World Sleep 2017

OCTOBER 7-11, 2017 • PRAGUE, CZECH REPUBLIC

A JOINT CONGRESS OF WORLD ASSOCIATION OF SLEEP MEDICINE AND WORLD SLEEP FEDERATION

worldsleepcongress.com

World Sleep 2017 organized by

WORLD SLEEP SOCIETY

ADVANCING SLEEP HEALTH WORLDWIDE

Final Program

14th WORLD SLEEP

World Sleep Society Founded by

WORLD SLEEP FEDERATION

WORLD FEDERATION OF SLEEP RESEARCH & SLEEP MEDICINE SOCIETIES

WASM

WORLD SLEEP SOCIETY

ADVANCING SLEEP HEALTH WORLDWIDE

Founded by WORLD ASSOCIATION OF SLEEP MEDICINE and WORLD SLEEP FEDERATION

Join us for the 15th World Sleep Congress

World Sleep 2019

SEPTEMBER 20-25, 2019 • VANCOUVER, CANADA

A JOINT CONGRESS OF WORLD ASSOCIATION OF SLEEP MEDICINE AND WORLD SLEEP FEDERATION

worldsleepcongress.com

Dear Colleagues and Friends,

Welcome to World Sleep 2017 the first congress of the new World Sleep Society, which represents combining together the World Association of Sleep Medicine (WASM) and World Sleep Federation (WSF). This year's program is extremely robust, featuring over 278 hours of scientific content that includes 16 keynotes, 102 symposia, 18 courses, 138 oral abstract presentations, and 1,168 accepted abstracts.

The congress is hosted by the historic-city Prague, located in the heart of Europe, and brings the best in sleep medicine to Prague by providing an international discussion forum of sleep professionals from the entire world. It focuses particularly on the interdisciplinary character of our field. Sleep scientists, clinicians, educators, technologists, and trainees from around the world will meet to advance knowledge on sleep science, chronobiology, sleep in public health, sleep health and the diagnosis and treatment of sleep-wake disorders. We seek to maximize learning both from formal presentations by the leading experts in their fields and from informal discussion groups emphasizing opportunities for your participation.

Your involvement in this congress is greatly valued. You may learn and share knowledge and skills that will advance sleep health throughout the world. We are very pleased you have joined us for the science, learning, collegiality, networking and social events at this historic first congress of the World Sleep Society, the 14th congress of the combined WASM and WSF.

Sincerely,

World Sleep 2017 Program Committee

Richard Allen, PhD
Colin Espie, PhD, DSc
Clete Kushida, MD, PhD
Soňa Nevšímalová, MD, DrS
Allan O'Bryan
Thomas Penzel, PhD
Claudia Trenkwalder, MD
Anthony Williams

5TH CONGRESS OF THE INTERNATIONAL PEDIATRIC SLEEP ASSOCIATION

IPSA2018.COM

April 27-29, 2018

IPSA 2018
PARIS · FRANCE

International Pediatric Sleep Association pedsleep.org

VENUE

IPSA 2018 will take place in The Palais des Congrès de Paris.

Address:
Palais des Congrès de Paris
2 place de la Porte Maillot,
75017 Paris

ABSTRACTS

Abstract submission starts November 1, 2017 with a **deadline of March 31, 2018**. Accepted abstracts will be published on IPSA's website as part of the congress proceedings.

SYMPOSIA

Symposium submission **deadline is January 31, 2018**. Symposium can be submitted and early acceptance will be sent.

KEYNOTE SPEAKERS

Carole L. Marcus, MBBCh
New developments in childhood OSAS
Director of the Sleep Center at Children's Hospital of Philadelphia, Perelman School of Medicine, University of Pennsylvania (US)

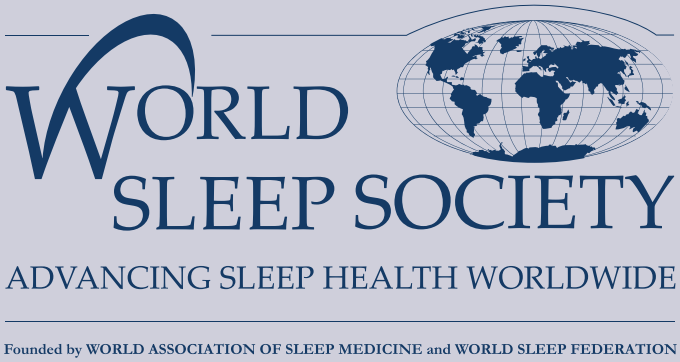
Monique K. LeBourgeois, PhD
Sleep Regulation in early childhood: Insights into the development of behavioral sleep problems
Department of Integrative Physiology, University of Colorado, Boulder (US)

Marion Leboyer, MD, PhD
Microbiote and psychiatric disorders in children
Hôpital Albert Chenevier,
University of Paris Est (UPEC) (France)

Mark S. Blumberg, PhD
REM sleep twitches and sensorimotor development
Director of The Delta Center,
University of Iowa, Iowa City (US)

International Pediatric Sleep Association

FOR MORE INFORMATION VISIT, IPSA2018.COM



Mission Statement

The fundamental mission of the World Sleep Society is to advance sleep health worldwide. World Sleep Society will fulfill this mission by promoting and encouraging education, research and patient care throughout the World, particularly in those parts of the world where the practice of sleep medicine is less developed. World Sleep Society will act as a bridge between different sleep societies and cultures, supporting and encouraging worldwide exchange of clinical information and scientific studies related to sleep medicine. World Sleep Society will seek to encourage development and exchange of information for world-wide and regional standards of practice for sleep medicine.

Goal and Purpose

The goal and purpose of the World Sleep Society is to advance knowledge about sleep, circadian rhythms, sleep health, and sleep disorders worldwide, especially in those parts of the world where this knowledge has not advanced sufficiently. This endeavor will promote clinical and scientific information for scientists, health care personnel, and the general public. The World Sleep Society will foster international exchanges among scientists, physicians, psychologists, nurses, physician assistants, technologists, and other medical and research personnel interested in the sleep field. In this manner it is expected that sleep science and medicine can advance for all populations to improve the quality of life of individuals throughout the world. The World Sleep Society will promote world-wide sleep health by advancing public education, supporting public policies related to sleep health, and supporting sleep research.

World Sleep Society currently represents over 823 individual members, 26 societies. It is located in over 64 countries and will continue to operate programs of both the World Sleep Federation (WSF) and World Association of Sleep Medicine (WASM). Programs consist of education, awareness, and member services.

Program Committee

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Biologist, Sleep Research and Chronobiology
Department of Pharmacology and Toxicology,
University of Zurich
(Switzerland)

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General Information

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3270 19th Street NW • Suite 109
Rochester, MN 55901
Ph: (507) 316-0084
Fax: 001-612-465-5357
www.worldsleepsociety.org

Meeting Location
Prague Congress Centre
5. kvetna 65
140 21 Prague 4
Czech Republic
630 80 24

Opening Ceremony Location
Municipal House
nám. Republiky 5
111 21 Praha 1-Staré Město
Czech Republic

Gala Dinner Location
Slovansky dum (Slavic House)
Na Příkopě 22, 110 00 Praha 1-Nové Město, Czechia

Closing Ceremony Location
Prague Congress Centre
5. kvetna 65
140 21 Prague 4
Czech Republic
630 80 24

Registration materials (including badges, final programs, tickets, etc.) will be provided at the registration counter located at the Prague Congress Centre. Tickets are required for entry for Saturday and Sunday's pre-congress courses, Monday's gala dinner. Tickets can be purchased online at www.worldsleepcongress.com or at the registration desk.

Badge Identification
All congress participants and guests must wear a World Sleep 2017 congress badge. Badges allow entrance to the scientific sessions and access to the convention center. Your cooperation with this policy is appreciated. Recycle your badge holder after the congress by dropping it off at the registration desk area. Replacement badges can be obtained at the registration desk for a fee.

Exhibition will be closed and monitored by security before 10:00AM and after 4:00PM. Exhibitor personnel are allowed in their booths one hour prior and one hour after exhibition times. Exhibitor space is jointly occupied by main keynote exit corridor and delegates will be using the corridor throughout the congress presentation times. We require that all exhibit booths be staffed only during exhibit hours.

Speaker Ready Room
Presenting speakers can use the Speaker Ready Room to upload their PowerPoint presentations, test software, and make changes and adjustments to their presentations. Speakers are required to upload their presentation in the speaker ready room 12 hours prior to their session. A technician will be available to assist as needed.

Press Room
Members of the press are encouraged to utilize the Speaker Ready Room to work on their stories, access the internet, and other resources that are needed. Press are encouraged to attend the Press Meeting on Monday, October 9, 2017 from 12:00-12:30pm.

Membership Booth
World Sleep Society/WASM membership records may be reviewed and purchased at the Registration Desk. Details about membership are available on www.worldsleepsociety.org.

On-site Registration Hours

Day	Date	Desk Opens	Desk Closes
Friday	October 6	5:00pm	8:00pm
Saturday	October 7	7:00am	5:00pm
Sunday	October 8	7:00am	5:00pm
Monday	October 9	7:00am	7:00pm
Tuesday	October 10	7:00am	7:00pm
Wednesday	October 11	7:00am	7:00pm

Exhibits and Exhibiting Hours

Day	Date	Desk Opens	Desk Closes
Monday	October 9	10:00am	4:00pm
Tuesday	October 10	10:00am	4:00pm
Wednesday	October 11	10:00am	4:00pm

Speaker Ready Room Hours

Saturday	October 7, 2017	7:00am – 5:00pm
Sunday	October 8, 2017	7:00am – 5:00pm
Monday	October 9, 2017	7:00am – 7:00pm
Tuesday	October 10, 2017	7:00am – 7:00pm
Wednesday	October 11, 2017	7:00am – 5:00pm

Recording Device Policy
No recording devices, audio or visual, may be used during CME activities. Duplication, distribution, or excerpting of this program, without the express written permission of World Sleep Society, is strictly prohibited. All of the proceedings of this program, including the presentation of scientific papers, are intended for limited publication only, and all property rights in the material presented, including common-law copyright, are expressly reserved by the Faculty, World Sleep Society, and/or CME provider. No statement of presentation made is to be regarded as dedicated to the public domain. Any sound reproduction, transcript or other use of the material presented at this CME activity without the permission of the World Sleep Society and CME provider is prohibited to the full extent of common-law copyright in such material.

Cameras and recording devices are not allowed to be used in the scientific meeting rooms at any time. Violation of this rule could result in removal from the World Sleep 2017 Congress on Sleep Medicine and Prague Congress Centre along with the confiscation of the film and/or recording device.

Electronic Devices
Please turn all electronic devices (cellular telephones, pagers, etc.) to silent mode. As a courtesy to the presenters and other participants, phone calls should be taken outside of the scientific sessions.

Seating
Scientific sessions are filled on a first-come, first-served basis. World Sleep Society along with the Program Committee reviewed the scientific sessions to anticipate demand to match the room size with expected seating. Occasionally, a talk will have higher demand than expected. Seating limits are strictly enforced by the Prague Congress Centre and city of Prague. We encourage delegates to arrive early for best possible seating.

Abstract Supplement
All accepted abstracts will be published in a supplement of Sleep Medicine (Elsevier). The Sleep Medicine abstract supplement should be published and available online (PubMed) by December 2017.

Opening Ceremony

Sunday, October 8, 2017: The Opening Ceremony of World Sleep 2017 will take place in the beautiful Municipal House located in the heart of Prague. This Art Nouveau treasure dominates the site of the former Royal Court. The ceremony will feature award presentations and classic Czech music played by a 65-piece orchestra. We expect over 1,000 delegates to attend this historic opening ceremony of the first joint congress of World Association of Sleep Medicine (WASM) and World Sleep Federation (WSF).

Agenda
6:00pm to 6:30pm
Opening Welcome, awards, and concert introduction
6:30pm to 7:30pm
Concert of the Prague Radio Symphony Orchestra, with conductor T. Brauner
7:30pm to 9:00pm
Reception

All attendees must have official congress badge to attend.

Gala Dinner

Monday, October 9, 2017: The Gala Dinner will feature entertainment, dinner, and beverages. **A ticket will need to be purchased to attend*

The gala will be held at Slovansky dum (Slavic House) in Prague. Join attendees to celebrate and network over dinner and drinks. This is an exclusive event limited to 320 tickets. Purchase a single ticket or a table of eight. Entertainment will be provided by a local jazz band followed by a black light theatre production.

Agenda
7:30pm – 8:00pm
Welcome & Reception
8:00pm – 9:45pm
Dinner & Black light theatre production
9:45pm – 10:30pm
Dessert, coffee & networking

Networking Opportunities

Plan to partake in networking events while attending congress to meet some of the names behind the research. Cost to attend events is included in the registration fee unless otherwise stated. Please make sure to wear your badge to non-ticketed events.

World Sleep Day Networking Event
Monday, October 9, 2017, 1 P.M., Room 221: Join several of our World Sleep Day 2017 delegates and past Distinguished Activity Award winners at the World Sleep Day Networking Event in Prague! You did not need to host an event to join us.

Coffee Breaks
Monday, October 9-Wednesday, October 11: Take a break from the exhibits and stop in for a cup of coffee. Available during exhibition hours at 10:00am and 2:00pm.

Poster Sessions
Monday, October 9-Wednesday, October 11: View young investigator and different abstracts each day during poster sessions.

Closing Ceremony

Wednesday, October 11: The Closing Ceremony will be at the main event venue, Prague Congress Centre. All participants and exhibitors are invited to join. Before leaving, enjoy discussing the latest science and research you discovered at World Sleep 2017.

CME

Continuing Medical Education (CME) Credit for Physicians Accreditation Statement:

This Live activity, World Sleep 2017, with a beginning date of 10/07/2017, has been reviewed and is acceptable for up to 42.25 Prescribed credit(s) by the American Academy of Family Physicians. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

CME is awarded for pre-congress courses, keynote lectures, symposia sessions and technologist workshops. A CME fee of US$25 is payable online with registration or in-person at the registration desk to obtain CME documentation.

CME Record of Attendance
A Record of Attendance is provided to all attendees at on-site registration. The Record of Attendance allows attendees to calculate their own credits of participation in the educational activity. The total number of credits participants can earn per day is noted on the Record of Attendance. Below each day is a line to record the actual number of credits during which you participated in the educational activity. It is recommended that you record your actual credits daily as you proceed through the CME activity. Upon conclusion of the CME activity, please total the number of credits you have recorded on the top half of the form, sign it, and return it to the registration desk.

The bottom half of the form represents your Record of Attendance, which you must retain for your records. Please make sure the number of credits claimed in both sections coincide. No other documentation is provided to you after this CME activity. The Record of Attendance has replaced the certificate. The Record of Attendance can be used for requesting credits in accordance with state licensing boards, specialty societies, or other professional associations.

CME Activity Evaluation
The overall CME activity evaluation will be emailed following the activity to the email address that was provided when you registered. The CME activity evaluation is brief and will only take a few minutes to complete. Faculty evaluation forms will be provided electronically via email to registrants. Completed faculty evaluation forms should be completed online at the conclusion of the CME activity and within one week of the congress. Your feedback is very important to us and will be used for planning future programs, as well as identifying faculty strengths and opportunity for growth.

CME Registration Required
Please complete the following steps to receive CME credit:

1. Purchase the $25 CME fee at registration desk.
2. Obtain the CME Record of Attendance at registration desk.
3. Turn in CME Record of Attendance once completed to registration desk or email to info@worldsleepsociety.org by November 1, 2017.
4. Complete the faculty, session, and congress evaluations. Evaluations will be collected electronically and sent via email each day.

Certificate of Attendance

All attendees of World Sleep 2017 will receive an official Certificate of Attendance.
Certificates will be available at the Registration Desk Monday, October 9 through Wednesday, October 11.

Elsevier Awards

The international panel of sleep specialists convened to score abstracts for the selection of the **Elsevier Awards for 2017** to recognize two young basic and clinical sleep specialists. The panel, integrated by Antonio Culebras, MD (United States), Liborio Parrino, MD (Italy), Elena Majano, MD (El Salvador), and Melissa Lipford, MD (United States), has made the following selection:

Christian Guilleminault Award
Gianluca Sesso
Electrophysiological and microstructural features of sleep in children at high risk for depression: a preliminary study.

Elio Lugaresi Award
Tatyana Mollayeva
Sleep stage distribution in persons with mild traumatic brain injury: A polysomnographic study according to American Academy of Sleep Medicine Standards.

Young Investigator Awards 2017

The following award winners will present during S38 Young Investigator: Sleep research in neurodegeneration:
Monday, October 9 from 5:30 to 7:00 P.M.

Carmen Gutierrez Muñoz (Spain)
DIFFERENT MARKERS IN IDIOPATHIC RAPID EYE MOVEMENT (REM) SLEEP BEHAVIOUR DISORDER (RBD), POSSIBLE PREDICTORS OF CONVERSION TO DIFFERENT TYPES OF ALPHA-SINUCLEINOPATHIES

Danay Clarissa Espinoza Castro (Chile)
ENVELOPE ANALYSIS OF ELECTROMYOGRAM IN REM SLEEP BEHAVIOR DISORDER PATIENT

Gohei Yamada (Japan)
STRIATAL DYSFUNCTION AND DIMINISHED FUNCTIONAL CONNECTIVITY IN IDIOPATHIC REM SLEEP BEHAVIOR DISORDER WITH SUBTLE MOTOR ALTERATION

Kangping Xiong (China)
INCREASED SERUM CYSTATIN C IN PARKINSON'S DISEASE WITH OBJECTIVE SLEEP DISTURBANCE

Michela Figorilli (Italy)
DIAGNOSING REM SLEEP BEHAVIOR DISORDER IN PARKINSON DISEASE WITHOUT A GOLD STANDARD: A LATENT CLASSES MODELS STUDY

Yaping Liu (Hong Kong)
FAMILIAL AGGREGATION OF REM SLEEP BEHAVIOR DISORDER AND NEURODEGENERATIVE BIOMARKERS: A CASE-CONTROL FAMILY STUDY

Young Investigator Awards 2017

The following award winners will present during S77 Young Investigator: Sleep research in respiratory sleep medicine:
Tuesday, October 10 from 5:30 to 7:00 P.M.

Anupama Gupta (India)
WHY IS OBSTRUCTIVE SLEEP APNEA IN PEOPLE WITH RESISTANT HYPERTENSION MISSED SO OFTEN – A CLINICAL AND POLYSOMNOGRAPHIC CASE-CONTROLLED STUDY

Iana Andreieva (Ukraine)
CARDIAC BIOMARKERS IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA SYNDROME AND HEART FAILURE WITH PRESERVED EJECTION FRACTION

Jennifer M Cori (Australia)
HYPOCAPNIA HAS MINIMAL INFLUENCE ON GENIOGLOSSUS MUSCLE AFTER-DISCHARGE ELICITED BY AROUSAL FROM SLEEP IN HEALTHY INDIVIDUALS

Rodrigo Tomazini Martins (Australia)
EFFECTS OF MORPHINE ON THE PHENOTYPIC CAUSES OF OBSTRUCTIVE SLEEP APNEA

Yu Sun Bin (Australia)
DOES MATERNAL SLEEP APNEA AFFECT CHILDHOOD HEALTH AND EDUCATIONAL OUTCOMES? A LONGITUDINAL STUDY USING POPULATION RECORD LINKAGE

Anabel Castro-Grattoni (Spain)
IMPACT OF INTERMITTENT HYPOXIA ON CARDIOVASCULAR REMODELING IN A MURINE MODEL OF SLEEP APNEA: EFFECT OF AGE

Young Investigator Awards 2017

The following award winners will present during S99 Young Investigator: Oral presentation:
Wednesday, October 11 from 5:30 to 7:00 P.M.

Anja Holm (Denmark)
THE EVOLUTIONARY CONSERVED microRNA miR-137 REGULATES GENE EXPRESSION AND DIURNAL RHYTHM OF THE WAKE-PROMOTING HYPOCRETIN NEUROPEPTIDES

Giovanni Piantoni (Netherlands)
ULTRA-SLOW (0.0002 Hz) FLUCTUATIONS IN HUMAN INTRACRANIAL RECORDINGS CORRELATE WITH SLEEP

Janet M.Y. Cheung (Canada)
TRAJECTORIES OF USE OF OVER-THE-COUNTER AND NATURAL PRODUCTS FOR SLEEP: A FIVE YEAR FOLLOW-UP

Lampros Perogamvros (Switzerland)
EXPERIENCING FEAR IN DREAMS RELATES TO BRAIN RESPONSES TO AVERSIVE STIMULI DURING WAKEFULNESS

Lucie Barateau (France)
EXPLORATION OF CARDIAC AUTONOMIC FUNCTION BY MYOCARDIAL 123-I-MIBG SCINTIGRAPHY IN NARCOLEPSY TYPE 1

Michael Prerau (United States)
SEEKING A NEW STANDARD: A NOVEL CHARACTERIZATION OF SLEEP SPINDLES THROUGH TIME-FREQUENCY PEAK ANALYSIS

World Sleep Day Distinguished Activity Awards

Dr. Elena Majano de Carías (El Salvador)
Dr. Nevin Zaki / Dr. Nesreen Elmorsy & Mansoura University
Safety Society (Egypt)
Montida Veeravigrom, M.D. / Sleep Society of Thailand (Thailand)
Dr. David Lira (Peru)
Dr. Laura Palagini / Italian Association of Sleep Medicine (Italy)
María Montserrat Sánchez Ortuño / School of Nursing University of Murcia (Spain)

2016 World Sleep Day Distinguished Activity Awardees
Portuguese Association of Chronobiology and Sleep Medicine
and the Portuguese Society of Hypertension (Portugal)
Brazilian Sleep Society (Brazil)
Liborio Parrino (Italy)
Matilde Valecia Flores (Mexico)
Am Life International / Lew Mun Yee (Malaysia)

2015 World Sleep Day Distinguished Activity Awardees
Australasian Sleep Association and Sleep Health Foundation / Sarah Biggs (Australia & New Zealand)
Elena Majano (El Salvador)
Kiril Terziyski (Bulgaria)
Peruvian Institute of Neurosciences – David Lira (Peru)
Russian Society of Somnologists Youth Committee of Russian Society of Somnologists (Russia)

World Sleep Day Honorable Mention
Portuguese Sleep Association (Portugal)
Khosro Sadeghniiat Haghighi / Baharloo Hospital (Iran)
Lenise Jihe Kim / Brazilian Sleep Society (Brazil)
Dr. Miguel Meira e Cruz (Portugal)
Patricia van Wijk / SleepNet-BreatheNet Sleep Clinic (South Africa)
Dr. Surya Prakash (India)
Dr. Ravi Gupta / ISSR (India)
Rayleigh Ping-Ying Chiang / International Sleep Science and Technology Association & AM Life International (Malaysia)
Kenya Felicíssimo / Brazilian Association of Sleep (Brazil)
Associação Brasileira de Sono (Brazil)
Arla Cinderella Stokes Brackett (Guatemala)
Dia Mundial Del Sueno (Bolivia)
Prerana Garg (India)
Slavko Jankovic (Serbia)
Wojciech Kukwa (Poland)
Olga Lyshova (Russia)
Lia Maisuradze (Georgia)
Pakistan Pediatrics Association – Pulmonary Group and Department of Pediatrics (Pakistan)
Sleep Medicine and Research Center (Saudia Arabia)
Tushar Patel (India)
Mary Beth Valiulis (U.S.A.)

