigue (as opposed to daytime sleepiness) can be correlated with amount of SWS and degree of sleep fragmentation. This finding could suggest management strategies of fatigue in sleep clinic patients.

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

Results: Of the 60 patients, 40 were males (age=56 ± 14) and 20 were females (age=57 ± 12). Statistical analysis was performed for males and females separately as well as males and females together. After CPAP treatment, there was a significant ($P<0.05$) decrease in fatigue, daytime sleepiness and arousal index (sleep fragmentation). SWS percentage, however, did not increase significantly after CPAP treatment. The decrease in fatigue was moderately correlated with the decrease in arousal index only in males ($P<0.05$, $r=0.4$, $r^2=0.16$). No significant correlations were observed in females and when the males and females were mixed. Daytime sleepiness did not have any significant correlation with neither SWS nor sleep fragmentation before and after treatment in any of the populations.

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

P066

FREQUENCY OF OBSTRUCTIVE SLEEP APNEA IN PAEDIATRIC PATIENTS WITH ADENOTONSILLAR HYPERTROPHY

CAPUA C, CHUNG SA, MARCU S, JOVANOVIC D, AND SHAPIRO CM.
Youthdale Child & Adolescent Sleep Centre, Ontario, Canada

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

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

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

p=0.077). Lastly, for children with enlarged tonsils, the incidence of OSA was over forty times greater than for children with normal sized tonsils (OR=40.9, 95%CI: 4.5-372.7).

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

P067

OBSTRUCTIVE SLEEP APNEA IS HIGHLY PREVALENT AMONG KIDNEY TRANSPLANTED PATIENTS: RESULTS OF THE SLEPT STUDY

Dunai A.[1], Molnar M. Zs.[2,3], Novak M.[3,4], Czira M. E.[3], Lindner A.[3], Lazar A. S.[3], Fornadi K.[3,5], Zoller R.[3], Szentkiralyi A.[3], Mucsi I.[1,3]

[1]1st Department of Internal Medicine, Semmelweis University, Budapest, Hungary; [2]Department of Transplantation and Surgery, Semmelweis University, Budapest, Hungary; [3]Institute of Behavioural Sciences, Semmelweis University, Budapest, Hungary; [4]Department of Psychiatry, University of Toronto, Toronto, Canada; [5] Department of Neurology, Semmelweis University, Budapest, Hungary

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

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

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

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

P068

PERIODIC LIMB MOVEMENTS IN SLEEP AND CARDIOVASCULAR RISK IN KIDNEY TRANSPLANTED PATIENTS

Novak M.[1,2,3], Mucsi I.[1,3], Czira E.M.[1], Lindner A.[1,] Fornadi K.[1,4], Lazar A.S.[1], Dunai A.[3], Zoller R.[1,3], Szentkiralyi A.[1], Madarasz Cs.[1], Kiss Z.[5],Molnar M. ZS.[1,6]

[1] Institute of Behavioural Sciences, Semmelweis University, Budapest, Hungary; [2]Department of Psychiatry, University Health Network, University of Toronto, Toronto, Canada; [3]1st Department of Internal Medicine, Semmelweis University, Budapest, Hungary; [4]Department of Neurology, Semmelweis University, Budapest, Hungary; [5]Amgen Limited Hungary, Budapest, Hungary. [6] Department of Transplantation and Surgery, Semmelweis University, Budapest, Hungary;

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

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

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

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

Support: The study was supported by the National Research Fund (OTKA, F -68841), the Janos Bolyai Research Scholarship of the Hungarian Academy of Sciences (Molnar MZs and Novak M) and SomnoMedics GmbH.