World Sleep Day
March 17, 2017 • Sleep Soundly, N

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[*] Xu L, Han F, Kristensen BT, Kristiansen-Simonsen S, Ye M, Dong X, Chang Y, Zhao L, Zheng X, Li J, Pan W, Kuna ST. Validation of the Nox-T3 portable monitor for diagnosis of obstructive sleep apnea in Chinese adults. J Clin Sleep Med. 2017;13(5):675-683.

World Sleep 2017 App

Swipe Schedules, Science & Speakers

Download the App that has it all.

Easy as 1...2...3...

1. Download

Visit the iTunes store or Google Play store to download the **World Sleep 2017 App**. View and search speaker names, sessions, rooms and topics in one easy App.

2. Connect

Don't forget to post your favorite science and selfies on Facebook and Twitter with the hashtag **#WorldSleep2017!**

3. Play

Pick up a **#WorldSleep2017 BINGO** card to share, post and win! Bingo winners will be entered to win a 1st Prize of $400 USD. Prizes for 2nd and 3rd place will be Complimentary Registration to World Sleep 2019 in Vancouver, Canada. Details on **worldsleepcongress.com/bingo.**

Keynote Speakers

Jerry Siegel, PhD
UCLA Center for Sleep Research (United States)
Monday, October 9
8:00am – 9:00am I Congress Hall

K01: The evolution of human sleep based on present-day hunter-gatherers
How did humans sleep before the modern era? Because the tools to measure sleep under natural conditions were developed long after the invention of the electric devices suspected of delaying and reducing sleep, we investigated sleep in three preindustrial societies. We find that all three show similar sleep organization, suggesting that they express core human sleep patterns, most likely characteristic of pre-modern era Homo sapiens. Sleep periods, the times from onset to offset, averaged 6.9-8.5 hr, with sleep durations of 5.7-7.1 hr, amounts near the low end of those industrial societies [4-7]. There was a difference of nearly 1 hr between summer and winter sleep. None of these groups began sleep near sunset, onset occurring, on average, 3.3 hr after sunset. The sleep period consistently occurred during the nighttime period of falling environmental temperature, was not interrupted by extended periods of waking, and terminated near the nadir of daily ambient temperature. The daily cycle of temperature change, largely eliminated from modern sleep environments, may be a potent natural regulator of sleep. Napping occurred on <7% of days in winter and <22% of days in summer. Mimicking aspects of the natural environment might be effective in treating certain modern sleep disorders.

Soňa Nevšímalová, MD, DSc
Charles University (Czech Republic)
Monday, October 9
10:30am – 11:15am I Congress Hall

K02: Central hypersomnias through the eyes of time
Central hypersomnias have a long tradition in our country thanks to Bedrich Roth. The first cases of idiopathic hypersomnia were described more than 60 years ago, and Prof. Roth collected in the 70s and 80s the largest clinical cohort of 1,000 patients with excessive daytime sleepiness. Later research confirmed many of his ideas. According to present views, narcolepsy type 1 is an autoimmune disease due to a focal neurodegenerative process, while narcolepsy type 2 is a less clear clinical entity. Changes in biomarkers (particularly hypocretin and histamine) together with increasing attention to children´s cases helped to improve our knowledge of narcolepsy etiology. In spite of the disappearance of secondary narcolepsy from the latest International Classification (ICSD-3), rare cases due to brain damage by metabolic disorder (particularly Niemann-Pick type C disease) and/or tumors still exist. Idiopathic hypersomnia with long nighttime sleep, disappearing from the ICDS-3 as well, seems to be another separate disease with a strong genetic predisposition worth molecular analysis. Idiopathic hypersomnia without long sleep is a little vague clinical entity reminiscent of narcolepsy type 2. Of much etiological interest is also Kleine-Levin syndrome including different phenotypes with a variety of biomarkers and therapeutic results. Hence, central hypersomnias seem to be a hot topic for a new design of ICSD-4 classification.

Yuichi Inoue, MD, PhD
Tokyo Medical University (Japan)
Monday, October 9
11:15am – 12:00pm I Congress Hall

K03: Racial difference in sleep disorders
It is widely accepted that various factors including social life schedule, genetic background and bed room environment may affect nocturnal sleep. Given this, we should consider a possibility that frequency and symptomatic characteristics of sleep problems/disorders may differ depending on racial or regional difference. In this presentation, with aims of promoting sleep health and sleep medicine we would like to focus on difference in sleep habits as well as the prevalence and clinical characteristics (symptoms and treatment responses) of insomnia, narcolepsy, sleep related breathing disorders, movement disorders and parasomnias among populations in countries around the world.

Keynote Speakers

Clement Cheng-Hui Lin, MD
Chang Gung Craniofacial Center, Surgery and orthodontics (Taiwan)
Monday, October 9
3:30pm – 4:15pm I Congress Hall

K04: Dental sleep medicine
Underdevelopment of craniofacial region can be accompanied by small skeletal framework, disproportion between structures, and narrowed pharyngeal airway. Segmental Maxillomandibular Rotational Advancement (SMMRA) is designed specifically for Far-East Asian OSA patients with underdeveloped maxillomandibular skeleton, featured by narrow maxilla with crowded upper dental arch, high mandibular plane angle, mandibular retrognathism, retruded chin and a generally narrowed pharyngeal airway. SMMRA advances maxilla by two segments, counterclockwisely rotates the maxillomandibular complex to improve the mandibular plane angle, advance the mandible to the optimal extent, and forward the anterior inferior mandible including chin and genioglossus tubercle. The surgery may normalize the airway, facial skeleton, occlusion, and facial aesthetics at the same time.

Oliviero Bruni, MD
Sapienza University of Rome, Pediatrics (Italy)
Monday, October 9
4:15pm – 5:00pm I Congress Hall

K05: History of pediatric sleep and the contribution of sleep microstructure
The aim of this presentation is to depict the discovery of sleep physiology and pathology in infants and the emergence of the discipline of Pediatric Sleep Medicine as a relatively autonomous entity. The gradual awareness regarding sleep disorders in infants and children began in the 19th century; children sleep had been neglected until the end of the last century with the main textbook of Pediatrics reporting none or only few paragraphs devoted to pediatric sleep, although the first observation that lead to the discovery of REM sleep was made on neonates and infants, as well as the first study on the negative behavioral consequences of sleep apnea was run in children. Researchers from different countries made important contributions for the development of the pediatric sleep medicine and actually different health providers (pediatric pulmonologists, otolaryngologists, neurologists, orthodontists and psychologists) recognize the fundamental role of sleep for the child health and development. In the last few decades, the analysis of sleep microstructure and of cyclic alternating pattern (CAP) allowed a better understanding of the neurophysiological mechanisms of sleep disturbance, especially in children. CAP can be considered as a window on pediatric sleep, allowing a new vision on how the sleeping brain is influenced by a specific pathology or how sleep protecting mechanisms try to counteract internal or external disturbing events.

Commemorative Posters

Posters commemorating World Sleep 2017 are available for purchase to congress attendees. **Pick up your poster at the Registration Desk!**

Keynote Speakers

Chiara Cirelli, MD, PhD
University of Wisconsin-Madison (United States)
Tuesday, October 10
8:00am – 9:00am I Congress Hall

K06: Sleep and synaptic homeostasis
Sleep is universal, tightly regulated, and many cognitive functions are impaired if we do not sleep. But why? Why do our brains need to disconnect from the environment for hours every day? The synaptic homeostasis hypothesis (SHY) states that sleep is the price we pay for brain plasticity and predicts that synaptic connections throughout the brain undergo net potentiation during wakefulness, while we learn new facts and regularities about the environment. Synaptic renormalization during sleep restores the homeostasis of energy and cellular supplies, with beneficial effects at the cellular and systems level, including memory acquisition, consolidation, integration, and smart forgetting. I will discuss the rationale underlying this hypothesis and summarize previous electrophysiological, molecular and genetic studies in flies, rodents and humans that confirmed SHY's main predictions. Synaptic size correlates with synaptic strength and most excitatory synapses in the cortex occur on spines. Thus, a strong prediction of SHY is that cortical spines should grow after wake and shrink after sleep, independent of circadian time. I will present new ultrastructural results obtained in mice using serial block face scanning electron microscopy that confirm this prediction, supporting the hypothesis that a core function of sleep is to renormalize overall synaptic strength increased by wake.

Mehdi Tafti, PhD
University of Lausanne, Basic Science (Switzerland)
Tuesday, October 10
10:30am – 11:15am I Congress Hall

K07: Sleep: From single neuron to behavior
Sleep is conserved throughout the evolution independent of the organization of the nervous system. This suggests that mechanisms regulating this complex behavior must also be conserved at the very basic molecular and cellular levels. We have shown that cortical cultures show robust similarities in terms of electrophysiology, transcription, and metabolism to the intact cortex of living animals. That sleep can occur in in vitro models is now reported by several groups. Such a simple model is very powerful for discovering the molecular and cellular bases of sleep. We recently showed that if cortical cultures are stimulated with waking neuromodulators, they show surprising homeostatic adaptations, very similar to the effects of sleep deprivation in animals. We also used this model to dissect the signaling pathways leading to the accumulation of plasticity-related transcripts during wakefulness. A major and highly evolutionary conserved pathway (Erk) was identified and we showed that Erk phosphorylation during wakefulness regulates sleep-related genes, sleep duration and consolidation in mice. Whether sleep is a self-sustained, cell autonomous, or a neural network property is under investigation.

Juliane Winkelmann, MD, PhD
Neurogenetics, Technische University Munich (Germany)
Chair, Institute of Neurogenomics, Helmholtz Zentrum München
Tuesday, October 10
11:15am – 12:00pm I Congress Hall

K08: Restless legs syndrome: Towards a new concept of the disease
Restless legs syndrome: towards a new concept of the disease. Genome-wide association studies led to the identification of first genetic risk variants for RLS and ongoing meta-analysis of large consortia yealed to many new risk loci. Variants detected by this approach are common genetic variants, which individually confer only a minor increase in risk of the disease. Large scale sequencing studies complemented the picture and showed that RLS is a complex genetic disorders where common and rare genetic variants contribute to the phenotype. This knowledge changed our idea about our pathophysiological concept. Furthermore, we are gaining new ideas towards the mode of action of drugs and potential new drugs.

Keynote Speakers

Michael R. Irwin, MD
UCLA Semel Institute for Neuroscience, and David Geffen School of Medicine at UCLA (United States)
Tuesday, October 10
3:30pm – 4:15pm I Congress Hall

K09: Chronic insomnia and the immune response
Insomnia is considered a public health epidemic, which contributes to increased risk of inflammatory disorders and all-cause mortality. Dr. Irwin will provide an integrated understanding of the reciprocal relationships between sleep and inflammation; present innovative findings on impact of sleep on the regulation of genomic, cellular, and systemic markers of inflammation, as well as molecular processes of cellular aging; and demonstrate the robust efficacy of insomnia treatment to reverse inflammatory activation in humans.

Allison Harvey, PhD
University of California – Berkeley (United States)
Tuesday, October 10
4:15pm – 5:00pm I Congress Hall

K10: Treating sleep and circadian problems: A transdiagnostic approach
Past treatments for sleep and circadian disturbances have been disorder-focused—they have treated a specific sleep problem (e.g., insomnia) in a specific diagnostic group (e.g., depression). However, real life sleep and circadian problems are often not so neatly categorized: Features of insomnia commonly overlap with features of hypersomnia, delayed sleep phase and irregular sleep-wake schedules. To address this complexity, the process of developing and testing the Transdiagnostic Intervention for Sleep and Circadian Dysfunction (TranS-C) will be described. TranS-C is transdiagnostic in two ways; it treats a range of the most common sleep and circadian problems across mental disorders.

Fang Han, MD
Peking University (China)
Wednesday, October 11
8:00am – 9:00am I Congress Hall

K11: H1N1, seasonality and childhood narcolepsy
Narcolepsy is recognized different across ethnic groups in many aspects including prevalence, predisposing factors and clinical presentations. In a series of 2500 narcolepsy cataplexy patients received over 20 years in a sleep lab in China, narcolepsy symptoms onset is highly correlated with seasonal and annual patterns of upper airway infections. More specially, a large rise in childhood onset cases associated with the pH1N1 outbreak, but independent of vaccination, was noted in China, and the increased incidence returned to previous levels in 2011 till to the end of 2016. This allows further cross-ethnic comparisons, and facilitate our understanding of the neurologic autoimmune mechanisms of narcolepsy.

Isabelle Arnulf, MD, PhD
Pitié-Salpêtrière University Hospital (France)
Wednesday, October 11
9:45am – 10:30am I Congress Hall

K12: Parasomnias: A window into dream
Parasomnias in REM sleep (RBD) are newly identified behaviors strongly associated with neurodegeneration. Researches are now focused on following other early signs of neurodegeneration and developing neuroprotective therapy. In contrast, NREM parasomnias (sleepwalking, sleep terrors) have been identified for a century, but somehow neglected and considered as benign or childish behaviors. We would like to raise attention on the fact that these two adult parasomnias correspond to enacted dreams. In addition to be treatable medical disorders, they open a brief but exceptional window into the dreaming cognitive and motor activity. The ethology of all behaviors during sleep is an open field for investigation, including sleep-associated speeches, facial expressions, and movements. These dreaming mental images are made visible for external investigators. This is a plea for giving as much importance to video and audio at night as to functional brain imaging, in order to access to the complex brain functioning during sleep.

Keynote Speakers

Michael Chee, MBBS
Duke-NUS Medical School (Singapore)
Wednesday, October 11
10:30am – 11:15am I Congress Hall

K13: Sleep restriction in adolescents: Cognitive effects and remedies'
Adolescents in the developed world are sleeping less compared to previous generations. Reduced sleep is most prominent in East Asian countries where scholastic achievement is highly venerated. A snapshot of current sleep behaviours and factors underlying short sleep in a representative country (Singapore) will be presented. Even in high performing adolescents, sleep restriction to 5h/night over a simulated school week causes degradation in vigilance, working memory, processing speed and mood. Such changes are exacerbated by a second cycle of sleep restriction. Topographical and declarative memory encoding are affected but problem solving skills are inconsistently affected. Napping for one hour in the afternoon can be of help for some cognitive operations. Starting school later has a positive effect on mood. Providing tools for evaluating time use can help shift behaviour and modifying study methods might also benefit sleep restricted students.

Debra Skene, BPharm, MSc, PhD
University of Surrey (United Kingdom)
Wednesday, October 11
11:15am – 12:00pm I Congress Hall

K14: Circadian rhythm sleep disorders: Challenges in diagnosis and treatment
The mismatch between the circadian timing system and behavioral rhythms in sleep/wake and feeding/fasting has both acute and chronic adverse effects on many physiological systems. Elucidation of the molecular clockwork, the melanopsin-mediated photic pathways and discovery of peripheral clocks throughout the body has provided not only new opportunities but also challenges in the diagnosis and treatment of circadian rhythm disorders. Accurate diagnosis of both central and peripheral clock timing in humans and the role of photic and nonphotic time cues (meals, melatonin) in synchronizing/resetting these rhythms will be discussed.

Mary Morrell, PhD
London, Sleep breathing disorders (United Kingdom), President of British Sleep Society
Wednesday, October 11
12:30pm – 1:15pm I Congress Hall

K15: Control of sleep-related breathing
This keynote lecture will explore the interactions between sleep and respiratory control that lead to sleep-related breathing disorders. In particular how measurement of physiological parameters such as upper-airway collapsibility, arousal threshold and loop gain may predict the development of sleep-related breathing disorders and responses to treatment. Such a personalised approach my improve adherence with therapy and help to target those who will benefit most from new treatments. Specifically in vulnerable groups such older people and patients with heart failure, in whom changes in sleep and respiratory control predispose to high rates of sleep-related breathing disorders.

Doug McEvoy, MD
Adelaide Institute for Sleep Health (Australia)
Wednesday, October 11
1:15pm – 2:00pm I Congress Hall

K16: Cardiovascular risk, OSA and CPAP (SAVE study)
Summary can be viewed online at www.worldsleepcongress.com.

Schedule at a Glance

Saturday

14th Czech-Slovak and 19th Congress of the Czech Society for Sleep Research and Sleep Medicine	8:00am - 11:00pm I Meeting Hall 1A & 1B
C01 Pediatric sleep medicine	8:00am - 5:00pm I Club E
C02 Insomnia therapeutics	8:00am - 5:00pm I Club A
C100 International RLS Study Group: RLS is not just leg kicking	8:00am - 5:30pm I Club H
C05 Technology and effective business models in sleep medicine	8:00am - 12:00pm I Club C
C06 Biology and pharmacology of sleep	8:00am - 12:00pm I Club B
C07 Circadian medicine	8:00am - 12:00pm I Club D
C08 Ambulatory sleep medicine	1:00pm – 5:00pm I Club C
C09 Sleep stages scoring and apnea scoring using computer lab equipment	1:00pm - 5:00pm I Room 221
C10 Differential diagnosis of sleep disorders: Video seminar of different sleep disorders and review of treatment options	1:00pm - 5:00pm I Club B
C101 Affiliated Meeting of Academy of Applied Myofunctional Sciences (AAMS)	12:30pm - 6:00pm I Club D

Sunday

C03 State of the field	8:00am - 5:00pm I Club E
C04 Modifying the upper airway for sleep apnea management	8:00am - 4:30pm I Club D
C11 Restless legs syndrome	8:00am - 12:00pm I Club A
C12 Pushing the envelope of sleep apnea medicine	8:00am - 12:00pm I Club C
C13 Heart and sleep	8:00am - 12:00pm I Club B
ISSTA 5th International Sleep Science and Medicine Expert Forum	8:00am - 12:00pm I Room 220
T101 Technologist Program	9:00am - 5:00pm I Club H
C14 Dental sleep medicine	12:30pm - 4:30pm I Club C
C15 Sleep and neurodegeneration	12:30pm - 4:30pm I Club A
C16 Sleep related movements: Standards for scoring, interpreting, reporting and publishing	12:30pm - 4:30pm I Club B
Romanian Association for Pediatric Sleep Disorders: How early is too early for education in pediatric sleep - beyond the influence of light and circadian biology	1:00pm – 2:30pm I Room 220
Opening Ceremony: Concert - Prague Orchestra	6:00pm - 10:00pm I Municipal House

Monday

K01 The evolution of human sleep based on present-day hunter-gatherers	8:00am - 9:00am I Congress Hall
S01 SERVE-HF and beyond	9:00am - 10:30am I Congress Hall
S02 Sleep and hypertension - causality and co-morbidities	9:00am - 10:30am I Meeting Hall IV
S03 Sleep and stress: A relationship lasting a lifetime	9:00am - 10:30am I Meeting Hall V
S04 The importance of sleep in children around the world: Factors which affect outcomes	9:00am - 10:30am I North Hall
S05 Sleep and circadian factors in metabolic risk: A translational perspective	9:00am - 10:30am I Club A and B
S06 Role of neuroimaging: Brain characteristics in sleep disorders	9:00am - 10:30am I Meeting Hall 1A
S07 The role of genetic biomarkers in sleep medicine	9:00am - 10:30am I Meeting Hall 1B
S08 Iron metabolism: Genetics, environment and restless legs syndrome (RLS)	9:00am - 10:30am I Club D and E

Monday

S09 Physiological responses of oromaxillofacial anatomy in obstructive sleep apneics undergoing maxillomandibular advancement (MMA)	9:00am - 10:30am I Terrace 1
T01 Hands-on head wiring, with paediatric aspects of polysomnography	9:00am - 10:30am I Club H
K02 Central hypersomnias through the eyes of time	10:30am - 11:15am I Congress Hall
S10 REM-sleep and depression: Research into clinically meaningful biomarkers	10:30am - 12:00pm I Meeting Hall IV
S11 The role of nocturnal eating on insomnia, diurnal sleepiness and obesity	10:30am - 12:00pm I Meeting Hall V
S12 The interplay between sleep and academic performance: From neural mechanisms to educational policy	10:30am - 12:00pm I North Hall
S13 OSA and atherogenesis: Reversible or not?	10:30am - 12:00pm I Club A and B
S15 Local sleep and local wake: From basic science to sleep arousal disorders	10:30am - 12:00pm I Meeting Hall 1B
S16 Nasal obstruction and its role in sleep disordered breathing	10:30am - 12:00pm I Terrace 1
O01 Sleep breathing disorders oral abstract presentations	10:30am – 12:00pm I Club D and E
O02 Insomnia oral abstract presentations	10:30am – 12:00pm I Club H
K03 Racial difference in sleep disorders	11:15am - 12:00pm I Congress Hall
Poster Abstracts 1	12:00pm - 12:30pm I Panorama Hall
TEVA Industry Satellite Symposium: Challenges of recognizing and treating excessive sleepiness	12:30pm - 2:00pm I Congress Hall
Merck Industry Satellite Symposium: New approaches to personalizing treatment of insomnia: Why and how?	12:30pm - 2:00pm I Meeting Hall IV
Philips Industry Satellite Symposium: Boosting slow wave sleep to improve cognitive outcomes	12:30pm - 2:00pm I North Hall
World Sleep Day Networking Event	1:00pm I Room 201
S17 New evidence on the treatment of insomnia comorbid with depression, pain, sleep apnea or circadian disorders	2:00pm - 3:30pm I Congress Hall
S18 Beyond academic walls: Society education as an essential field in sleep science	2:00pm - 3:30pm I Meeting Hall IV
S19 Sleep disorders in the adolescent population: The missing link	2:00pm - 3:30pm I North Hall
S20 Advances in obstructive sleep apnea pathogenesis and non-CPAP therapies	2:00pm - 3:30pm I Club A and B
S21 European Narcolepsy Network (EU-NN) - Narcolepsy: From etiology to treatment	2:00pm - 3:30pm I Meeting Hall 1A
S22 The waking, sleeping and dreaming brain: New circuits and insights	2:00pm - 3:30pm I Meeting Hall 1B
S23 In search of alternatives to dopaminergic ligands in RLS/WED: The emerging role of glutamate and adenosine	2:00pm - 3:30pm I Club D and E
S24 Personalization of mandibular advancement devices: Digital analysis of the movements achieved and mathematical model for the study of the jaw Kinematics.	2:00pm - 3:30pm I Terrace 1
O03 Sleep breathing disorders oral abstract presentations	2:00pm – 3:30pm I Meeting Hall V
T02 Cardiopulmonary resuscitation for sleep technologists part 1	2:00pm - 3:30pm I Club H
K04 Dental Sleep Medicine	3:30pm - 4:15pm I Congress Hall
S25 Measuring quality in the delivery of sleep medicine: Metrics and patient reported outcomes	3:30pm - 5:00pm I Meeting Hall IV
S26 Factors in night and rotating shift work associated with poor sleep and health	3:30pm - 5:00pm I Meeting Hall V

S27 Functioning of the restless legs syndrome (RLS) brain: Excitability and control	3:30pm - 5:00pm I North Hall
S28 Arousability and loop gain: The factors that bridge insomnia and sleep-disordered breathing	3:30pm - 5:00pm I Club A and B
S29 New developments in narcolepsy diagnosis, research and therapeutics	3:30pm - 5:00pm I Meeting Hall 1A
S30 Behavioral and neurophysiological influences of waking system on sleep	3:30pm - 5:00pm I Meeting Hall 1B
O04 Neurological sleep disorders affecting sleep oral abstract presentations	3:30pm – 5:00pm I Club D and E
O05 Psychiatric disorders affecting sleep/wake oral abstract presentations	3:30pm – 5:00pm I Terrace 1
O06 Neural plasticity, memory, parasomnia and pharmacology oral abstract presentations	3:30pm – 5:00pm I Club H
K05 History of pediatric sleep and the contribution of sleep microstructure	4:15pm - 5:00pm I Congress Hall
Poster Abstracts 2	5:00pm - 5:30pm I Panorama Hall
S31 Parasomnias: Recent advances in etiology, assessment and treatment	5:30pm - 7:00pm I Congress Hall
S32 Novel treatments for age-related sleep disruption	5:30pm - 7:00pm I Meeting Hall IV
S34 Pediatric OSA: Diagnostic and treatments involving a multidisciplinary team	5:30pm - 7:00pm I North Hall
S35 Minimally invasive implantable approaches for OSA	5:30pm - 7:00pm I Club A and B
S36 Understanding the potential role for Mn in RLS etiology using novel human and animal models	5:30pm - 7:00pm I Meeting Hall 1A
S37 Basic research & new treatment approaches in sleep related breathing disorders	5:30pm - 7:00pm I Meeting Hall 1B
S38 Young Investigator: Sleep research in neurodegeneration	5:30pm - 7:00pm I Club D and E
S39 Obstructive sleep apnea severity and the role of oral appliances	5:30pm - 7:00pm I Terrace 1
O07 Narcolepsy Oral Abstract Presentations	5:30pm – 7:00pm I Meeting Hall V
T03 Cardiopulmonary resuscitation for sleep technologists part 2	5:30pm - 7:00pm I Club H
Gala Dinner	7:30pm - 10:30 pm I Slovansky dum (Slavic House)

Monday

K06 Sleep and Synaptic homeostasis	8:15am - 9:00am I Congress Hall
S41 Sleep and sex: What can go wrong?	9:00am - 10:30am I Meeting Hall IV
S42 Sleep dependent brain oscillations as early markers of neurodegeneration	9:00am - 10:30am I Meeting Hall V
S43 Sleep related learning and behavioural functioning in children with developmental disorders	9:00am - 10:30am I North Hall
S44 Myofunctional therapy as an adjunct treatment for sleep disordered breathing: Validation of screening tools and objective measurements of progress for an emerging standard of care	9:00am - 10:30am I Club A and B
S46 New approaches to studies of genetics of sleep and its disorders	9:00am - 10:30am I Meeting Hall 1B
S47 Insomnia phenotypes: Identification and treatment response	9:00am - 10:30am I Club D and E
S48 NON-PAP treatment of obstructive-sleep-apnea in late teen-agers and early adulthood: What can be done, orthodontia and surgery?	9:00am - 10:30am I Terrace 1

Tuesday

Tuesday

T04 Group scoring discussion: Respiratory related breathing disorders	9:00am - 10:30am I Club H
K07 Sleep: From single neuron to behavior	10:30am - 11:15am I Congress Hall
S49 Daytime sleepiness: Newest research results & experts' opinions	10:30am - 12:00pm I Meeting Hall IV
S50 Multiple sclerosis, sleep and sleep disorders	10:30am - 12:00pm I Meeting Hall V
S51 Diagnosis, morbidity and treatment of pediatric OSA: What's new?	10:30am - 12:00pm I North Hall
S52 Dynamic circuit connecting the circadian clock and sleep/wakefulness	10:30am - 12:00pm I Club A and B
S53 How the reticular activating system (RAS) modulates perception and movement	10:30am - 12:00pm I Meeting Hall 1A
S54 Menopause matters: Hormones, hot flashes and sleep disorders	10:30am - 12:00pm I Meeting Hall 1B
S55 OSA treatment in adult [men and women] non compliant with nasal PAP, indication and potential treatment: surgical approaches and nerve implantation	10:30am - 12:00pm I Terrace 1
O08 REM behavior disorders oral abstract presentations	10:30am – 12:00pm I Club D and E
O09 Sleep breathing disorders oral abstract presentations	10:30am – 12:00pm I Club H
K08 Restless legs syndrome: Towards a new concept of the disease	11:15am - 12:00pm I Congress Hall
Poster Abstracts 3	12:00pm - 12:30pm I Panorama Hall
Cataplexy: From fundamentals to the clinic	12:30pm - 2:00pm I Congress Hall
Evolution of sleep medicine – Challenging the status quo	12:30pm - 1:15pm I North Hall
Member Meetings of WASM and WSS	1:00pm - 2:00pm I Club A and B
S56 What can we learn from recent large negative clinical trials in sleep-disordered breathing?	2:00pm - 3:30pm I Congress Hall
S57 Ascertainments of RBD and its clinical implications for neurodegeneration	2:00pm - 3:30pm I Meeting Hall IV
S58 Environmental challenges: The impact of artificial light on sleep and circadian biology	2:00pm - 3:30pm I Meeting Hall V
S59 Developmental aspects of sleep's influence on memory and general cognitive abilities	2:00pm - 3:30pm I North Hall
S60 Sleep loss and socio-emotional functioning	2:00pm - 3:30pm I Club A and B
S61 Idiopathic hypersomnia: A neglected disorder	2:00pm - 3:30pm I Meeting Hall 1A
S62 Sleep slow waves: From cells to consciousness	2:00pm - 3:30pm I Meeting Hall 1B
S63 OSA, Upper-airway surgery and MAD: Techniques to select location, surgical techniques to be used, personalization of mandibular-advancement-device	2:00pm - 3:30pm I Terrace 1
O10 Restless legs syndrome (RLS/WED) oral abstract presentations	2:00pm – 3:30pm I Club D and E
T05 Breathing Re-Education to achieve nasal and normal minute volume for sleep	2:00pm - 3:30pm I Club H
K09 Chronic insomnia and the immune response	3:30pm - 4:15pm I Congress Hall
S64 Understanding the pathophysiology of RBD and REM sleep	3:30pm - 5:00pm I Meeting Hall IV
S65 Sleep-related hypermotor epilepsy (SHE): From the basic mechanisms to the differential diagnosis	3:30pm - 5:00pm I Meeting Hall V
S66 Deficient sleep in children and adolescents: Generating solutions for a global epidemic	3:30pm - 5:00pm I North Hall

Tuesday

S67 Respiratory muscle function and intervention of upper airway in patients with sleep disordered breathing	3:30pm - 5:00pm I Club A and B
S68 Sleep disorders in post-menopausal women: The impact on health	3:30pm - 5:00pm I Meeting Hall 1A
S69 Effects of sleep deprivation: Novel agents and mechanisms	3:30pm - 5:00pm I Meeting Hall 1B
O11 Sleep breathing disorders oral abstract presentations	3:30pm – 5:00pm I Club D and E
O12 Behavior, cognition and dreaming and neurological sleep disorders affecting sleep oral abstract presentations	3:30pm – 5:00pm I Terrace 1
O13 Basic research oral abstract presentations	3:30pm – 5:00pm I Club H
K10 Treating sleep and circadian problems: A transdiagnostic approach	4:15pm - 5:00pm I Congress Hall
Poster Abstracts 4	5:00pm - 5:30pm I Panorama Hall
S71 Sleep, clocks and neurodegeneration	5:30pm - 7:00pm I Meeting Hall IV
S72 The duality of sleep movement	5:30pm - 7:00pm I Meeting Hall V
S73 Practical aspect of pediatric sleep medicine	5:30pm - 7:00pm I North Hall
S74 Extracting sleep breathing phenotypes from lab and home data	5:30pm - 7:00pm I Club A and B
S75 Sleep and mental health in a changing society	5:30pm - 7:00pm I Meeting Hall 1A
S76 Sleep, slow waves and brain temperature: Insights from hibernators	5:30pm - 7:00pm I Meeting Hall 1B
S77 Young Investigator: Sleep research in respiratory sleep medicine	5:30pm - 7:00pm I Club D and E
O14 Aging and excessive daytime sleepiness oral abstract presentations	5:30pm – 7:00pm I Terrace 1
T06 The 3 C's: Credentialing, Certification, CECs	5:30pm - 7:00pm I Club H
RS1 Society Symposium ASRS and IASSA: Sleep medicine in Asia: Across the discipline	7:00pm - 9:00pm I Meeting Hall IV
RS2 Society Symposium ASA: Biomarkers for sleep disordered breathing: Clinical, physiological, neurocognitive and genetic	7:00pm - 9:00pm I Terrace 1
RS3 Society Symposium ESRS and WSS: Sleepiness and accidents	7:00pm - 9:00pm I North Hall
RS4 Society Symposium SRS: Circadian rhythm sleep-wake disorders: Looking to the future	7:00pm - 9:00pm I Meeting Hall V

Wednesday

K11 H1N1, seasonality and childhood narcolepsy	8:00am - 9:00am I Congress Hall
S78 Sleep at high altitude	9:00am - 10:30am I Meeting Hall IV
S79 Cerebral networks during sleep and after sleep deprivation	9:00am - 10:30am I Meeting Hall V
S80 Sleep during early stage of life affects long-term outcomes	9:00am - 10:30am I North Hall
S81 Sleep, brain-heart relationships and sudden death risk	9:00am - 10:30am I Club A and B
S82 Animal models for restless legs syndrome: New developments and future challenges	9:00am - 10:30am I Club D and E
T07 Group scoring discussion: PLMD	9:00am - 10:30am I Room 221
K12 Parasomnias: A window into dream	9:45am - 10:30am I Congress Hall
K13 Sleep restriction in adolescents: Cognitive effects and remedies'	10:30am - 11:15am I Congress Hall
S83 The relationship between sleep, pain and fatigue following traumatic brain injury: From bench to bedside	10:30am - 12:00pm I Meeting Hall IV
S84 Cortical nNOS neurons: A nexus between homeostatic sleep drive and EEG slow wave activity?	10:30am - 12:00pm I Meeting Hall V
S85 Suicide, sleep and circadian rhythms in adolescents	10:30am - 12:00pm I North Hall
S86 Vitamin D and sleep	10:30am - 12:00pm I Club A and B
O15 Chronobiology/circadian disorders oral abstract presentations	10:30am – 12:00pm I Club D and E

Wednesday

O16 Sleep breathing disorders oral abstract presentations	10:30am – 12:00pm I Terrace 1
O17 Sleep health and other Issues oral abstract presentations	10:30am – 12:00pm I Club H
K14 Circadian rhythm sleep disorders: Challenges in diagnosis and treatment	11:15am - 12:00pm I Congress Hall
Poster Abstracts 5	12:00pm - 12:30pm I Panorama Hall
K15 Control of sleep-related breathing	12:30pm - 1:15pm I Congress Hall
K16 Cardiovascular risk, OSA and CPAP (SAVE study)	1:15pm - 2:00pm I Congress Hall
S87 Phenotyping and genotyping sleep apnea	2:00pm - 3:30pm I Congress Hall
S88 Novel biomarkers for sleep insufficiency and sleep disorders	2:00pm - 3:30pm I Meeting Hall IV
S89 Restless legs syndrome, augmentation and dopamine treatment - clinical data and emerging new models	2:00pm - 3:30pm I Meeting Hall V
S90 Sleep across cultures in young children from around the world	2:00pm - 3:30pm I North Hall
S91 Improving insomnia treatments: Less pain, more gain?	2:00pm - 3:30pm I Club D and E
O18 Technology and technical oral abstract presentations	2:00pm – 3:30pm I Club A and B
O19 Sleep breathing disorders oral abstract presentations	2:00pm – 3:30pm I Terrace 1
T08 Oximetry interpretation	2:00pm - 3:30pm I Club H
S92 Creating a framework for analyses of movement patterns of challenging/disruptive sleep and wake behaviours	3:30pm - 5:00pm I Congress Hall
S93 Infra-slow (< 0.1 Hz) oscillations: from the cell to the clinic	3:30pm - 5:00pm I Meeting Hall IV
S94 Circadian rhythm sleep-wake disorders and insomnia: What are the consequences and how do we optimize treatment?	9:00am - 10:30am I Terrace 1
S95 Hypertrophic cardiomyopathy and sleep disordered breathing: Implications for atrial arrhythmias and sudden cardiac death	3:30pm - 5:00pm I North Hall
S96 Autonomic disorders in sleep medicine	3:30pm - 5:00pm I Club A and B
S33 Sleep and the kidney	3:30pm - 5:00pm I Club H
O20 Basic research oral abstract presentations	3:30pm – 5:00pm I Club D and E
O21 Insomnia oral abstract presentations	3:30pm – 5:00pm I Meeting Hall V
Poster Abstracts 6	5:00pm - 5:30pm I Panorama Hall
S97 Sleep and sexual dysfunction	5:30pm - 7:00pm I Meeting Hall V
S99 Young Investigator: Oral presentation	5:30pm - 7:00pm I Club D and E
S98: Sleep and interventions in children and young people with autism spectrum disorder	5:30pm - 7:00pm I North Hall
S100 The characteristics of type 2 narcolepsy in Asian patients	5:30pm - 7:00pm I Congress Hall
O23 Sleep breathing disorders and research oral abstract presentations	5:30pm – 7:00pm I Terrace 1
T09 Experiences in sleep medicine around the world	5:30pm - 7:00pm I Club H
Closing Ceremony	7:00pm - 7:30pm I Congress Hall

Prague Metro Pass

All attendees of World Sleep 2017 will receive a complimentary Metro & Bus Pass for any public transportation between the dates of October 7-11, 2017.

Passes are included in your delegate bag.

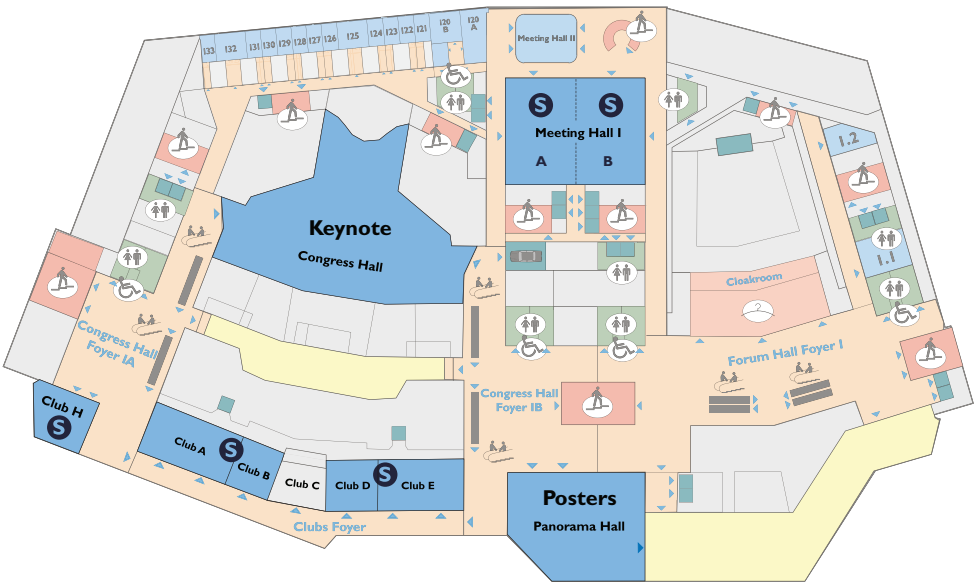
Prague Congress Centre

Ground Level

Registration Desk
Coat Check

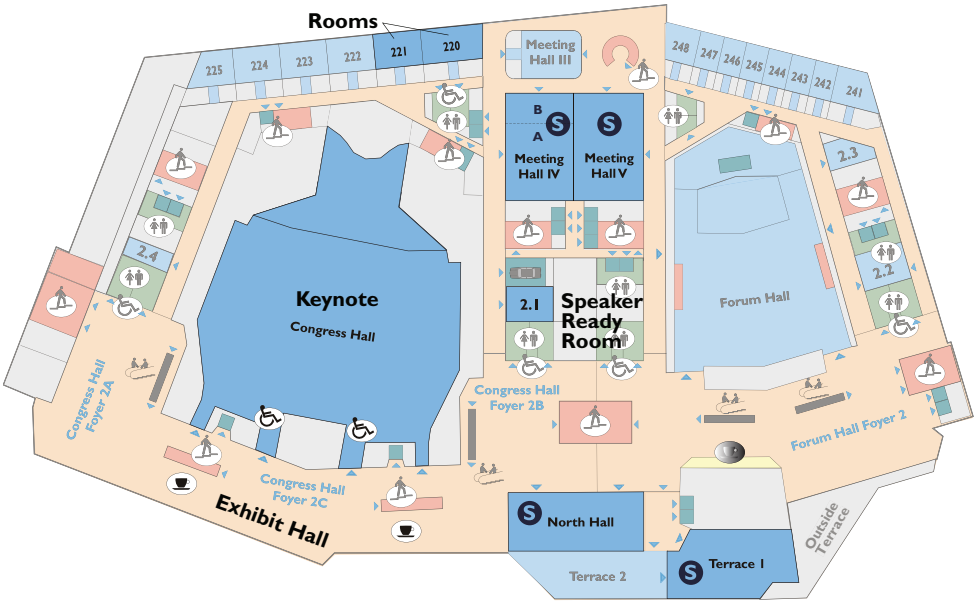
First Floor

Congress Hall – Keynote
Club AB – Breakout Sessions
Club DE – Breakout Sessions
Club H – Breakout Sessions
Panorama Hall – Abstract Posters
Meeting Hall IA – Breakout Sessions
Meeting Hall IB – Breakout Sessions



Second Floor

Exhibit Hall
Congress Hall – Keynote
Room 220 – Executive Office
Room 221 – Board Room
Room 2.1 – Speaker Ready Room
North Hall – Breakout Sessions
Meeting Hall IV – Breakout Sessions
Meeting Hall V - Breakout Sessions
Terrace 1 - Breakout Sessions



Scientific Content

Saturday 25

Courses I Affiliate Meetings

Sunday 36

Courses I Technologist Sessions I Affiliate Meetings

Monday 48

Symposia I Oral Abstracts I Technologist Sessions I Poster Presentations

Tuesday 92

Symposia I Oral Abstracts I Technologist Sessions I Poster Presentations

Wednesday 139

Symposia I Oral Abstracts I Technologist Sessions I Poster Presentations

14th Czech-Slovak and 19th Congress of the Czech Society for Sleep Research and Sleep Medicine

8:00am - 5:00pm I Meeting Hall 1A & 1B

8:00am - 9:00am	**Registration**
9:00am - 10:30am	**World Sleep - Czech Sleep Society Symposium**
	The significance of periodic and non-periodic limb movements during *L. Ferini-Strambi (Italy)*
	Actigraphy and periodic limb movements *D. Kemlink (Czech Republic)*
	Idiopathic REM sleep behavior in Parkinson disease? *A. Iranzo (Spain)*
	Rhythmic movement disorder *I. Příhodová (Czech Republic)*
10:30am - 11:00am	**Coffee + Posters**
11:00am - 12:00pm	**Sleep in neurological diseases**
12:00pm - 1:00pm	**Sleep disordered breathing and cardio-metabolic diseases**
1:00pm - 2:00pm	**Lunch**
2:00pm - 4:00pm	**Diagnosis and treatment of sleep disordered breathing**
4:00pm - 5:00pm	**Plenary meeting of the Czech Sleep Society**
5:30pm - 6:30pm	**Industry sponsored symposium**
8:00pm - 11:00pm	**Dinner**

C01 Pediatric sleep medicine

8:00am - 5:00pm I Club E

Chairs:

D. Gozal (United States), O. Bruni (Italy), R. Silvestri (Italy)

Summary

A comprehensive review of key features of pediatric sleep medicine. The prevalent expertise in pediatric aspects of sleep medicine is relatively poor, so the topics and speakers are expected to provide basic and advanced aspects on individual topics. Important areas to cover, besides the usual (e.g., sleep apnea, narcolepsy, insomnia) include the use of media and impact on the sleep of children, sleep in developmental disorders, and the inter-relationship of sleep and psychiatric disorders.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Recognize the most important sleep disorders in children
- Diagnose the main sleep disorders (i.e. insomnia, OSAS, parasomnias, hypersomnias)
- Identify better treatment options for the different sleep disorders in typically developing children and in children with neurodevelopmental disabilities
- Describe the comorbidity between insomnia different neuropsychiatric conditions

Target Audience

This course is intended for pediatricians, sleep clinicians, nurses, fellows, and students involved in the management of sleep disorders in children.