P069

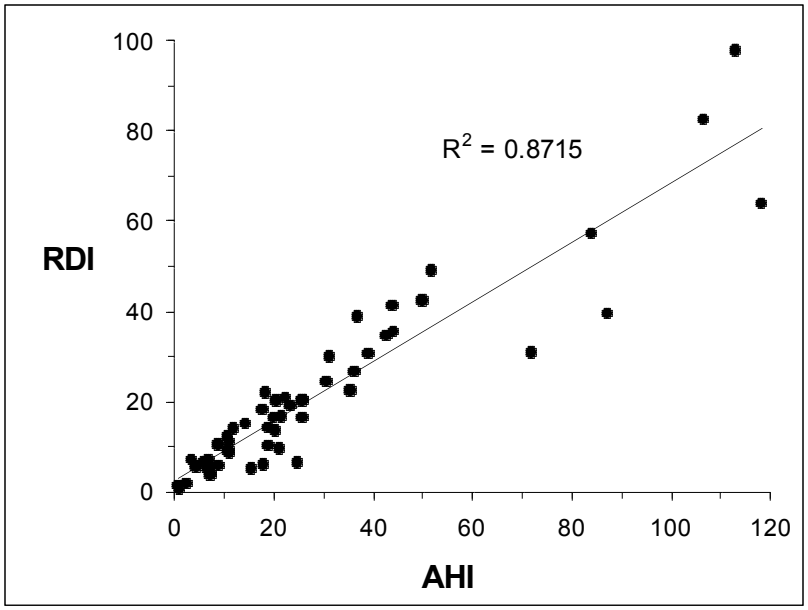
EVALUATION OF A PORTABLE MONITOR COMPARED WITH POLYSOMNOGRAPHY FOR THE DIAGNOSIS OF OBSTRUCTIVE SLEEP APNEA

Driver HS.[1,2], Bjerring K.A.[2], Toop F.[2], Pereira E.[2], Stewart S.C., Munt P.[1], Fitzpatrick M.F.[1]
Sleep Disorders Laboratory, Kingston General Hospital and Departments of Medicine[1] and Psychology[2], Queen's University, Kingston, Ontario, Canada

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

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

Results: For 53 patients (20M/33F) aged 20 to 73 years (mean ± SD: 52 ± 12) and BMI 33.4 ± 7.3 kg/m^2 (range 21.4 - 52.7), the AHI was 28.7 ± 28.3 while the RDI was 21.5 ± 20.1. There was good correlation between the RDI and AHI (Pearson correlation r = 0.93) which accounted for 87% of the variance (R^2 = 0.872). The mean difference AHI - RDI (PSG versus PM) showed under-reporting using the PM by 7.2 ± 11.9 events per hour. For an AHI ≥10 the sensitivity (true-positive), as well as the specificity (true-negative), of the PM were 92% . For severe OSA (AHI ≥ 30), the PM sensitivity was 82% and specificity 100%. All 17 cases of severe OSA had an RDI > 15 on the PM.



Conclusion: Tested in the laboratory, the PM was highly sensitive and specific in evaluating moderate to severe OSA.

Support: MediByte portable monitors and the associated consumables were provided by Braebon Medical Corporation.

P070

ANALYSIS OF HOSPITAL DISCHARGE DATA TO DETERMINE THE USE OF CONTINUOUS POSITIVE AIRWAY PRESSURE IN CANADIAN HOSPITALIZED PATIENTS WITH SLEEP APNEA

Kathy Spurr, RRT, RPSGT, MHI (c)[1], Adam Webber, MSc.[2], Debra Morrison, MD, FRCPC[3], Robert W Gilbert, PhD[1]

[1] School of Health Sciences, Dalhousie University, Halifax, Nova Scotia, Canada
[2] Addiction Prevention and Treatment Services, Capital Health, Halifax Nova Scotia, Canada
[3] Faculty of Medicine, Dalhousie University, Halifax Nova Scotia, Canada

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

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

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

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

P071

THE MOUTH LEAK SYNDROME IN PATIENTS INITIATING NCPAP FOR OSAS

Baltzan M.A.,[1,2,3] Garcia-Asensi A.,[3] Parenteau M,[3] Dabrusin R.,[1] Tanzimat G,[3] Kassissia I.,[3] Wolkove N.[1]

[1] Mount Sinai Hospital Center; [2]Department of Epidemiology, Biostatistics & Occupational Health, McGill University; [3] OSR Medical Sleep Disorders Center; all in Montreal, Canada.

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

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

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

Polysomnographic Findings

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

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

Conclusions: Patients with OSAS treated with nCPAP who develop mouth leak syndrome demonstrate less clinical improvement in OSAS measures as well as more mouth leaks and more sleep disruption due to these mouth leaks.