8:00am - 8:20am	**Introduction** *R. Silvestri (Italy)*
8:20am - 9:00am	**Insomnia: Clinical and diagnostic aspects** *O. Bruni (Italy)*
9:00am - 9:30am	**Comorbidity of insomnia** *J. Owens (United States)*
9:30am - 10:00am	**The effects of media on sleep in pediatric age** *J. Van den Bulck (United States)*
10:00am - 10:20am	**Coffee break**
10:20am - 11:00am	**Pediatric sleep apnea: Clinical and diagnostic aspects** *D. Gozal (United States)*
11:00am - 11:30am	**Morbidity of OSA in children** *L. Kheirandish-Gozal (United States)*
11:30am - 12:00pm	**T&A and beyond** *H.-L. Tan (United Kingdom)*
12:00pm - 12:30pm	**Group discussion**
12:30pm - 1:30pm	**Lunch**
1:30pm - 2:10pm	**Narcolepsy and REM related parasomnia** *M. Lecendreux (France)*
2:10pm - 2:50pm	**Disorders of arousal and nocturnal epileptic seizures** *L. Nobili (Italy)*
2:50pm - 3:30pm	**PLMD-RLS: diagnosis, clinical aspects and therapy** *R. Silvestri (Italy)*
3:30pm - 3:50pm	**Coffee break**
3:50pm - 4:20pm	**Rhythmic movement disorder, bruxism, enuresis** *I. Příhodová (Czech Republic)*
4:20pm - 4:50pm	**Sleep in neurodevelopmental disorders** *S. Miano (Italy)*
4:50pm- 5:00pm	**Question and answer**

C02 Insomnia therapeutics

8:00am - 5:00pm I Club A

Chairs:
C. Espie (United Kingdom), C. Morin (Canada)

Summary
This course offers a comprehensive overview of insomnia with a focus on best clinical evidence-based practice. The pros and cons of treatment using medications and Cognitive Behavioral Therapies (CBT) wil be discussed, with reference both to the literature and to real world time-constrained clinical practice. The intention is to make the course practically useful for those working in non-specialized health settings as well as informative in relation to contemporary theory and more advanced practice.

Learning Objectives
Upon completion of this CME activity, participants should be able to:

- Recognize the contemporary evidence-base for pharmacotherapy (PCT) and psychological therapy (CBT) for insomina and to understand the recommendations made in authoritative clinical guidelines concerning how insomnia may best be managed and treated.
- Identify how to assess insomnia disorder in clinical practice and how to develop a treatment plan for the different insomnia phenotypes including adapting treatment for more complex cases involving illness comorbidities, and the use in tendem or sequentially of PCT and CBT.
- Recognize the elements of CBT and modes of delivery of CBT such as individual and small group formats and how to apply CBT for insomnia.

Target Audience
This course will be of particular interest to practicioners from medical, psychology, nursing and technology backgrounds. The course does not assume a high level of background knowledge about insomnia, nor experience of working in a sleep center or laboratory. Some familiarity with the clinical presnetation and challenges of managing insomnia and an interest in extending clinical skills in this area would be helpful.

8:00am - 8:15am	**Introduction** *C. Espie (United Kingdom), C. Morin (Canada)*
8:15am - 9:00am	**The contemporary evidence base and current clinical guidelines for the treatment of insomnia disorder** *D. Riemann (Germany)*
9:00am - 9:45am	**Selecting and using pharmacotherapy for insomnia: An everyday clinical practice update** *R. Benca (United States)*
9:45am - 10:15am	**Coffee break**
10:15am - 11:00am	**Applications of CBT for insomnia in medical populations: The example of cancer** *J. Savard (Canada)*
11:00am - 11:45am	**Rational combination of CBT and PCT in insomnia management** *C. Morin (Canada)*
11:45am - 12:30pm	**Incorporating light therapy into the treatment of insomnia** *B. Bjorvatn (Norway)*
12:30pm - 1:30pm	**Lunch**
1:30pm - 2:15pm	**'Third wave' psychological therapies (e.g. mindfulness) and their role in the management of insomnia** *M.R. Irwin (United States)*
2:15pm - 3:00pm	**Digital medicine for insomnia: the potential of technology in the management of insomnia** *C. Espie (United Kingdom)*
3:00pm - 3:15pm	**Coffee Break**
3:15pm - 4:00pm	**Novel and emerging behavioural therapeutics for insomnia** *S.D. Kyle (United Kingdom)*
4:00pm - 5:00pm	**Discussion panel**

C100 International RLS Study Group: RLS is not just leg kicking

8:00am - 5:30pm I Club H

Chairs:

D. Sharon (United States), F. Provini (Italy), S. Zak (United States)

This year's educational program will introduce new and expanded data on RLS and limb movements. Several presentations will discuss the association of weight and RLS with and without PLMS. Recent data from imaging studies contribute to the understanding of the pathophysiology of RLS. More presentations will debate the unequivocal association of sleep related limb movements and RLS. Clinical aspects of RLS in adults and children will be reviewed. Finally, treatment conundrums will be discussed. New this year, we will present opportunities for international collaboration.

	Moderator: *F. Provini (Italy)*
8:00am - 8:10pm	**Welcome to IRLSSG in Prague** *K. Sonka (Czech Republic)*
8:10am - 8:30am	**Obesity and RLS** *X. Gao (United States)*
8:30am - 8:50am	**RLS severity and BMI** *F. Provini (Italy)*
8:50am - 9:10am	**RLS and BMI in stroke patients** *M. Manconi (Switzerland)*
9:10am - 9:30am	**Cerebral hemodynamic disturbances in RLS with PLMs** *K.Y. Jung (Republic of Korea)*
9:30am - 9:50am	**Resting state and thalamic role in RLS** *Y.W. Cho (Republic of Korea)*
9:50am - 10:20am	**Coffee break**
	Moderator: *D. Sharon (United States)*
10:20am - 10:40am	**Beyond PLMS: The neurophysiology of short-interval leg movements during sleep** *R. Ferri (Italy)*
10:40am - 11:00am	**Periodic vs. respiratory related leg movements** *S. Fulda (Switzerland)*
11:00am - 11:20am	**PLMS mimicking RBD** *A. Iranzo (Spain)*
11:20am - 12:10am	**Wayne Hening Award Abstract presentations**
12:10pm - 1:10pm	**Lunch break**
	Moderator: *S. Zak (United States)*
1:10pm - 1:30pm	**Actigraphy and PLMS** *D. Kemlink (Czech Republic)*
1:30pm - 1:50pm	**Ped RLS TF report** *A. Walters (United States)*
1:50pm - 2:10pm	**Update on the sIRLS validation** *D. Sharon (United States)*
2:10pm - 3:10pm	**Opiates vs. alpha 2 delta in RLS** *D. Garcia-Borreguero (Spain), J. Winkelmann (Germany)*
3:10pm - 3:40pm	**Coffee break**
	Moderator: *D. Garcia-Borreguero (Spain)*
3:40pm - 3:50pm	**International projects – our experience** *J. Winkelmann (Germany), A. Walters (United States)*
3:50pm - 4:30pm	**International projects – networking for science group session**
4:30pm - 5:30pm	**IRLSSG business meeting**

C05 Technology and effective business models in sleep medicine

8:00am - 12:00pm I Club C

Chairs:
N. Ramakrishnan (India), M. Bianchi (United States)

Summary
This course will focus on best use of personnel time and technology. Included are an update on technology used in the practice of sleep medicine, including data archiving, remote viewing of data, software running laboratory devices, positive pressure therapy compliance tracking devices, patient therapy applications, and telemedicine. The course will present diverse models in the practice of sleep medicine, with limits imposed by resource, personnel, biology and technology. "Minimal" (individual physicians in private practice in resource limited environment), "maximal" (classic tertiary sleep centers in academic programs) and models in between will be compared, with active engagement from audience members.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Appraise the spectrum of technological advances in the field of sleep apnea
- Summarize the concepts of value-based sleep medicine
- Ascertain the role of telemedicine for diagnosis and management of sleep disorders
- Compose a plan for applying big data analytics to clinical databases
- Develop methods of training within a business framework

Target Audience
Clinicians and researchers in sleep medicine

8:00am - 8:10am	**Introduction** *M. Bianchi (United States), N. Ramakrishnan (India)*
8:10am - 8:50am	**Technology in diagnostic and therapeutic devices in OSA: Where to from here?** *W. Randerath (Germany)*
8:50am - 9:30am	**Value based sleep: What is it, why it matters and how to maximize it?** *E. Wickwire (United States)*
9:30am - 9:50am	**Coffee break**
9:50am - 10:30am	**Telemedicine to diagnose and manage sleep disorders** *J. Teran-Santos (Spain)*
10:30am - 11:10am	**Turning your clinical sleep lab into a big-data enterprise** *M. Bianchi (United States)*
11:10am - 11:50am	**Novel methods of teaching and training: The business of sleep education** *R. Grunstein (Australia)*
11:50am - 12:00pm	**Question and answer** *N. Ramakrishnan (India), M. Bianchi (United States)*

C06 Biology and pharmacology of sleep

8:00am - 12:00pm I Club B

Chairs:

T. Roth (United States), P. Shiromani (United States), T. Kilduff (United States), S. Veasey (United States)

Summary

Dr. Shiromani will summarize the evidence that specific neurons distributed throughout the brain regulate the daily levels of wake, non-REM and REM sleep. Emerging evidence indicates that neurons may in fact participate with glia in regulating sleep. Emphasis will be on the use of new tools such as optogenetics, pharmacogenetics and deep-brain imaging that have made it possible to identify the brain network regulating wake, nREM and REM sleep. The presentation will summarize the evidence that there are arousal (acetylcholine, dopamine, histamine, orexin, serotonin) and sleep-active neurons (GABA, galanin, NOS, MCH), and they interact with local glia to regulate sleep.

Dr. Kilduff will extend the focus on brain circuitry by discussing the molecular targets that have been exploited for treatment of sleep disorders to date. The receptors targeted for treatment of insomnia (histamine H1, benzodiazepine/GABA-A, and hypocretin/orexin receptors) and narcolepsy (monoamine transporters, GABA-B receptors, histamine H3 receptors), in particular, will be presented. The prospect of newer, little understood targets, such as trace amine-associated receptor 1 (TAAR1), will also be discussed.

Dr. Roth will discuss the various pharmacological agents used to treat disturbed sleep. Special emphasis will be placed on issues differences in efficacy and safety of drugs targeting wake versus sleep signaling. The lecture will discuss also discuss the development of new therapeutic endpoints in evaluating efficacy of sleep promoting agents, including augmenting antidepressant and anxiolytic response and pain. Safety issues such as arousal response and its clinical implications will be discussed.

Dr. Veasey will discuss studies demonstrating weekend recovery sleep may not be sufficient in all persons to fully reverse neurobehavioral impairments observed with chronic sleep loss. Dr. Veasey will present evidence of persistent injury to and loss of specific neuron types in response to chronic short sleep with lasting effects on sleep/wake patterns. In developing animals, sleep loss in a critical window shapes behaviors critical to the survival of the species and irreversibly disturbs neuronal plasticity. Sleep serves vital roles in critical aspects of brain development, health, function and aging, and when shortened, the consequences are far worse than once imagined.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Review the use of 21st Century tools to identify the brain circuits responsible for wake, NREM and REM sleep
- Assess new drugs that target specific receptors
- Describe safety and efficacy of current hypnotics
- Recognize the adverse consequences of sleep loss

Target Audience

Clinicians engaged in the practice of treating sleep disorders, and basic scientists conducting research on the neurobiology of sleep.

8:00am - 8:05am	**Introduction: Summary of panel and objectives** *P. Shiromani (United States)*
8:05am - 8:45am	**Brain circuits regulating sleep-wake states** *P. Shiromani (United States)*
8:45am - 9:40am	**Molecular targets for sleep/wake pharmaceuticals** *T. Kilduff (United States)*
9:40am - 10:00am	**Coffee break**
10:00am - 10:55am	**Pharmacological treatment of sleep disorders** *T. Roth (United States)*
10:55am - 11:50am	**Lasting effects of chronic short sleep on brain health and aging** *S. Veasey (United States)*
11:50am - 12:00pm	**Panel discussion**

C07 Circadian medicine

8:00am - 12:00pm I Club D

Chairs:

P. Zee (United States), A. Sumova (Czech Republic), D. Skene (United Kingdom)

Summary

This course will cover an update on the neural and systemic biology of circadian rhythms, challenges in clinical diagnosis and management, focusing on new data. The intent is to move away from circadian disorders as 'sleep disorders' to be viewed more as systemic disorders. This is the first step for sleep medicine physicians taking ownership of circadian medicine.

Learning Objectives

Upon completion of this CME activity, participant should be able to:

- Identify the molecular, cellular and physiological regulation of central and peripheral circadian rhythms.
- Understand the impact of circadian disruption and the risk for cardio-metabolic and neuropsychiatric disorders.
- Recognize the importance of diagnosis and treatment of circadian dysfunction and misalignment for cardiovascular, metabolic and neurological health.
- Apply circadian based approaches to the treatment of circadian rhythm sleep-wake disorders and associated medical co-morbidities.
- Discuss the potential of integrating the time domain into the practice medicine.

8:00am - 8:10am	**Introduction** *P. Zee (United States)*
8:10am - 8:40am	**Human circadian rhythms and entrainment** *T. Roenneberg (Germany)*
8:40am - 9:10am	**Clocks, melatonin and diabetes** *J. Johnston (United Kingdom)*
9:10am - 9:40am	**Clocks, metabolism and cardiovascular regulation** *K. Wright (United States)*
9:40am - 10:10am	**Coffee Break**
10:10am - 10:40am	**Clocks and mental health** *C. McClung (United States)*
10:40am - 11:10am	**Clocks and immunity** *P. Zee (United States)*
11:10am - 11:50am	**Challenges in clinical diagnosis and treatment of CRSWD** *P. Zee (United States), S. Abbott (United States)*
11:50am - 12:00pm	**Question and answer**

C08 Ambulatory sleep medicine

1:00pm – 5:00pm I Club C

Chairs:

S. Keenan (United States)

This course will cover the gathering of data outside the sleep lab, across medical and consumer devices and telemedicine, including data from therapy devices. The future and ongoing development of sleep medicine in the world lies in the power of our tools and the management of data. The focus will be on how to best use (and not use) technology, with an aim for greatest impact on society. What if we had the tools we needed to shift the paradigm? How can we empower individuals to optimize their sleep and achieve optimal health? How can we maximize efficiency of sleep health care delivery and maintain the highest standards of patient care? Please join us and be part of the discussion.

C09 Sleep stages scoring and apnea scoring using computer lab equipment

1:00pm - 5:00pm I Room 221

Chairs:
T. Penzel (Germany)

Summary
In this course there are the most common and frequently requested topics to be covered in order to get familiar with the latest and most updated guidelines as per the AASM. The topics will discuss very interesting practical and clinical examples where most scorers find these very confusing. Each participant will have the opportunity to do hands on under the supervision of the experienced trainer, whom is certified by the American Board of Sleep Medicine and the Board of Sleep Technologists.

Participants will be using computer software within the course.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Summarize the updated AASM guidelines for scoring sleep stages and arousals
- Summarize the updated AASM guideliness for scoring respiratory events
- Identify the sleep related motor disorders
- Recognize sleep related events and score them accourding to the AASM guidelines

Target Audience
Health professionals interested in getting updated about the latest AASM scoring rules and to increase their confidence in scoring by understanding the methodology of applying these rules on real demo traces

1:00pm - 1:10pm	**Introduction** *T. Penzel (Germany)*
1:10pm - 1:50pm	**Recommended & alternative sensors in PSG** *A. Obeidat (United States)*
1:50pm - 2:30pm	**AASM guidelines for scoring sleep stages in adults** *A. Obeidat (United States)*
2:30pm - 2:50	**Coffee break**
2:50pm - 3:30pm	**AASM guidelines for scoring respiratory events in adults** *A. Obeidat (United States)*
3:30pm - 4:10pm	**AASM guidelines for motor related events in adults** *A. Obeidat (United States)*
4:10pm - 4:50pm	**Group scoring** *A. Obeidat (United States)*
4:50pm - 5:00pm	**Question and answer** *A. Obeidat (United States)*

C10 Differential diagnosis of sleep disorders: Video seminar of different sleep disorders and review of treatment options

1:00pm - 5:00pm I Club B

Chairs:

B. Högl (Austria), A. Iranzo (Spain)

Summary

This course is aimed to increase participants' knowledge and skills on the diagnosis and differential diagnosis of some of the most frequent sleep disorders, namely narcolepsy, idiopathic REM sleep behavior disorder, NREM sleep parasomnia (sleepwalking, nocturnal terrors, confusional awakenings), restless legs syndrome, periodic leg movements in sleep and obstructive sleep apnea. The use of clinical history, polysomnography, video analysis and ancillary tests will be discussed. Pharmaceutical and non pharmaceutical therapeutic options will also be addressed.
The course will be enriched by case presentations, slides of polysomnograms and video presentations to illustrate the clinical and polysomnographic features of these sleep disorders and their mimics.
The course will be highly interactive between the speakers and the audience. Each talk is 30 minutes long and the next 10 minutes will be dedicated to comments, questions and answers.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Identify the clinical features, the video-polysomnographuc features, diagnosis, differential diagnosis, potential mimics and therapeutic options of:
  - Narcolepsy and cataplexy
  - Idiopathic REM sleep behavior disorder
  - Sleepwalking, sleep terrors and confusional awakenings
  - Restless legs syndrome and periodic leg movements in sleep
  - Obstructive sleep apnea

Target Audience

Intended audience includes sleep specialists, neurologists, pulmonologists, psychologists, and scientists interested in the field of the clinical and video-polysomnohraphic aspects of sleep disorders.

1:00pm - 1:10pm	**Introduction** *B. Högl (Austria)*
1:10pm - 1:50pm	**Differential diagnosis of narcolepsy and cataplexy** *A. Iranzo (Spain)*
1:50pm - 2:30pm	**Differential diagnosis on sleep apnea** *W. Randerath (Germany)*
2:30pm - 2:50pm	**Coffee break**
2:50pm - 3:30pm	**Differential diagnosis on restless legs syndrome and periodic limb movements in sleep** *B. Högl (Austria)*
3:30pm - 4:10pm	**Differential diagnosis of REM behavior sleep disorder** *A. Stefani (Austria)*
4:10pm - 4:50pm	**Differential diagnosis of the NREM parasomnias** *L. Nobili (Italy)*
4:50pm - 5:00pm	**Question and answer** *A. Iranzo (Spain)*

C101 AAMS: Myofunctional therapy in modification of the upper airway in OSA

12:30pm - 6:00pm I Club D

Chairs:

C. Guilleminault (United States), M. Moeller (United States)

Summary

The field of Orofacial Myofunctional Therapy (OMT) has recently gained more attention as an emerging adjunct treatment for sleep disordered breathing (SDB). With early randomised studies consistently showing efficacy of OMT in the reduction of AHI and the increase in SaO2 (Guimares 2009, Villa 2014, Camacho 2015), significant reduction of snoring (Ieto 2015), and improvement in CPAP adherence and performance (Diaferia 2016, Cao 2017). Additional recent work considering the potential correlation of orofacial myofunctional disorders (OMDs) to OSA pathogenesis such as mouth breathing (Guilleminault 2014, 2015, 2016, 2017, Lee 2015, Torre 2017) and ankyloglossia (Huang 2015, Guilleminault 2016, Yoon 2017) holds great promise for early identification of patient risk and successful and stable intervention in treatment and potential reversal of SDB. Such early work has led to the increasing adoption of OMT as a standard of care, both adjunct and as a 1st line treatment for SDB (Brazilian Sleep Association/ABSONO 2013, Asian Paediatric Pulmonary Society/APPS 2016, French Society of Research and Sleep Medicine/SFRMS 2016, Italian Ministry of Health 2016), but a great deal more work must be done to establish the science necessary for OMT to become widely accepted. This symposium will review the current uses of OMT related to SDB around the world and also look to ask what is necessary for this promising field to gain greater prominence and use. We will review the efficacy of OMT with SDB with standard approaches to treatment including sleep surgery and orthodontics. This affiliate meeting is a satellite symposium of the Academy of Applied Myofunctional Sciences, a USA registered non-profit (501c3) that is the leading world body in the field of orofacial myofunctional therapy (OMT).

Learning Objectives

Upon completion of this CME activity attendees should be able to:

- Identify orofacial myofunctional disorders (OMDs) that are phenotypically related to SDB
- Assess potential SDB patients who could benefit from OMT
- Explain the potential pathogenesis of OMDs in the development of OSA
- Evaluate potential SDB patients for OMT

12:30pm – 12:45pm	**Opening remarks: Myofunctional therapy and sleep disordered breathing** *C. Guilleminault (United States)*
12:45pm – 1:05pm	**A survey of myofunctional therapy and sleep disorders** *M. Moeller (United States)*
1:05pm – 1:30pm	**Targeting the pharyngeal muscles to treat OSA: A phenotyping perspective** *D. Eckert (Australia)*
1:30pm – 1:55pm	**Patients' perspectives on myofunctional therapy and OSA: A call for action** *J. Moeller (United States)*
1:55pm – 2:20pm	**Pathways to update standards of care: How myofunctional therapy works in OSA** *E. Bianchini (Brazil)*
2:20pm – 2:45pm	**AAMS - Interdisciplinary approaches in the treatment of orofacial myofunctional anomalies in sleep** *H. Vaher (Estonia), T. Jagomagi (Estonia)*
2:45pm – 3:10pm	**From emerging adjunct treatment to 1st line treatment: The OMT journey for OSAS in Italy** *M.P. Villa (Italy)*
3:10pm – 3:25pm	**Break**
3:25pm – 3:50pm	**From reconstruction to re-education: The evolution of a sleep surgery protocol with DOME, MMA, hypoglossal nerve stimulation, and myofunctional therapy** *S. Liu (United States)*
3:50pm – 4:15pm	**A missing link in OSA pathogenesis: Low tongue posture as a phenotype in OSA** *A. Yoon (United States)*
4:15pm – 4:40pm	**Rapid return function after MMA: OMT, recovery & QOL** *Y.-F. Chen (United States)*
4:40pm – 5:05pm	**The impact of orofacial myofunctional therapy in the upper airway: Wake and sleep** *M. Moeller (United States)*
5:05pm – 5:30pm	**From the needles of dionysius: Evolution in the treatment of sleep apnea** *M. Kryger (United States)*
5:30pm – 6:00pm	**Panel discussion**

Learn about sleep disorders through Oxford's Online Programme in Sleep Medicine

www.ndcn.ox.ac.uk/oxford-online-programme-sleep-medicine

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- Hosted by world-leading Sleep & Circadian Neuroscience Institute
- Includes modules on insomnia, circadian rhythm disruption and sleep-related breathing disorders
- Teaching delivered online and via a summer school in Oxford
- Standalone modules can also be completed as part of CPD

C03 State of the field

8:00am - 5:00pm I Club E

Chairs:

A. Pack (United States), R.J. Thomas (United States)

Summary

This course will integrate research and clinical practice, bringing together basic science and clinical advances, putting together the best of a "year in review" and a "basic science/methods" update for the sleep physician". The span of topics should include technology, controversial areas, recent (2-3 years) literature. The course will aim to provide a substantial sweep across multiple topic areas, nearly a data blitz, and reach into areas outside the traditional sleep journals but of direct relevance to the practice and science of sleep medicine.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Recognize the biological interface of clinical practice and research in sleep medicine
- Identify translational aspects of cutting edge research in sleep sciences
- Review the current thinking in the field, with relevance to clinical practice
- Recognize research methodology of relevance to research that impacts clinical practice

Target Audience

Sleep medicine physician and researchers primarily, though trainees and technologists will find much of interest.

8:00am - 8:05am	**Introduction** *R.J. Thomas (United States)*
8:05am - 8:40am	**Genetic approaches to sleep science** *E. Mignot (United States)*
8:40am - 9:20am	**Mapping and biology of sleep neurocircuitry** *A. Adamantidis (Switzerland)*
9:20am - 10:00am	**Homeostatic mechanisms in sleep regulation** *V. Vyazovskiy (United Kingdom)*
10:00am - 10:15am	**Coffee break**
10:15am - 10:45am	**Sleep apnea: New concepts, mechanisms, and therapies** *A. Pack (United States)*
10:45am - 11:15am	**Sleep pathology as an oncogenetic factor** *D. Gozal (United States)*
11:15am - 11:45am	**Wearable devices and sleep science/practice** *R.J. Thomas (United States)*
11:45am - 12:00am	**Question and answer** *R.J. Thomas (United States)*
12:00pm - 12:45pm	**Lunch**
12:45pm - 1:15pm	**Surgical approaches to sleep apnea: 2017 update** *J. Suri (India)*
1:15pm - 1:45pm	**Mechanisms and management of insomnia** *C. Morin (Canada)*
1:45pm - 2:15pm	**Current concepts and management of hypersomnia** *D. Rye (United States)*
2:15pm - 2:45pm	**Neurodegeneration and sleep: Bidirectional engagement** *E. Musiek (United States)*
2:45pm - 3:00pm	**Coffee break**
3:00pm - 3:45pm	**Translational circadian sciene** *K. Wright (United States)*
3:45pm - 4:15pm	**Oral appliance therapy** *P. Cistulli (Australia)*
4:15pm - 4:45pm	**Sleep and brain disorders** *C. Bassetti (Switzerland)*
4:45pm - 5:00pm	**Question and answer** *A. Pack (United States)*

C04 Modifying the upper airway for sleep apnea management

8:00am - 4:30pm I Club D

Chairs:
C. Guilleminault (United States)

Summary
This course will bring together dental and surgical approaches to the upper airway, which is of special interest to countries where sleep medicine is less developed or too expensive. The course should range from airway development to structural and functional modification, with presentation of long-term outcomes when available.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Review the new findings related to the collapsibility of the upper airway
- Recognize the critical role of the nasal passage in impacting the upper-airway collapsibility
- Describe the new techniques available to investigate the upper airway
- Cite the surgical approaches recommended including their risks and complications when performing upper airway surgeries
- Recognice the new combination of orthodontic and surgical approaches available to treat OSA in teen-agers and young adults, their risks and complications (types and rates)
- Recall the results of the stimulation approaches of the upper airway during sleep

Target Audience
Sleep specialists of all disciplines- from surgeons to pulmonary specialists and neurologists or pediatricians involve in diagnosis and treatment of obstructive sleep apnea and abnormal resistance in the upper airway during sleep. Specialists who may be interested are not only MDs, but also Ortodontists, Oral muscle reeducators, and physiologists.

8:00am - 8:20am	**Introduction: Importance of UA for OSA** *C. Guilleminault (United States)*
8:20am - 9:10am	**The nose and SDB-50** *C. Torre (United States)*
9:10am - 10:00am	**The role of pharyngeal anatomy and airway collapsibility in the pathogenesis of OSA** *A. Schwartz (United States)*
10:00am - 10:20am	**Coffee break**
10:20am - 11:10am	**Evaluation of the upper-airway: Different approaches and comparison of results** *C.C.-H. Lin (Taiwan)*
11:10am - 12:05pm	**Surgical techniques for palatal and pharyngeal obstruction: Advantages, disadvantages, complications, and long term follow-up** *P. Baptista (Spain)*
12:05pm - 12:35pm	**Lunch**
12:35pm - 1:25pm	**Maxillo-Mandibular Advancement surgery: Indication, technique, followup, complications** *J. Cifuentes (Chile)*
1:25 - 2:15	**OSA, glossectomy and usage of robot** *S.-T. Toh (Singapore)*
2:15pm - 2:35pm	**Coffee break**
2:35pm - 3:25pm	**Distraction Osteogenesis Maxillary Expansion in young adult: Indication and results** *S. Liu (United States)*
3:25pm - 4:15pm	**XII nerve stimulation surgical approach, challenges and long term followup** *N. de Vries (The Netherlands)*
4:15pm - 4:30pm	**Question and answer** *C. Guilleminault (United States)*

C11 Restless legs syndrome

8:00am - 12:00pm I Club A

Chairs:
R. Allen (United States), D. Garcia-Borreguero (Spain)

Summary
This course will deal with new concepts in diagnosis and management of RLS, with a special focus on iron therapy, augmentation, long term outcomes including impulse control disorders, new guidelines, and update on pathophysiology.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Evaluate complex cases of RLS
- Describe the complications of long-term treatment with dopaminergic drugs
- Recognize the potential use of emerging therapies such as intravenous iron or the use of opiates
- Describe how to better prevent treatment failure
- Review the latest treatment algorithms for RLS

Target Audience
Sleep experts, neurologist, general practicioners, nurses, and sleep technicians

8:00am - 8:05am	**Introduction** *D. Garcia-Borreguero (Spain)*
8:05am - 8:35am	**Difficult cases in RLS diagnosis** *B. Högl (Austria)*
8:35am - 9:05am	**Main advantages and problems in dopaminergic treatment** *J.W. Winkelman (United States)*
9:05am - 9:35am	**Using glutamatergic drugs** *D. Garcia-Borreguero (Spain)*
9:35am - 10:00am	**Coffee break**
10:00am - 10:30am	**The role of opiates in RLS** C.J. Earley (United States)
10:30am - 11:00am	**New IRLSSG standards of practice for iron treatment** *R. Allen (United States)*
11:00am - 11:30am	**IV iron treatment for treatment resistant RLS** *W. Ondo (United States)*
11:30am - 12:00pm	**Towards an integrated treatment alghorithm** *S. Chokroverty (United States)*

C12 Pushing the envelope of sleep apnea medicine

8:00 - 12:00 I Club C

Chairs:
J. Puertas (Spain), J. Verbraecken (Belgium)

Summary
Knowledge and skills in the field of sleep disordered breathing is of paramount importance for the modern clinician and healthcare professional, who are confronted with an increasing number of patients with mild but also more complex breathing disorders during sleep. More insight in the pathophysiological mechanisms and interactions will lead to a better approach to manage these problems.

This course will deal with difficult, cutting edge and controversial aspects of sleep apnea management, including high loop gain sleep apnea, hypoventilation, management of associated symptoms, and use of data from therapy devices.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize standard therapies (and alternatives to CPAP) and their long-term results
- Describe how to deal with mild but also more complex sleep breathing disorders
- Recognize the opportunities and limitations of telemedicine applications in sleep apnea management
- Identify the problem of residual sleepiness and how to cope with it
- Recall pathophysiological mechanisms and interactions, which will lead to a better approach to manage these problems

Target Audience
Pulmonologists, sleep specialists, nurses, technicians, healthcare scientists, sleep medicine scientists involved in the treatment of sleep disordered breathing

8:00am - 8:05am	**Introduction** *J. Verbraecken (Belgium), J. Puertas (Spain)*
8:05am - 8:40am	**Standard treatments for obstructive and central sleep apnea and their cardiovascular outcome** *M. Eijsvogel (The Netherlands)*
8:40am - 9:15am	**Treatment of complex breathing disorders during sleep** *W. Randerath (Germany)*
9:15am - 9:50am	**Routine and advanced monitoring of sleep apnea therapies** *J. Montserrat (Spain)*
9:50am - 10:10am	**Coffee break**
10:10am - 10:45am	**CPAP and cognitive and metabolic functions** *M. Bonsignore (Italy)*
10:45am - 11:20am	**Treatment of mild OSA: Treat it or not, and how** *W. McNicholas (Ireland)*
11:20am - 11:55am	**How to deal with residual sleepiness and CPAP intolerance** *S. Launois (France)*
11:55am - 12:00pm	**Question and answer** *J. Verbraecken (Belgium), J. Puertas (Spain)*

C13 Course 13: Heart and sleep

8:00 - 12:00 I Club B

Chairs:
D. McEvoy (Australia), D. Bradley (Canada)

Summary
This course is designed to provide attendees with up-to-date information on a broad spectrum sleep apnea-cardiovascular interactions, from basic mechanisms through to epidemiological studies and large scale randomized trials from experts in the field. With respect to basic mechanisms, the effects of intermittent hypoxia, mimicking sleep apnea, on cardiovascular function and biomarkers will be explored. The relationship between sleep apnea and cardiac arrhythmias will be discussed. The influence of exercise on sleep apnea severity through its effects on fluid accumulation in the legs, its overnight rostral shift and effects on the upper airway will be discussed in detail. The results and interpretation of large scale randomized trials testing the effects of sleep apnea treatment on cardiovascular outcomes, including SAVE and others will be reviewed. Finally, the potential of future novel mechanistic studies and clinical trials will be considered.

Learning Objectives
Upon completion of this CME activity, particpants should be able to:
- Identify basic mechanisms through which sleep apnea can have adverse effects on the cardiovascular system
- Recognize the relationship between sleep apnea and various cardiac arrhythmias, especially atrial fibrillation
- Recognize how exercise can improve sleep apnea in patients with cardiovascular diseases, and the role that alterations in fluid accumulation and nocturnal rostral fluid shift play in this effect
- Review he generally negative results of recent randomized trials of treating sleep apnea on mortality and cardiovascular events
- Identify the types of clinical trials in sleep apnea that could be considered to determine whether there are certain cardiovascular outcomes that can be improved by treating sleep apnea

Target Audience
Respiratory and sleep physicians, cardiologists, clinical trialists, medical trainees, cardio-respiratory physiologists and allied health professionals.

8:00am - 8:10am	**Introduction** *D. Bradley (Canada)*
8:10am - 8:30am	**The current state of play - SAVE and a meta-analysis of CV event trials** *D. McEvoy (Australia)*
8:30am - 9:10am	**OSA: Atrial fibrillation and other arrhythmias** *D. Linz (Australia)*
9:10am - 9:50am	**Cardiovascular effects of intermittent hypoxia** *C. Arnaud (France)*
9:50am - 10:10am	**Coffee break**
10:10am - 10:50am	**Biomarkers of cardiovascular risk in OSA** *M. Sanchez-de-la-Torre (Spain)*
10:50am - 11:30am	**Effect of exercise on sleep apnea in patients with cardiovascular diseases** *M. Mendelson (France)*
11:30am - 12:00pm	**Future opportunities for trials and experimental studies** *D. Bradley (Canada)*

ISSTA 5th International Sleep Science and Medicine Expert Forum

8:00am - 12:00pm I Room 220

2017 Annual Assembly of Members of International Sleep Science & Technology Association (ISSTA) / 5th International Sleep Science and Medicine Expert Forum

Separate registration required. Visit https://www.ifessweb.com for registration and details.

T101 Technologist Program

9:00am - 5:00pm I Club H

Chairs:
S. Keenan (United States), O. Ludka (Czech Republic)

9:00am - 9:05am	**Opening address**
9:05am - 9:50am	**Parasomnias, movement disorders and forensics in sleep medicine: Sleep technology at its most exciting and most challenging** *M. Mahowald (United States)*
9:50am - 10:20am	**Variability in scoring of respiratory events during sleep** *E. Sif Arnardottir (Iceland)*
10:20am - 10:40am	**Coffee break**
10:40am - 11:10am	**Cognitive behavioral therapy** *V. Castronovo (Italy)*
11:10am - 11:40am	**Orofacial myofuntional therapy** *J. Moeller (United States)*
11:40am - 12:10pm	**Overview of normal human sleep: Clinical and technical considerations** *S. Keenan (United States)*
12:10pm - 12:15pm	**Question and answer**
12:15pm - 1:15pm	**Lunch break**
1:15pm - 2:00pm	**Overview of childhood sleep disorders** *S. Sullivan (United States)*
2:00pm - 2:30pm	**Driving and sleep apnea: European driver's license** *M. Gonçalves (Portugal)*
2:30pm - 3:00pm	**New technology based on sound collection** *K. Melehan (Australia)*
3:00pm - 3:20pm	**Coffee break**
3:20pm - 3:50pm	**The impact of lack of early diagnosis of sleep disorders: The need for community outreach** *S. Keenan (United States)*
3:50pm - 4:20pm	**Sleep disordered breathing in adults with down syndrome** *L. Hill (United Kingdom)*
4:20pm - 4:30pm	**Question and answer**

C14 Dental sleep medicine

12:30pm - 4:30pm I Club C

Chairs:
C. Guilleminault (United States), G. Lavigne (Canada)

Summary
In the management of sleep disordered breathing, dental, and medical sleep medicine societies and members have built a strong collaboration in the last three decades. Early interventions in children and teenagers are avenues that may concur to prevent development of long term risks related to sleep apnea. What we should know and avoid with such interventions remains an open field of clinical research.

Among management strategies, orthodontics or rapid maxillary expansion, or ENT surgical interventions are recognized to be successful approaches in many young patients. It seems that stabilisation over time may request myofunctional therapy and/or oropharyngeal re-education. Although this is intuitively attractive and supported by recent data, few issues needs to be identified such as: case selection, ideal age for intervention, best surgical techniques, benefit of myofunctional therapy on long term, etc. This is more relevant since use of oral appliance in children and teenagers is not a first line approach. The challenge of the future resides in building algorithm for case success prediction.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Identify success chances of orthodontics-rapid maxillary/palatal expansion-ENT surgeries
- Recognize that age/type of intervention are critical factors
- Recognize limitation of orthodontic, surgery, and oral appliance for children and teenager in managing sleep disordered breathing
- Review the benefits and drawbacks of oral appliances in young and not so young adults
- Assess the benefits and limits of re-education / myofunctional therapy to achieve long term success
- Review and exchange on future avenues in dental sleep medicine

Target Audience
Dentist, orthodontist, ENT, respiratory physician, pediatrician, dental and medical sleep doctors, psychologist, speech therapist and myofunctional therapist, and others in the field

12:30pm - 12:35pm	**Introduction** *G. Lavigne (Canada)*
12:35pm - 1:15pm	**Pediatric RME and childhood OSA including long term follow-up** *P. Pirelli (Italy)*
1:15pm - 1:55pm	**Bone to bone rapid maxillary expension** *B. Vande Vannet (Belgium)*
1:55pm - 2:25pm	**Distraction Osteogenesis Maxillary Expansion: New Surgical RME for OSA** *A. Yoon (United States)*
2:25pm - 2:45pm	**Coffee break**
2:45 - 3:25	**Dental devices and obstructive sleep-apnea** *M. Marklund (Sweden)*
3:25pm - 3:55pm	**MFT and orthodontics** *J. Moeller (United States)*
3:55pm - 4:25pm	**Conclusion: The developing field of dental sleep medicine** *N. Huhn (Canada)*
4:25pm - 4:30pm	**Question and answer** *G. Lavigne (Canada)*

C15 Sleep and neurodegeneration

12:30 - 4:30 I Club A

Chairs:
L. Ferini-Strambi (Italy), A. Iranzo (Spain)

Summary

This course aims to increase paticipants' knowledge and skills on the impact on sleep and sleep disorders in subjects with neurodegenerative diseases. We will see how a sleep disorder (the idiopathic form REM sleep behavior disorder) indeed represents the first manifestation of a neurodegenerative disease such as Parkinson disease. Thus, its correct diagnosis and the design of a neuroprotective trial in idiopathic REM sleep behavior is a real need today.

Sleep disorders are very common in subjects affected already by Parkinson disease and disease duration, comorbid medical issues such as the motor phenotype, and coexistent medications play a crucial role. A similar issue occurs in patients with Alzheimer disease, where the identification of sleep disorders may help in their management and offers an opportunity for prevention in its early stage. Patients with multiple system atrophy may present insomnia, REM sleep behavior disorder and nocturnal stridor, which corresponds to vocal cord paralysis and can cause sudden death during sleep.

Finally, we will discuss how a complex NREM and REM parasomnia may be the clue of a new entity, the IgLON5 disease that is characterized by abnormal tau deposits in the hypothalamus and brainstem, and antibodies against a neural protein.

The course will be enriched by case presentations, slides of polysomnograms and video presentations to illustrate the clinical and polysomnographic features of sleep disorders in subjects with neurodegenerative diseases. The course will be highly interactive between the speakers and the audience.

Learning Objectives

Upon completion of this CME activity, participants should be able to:
- Diagnose and manage idiopathic REM sleep behavior disorder
- Recognize the need of a neuroprotective trial in subjects with idiopathic REM sleep behavior disorder
- Identify and manage the sleep disorders occurring in Parkinson disease with emphasis on disease duration and motor phenotype
- Identify the IgLON5 disease in subjects with a sleep disorders
- Identify and manage the sleep disorders occurring in Alzheimer disease with emphasis on possible opportunity for prevention
- Identify and manage the sleep disorder occurring in multiple system atrophy with emphasis on nocturnal stridor

Target Audience

Sleep specialists, neurologists, pulmonologists, psychologists and scientists interested in the field of the clinical and video-polysomnohraphic aspects of sleep disorders occurring in subjects suffering from a neurodegenerative disease

12:30pm - 12:40pm	**Introduction** *L. Ferini-Strambi (Italy)*
12:40pm - 1:20pm	**Idiopathic REM sleep behavior disorder: Diagnosis, management and an opportunity for neuroprotective trials** *A. Stefani (Austria)*
1:20pm - 2:00pm	**Sleep disorders in Parkinson disease: Disease duration and phenotype effects** *M.L. Fantini (France)*
2:00pm - 2:40pm	**Sleep disorders in IgLON5 disease** *A. Iranzo (Spain)*
2:40pm - 3:00pm	**Coffee break**
3:00pm - 3:40pm	**Sleep disorders in Alzheimer disease: From the possible opportunity for AD prevention to the clinical management** *L. Ferini-Strambi (Italy)*
3:40pm - 4:20pm	**Sleep disorders and nocturnal stridor in multiple system atrophy** *V. Cochen DeCock (France)*
4:20pm - 4:30pm	**Conclusion** *A. Iranzo (Spain)*

C16 Sleep related movements: Standards for scoring, interpreting, reporting and publishing

12:30pm - 4:30pm I Club B

Chairs:
R. Allen (United States), R. Ferri (Italy), S. Fulda (Switzerland)

Summary
Practical learning of manual and automated scoring techniques and methods for PLMS, RBD, and bruxism including updates on international guidelines and publication standards for reporting of movement.
The course will contain hands on practice elements for both leg movement detection and scoring, using new, freely available scoring programs. Participants should bring their own computers (not tablets). Materials including sample recordings and scoring programs will be available for download.
International experts will discuss the significance of the scoring of sleep related movements with implications for research and publications standards.

Learning Objecitves:
Those attending will learn how to:
- Identify leg movements and score these for PLMS using the new scoring rules
- Use computerized automatic PLMS scoring routines and leg movement identification routines available for download from the internet
- Identify and score PSG features indicating RBD
- Identify and score fragmentary leg movements
- Identify and score bruxism with appropriate electrode placements

Target Audience
Clinicians reading, interpreting or scoring movement analyses from sleep tracings for PLMS, RLS, RBD, fragmentary myoclonus and bruxism. Sleep technologist and technicians responsible for scoring and logging data of movements during sleep for any cause. Sleep researchers who want to conduct research and publish in the field of sleep related movement disorders.

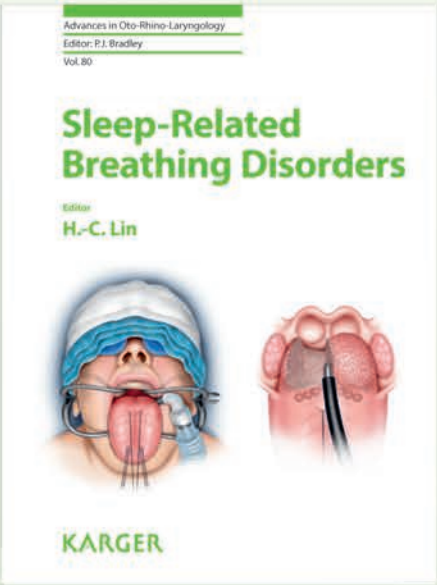
12:30pm - 1:00pm	**Periodic leg movements scoring: Updated 2016 international rules for the scoring of periodic leg movement issues and importance of respiratory event related leg movements** *S. Fulda (Switzerland)*
1:00pm - 1:45pm	**Practice scoring examples by new criteria- Participants computer files using automatic scoring programs – with practical examples** *S. Fulda (Switzerland), R. Allen (United States), R. Ferri (Italy), D. Alvarez Estevez (Spain)*
1:45pm - 2:15pm	**Automatic detection, evaluation of leg movements moving beyond PLM, with examples** *R. Allen (United States), D. Alvarez Estevez (Spain)*
2:15pm - 2:45pm	**Fragmentary leg movements and high frequency leg movements scoring: Fragmentary leg movements and high frequency leg movements scoring. Review presentation with practice scoring.** *B. Högl (Austria)*
2:45pm - 3:05pm	**Coffee break**
3:05pm - 3:35pm	**REM sleep behavior disorders scoring: Visual scoring of REM sleep without atonia and complex behaviour in RBD with practical examples** *F. Sixel-Döring (Germany)*
3:35pm - 4:00pm	**Automatic scoring of REM sleep without atonia** *R. Ferri (Italy)*
4:00pm - 4:30pm	**Sleep related bruxism scoring: Electrode placements, Visual scoring and reporting** *T. Kato (Japan)*

A multidisciplinary approach
by leading international experts

Sleep-Related Breathing Disorders

Editor: **Hsin-Ching Lin**

Many sleep-related breathing disorders (SRBD), especially obstructive sleep apnea, originate from upper airway abnormalities. The connection to cardio- and cerebrovascular comorbidities is significant and the impact on the general health of patients is noteworthy. In recent years, important advances have been made in the research, diagnosis, and treatment of SRBD due to a multidisciplinary approach. This volume incorporates contributions in which the efforts and expertise of more than thirty outstanding experts are shared. It provides a concise, practical, and comprehensive review of sleep medicine and will enable researchers and physicians to stay updated on the latest developments.