Support: OSR Medical & the Mount Sinai Hospital Research Foundation

P072

WHAT IS THE LEVEL OF SLEEP KNOWLEDGE IN FAMILY MEDICINE RESIDENTS?

Samuels C.H[1,2], Cohen R[1], Fryer S[1]
Centre for Sleep and Human Performance, Calgary, AB, Canada[1]
Faculty of Medicine, University of Calgary, Calgary, AB, Canada[2]

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

Methods: Family Medicine Residents (N=56) were surveyed using the15-item Sleep Knowledge questionnaire from the SAFER program which assesses knowledge of basic concepts of sleep and circadian biology. Fifty-five percent (21/38) were first year residents; average age was 29 years (range of 25-40), and majority were female (77%). Participants indicated True, False or Don't Know for each item. The authors reviewed and categorized the items into four domains to better differentiate the scope of sleep knowledge. The four domains are 1) Sleep Deprivation *(items 1,3,4,7,8,9,14)* 2) Sleep Hygiene *(items 2,3,6,10,13)* 3) Counter Measures *(items 2,5,10,11,13)* 4) Circadian Biology *(items 11,12,15)*. Items relevant to more than one domain were counted within each appropriate domain. The number (percent correct) of correct responses for each item were tabulated.

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

Table 1 Sleep Knowledge Domains

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

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

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

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

P073

WHAT MAKES SOME PATIENTS WANT TO INVESTIGATE THEIR SLEEP?

Rizzo, D.[1], Bailes, S.[1], Baltzan, M.[2,7,8], Grad, R.[3,5], Kassissia, I.[4,8], Fichten, C.[1,5,6], Creti, L.[1], Libman, E[1,7].

[1]Department of Psychiatry, SMBD-Jewish General Hospital, Montreal, Quebec, Canada; [2]Mount-Sinai Hospital, Montreal, Quebec, Canada; [3]Herzl Family Practice Centre, SMBD-Jewish General Hospital, Montreal, Quebec, Canada; [4]Clinique Plein-Ciel, Montreal, Canada; [5]Department of Psychology, Concordia University, Montreal, Quebec, Canada; [6]Department of Psychology, Dawson College, Montreal, Quebec, Canada; [7]McGill University, Montreal, Quebec, Canada; [8]OSR Medical, Montreal, Quebec, Canada.

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

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

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

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

Support. Canadian Institutes of Health Research

P074

COMPARISON OF SYNCHRONIZED LEVEL 1 AND LEVEL 3 SLEEP STUDIES

Sullivan, G. E.; Morehouse, R.; Savoy, A.
Department of Psychiatry, Atlantic Health Sciences Sleep Centre, Saint John, NB, Canada

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

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

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

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

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

P075

A RETROSPECTIVE, OBSERVATIONAL STUDY SHOWING PATIENTS WITH A NORMAL LEVEL III SLEEP STUDY AND NORMAL OSA PRETEST PROBABILITY FACTORS MAY STILL REQUIRE ADDITIONAL INVESTIGATIONS

Fordyce L[1], Samuels C.H[1,2], Oram C[1], Wallins B[1]
Centre for Sleep and Human Performance, Calgary, AB, Canada[1]
Faculty of Medicine, University of Calgary, Calgary, AB, Canada[2]

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

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

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

Table 1

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

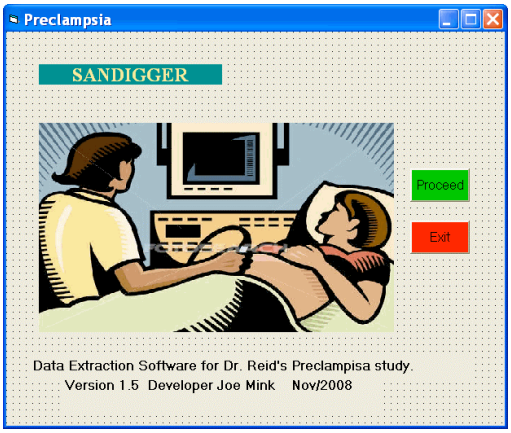
Conclusions: The results were limited due to a small sample size. However, the results did show that 5/113 (5%) of patients having only a level III study would not have been identified and treated for SDB. This would have had an impact on the patients' quality of life. More work is needed in this area.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

P076

SPECIALIZED NEW PROGRAMMING TO FACILITATE RESEARCH ORIENTED ANALYSIS OF POLYSOMOGRAPHIC STUDIES

Mink J, Skomro R. and Reid J. Sleep Disorders Centre, University of Saskatchewan, Saskatoon, SK.