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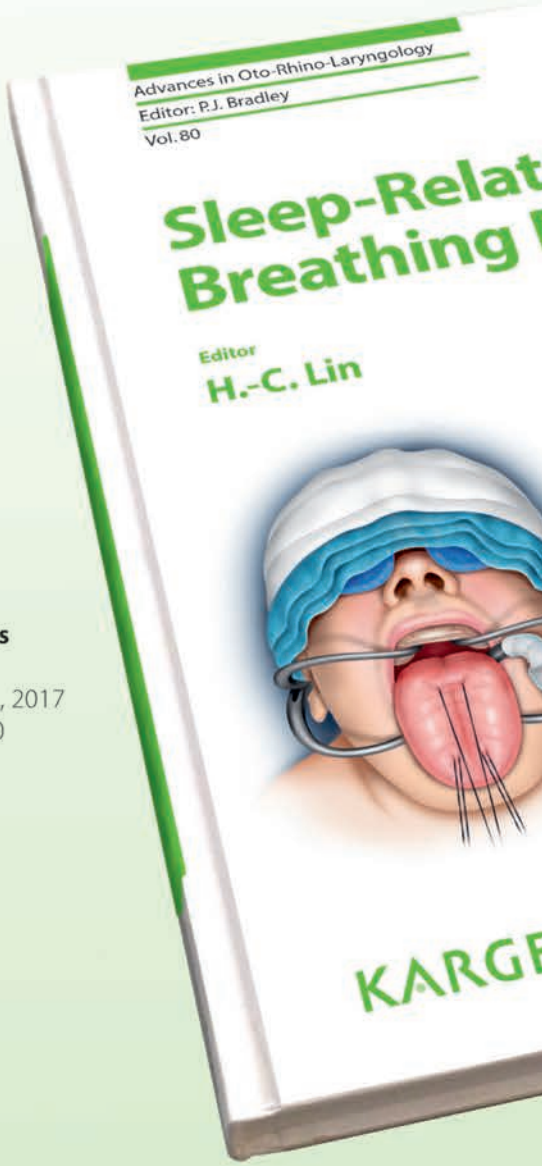

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Novel Ways to Enhance Slow Wave Sleep
Phyllis Zee, MD, PhD. Chief of Sleep Medicine, Northwestern University

Acoustical Enhancement of Slow Wave Sleep
Clare Anderson, PhD. Associate Professor, Monash University

Chair: Teofilo Lee-Chiong, MD
Date: Monday, 9th October 2017
Time: 12:30 - 14:00
Location: "North Hall" room

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Chen • Yung-Ting Chen • Hui Chen • Hsiao-Ling Chen • Rongrong Chen • Yu-Ting Cheng • Yuan Chenssai • Janet Mun Yee Cheung • Shintaro Chiba • Aleksi Chikadze • Weichih Chin • Petar Chipev • Haiao-Yean Chiu • Yong Won Cho • Yang-Ae Cho • Nayoung Cho • Su Jung Choi • Hayun Choi • Byeongji Choi • Seongjun Choi • Ji Eun Choi • Youngju Choi • Jinyoung Choi • Yun Ho Choi • Sim Chol-Kwon • Sudhansu Chokroverty • Akima Choudhary • Julie Anja Engelhard Christensen • Catherine Chua • Somchit Chumpan • Seokchoon Chung • Julio Cifuentes • Fatih Mehmet Cilingir • Chiara Cirelli • Peter Claluiti • Stefan Clemens • Valérie Cochen DeCock • Rute Coelho • Virginia Cojocaru • James Connor • Flavia Consens • Andrew Coogan • Georgia Cook • Jennifer Corl • Camila Correa • Pietro Cortelli • Ilaria Cosentino • James Coveney • Graciela Cristina Silva • Eszter Csabi • Ivan Cundrle Jr. • Jasmyn E. A. Cunningham • Milada Cvancarova Smerdova • Paolo D'Onofrio • Annalisa Da Ros • Chloe Dallen • Anna Dahlgren • Orrie Dan • Heidi Danker-Hopfe • Anshul Daradiyan • Nato Darchia • Aurora D'Atri • Subimal Datta • Kanuni Datta • Dipankar Datta • Alexandre N. Datta • Yves Dauvilliers • Raphaëlle Daville-Bicq • Carlos Mauricio Oliveira de Almeida • Tom de Boer • Ed De Bruin • Philip de Chazal • Joseph De Koninck • Luis de Lecea • Daniela Leite de Oliveira • Nico de Vries • Gana-Claudia Deleanu • Christopher Derry • Divya Devaraj • Uma Devaraj • Mario Diaz • Javier Diaz • Maria Didriksen • Derk-Jan Dijk • Dagmara Dimitriou • Ning Ding • Lars Dittrich • Karl Doghramji • Laja Dolenc Grmselj • Xiaoxiang Deng • Willem Denic • Tracy Jill Dory • Susan Dowd • Christopher Drake • Kelly Drew • Ying Duan • Brett Duce • Jeanne Duffy • Angela Duggins • Silvia Dumitru • Joaquin Durán-Cantolla • Thomas Dye • Henry Dyrberg • Christopher J. Earley • Charmaine Eastman • Rouyan Ebrahimbabaie Varnosfaderani • Alan Luiz Eckeli • Danny Eckert • Hideo Eda • Jack Edinger • Bradley Edwards • Michael Eisvogel • Mustapha El Fitouh • Mohammed El Ibrahimi • Maxime Elbaz • Mohamed El-Begermy • Dawn Elder • Ahmed Elsobki • Ayenchindorj Erkhembayar • Frédérique Es[illegible] • Colin Espie • Cynthia Espinosa Martinez • Danay Espinosa • Carla Estivill-Domènech • Hun Jeong Eun • Elisa Evangelista • Melanie Evangelisti • Liese Exelmans • Laura Facchin • Sayonara Fagundes • Maria Livia Fantini • Priscila Farias Tempaku • Eva Farkova • Sara Fattinger • Peng Fei • Eva Feketova • Luigi Ferini-Strambi • Laura M.J. Fernandez • Sergi Ferre • Rosario Ferreira • Ana Isabel Ferreira • Ana Cristina Ferreira • Raffaele Ferri • Karim Fifel • Michela Figorilli • Mariana Figueiro • Lina Fine • Dorothee Fischer • Sebastian Foellner • Stuart Fogel • Adela Paile Pereda • Karyn France • Aline Marques Franco • Clémentine Francois • Paul Franken • Peter Franzén • Carla Frare • Rolf Fronczek • Simone Frost • Nicole Fuegge • Koichi Fujiwara • Stephany Fulda • Patrick M. Fuller • Kristinn Gabric • Agata Gabryelska • Valery Galarov • Jean-François Gagnon • Maria Therese Galang-Boquiren • Artemis Galanou • Andrea Galbiati • Tea Galic • Xiang Gao • Xuemei Gao • Aida Garcia • Marcos Garcia Reyes • Diego Garcia-Borreguero • Vanessa Garcia-Larrea • Edgar Garcia-Hill • Heinrich Gerr • Rosa Gierlachlitz • Dimitri Gavriloff • Peter Geisler • Thomas Gerst • Samuel Genzor • Andreas Gerhardsson • Rachel Gimenes Albuquerque • Sandra Gimenez • Thierrorin Gislason • Martin Gloe • Nick Glozier • Barbara Gnidovec Strazisar • Lih Khiang Go • Luciana Godoy • Laurent Goetz • Mariam Gogichadze • Daniel Goh • Aimee Goldstone • Marta Gonçalves • Alain Gonfalone • Xu Gong • Jan Kyllim Good • Joanna J. Gooley • Maurizio Gorgoni • Hanampos Gouveris • David Gozal • Siqna Gradinaru • Maryne Graham • Michael Grandner • Amit Greenberg • Amanda Griffiths • Paul Gringras • John A. Groeger • Brooks Gross • Raul Gruber • Georg Gruber • Noh Gruenstein • Bianca Maria Guarneri • Mathilde C. G. Guillaumin • Christian Guilleminault • Banu Gulbay • Kamalesh K Gulia • Vivian Guo • Ravi Gupta • Marisa Gupta • Anupama Gupta • Candan Gurses • Carolina Gutierrez Romera • Carmen Guilleme Muñoz • Andrea Guzzetta • Jose Haba-Rubio • Fi[illegible]akhsri Habibi • Helena Hachul • Erika Hagen • Ins Hanov • Shahul Hameed • Fang Han • So-Ra Han • Patrick Hanly • Berit Heide Hansen • Sarah Harley • Lorenz Hartner • Allison Harvey • Emi Hasegawa • Katherine Hay • Jessica Hayton • Shan He • Jan Hedner • Jin Hee Jin • Anna Heidbreder • Raphael Heinzer • Amanda Hellebrion • Natalie Catherine Heyse • Cristina Hidalgo Chaves • Catherine Hill • Dennis C.Y. Ho • Erica Hodgson • Kerstin Hoedlmoser • Marieke Mathilde Breckle Hoekstra • Benedikt Holzer • Sigrid Högl • Benjamin Charles Holding • Ingvild Holde • Anja Holm • Seung Bang Hong • Seung-Chul Hong • Ji-Hee Hong • Kenichi Honma • Seiko Honma • Kimberly Honn • Eiko Horikawa • Rosemary Horne • Kazuaki Horikawa • Jun Horomidi • Kinnik Hovhannisyan • Mark Howard • Camila Hoyos • Silvia Hrubá • Hung-Ta Hsiao • Fan-Chi Hsiao • Sam SP Hsu • Ya-shu Huang • Renjung Huang • Minfang Huang • Junying Huang • Qiaoting Huang • Shou-Hung Huang • Ren-Jin Huang • Ya-Chuan Huang • Reto Huber • Stuart Hughes • Sung Young Huh • Nelly Huhn • Joseph T. Hull • Ching-Sai Hung • Lin-Kiong Huon • Stella Iacovides • Cor Iber • Muhammad Amin Ibrahim • Mito Imamura • Gonca Inanc • Francesca Ingravallo • Yuichi Inoue • Shoichiro Inoue • Osman Ipsiroglu • Alex Ivanzo • Michael R. Irwin • Gunnar Isacson • Toru Ishii • Alexandra Nadolyscaia • Hiromi Izawa • Muhammed J Nishadh • Joke Jaarsma • Chandra Jackson • Oter Jacobowitz • Shabnam Jalilolghadr • Yu-Wen Jan • Markus Jansson-Fröjmark • Mari Jarnefelt • Denise C. Jamin • Isabelle Jaussent • Shahrokh Javaheri • Aisha Jawed • Mackenzie E Jenkins • Poul Jennum • Ji Ye Jeon • Susanna Jemeric • Ulises Jiménez-Correa • Sakdy Jin • Kyung-Lim Joa • Jonathan Johnston • Daniel Jonasson • Byron C. Jones • Gwang Ju • Ki Young Jung • Yu Jin Jung • Hilde Therese Juvodden • Hiroshi Kadotani • Philippe Kahane • Ulf Kallwelt • Telly Kamelia • Eun-Ju Kang • Seung-Gul Kang • Ki-Woon Kang • Sandor Kardos • Manpreet Singh Kanwar • Tomas Kara • Bilguzar Kara • Jim Kasebusk • T. Kato • Takafumi Kato • Kirk Kee • Sharon Keenan • Yvonne Kelly • David Kemmerk • Jessica Kennedy • Sofia Kerrisk • Sam Kerr • Samson Khachatryan • Pierre Khoury • Habibolah Khazaie • Sepideh Khazaie • Lada Kheirandish-Gozal • Samar Khoury • Thomas Kilduff • Lenise Kim • Sung-Wan Kim • Regina Kim • Sang-Wook Kim • Kyu Dong Kim • Sang-Ha Kim • Jeong-Min Kim • Se Joong Kim • Jeongsik Kim • Daeyoung Kim • Jiyoung Kim • Tae Won Kim • Jeong-Whan Kim • HYEON JIN KIM • Mayumi Kimura • Bernie Kirkpatrick • Roumen Kirov • Elizabeth Klerman • Katarina Klobucníková • Gerhard Klosiski • Frances Knight • Stine Knudsen • Mi Ae Ko • Malcolm Kohler • Meaghan Kohli • Jun Kohyama • Venkata Koka • Melanie Köbel • Konstantinos Komotis • Kazuhiro Kon • Nemas Konecny • Angelika Konda • Anna Kontoo • Dae Um Koo • Suresh Kotagal • Sanjeev Kothare • Sylvia Kotterba • Stefan Thomas Kotzian • Petra Kovalska • Mette Kragh • Jaroslav Kraus • P. Vijaya Krishnan • Anuk Kruavit • Elena Kruglikova • Mari Krygie • Antoni Krzeski • Dmitry Kudryashov • David Kuhns • Antti Kulkas • Suresh Kumar • Chang-Pyng Kuo • Kenichi Kuriyama • Salome Kurth • Cinia Kushida • Dindu M. Kutty • Lilia Kuula • Benjamin Kwan • Simon D. Kyle • Luc Laberge • ILARIA LACCU • Joana Lagos • Ching-Hsiang Lai • Nushrotul Laliyya • Tea Lallukka • Yeue Ching Lam • Isabelle Lambert • Gert Jan Lammers • Kallimol Lamprou • Jaap Lancee • Hans-Peter Landolt • Supawan Landsiriwong • Philippe Latour • Yael Latzer • Esther Yuet Ying Lau • Kristy Nga Ting Lau • Sandrine Laumois • Gilles Lavigne • Victoria Lavrova • Aiper Lazar • Michael Lazarus • Frances Le Comu Knight • Michel Leonardus • Yu-Jin Lee • Sang Sup Lee • Yoon-Hee Lee • Eun Ju Lee • Ho-Won Lee • Alice Lee • Jung Lee • Hsin-Chien Lee • Sui L. Leo • Hanbyul Leo • Ramona Leonards • Philippe Lehert • Zoltán Lelkes • Francisco Leon • Francisco Xavier Leon Romero • Luong Chi Leow • Timo Leppanen • Daniel Lewandowski • Patrick Levy • Monika Levy Andersen • Albert Li • Yuqing Li • Kasey Li • Mingxian Li • Taomei Li • Gen Li • Ya-Dong Li • Zhanjiang Li • Shirley Xin Li • Gui-Xia Li • Yin Li • Shi Li • Wen-Chun Liao • Claudio Liguori • Laura Lilla-Triguero • Jung Hwa Lim • Namunda Limba • Clement Cheng-Hui Lin • Chien-Heng Lin • Chia Mo Lin • Jean-Marc Lina • Eva Lindberg • Jiefan Ling • Altay Lino de Souza • Dorina Linz • Melissa Lipford • Monika Liskova • Stanley Liu • Jue Liu • Yaping Liu • Shanshan Liu • Shuai Liu • Ing-Hsian Lo • Carolina Lombardi • Sally Loomis • Maria-Cecilia Lopes • Régis Lopez • Isabel Louzão de Amorim • Helena Cristina Loureiro • Arne Lowden • Shang-Yao Lu • Ondrej Ludha • Sylvia Ludwig • Yuan-Ming Luo • Pierre-Hervé Luppi • Adrian Lupusor • Kurt Lusi[illegible] • Anita Lüthi • Faith Luyster • Owen Lyons • Olga V. Lys[illegible] • Yan Ma • Jianfang Ma • Andrea Maciuano Estrada • Inna Mafalesca • Tovelli Maede • Ulysses Magalang • Mayra Magalhães Silva • Neil Magladdery • Mark Mahowald • Olivier Maillesse • Tiina E. Makela • Atul Malhotra • Nodira Malik • Bishnita Mallick • Hridananda Mallick • Mauro Manconi • Anastasia Manganaro • Denisa Mankova • Tadaski Mano • Satya Padmaja Mantha • Susanna Manuwami • Luisa Mar[illegible] • Angelina Maric • Jose Maria Marin • Marie Markland • Azad Marouli • Sandra Marques • Inés Marques Carneiro • Lenka Martinec Novakova • Olga Martynova • Midori Mashita • Khayram Masiyev • Jana Matlova • Ahmed Masoud • Stijn Massar • Marcello Massimini • Maria João Matos • Takeo Matsuzawa • Yuuki Matsumoto • Masahiro Matsuo • Keotani Matsuura • Raymond Matthews • Joachim Maurer • Geert Mayer • Pedro Mayoral • Pedro Mayoral Sanz • S M Khorshed Alam Mazumder • Paolo Muzzone • Diego Mazzotti • Cathy A. McCall • Colleen McClung • Doug McEvoy • Jennifer J. McGrath • Laura E McKillop • Laurie McLay • Walter McNicholas • Pablo Medrano-Martinez • Peter Mavrio • Marie Meintjes • Miguel Meira e Cruz • Jaidan Mekky • Kuni Melathan • Francisco Venissimo Mello-Filho • Luciano Mello-Fujita • Monique Mendelson • Armand Mensen • Maria Merchant • Ilona Menkanto • Mila Merino Andreu • Silvia Merino • Masanori Mayuchi • Monika Meralek • Michal Michalik • Mitchell Miglis • Emmanuel Mignot • Yulia Migunova • Thorsten Mikoteit • Francesca Milano • Eva Milankova • Jodi A. Mindell • Melissa Mindha • Elena Mind • Kazuo Mishima • Irina Mishkova • Seiko Mitachi • Ranju Mi[illegible] • Takui Miyagawa • Seohiya Miyagawa • Masayuki Miyamoto • Seiko Miyata • Rie Miyata • Makoto Miyazaki • Marc Moeller • Jay Moeller • Jessica Mogg • Shenandoah Montamat • Nestor Montesdeoca • Jose Montserrat • Marta Moralejada-Ottman • Marta M Moskwska • Gustavo Antonio Moreira • Kevin Morgan • Charles Morin • Mary Morrell • Jennifer Morton • Rupya Mughal • Peter Mugo Muthithi • Subuja Mukherjee • Marie Muller • Maria-Lucia Muntanar • Hisae Murai • Kazuhiro Musamatsu • Erik Musiek • Martina Muskova • Benny Mwenge • Sami Myllymaa • Hyo-Seok Na • Nargisi Nachkebia • Prabhu P. Nagendra • Kalpana Nagpal • Artur Nahorecki • Anatu Najeli • Mizuki Nakajima • Chikao Nakamura • Yukari Nakazato • Eun Nanghang • Shenooka Nanthakumar • Jiri Nepobitek • Daniel Neu • Song Nevsimalova • Johan Newell • Danilo Kwok Keung Ng • Xiaojia Ni • Yuval Nir • Arto C. Nirkko • Seiji Nishino • Gillian Nixon • Daniela Nogan • Lina Nohili • Kyung-Ha Noh • Marta Novak • Jan Novak • Manu Ntafouh • Carlos O'Donnell • Rena • Conor O'Dochartaigh • Amr Obeidat • Siobhan O'Daly • Jin Oh • Seung Min Oh • Masako Onuki • Yo Oishi • Izabela Okujava • Mutsumi Okura • Olafunji Olaoye • Humberto Olcese • Olaf Oldenburg • Maria João Oliveira • Luis Vicente Franco Oliveira • Vanda Olivera Clemente • William Ondo • Martina Ondrova • Manuela Oros • Marina Orth • Masri Otto • Qiong Ou • Simon Overland • Judith Owens • Murat Ozgeroglu • Iva Pihlodova • Allan Pack • Sang Ho Pae • Evelina Pajedienie • Dinesh Pal • Laura Palagini • Andreas Palm • Jose Alberto Palma • Luciana Palombi • Maria Panagiotoui • Vikram Pandit • Efrosini Papaconstantinou • Nelly A Papadimitriou • Chan Soon Park • Dae-Yang Park • Jong-Uk Park • Arpita Parmar • Libono Parreo • Claudia Pascovich • Stephanie Patton • Walter Paulus • Václav Pavelec • Georgia Pavlopoulou • Pavla Pernova • Antonio Perrotta-Mazano • John Pepper • Anna Pettke • Simon Pelmer • Patricio Peirano • Yüksel Peker • Zerrin Pelin • Erika Pellerin • Thomas Penzel • Paul Peppard • Claudia Pereira • Sandra Pérez Ramos • Lampros Perogamvros • Erik Pevernagie • Christelle Peyron • Luc Pham • Pierre Philip • Rahul Philip • Maria Pia Villa • Giovanni Plantoni • Andres Pinto • Ronaldo Piovesan • Paola Pirelli • Patrizia Pisanu • Fabio Pizza • Fabio Placidi • Bettina Platt • Giuseppe Plazzi • Roxana Maria Pleava • Alexandra Polishchuk • Lorenzo Polizzi • Michael Polkey • Pálvi Poo-Kantola • Vsevolod Polotsky • Mikhail Poluektov • Mihael H. Polymeropoulos • Tanja Porkka-Heiskanen • Elizabeth Portegal Velloso • Katarzyna Pretzsch-Artemczyk • Gregory Potter • Marc Poulin • Catherine Poulin-Collins • Paolo Pozzi • Brijesh Prajapati • Surya Prakash • Michael Prerau • Uni Gamayani Primadi • Anshu Priya • Federica Provini • Javier Puertas • F. Javier Puertas • Arunkumar Pugalenthi • Monica Puligheddu • Bathsaiyi Purbasari • Konstantin Pychov • Yingjun Qian • Minja Quantic • Maria Antonia Guerra-Salva • Stacey Quo • Shantha Rajaratnam • Naganjan Ramakrishnan • Candelaria Ramirez • Alberto Ramos • Alberto Ramos, MD, MSPH • Wilfried Randerath • Pietro Luca Ratti • Laura B Ray • Mark Rea • Catia Reis • Ricardo Reis • Rong Ren • Seonkyeong Rhee • Kathy Richards • Amanda Richdale • Dieter Riemann • Marta Rios • Giovanni Rizzo • Geraldo Rizzo • Seung Hoon Ro • M. Carmen Rodriguez Morales • Beatriz Rodriguez-Morilla • TA Roehrs • Till Roennberg • Jae Hoon Roh • Eva Rolfmans • Michele Romano • Karina Rossa • Thomas Roth • Marine Roussel • Sylvie Royant-Parola • Perrine Ruby • Noelia Ruiz Herrera • Lukas Ruzek • David Rye • Tiago Sa • Outi Saarenpää-Heikkilä • Schrab Sabet Moghadam • Abdelkebir Sahli • Dena Sadeghi • Khosro Sadeghniiat Haghighi • Patricia Sagaspe • Cwin Sahlin • Bandana Saini • Yann Saint-Georges-Chaumet • Katarina Sajgalikova • Egle Sakadauskaite-Juodelikiene • Dimitros Sakellariou • Inna Sakhatashvili • Rachel Salas • Michael Salatu • Aaro Salminen • Alessandro Salvatori • Walkyria Sampaio • Manuel Sanchez-de-la-Torre • Christina Sandlund • Pilar Santelices • Taeko Sasai-Sakuma • Ryo Sasaki • Toshihiko Sato • Mayuto Satoh • Cornelius Sauder • Jose Savard • Hermant Sawnani • Eugenio Scarnati • Serena Scarpelli • Carlos Schenck • Karine Schieuermaier • Mirk Schmeisbeck • Alessandro Schirru • Angelika Schlarb • Markus Schmidt • Sarah Fiona Schoch • Prestam Schramm • Brittany J Schultz • Richard Schwab • Alan Schwartz • Jean-Charles Schwartz • Johanna Schwarz • Natalia Schwarzkopf • Paula K Schweitzer • Holly Scott • Anna J. 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Sweetman • Zoltan Szakacs • Andras Szemkovics • Naoko Tachibana • Mehdi Tafti • Jacques Taillard • Yoshikazu Takaesu • Pradeep Talwalkar • Hui-Leng Tan • Lu Tan • Toksen Tanahashi • Kosuke Tamoka • Leila Tanoeh • Zarehi Tavadyan • Jin Yu Tay • Selver Burcu Telloglu • Priscila Tempaku • Joaquin Terán-Santos • Jerry Theorell-Haglöw • Robert Joseph Thomas • Michael J Thorpy • Herning Tiemeier • Hua Ting • Vikas Kumar Tiwari • Tomoyuki Tobushi • Sonia Togeiro • Song-Tar Toh • Rodrigo Tomazini Martins • Carlos Torre • Pablo Torterolo • Claudia Trenkwalder • Manjari Tripathi • Ivana Trojova • Philip Tucker • Sergio Tufik • William J. Tyler • Orna Tzachanis • George Uhl • Jan Ulfberg • Doo Hyun Um • Yasemin Unal • Chandimani Undugodage • Amaia Urrutia Gajate • Andrew Vakulin • Pablo Valdez • Matilde Valencia-Flores • Carlos Valera-Davila • Stella Valiensi • Raphael Vallat • Jan Van den Bulck • Vincent T van Hees • Wessel M.A. van Leeuwen • Emer Van Ryswyk • Eus Van Someren • Bart Vande Vannet • Moya Vandeleur • Gilles Vandewalle • Pilar Varillota • Maria Varendh • Andrew W. Varga • Anne Vassalli • Sigrid Veasey • Olivia Veatch • Christian Veauthier • Karolina Veldova • Andreas Velten • Anne Venner • Rahul Venugopal • Johan Verbraecken • Joris Verster • Aleksandar Videnovic • Javier Vila • Maria Pia Villa • Pierguiseppe Vinai • Pedro Pilegge Vinta • Lal Chandra Vishwakarma • Bogdan Voinescu • Carina Volk • Bruno von Amann • Dimitrios Vourdas • Victor Voxo • Vladyslav Vyazovskiy • Hamnet Walla • Douglas Wallace • Arthur Walters • Yuqing Wang • Bei Wang • Peipei Wang • David Wang • Tao Wang • Allan Wang • Han Wang • Yulin Wang • Nono Watanabe • Frederik Weber • Silke Weber • Shelly Weiss • Andrew Wellman • Adam Wichniak • Emerson Wickwire • Tomasz Wieczorek • Hanna-Kaisa Wigren • Veronica Wilbor • Ines Willem • Rhiannon Williams • Yun Kwok Wing • John W. Winkelman • Juliane Winkelmann • Catherine Wiseman-Hakes • William Wohlgemuth • Mark Lawrence Wong • Kenneth Wright • Yanxiao Wu • Fei-Ling Wu • Melanie Wust • Khamaphaphon Wuttamporn • Yanyan Xia • Fulong Xiao • Kang-Ping Xiong • Ying Xu • Liyue Xu • Zhifei Xu • Huaqun Xu • Zhou Xuan Zi • Gohei Yamada • Tomoko Yamagata • Toshitaka Yamakawa • Akihiro Yamanaka • Madoka Yamazaki • Tang Yan • Xue Yan • Timothy Yang • Liu Yanjiao • Chun Yao • Nakadai Yazihan • Garik Yeganyan • Pei-Lu Yi • Gokce Yilmaz Akdogan • Shanlan Yin • Akihiko Yokoo • Audrey Yoon • In-Young Yoon • Dae Wui Yoon • Vladimir Yordanov • Takuya Yoshiike • Yasuko Yoshioka • Arika Yoshizaki • Peter Young • Haibo Yuan • Xiang-Shan Yuan • Merai Yuksel • Nevin FW Zaki • Dora Zalai • Abigail Zarazpe • Helmut Zarbl • Phyllis Zee • Feng Zhai • Xilong Zhang • Zhang-Jin Zhang • Jingjing Zhang • Ze Zhang • Yingying Zhao • Xue Zhou • Xindi Zhu • Nataly Zion

K01 The evolution of human sleep based on present-day hunter-gatherers

J. Siegel (United States)

Keynote 8:00am - 9:00am I Congress Hall

How did humans sleep before the modern era? Because the tools to measure sleep under natural conditions were developed long after the invention of the electric devices suspected of delaying and reducing sleep, we investigated sleep in three preindustrial societies. We find that all three show similar sleep organization, suggesting that they express core human sleep patterns, most likely characteristic of pre-modern era Homo sapiens. Sleep periods, the times from onset to offset, averaged 6.9-8.5 hr, with sleep durations of 5.7-7.1 hr, amounts near the low end of those industrial societies [4-7]. There was a difference of nearly 1 hr between summer and winter sleep. None of these groups began sleep near sunset, onset occurring, on average, 3.3 hr after sunset. The sleep period consistently occurred during the nighttime period of falling environmental temperature, was not interrupted by extended periods of waking, and terminated near the nadir of daily ambient temperature. The daily cycle of temperature change, largely eliminated from modern sleep environments, may be a potent natural regulator of sleep. Napping occurred on <7% of days in winter and <22% of days in summer. Mimicking aspects of the natural environment might be effective in treating certain modern sleep disorders.

S01 SERVE-HF and beyond

9:00am - 10:30am I Congress Hall

Chairs:

C. Kushida (United States)

Summary

The management of central sleep apnea (CSA) in patients with heart failure is a major therapeutic challenge for sleep clinicians. In 2015, the results of The Treatment of Predominant Central Sleep Apnoea by Adaptive Servo Ventilation in Patients With Heart Failure (SERVE-HF) trial were released; this trial tested the hypothesis that adaptive servoventilation (ASV) would reduce the incidence of the composite primary end point of all-cause mortality, life-saving cardiovascular interventions, or unplanned hospitalizations for worsening heart failure in 1,325 patients with heart failure with reduced ejection fraction and co-existing CSA. However, the intention-to-treat analysis showed no significant difference between individuals randomly assigned to ASV and those randomly assigned to control for the primary end point, and post hoc analysis found excess cardiovascular mortality in treated patients.

The proposed symposium is intended to explore the management of central sleep apnea since the results of the SERVE-HF trial, to discuss lessons learned, to describe newer studies such as the Effect of Adaptive Servo Ventilation on Survival and Hospital Admissions in Heart Failure (ADVENT-HF), and to assess CSA management by oxygen therapy and phrenic nerve stimulation in heart failure patients.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Recognize the pathophysiology and relationship of central sleep apnea in the setting of patients with heart failure
- Analyze the results and limitations of The Treatment of Predominant Central Sleep Apnoea by Adaptive Servo Ventilation in Patients With Heart Failure (SERVE-HF) trial
- Review new treatments and trials to explore the management of central sleep apnea in patients with heart failure

Target Audience

Clinicians or researchers who evaluate or study patients with obstructive and central sleep apnea, including sleep specialists, pulmonary physicians, and respiratory physiologists

9:00am - 9:03am	**Introduction** *C. Kushida (United States)*
9:03am - 9:20am	**Central sleep apnea in heart failure** *W. Randerath (Germany)*
9:20am - 9:37am	**SERVE-HF update** *P. Levy (France)*
9:37am - 9:54am	**ADVENT-HF update** *D. Bradley (Canada)*
9:54am - 10:11am	**Oxygen therapy for central sleep apnea in heart failure** *M. Kryger (United States)*
10:11am - 10:28am	**Phrenic nerve stimulation to treat central sleep apnea in heart failure** *A. Malhotra (United States)*
10:28am - 10:30am	**Question and answer** *C. Kushida (United States)*

S02 Sleep and hypertension - causality and co-morbidities

9:00am - 10:30am I Meeting Hall IV

Chairs:
G. Shukla (India)

Summary
Both Hypertension as well as sleep disorders are highly prevalent among populations worldwide. Population based studies have shown that short nightly sleep duration is associated with increased incidence of various cardiovascular diseases. Data from a recent epidemiological study suggest that the risk for hypertension was nearly one and a half times higher among patients with sleep disorders, in comparison with those without any sleep problems. Obstructive sleep apnea is among the most prevalent and well characterised sleep disorders. Its independent association with cardiovascular disease, especially hypertension, has been demonstrated through a large number of studies; although much work is still ongoing to explore the extent of risk and underlying mechanisms for this association. The effects of treating sleep apnea on blood pressure control as well as reducing incidence of Hypertension are subjects of much healthcare research interest. Much interesting data is also emerging on treatment strategies like renal denervation, for patients with hypertension, who also have sleep apnea. The first talk of the proposed symposium will elucidate these important points. Sleep apnea and other sleep disorders have now been recognized to be an extremely commonly occurring part of the metabolic syndrome (co-existing abdominal obesity, hypertension and carbohydrate as well as lipid metabolism abnormalities); this association often termed as the 'Syndrome Z'. The interlinking of hypertension with sleep apnea, in this very specific population is a matter of much research and it has been established that even within this narrowly well-defined population, the prevalence of sleep apnea is much higher among patients with treated and controlled hypertension in comparison with normotensive patients with other components of the metabolic syndrome. The sleep apnea-hypertension within the specific population of metabolic syndrome, will be highlighted, in the second talk. There is a much stronger relationship of sleep disorders, especially sleep apnea with pharmacological treatment resistant hypertension. Data from a recent meta-analysis suggest significant reduction in blood pressure among people with sleep apnea and resistant hypertension, treated with positive airway pressure therapy. The role of various sleep disorders among patients with resistant hypertension has been highlighted and compilation of this evidence, systematically studying the relationship between specific sleep disorders, is much needed. Some original data on this aspect of looking at the association of common sleep disorders among treatment resistant hypertension will form the gist of the third talk of the proposed symposium. Several mechanisms connected with RLS can influence blood pressure, including insomnia and especially PLMS. The latter are accompanied by evident transient rises in heart rate (higher than those found in normal controls), blood pressure and cerebral hemodynamic changes. The repetition of these events for hundreds of times every night and for many years might represent itself a state of repetitive transient nocturnal hypertension that might favor the establishment of permanent functional and/or morphologic changes in RLS patients. The concluding talk of the symposium aims to present an appraisal on this fascinating relationship, which is an area of much ongoing research.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the prevalence of sleep apnea in patients with hypertension and the underlying mechanisms
- Recognize effects of treating OSA on BP control and on hypertension incidence
- Identify the close link between sleep apnea and hypertension in the specific population suffering from the metabolic syndrome
- Review the prevalence and impact of different sleep disorders among patients with pharmacological treatment resistant hypertension
- Define the relationship between restless legs syndrome and associated periodic limb movements with hypertension

Target Audience
Sleep physicians, internal medicine physicians, cardiologists, fellows from these specialties, general practitioners, nurse practitioners

9:00am - 9:05am	**Introduction** *G. Shukla (India)*
9:05am - 9:25am	**Obstructive sleep apnea in hypertension - mechanisms and therapy** *V. Somers (United States)*
9:25am - 9:45am	**'Syndrome Z' - teasing out the interlinking of obstructive sleep apnea and hypertension** *J. Suri (India)*
9:45am - 10:05am	**Sleep disorders in resistant hypertension - how big is the burden?** *G. Shukla (India)*
10:05am - 10:25am	**Restless legs syndrome and periodic limb movement disorder and the link with hypertension** *R. Ferri (Italy)*
10:25am - 10:30am	**Question and answer** *G. Shukla (India)*

S03 Sleep and stress: A relationship lasting a lifetime

9:00am - 10:30am I Meeting Hall V

Chairs:
L. Palagini (Italy)

Summary

The bidirectional relationship between sleep and stress has been widely investigated (McEwen 2015, Juster 2015). However, this relationship need to be better understood for important clinical implications, especially, regarding insomnia and circadian disorders, in order to prevent and treat their important negative impact on health. Here, we want to include "periods of life" as a new element of this equation. Indeed, this symposium aims at investigating the role of acute and chronic stress in determining sleep derangements from a life course perspective by integrating basic and clinical research. The first speaker (Dr. Palagini, University of Pisa, Italy) will introduce this topic reviewing the findings about the bidirectional relationship between sleep and stress and focusing on how acute, chronic and even perinatal stress can lead to insomnia [Palagini et al. Sleep Med. 2015]. With "Roads leading to insomnia: the role of stress from the perinatal period through adult life", Dr. Palagini will introduce the possible role of clock genes and epigenetic mechanisms underlying this relationship from perinatal through adult life. The second speaker (Dr. Bastianini, University of Bologna, Italy) will review animal studies investigating the link between perinatal stress and wake-sleep cycle derangements in adults. With "Perinatal stress and hypnic derangements in adults: news from animal models", Dr. Bastianini will also analyze the possible epigenetic mechanisms underlying this relationship [Bastianini et al. J Sleep Res. 2016]. The third speaker (Dr. Franken, University of Lausanne, Switzerland) will focus on how acute sleep loss through the alteration of glucocorticoids can modify both central and peripheral clock gene/protein expression. With "Acute stress and modulation of central and peripheral circadian clocks in mice", Dr. Franken will also address the independent modulation of peripheral and central (i.e. within the SCN) circadian rhythms in clock genes [Franken et al. Sleep. 2015]. In line with Dr. Franken's data, the fourth speaker (Dr. Meerlo, University of Groningen, The Netherlands) will deal with the consequences of chronic (social) stress on periphery and central circadian oscillators in animal models. With "Social defeat in rats as a model to study stress-induced changes in the circadian function and sleep", he will emphasize the theory that stress-induced changes in sleep-architecture may affect neuronal plasticity thereby contributing to cognitive and psychiatric disorders [Meerlo et al. Curr Top Behav Neurosci. 2015]. Accordingly, the fifth speaker (Dr. Drake, University of Detroit, USA) will conclude this symposium exposing clinical data linking stress-related sleep reactivity and insomnia-circadian disorders. In particular, with "The role of stress reactivity in insomnia and circadian disorders", Dr. Drake will deal with the theory that there is a vulnerability to the development of insomnia and circadian disorders in response to stress and a sensitization of the sleep system [Drake et al Sleep 2014, Kalmbach et al Sleep Med 2015]. The present session proposes the special format of combining junior and senior presenters to provide an integrated review of findings on the relationship between sleep and stress from a life course perspective. It also has this particular format of combining clinical and basic research data on insomnia and circadian rhythms.

Learning Objectives

Upon completion of this CME activity, participants should be able to:
- Summarize the relationship between sleep and stress in different periods of life
- Compare basic and clinical data relative to the effect of stress on sleep and circadian rhythms
- Combine information coming from animal and clinical research
- Translate animal data to clinical applications
- Interpret sleep and sleep-related disorders on the basis of latest animal and clinical data.

Target Audience

Psychiatrists, psychologists, basic sleep researchers, and neurologists

9:00am - 9:03am	**Introduction** *L. Palagini (Italy)*
9:03am - 9:20am	**Roads leading to insomnia: The role of stress from the perinatal period through adult life** *L. Palagini (Italy)*
9:20am - 9:37am	**Perinatal stress and hypnic derangements in adults: News from animal models** *S. Bastianini (Italy)*
9:37am - 9:54am	**Acute stress and modulation of central and peripheral circadian clocks in mice** *P. Franken (Switzerland)*
9:54am - 10:11am	**Social defeat in rats as a model to study stress-induced changes in the circadian function and sleep** *P. Meerlo (The Netherlands)*
10:11am - 10:28am	**The role of stress reactivity in insomnia and circadian disorders** *C. Drake (United States)*
10:28am - 10:30am	**Question and answer** *L. Palagini (Italy)*

S04 The importance of sleep in children around the world: Factors which affect outcomes

9:00am - 10:30am I North Hall

Chairs:
R. Horne (Australia)

Summary
During childhood sleep is at a life time maximum and is essential for normal development. Sleep disorders are common and it is estimated that these affect around 40% of all children. Adequate sleep is important for all aspects of daytime functioning including maintenance of alertness, memory and school performance. Unlike adults where sleep disruption results in daytime sleepiness, in children this can manifest as disruptive behaviour and inattention at school. Disruption of sleep due to sleep disordered breathing not only affects daytime functioning but also has significant effects on the cardiovascular system and behaviour and school performance. This symposium will discuss the broad spectrum of sleep habits and sleep disorders that occur in children and new research which aims to predict those at most risk. Prof Elder will present data parent knowledge of sleep and child sleep practices and symptoms in both Māori and New Zealand European children and compare those of each ethnicity who attend low decile schools (deprived) with those who attend high decile schools (not deprived). The presentation will examine how sleep problems might influence learning in this context and will discuss factors that affect sleep in children in these diverse New Zealand population groups and consider child, parent and school teacher perspectives of child sleep in this context. A/Prof Hill will present data on sleep patterns and habits of children aged 6 months to 16 years in Bolivia recruited from two urban populations at Santa Cruz (500m) and a high altitude setting, La Paz (3700m). This presentation will further explore these differences between South American parental report and data from other countries and consider whether cultural norms may be biologically mal-adaptive. Prof Li will examine the evidence linking chronically sleep deprivation, with abnormal blood pressure control. Sleep deprivation is a world-wide phenomenon and the issue is even more concerning for adolescents. Sleep deprivation is common in this age group due to delayed sleep circadian phase and is compounded by environmental and lifestyle/social demands such as extracurricular activities and home-work. The presentation will also report findings of an on-going study to evaluate the effects of prolonging sleep on blood pressure. Prof Horne will discuss new research examining the cardiovascular and behavioural and neurocognitive effects of obstructive sleep apnoea (OSA) in overweight/obese children. Up to 50% of overweight or obese children have OSA compared to 6% in normal weight children. Both childhood overweight/obesity and OSA have independent adverse cardiovascular effects. This presentation will describe the independent and combined effects of obesity and OSA in children and discuss implications for long term outcomes and whether treatment has the potential to improve these.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the importance of adequate sleep in children
- Identify how inadequate sleep affects children in different cultures
- Recognize how inadequate sleep affects children with different sleep practices and disorders

Target Audience
Sleep and respiratory physicians and researchers

9:00am - 9:05am	**Introduction** *R. Horne (Australia)*
9:05am - 9:25am	**Sleep practices and sleep problems in Māori and New Zealand children from different socioeconomic backgrounds** *D. Elder (New Zealand)*
9:25am - 9:45am	**The effects of altitude on sleep patterns and habits in children** *C. Hill (United Kingdom)*
9:45am - 10:05am	**Adverse consequences of sleep deprivation in Hong Kong adolescents – cardiovascular perspective** *A. Li (Hong Kong)*
10:05am - 10:25am	**Are the consequences of obstructive sleep apnoea compounded by obesity in children?** *R. Horne (Australia)*
10:25am - 10:30am	**Question and answer** *R. Horne (Australia)*

S05 Sleep and circadian factors in metabolic risk: A translational perspective

9:00am - 10:30am I Club A and B

Chairs:
M. Grandner (United States)

Summary
Obesity and diabetes are major public health epidemics throughout most of the developed world. They play major roles in preventable morbidity and mortality. Substantial scientific work has been undertaken to better understand the role of sleep and circadian factors in the physiologic processes that contribute to obesity and diabetes (and related conditions within the cardiometabolic domain). This work has included basic science at the cellular and animal level, towards the goal of discerning basic molecular and genetic factors by which circadian and sleep-related processes exert influence over metabolism and related functions, such as inflammation, adipocyte function, and insulin signaling. This work also involves human laboratory work both in the basic and clinical domains. This line of research has generally involved highly-controlled laboratory studies in humans that have not only shown that sleep and circadian factors both exert influence on aspects of energy balance at both the mechanistic and phenomenologic levels, but that manipulation of sleep and circadian factors can produce both beneficial and adverse changes in metabolic outcomes that are known to be related to obesity and diabetes. Finally, a number of studies have taken a population science approach to this issue, documenting population trends in obesity and diabetes as they relate to sleep and circadian factors. Although these studies generally lack the precision of laboratory studies, they present an opportunity for a high degree of generalizability, as well as the statistical power to discern complex and subtle relationships at the population level (in the real world). Increasingly, as these lines of research converge on the issue of sleep/circadian factors in obesity and diabetes, it is clear that a more translational approach is needed to optimally understand and eventually combat these problems. The proposed session presents cutting-edge research on the relationship of sleep/circadian factors to metabolic disease risk while taking a translational perspective. The format of the session will start in cellular/animal studies, move to "first-in-human" basic science approaches, to clinical approaches where interventions are tested, finally to population and community-focused approaches. The session starts with the basic cellular/animal science perspective, where physiologic insights that can only be discovered this way have the opportunity to be brought to humans. Dr. Albrecht will present recent data on circadian regulation of metabolic processes. Then, the session focuses on basic human laboratory research, which takes the principles and processes examined in the basic science realm and applies them to the study of humans in a highly-controlled environment. Dr. Wright will present data from his lab, showing that sleep and circadian factors exert influence on energy balance systems at the physiologic level. Then, Dr. Spiegel will present data from her laboratory showing that not only does sleep deprivation likely increase diabetes risk, but sleep extension may ameliorate some of that risk. Finally, Dr. Grandner will extend these findings to the community and population level, discussing population trends and public health interventions for sleep health. Taken together, this session will present new information on an important topic, while taking a novel, translational perspective.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Describe the role of sleep and circadian processes in the basic mechanisms of metabolism and how that relates to roles of sleep/circadian processes in clinical metabolic disease risk
- Describe the role of sleep and circadian processes in metabolic risk across a translational spectrum,from basic science with cells and animals, to basic science in humans, to clinical research, and finally to population science
- Describe how laboratory studies of sleep and metabolism relate to both basic cellular/animal studies from which they can be informed and clinical/population studies to which they can be extended
- Design research projects around the issue of sleep/circadian science in metabolism by considering a translational framework

Target Audience
Scientists working at all places within the translational spectrum: Basic scientists who work with cells and animals, basic scientists who work with humans, laboratory experimental and clinical researchers, and community/epidemiology researchers. Also, clinicians treating both sleep and metabolic disorders should benefit from this symposium

9:00am - 9:05am	**Introduction** *M. Grandner (United States)*
9:05am - 9:25am	**Basic circadian regulation of metabolism** *U. Albrecht (Switzerland)*
9:25am - 9:45am	**Sleep and energy balance in humans** *K. Wright (United States)*
9:45am - 10:05am	**Clinical impact of sleep deprivation and extension on metabolic risk** *K. Spiegel (France)*
10:05am - 10:25am	**Impact of sleep and circadian processes on population health** *M. Grandner (United States)*
10:25am - 10:30am	**Question and answer** *M. Grandner (United States)*