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



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

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

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

(Acknowledgement: Saskatoon Region Health Authority and the Saskatchewan Health Research Foundation).

P077

ARTIFICIAL NEURAL NETWORK SCORING OF HUMAN SLEEP-WAKE STAGES COMBINING SHORT-EPOCH FEATURE EXTRACTION AND POST-PROCESSING INFERENCE RULES

Florian Chapotot Ph.D.[1], Lukas Zoubek B.M.E.[2] and Guillaume Becq Ph.D.[3]
[1] Sleep, Neuroendocrinology and Chronobiology Laboratory, Department of Medicine, The University of Chicago, USA
[2] GIPSA-Lab, Control Systems Department, BP 46, 38402 Saint Martin d'Heres Cedex, FRANCE
[3] PhiTools, Strasbourg, FRANCE

The use of learning machines in the automatic analysis of human sleep-wake stages has shown performance near to inter-expert agreement. However, automatic classifiers are sensitive to small differences in the signal conditioning inherent to the existence of various polysomnographic collection systems and digital file formats. In addition, automatic analysis yields some incoherent results and doesn't always provide the requested flexibility in time resolution, which may vary between countries and species.
Using candidate features selected for their relative independence to biosignal collection parameters, we have developed a new method allowing 1) to train artificial neural network (ANN) from a database of short duration prototypic sleep/wake stage epochs and 2) to infer final scores at a variable duration using a set of implemented expert rules. The PRANA biosignal processing software was used to compute and extract a set of 16 different features from the electroencephalographic and electromyographic signals collected during 48 night recordings performed in 11 healthy adults using ambulatory recorders. Two independent human experts scored sleep/wake stages into 20-s epochs according to the conventional criteria. A database of 1 029 125 2-s epochs including the expert scores and the computer-extracted features was created. Five hundred and six 2-s epochs representative of each sleep/wake stage were manually selected by one expert from a subset of 7 individuals and further used for ANN learning.

Simulation of the automatic scoring system using 20-s epochs showed a 30±10% error rate as compared to consensual expert scores and a Cohen's kappa of 0.56±0.16. The global performance of the sleep-wake stage classification system ranges slightly below inter-scorer agreement (82.8±3.3%).
This new classification method can perform automatic sleep/wake staging with various epoch durations. Improved performance can reasonably be expected by selecting a larger amount of learning epochs and by introducing additional rules mimicking expert decision-making strategies.

This work received financial support from PhiTools SARL (Strasbourg, FRANCE, www.phitools.com) covering Dr. Guillaume Becq salary and providing the PRANA software.

Keywords: Human, Sleep, automatic analysis

P078

THE INFLUENCE OF SLEEP QUALITY ON MENTAL AND PHYSICAL HEALTH IN THE CANADIAN COMMUNITY HEALTH SURVEY

Bauermann, T.M.[1] & Najbor, R.M. [2]
[1]Department of Psychology, Queen's University; [2]Frontenac Community Mental Health Services, Kingston, Ontario, Canada

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

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

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

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

P079

THE DISCRIMINANT VALIDITY OF THE SLEEP PROBLEMS INVENTORY

Bauermann, T.M.[1], Parker, J.D.A. [2], Wood, L.M. [2]
[1]Department of Psychology, Queen's University, Kingston, Ontario; [2] Department of Psychology, Trent University, Peterborough, Ontario, Canada.

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

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

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

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

P080

THERE MAY BE NO FIRST NIGHT EFFECT FOR AMBULATORY POLYSOMNOGRAPHY IN OLDER ADULTS

McInrue, E.[1], Hoehn, J. [1], Cosenza, S. [1], Buenaver, L.[1], Smith M.T.[1]
[1]Behavioral Sleep Medicine Program, Department of Psychiatry and Behavioral Sciences, Johns Hopkins School of Medicine, Baltimore, MD, USA.

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

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

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

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

Support (optional): This work was supported by NIH R0190031365 (MTS).

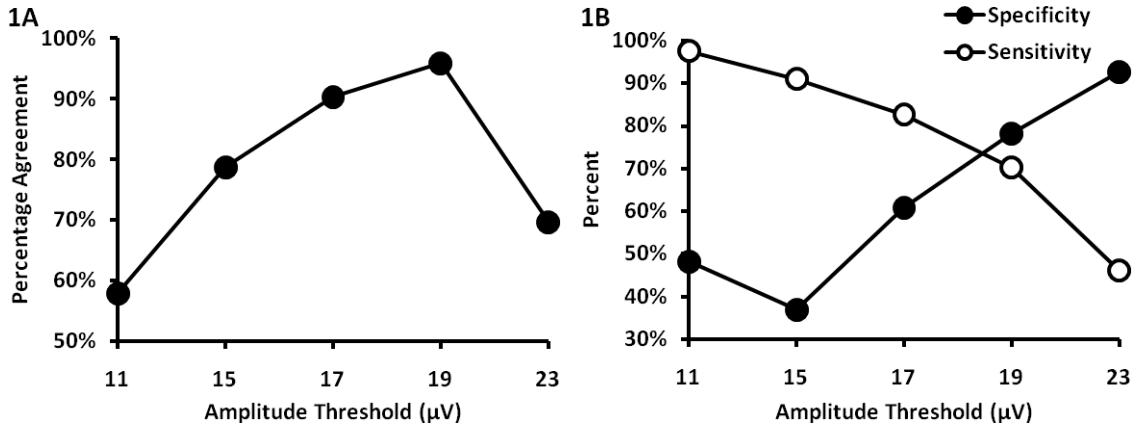
P081

SYSTEMATIC OPTIMIZATION OF AUTOMATED SLEEP SPINDLE DETECTION

Ray LB.[1], Peters KR.[1], Fogel SM.[2], Smith CT. [1]
[1]Department of Psychology, Trent University, Peterborough, Ontario, Canada; [2]Neuroscience Department, Queens University, Kingston, Ontario, Canada

Introduction: The sleep spindle has been implicated in memory consolidation[1] and is thought to serve as a physiological index of intelligence[2]. To identify spindles, most studies utilize manual or automatic spindle detection. Manual scoring methods are labour intensive and may be less reliable due to subjectivity or the expertise of the scorer. To be reliable, automatic methods should have adequate sensitivity (true positive/true positive + false negative) and specificity (true negative/true negative + false positive). Only a few methods for automatic spindle detection are commercially available and validation data are limited[3]. This warrants the need for validation of automatic spindle detection methods. The goal of the current investigation was to develop a systematic method to optimize the use of automated spindle detection.
Methods: Ten polysomnograms (adults: 19-22yr, 5 females) were randomly selected. Spindles were manually scored in the first 25 consecutive epochs of stage 2 sleep for each quartile of the night. Spindles were identified at the C3 scalp location based on two criteria: 1) minimum of 0.5sec in length, and 2) between 12-16Hz. Subsequently, the PRANA® software (PhiTools, Strasbourg, France) spindle detection algorithm was employed to automatically detect spindles at least 0.5sec in length for the same epochs using six different minimum amplitude thresholds (11, 15, 17, 18, 19 & 23μV).
Results: Figure1A shows the percent agreement between manual and automatic spindle detection at varying amplitude thresholds. Percent agreement was highest (95.9%) at 19μV. The sensitivity (70.1%) and specificity (78.2%) were most balanced at this point (figure1B).
Conclusions: Across thresholds, an inverse relationship between sensitivity and specificity was observed. Sensitivity and specificity were balanced at the point where manual and automatic detection totals had the highest percent agreement. Selecting amplitude thresholds for spindle

detection based on systematic benchmarking data may validate methods and improve reproducibility of experimental results.



[1] Fogel S & Smith C (2006) Learning-dependent changes in sleep spindles and stage 2 sleep. *J Sleep Res* 15:250-5.
[2] Fogel S, et al (2007) Sleep spindles and learning potential. *Behav Neuro* 121(1):1-10.