S06 Role of neuroimaging: Brain characteristics in sleep disorders

9:00am - 10:30am I Meeting Hall 1A

Chairs:
S.B. Hong (Republic of Korea), Y. Inoue (Japan)

Summary
Neuroimaging is a very useful technique for investigating brain structural or functional changes in neurological disorders and sleep disorders. In addition to widely reported cognition decline with OSA, diminished regional and often unilateral gray matter loss has been reported in several neuroimaging studies of OSA. An assessment of white matter integrity using diffusion weighted imaging in OSA patients showed multiple regions of lower fractional anisotropy in the wide spread area of brain. While these studies report brain abnormalities, e.g., atrophicor hypertrophic comparison to normal control, there are still many results remain controversial. This symposium will review on-going neuroimaging studies in OSA Korean elderly population and initiate the discussion about how neuroimaging can help the OSA research. The quantitative neuroimaging measures can be associated with other clinical variables (e.g., cognition test or behavior test) to better understand the OSA mechanism. With appropriate understanding of the neuroimaging and its application to sleep research, researchers can effectively utilize neuroimaging data and tools to investigate the specific association between OSA and brain. Structural brain changes have been investigated in patients with narcolepsy and insomnia. The lecture will present recent results of structural brain abnormalities in narcolepsy and insomnia. Quantitative electroencephalographic (QEEG) analysis has been used to measure cortical activity and assess an underlying pathological process in neurodegenerative disorders, because EEG power analysis provides high temporal resolution and is useful in detecting subtle changes of brain function. In this presentation, we will discuss alterations of awake EEG in idiopathic REM sleep behavior disorder without cognitive impairment and effects of benzodiazepines on EEG in patients with insomnia disorder.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the importance of neuroimaging study in obstructive sleep apnea study
- Recognize the role of different neuroimaging modalities in OSA study
- Use independent study design involving neuroimaging

Target Audience
Neuroscientists, sleep medicine specialists, and sleep scientists

9:00am - 9:05am	**Introduction** *Y. Inoue (Japan)*
9:05am - 9:25am	**The association between brain structure and cognition in OSA: Largescale Korean MRI (n>1200) study** *R. Kim (Republic of Korea)*
9:25am - 9:45am	**Accelerated brain aging in OSA: Korean population study** *C. Shin (Republic of Korea)*
9:45am - 10:05am	**Brain structural changes in narcolepsy and insomnia** *S.B. Hong (Republic of Korea)*
10:05am - 10:25am	**Quantitative analysis of EEG and brain dysfunction in sleep disorders** *I.-Y. Yoon (Republic of Korea)*
10:25am - 10:30am	**Question and answer** *S.B. Hong (Republic of Korea)*

S07 The role of genetic biomarkers in sleep medicine

9:00am - 10:30am I Meeting Hall 1B

Chairs:
S. Tufik (Brazil)

Summary
Sleep-wake patterns as well as sleep disorders are complex traits influenced by variations within multiple genes and their interaction with behavioral and environmental factors. Previous studies in twins and families have established the influence of genetic factors on restless legs syndrome, insomnia, obstructive sleep apnea, narcolepsy and sleep bruxism. However, studies have identified only a few genetic variants that seem to contribute to greater risk in the development of sleep disorders. The identification of new risk factors is enduring continuous issue in sleep medicine research to increase our understanding about the etiopathogeny of most sleep disorders, and to improve methods for identifying people who are in the early stages of, or at high risk for, them. Interindividual differences are reflected in differential molecular activity of genes or proteins, metabolites and signaling pathways. Approaches to identify genomic biomarkers consist of several methods to evaluate gene polymorphisms (GWAS) and the expression of genes. These tools are critical for a better understanding of the molecular mechanisms involved in sleep regulation. A number of GWAS have successfully identified single-nucleotide polymorphisms associated with sleep characteristics, such as sleep duration and chronotype, as well as sleep disorders like restless leg syndrome and narcolepsy. However, for obstructive sleep apnea, insomnia and sleep bruxism, such common sleep disorders, genetic investigations are still in development. Advances in molecular biology, genetics and computational biology are very important, since they can also help to optimize clinical practice in sleep medicine by the identification and treatment of at risk individuals. Additionally, genetic strategies may open new lines of research to understand the basic mechanisms of sleep regulation and the pathophysiology of sleep disorders. This symposium aims to provide the most recent advance regarding the importance of genetic approaches in sleep and sleep medicine.

Learnng Objectives
Upon completion of this CME activity, participants should be able to:
- Review the genetic basis of sleep and sleep disorders
- Assemble the genomic tools to identify sleep-related biomarkers
- Compare recent potential biomarkers associated with sleep disorders

Target Audience
This session is proposed for a general audience since it is a relatively new field for Sleep Medicine, which is growing and needs to be explored. In particular, it may be of great interest for attendees whose research is focused on molecular pathways of sleep and the identification of genetic risk factors for sleep disorders.

9:00am - 9:05am	**Introduction** *S. Tufik (Brazil)*
9:05am - 9:25am	**Large-scale genomic studies to identify sleep-related risk factors** *M. Tafti (Switzerland)*
9:25am - 9:45am	**Genetics of insomnia** *H. Tiemeier (The Netherlands)*
9:45am - 10:05am	**Genome-wide association study of sleep bruxism in the episono cohort** *R. Amaral Jr. (Brazil)*
10:05am - 10:25am	**Obstructive sleep apnea syndrome and genetics: Data from a population based cohort in Brazil** *P. Farias Tempaku (Brazil)*
10:25am - 10:30am	**Question and answer** *S. Tufik (Brazil)*

S08 Iron metabolism: Genetics, environment and restless legs syndrome (RLS)

9:00am - 10:30am I Club D and E

Chairs:
R. Allen (United States)

Summary
Reduced brain iron remains the best-documented biological abnormality for RLS. Studies of the genetic and environmental factors affecting RLS brain iron have produced new insights into iron biology that has implications for advancing RLS treatments and prevention. This symposium integrates the complex genetics of iron management with new studies of RLS blood brain barrier (BBB), brain imaging of iron in RLS and the unexpected complex results of evaluating iron deficiency in RLS and the very mixed response to IV iron treatment. The concepts of brain iron regulation have dramatically changed with the recent discovery of circadian variation in brain iron. Brain iron is not static but rather involves a continual flux with stores in other tissues, presumably involving interchange with the blood brain barrier (BBB). This raises several questions regarding iron in RLS: First, how does the genetic regulation of brain and peripheral iron impact the iron deficiency state of RLS? Second, how does changes in iron status impact iron delivery to the brain for RLS? Third, what is the pattern of the brain iron deficiency in RLS and how does this relate to the RLS symptoms? Fourth, what are the effects of iron deficiency on RLS symptoms and response to treatments and Finally what are the factors we can gain from these studies that help explain the puzzling extremely mixed response to IV iron treatments with 40 -50% of RLS patients showing dramatic improvements but another at least 40% showing no response. In contrast about 75% of patients with iron deficiency anemia show a strong positive response to treatment. Moreover, why the need for repeated IV iron treatments in some patients. This symposium starts with some in depth exploration of the advances in the science of iron management providing a framework for understanding the treatment questions and possibly developing better iron treatments. The first talk by Dr. Jones presents what we have learned from a large panel of genetically crossed mice about the complex genetic regulation of iron in response to iron deprivation. The variation between strains and between peripheral and brain iron changes indicate the limits on assuming peripheral iron informs on brain iron status and it flags particular murine strains likely to match the iron brain changes. Given the basic iron flux over the day and the marked genetic separation of peripheral and brain iron status an important question involves factors affecting the transport of iron across the BBB. New data from experimental BBB models using RLS and control tissue will be reviewed by Dr. Connor. The regional brain iron pattern seen in RLS along with possible relation to RLS symptoms will be reviewed by Dr. Rizzo. The clinical effects of iron deficiency anemia producing RLS and the response to treatment will be presented by Dr. Earley. Finally, Dr. Allen will discuss the challenges and some limited knowledge about predicting response to IV iron treatment for RLS patients. These presentations set the table for a discussion of why the IV iron treatment is often dramatically effective for some time but not more consistently effective for a longer time?

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Evaluate the significant features of peripheral iron status for RLS treatments
- Integrate clinical history of environmental status and results of blood tests for consideration of iron treatment options for RLS
- Evaluate and possibly propose further iron studies and iron treatment options for RLS
- Recognize the interaction of genetic and environmental factors producing RLS

Target Audience
Clinicians treating sleep disorders, especially RLS; Scientists studying RLS biology or iron management biology

9:00am - 9:02am	**Introduction** *R. Allen (United States)*
9:02am - 9:19am	**Complex genetics of brain iron regulation** *B.C. Jones (United States)*
9:19am - 9:36am	**Iron uptake into the RLS brain: The Blood-Brain-Barrier** *J. Connor (United States)*
9:36am - 9:53am	**Brain imaging - iron status relation to RLS symptoms** *G. Rizzo (Italy)*
9:53am - 10:10am	**Iron deficiency anemia and RLS** *C.J. Earley (United States)*
10:10am- 10:27am	**RLS IV iron treatment - markers of success** *R. Allen (United States)*
10:27am - 10:30am	**Question and answer** *R. Allen (United States)*

S09 Physiological responses of oromaxillofacial anatomy in obstructive sleep apneics undergoing maxillomandibular advancement (MMA)

9:00am - 10:30am I Terrace 1

Chairs:
C.C.-H. Lin (Taiwan)

Summary
Maxillomandibular advancement (MMA) is the most effective surgical treatment option for non obese OSA patient with maxillofacial retrusion. After the surgery, facial skeleton is advanced, oral cavity is enlarged, and pharyngeal airway is dilated. Yet, detailed physiological responses of oromaxillofacial structures (such as tongue, soft palate, and oral muscles) are still infamiliar and keen for more understanding. In the symposium, Dr. Dennis CY Ho will demonstrate the differences between conventional MMA and MMA with counterclockwise rotation of maxillomandibular complex. Not only an aesthetic reason, but also a physiological meaning can be seen in the movement. During the surgery, the maxillomandibular skeleton was osteotomized. Further myofunctional management is taken to realese the constrain of oral muscles, and set the maxilla and mandible free. Dr. Vikram Pandit will reveal the myofuntional management during surgery, and how the skeleton is freed to enlarge the oral cavity. Dr. Ryo sasaki will illustrate the physiological responses of tongue and soft palate along with the maxillomandibular advancement. From the changes of this two fundamental organs, we can have a more comprehensive picture of how the MMA modify the pharyngeal airway. Dr. Edmund CK Chan will depict the 3D changes in the pharyngeal airway after MMA. Ultimately, the enlargement of pharyngeal airway may be a secondary response to the skeletal movement, but holds the direct and crucial effect on the reduction of pharyngeal airway resistance. Dr. Clement CH Lin will summarize the session and discuss fundamental philosophy of Segmental Maxillomandibular Rotational Advancement (SMMRA). The overall goal is to enhance the understanding of the physiological effect of MMA on the surrounding muscles, soft tissue organs, and pharyngeal airway through indiviual observation on each target structure, and to compose a general picture of physiological responses of maxillofacial anatomy in OSA patients undergoing MMA.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the surgical design of counterclockwise rotation of maxillomandibular complex in MMA
- Identify the muscular management during surgery and the related functional physiologic effect
- Identify the translation and transformation of tongue and soft palate after MMA
- Describe the 3D changes in pharyngeal airway after MMA
- Review the fundamental philosophy of Segmental Maxillomandibular Rotational Advancement

Target Audience
Sleep physicians who take care of patient with OSA, evaluate upper airway anatomy, and prescribe treatment options; dental doctors who provide services in upper airway evaluation and treatment for snore or OSA through oral appliance application; and sleep surgeons (craniofacial, maxillofacial, and ENT surgeons) who consult and perform surgeries in pharyngeal airway and craniomaxillofacial regions on patients with OSA.

9:00am - 9:02am	**Introduction** *C.C.-H. Lin (Taiwan)*
9:02am - 9:16am	**To rotate or not to rotate: The surgical planning in MMA** *D.C. Y. Ho (Taiwan)*
9:16am - 9:30am	**Myofunctional management during MMA** *V. Pandit (India)*
9:30am - 9:44am	**Translation and transformation of tongue and soft palate after MMA** *R. Sasaki (Japan)*
9:44am - 9:58am	**Three-dimensional changes in pharyngeal airway in OSA patients undergoing MMA** *E.C. K. Chan (Hong Kong)*
9:58am - 10:12am	**Segmental Maxillomandibular Rotational Advancement: why, when and how?** *C.C.-H. Lin (Taiwan)*
10:12am - 10:30am	**Question and answer** *C.C.-H. Lin (Taiwan)*

T01 Hands-on head wiring, with paediatric aspects of polysomnography

9:00am - 10:30am I Club H

Chairs:
S. Keenan (United States), O. Ludka (Czech Republic)

Speakers:
L. Hill (United Kingdom), S. Briscoe (United Kingdom)

K02 Central hypersomnias through the eyes of time

S. Nevšímalová (Czech Republic)
Keynote 10:30am - 11:15am I Congress Hall

Central hypersomnias have a long tradition in our country thanks to Bedrich Roth. The first cases of idiopathic hypersomnia were described more than 60 years ago, and Prof. Roth collected in the 70s and 80s the largest clinical cohort of 1,000 patients with excessive daytime sleepiness. Later research confirmed many of his ideas. According to present views, narcolepsy type 1 is an autoimmune disease due to a focal neurodegenerative process, while narcolepsy type 2 is a less clear clinical entity. Changes in biomarkers (particularly hypocretin and histamine) together with increasing attention to children´s cases helped to improve our knowledge of narcolepsy etiology. In spite of the disappearance of secondary narcolepsy from the latest International Classification (ICSD-3), rare cases due to brain damage by metabolic disorder (particularly Niemann-Pick type C disease) and/or tumors still exist. Idiopathic hypersomnia with long nighttime sleep, disappearing from the ICDS-3 as well, seems to be another separate disease with a strong genetic predisposition worth molecular analysis. Idiopathic hypersomnia without long sleep is a little vague clinical entity reminiscent of narcolepsy type 2. Of much etiological interest is also Kleine-Levin syndrome including different phenotypes with a variety of biomarkers and therapeutic results. Hence, central hypersomnias seem to be a hot topic for a new design of ICSD-4 classification.

S10 REM-sleep and depression: Research into clinically meaningful biomarkers

10:30am - 12:00am I Meeting Hall IV

Chairs:
T. Mikoteit (Switzerland)

Summary
The relevance of rapid eye movement (REM) sleep for affective disorders derives from well-established abnormalities in depressed patients. REM sleep overdrive with an increased frequency of rapid eye movements (REM density) is a trait marker of major depression. From a clinical point of view, the major question is whether REM sleep features in depression are reliable biomarkers to predict clinical trajectories or treatment outcomes. To start with preclinical insights from rodent models of depression, Kimura will describe the neuroendocrine background of REM sleep alterations in depression focusing on brain-specific CRH overexpression and a depression-associated gene variant P2RX7. Mikoteit will present clinical studies evaluating novel REM-sleep derived biomarkers for prediction of occurrence and treatment outcome of major depression. Alterations of the heart rate variability in REM sleep, especially an increased predominance of very low frequency power over vagus related high frequency power, correlate with REM density and represent a trait marker of depression. In contrast, prefrontal theta cordance derived from QEEG-analysis of REM sleep predicts response to antidepressant treatment and may be a clinical meaningful biomarker for the management of depression. Finally, turning to clinical research, Sadeghi Bahmani will present a cohort study of patients with multiple sclerosis, who very often suffer from both, sleep disturbances and depression. If sleep problems occur at disease onset they are likely to persist and may implicate both, risk for depression and course of disease.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the role of REM sleep in depressive disorders
- Identify its source for meaningful biomarkers for clinical practice
- Recognize the implications of REM sleep for treatment strategies

Target Audience
Sleep researchers, neuroscientists, neurologists, psychiatrists, clinical physicians, psychologists

10:30am – 10:35am	**Introduction** *T. Mikoteit (Switzerland)*
10:35am – 11:00am	**Preclinical models to understand REM sleep in depression** *M. Kimura (Germany)*
11:00am – 11:25am	**REM sleep derived biomarkers for depression: heart rate variability and prefrontal theta cordance** *T. Mikoteit (Switzerland)*
11:25am – 11:50am	**Depression and sleep in patients with multiple sclerosis** *D. Sadeghi (Islamic Republic of Iran)*
11:50am – 12:00pm	**Question and answer** *T. Mikoteit (Switzerland)*

S11 The role of nocturnal eating on insomnia, diurnal sleepiness and obesity

10:30am - 12:00pm I Meeting Hall V

Chairs:
P. Vinai (Italy)

Summary
The influence of sleep disturbances on excessive food intake has been widely hypothesized but the complex link between these factors is so far to be clarified.In the last years a relationship betweeen a delayed rhythm of food intake daytime sleepiness and a increased sensitivity to food reward inducing overeating has been reported, but the influence of Night Eating Syndrome (NES), an eating disorder mainly charachterized by a delayed food intake, on this mechanism, has been poorly studied. Aim of this symposium is to show thmost recent data of the literature on the topic, and the results of our recent studies on NES, insomnia, day time sleepiness, and weight gain. Annalisa da Ros, working in a bariatric center in Venice (Italy) , will present a review of the data on the relationship between insomnia, sleepiness and sensitivity to reward. Piergiuseppe Vinai will present the results of a study on the relationship between nocturnal eating diurnal sleepiness and obesity. The results of this research suggest that NES induce a an increased daytime sleepiness and an excessive weight gain, Orna Tzischinsky and Yael Latzer from Israel will show the results of of their studies on psychopathology and sleep disturbances of patients either affected by NES or by both NES and Binge Eating Disorder. Both the syndromes are characterized by an increased food intake but NES patients have higher levels of psychopathology and sleep disturbances. The results evidences the important role of nocturnal eating in both sleep quality and diurnal eating behavior and excessive food intake.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the influence of nocturnal eating on daytime food intake and obesity
- Identify patients at risk for day time sleepiness
- Identify patients at risk for obesity among insomniac patients

Target Audience
All the researchers and the clinicians interested to sleep and obesity

10:30am - 10:35am	**Introduction** *P. Vinai (Italy)*
10:35am - 10:55am	**Insomia, diurnal sleepiness and sensivity to reward: A review** *A. Da Ros (Italy)*
10:55am - 11:15am	**Sleep monitoring in patients with night eating syndrome with and whitout binge eating disorder** *Y. Latzer (Israel)*
11:15am - 11:35am	**Psychopathology of patients with NES either affected or not by binge eating disorders** *O. Tzischinsky (Israel)*
11:35am - 11:55am	**Obesity, sleepiness and nocturnal eating: What drives what?** *P. Vinai (Italy)*
11:55am - 12:00pm	**Question and answer** *P. Vinai (Italy)*

S12 The interplay between sleep and academic performance: From neural mechanisms to educational policy

10:30am - 12:00pm I North Hall

Chairs:
R. Gruber (Canada)

Summary
Academic success critically contributes to improving future lifetime opportunities for students. The key functional domains that are required for academic success have been shown to be affected by sleep, but unfortunately the role of sleep in academic performance has been largely ignored by educators and policy makers around the world. A better understanding of the relationship between sleep and academic performance would allow to equip policy makers and educators with knowledge that could be used to optimize children's academic performance.
This symposium will first present an overview of the mechanisms that underlie the associations between sleep and academic performance. These include executive functions, memory consolidation, sustained attention,—all of which are essential for academic performance. We will then specifically focus on the presentation of experimental studies that exhibit the impact of sleep on these processes in children, adolescents and university students. We will examine and integrate data from empirical studies in which interventions were used to manipulate sleep duration (i.e., via extension and restriction of sleep) and sleep efficiency (i.e., via cognitive behavioral therapy for insomnia), as a way to test the impacts of these aspects of sleep on the cognitive and emotional processes that are presumed to underlie the associations between sleep and academic performance. Finally, we will extract practical and clinical implications that could be used to guide researchers, educators and policy makers in planning how to move this created knowledge into effective action in educational settings. This is expected to pave the way to enabling the use of sleep improvement as a means for maximizing academic success of students around the world.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the interplay between sleep and academic performance
- Identify key empirical findings that demonstrate the impact of sleep on the processes that underlie the interplay between sleep and academic performance
- Produce innovative strategies that could be used to modify sleep in order to optimize academic performance in students

Target Audience
Sleep researchers, educators interested in developing and implementing innovative strategies to improve academic success, policy makers interested in students success and/or in sleep and health

10:30am - 10:32am	**Introduction** *R. Gruber (Canada)*
10:32am - 10:49am	**Overview of mechanisms that underlie the interplay between sleep and academic performance** *R. Gruber (Canada)*
10:49am - 11:06am	**The impact of improved and extended sleep on key processes needed for academic success** *E. De Bruin (The Netherlands)*
11:06am - 11:23am	**The impact of sleep restriction on key processes needed for academic success** *J.J. Gooley (Singapore)*
11:23am - 11:40am	**The role of sleep in memory consolidation and cognitive abilities – implications to academic success** *S. Fogel (Canada)*
11:40am - 11:57am	**Translating the evidence regarding the interplay between sleep and academic performance into practical strategies for improving academic performance around the world** *J. Owens (United States)*
11:57am - 12:00pm	**Question and answer** *R. Gruber (Canada)*

S13 OSA and atherogenesis: Reversible or not?

10:30am - 12:00pm I Club A and B

Chairs:
D. Gozal (United States), J.M. Marin (Spain)

Summary
Obstructive sleep apnea is a common and highly prevalent disorder affecting 8-10% of the general population. Obesity constitutes a major risk factor of OSA, the latter being characterized by recurrent upper airway collapse that occurs during sleep, and leads to episodic hypoxemia and sleep fragmentation. Both epidemiologic and intervention-based studies have provided conclusive evidence indicating a causative link between OSA and cardiovascular morbidity, independently from associated obesity. However, the mechanisms underlying the accelerated atherogenesis that is putatively ascribed to OSA remain elusive. Chronic intermittent hypoxia (CIH) during the sleep period, has been used as a useful murine model of OSA, and promotes the presence of increased atherogenesis, particularly in conjunction with other predisposing risk factors such as transgenic ablation of LdlR or ApoE, or concurrent feeding of a high fat diet. Macrophages are central to the processes mediating atherogenesis, and have been implicated in the initiation and progression of atherosclerosis through intrinsic activation, changes in polarity and phenotype, production of cytokines, and signaling to other cells in the vessel wall. It is now well established that the presence of a pro-inflammatory M1-like macrophage phenotype is associated with increased plaque burden and adverse outcomes. Specifically, activation of aortic macrophages through scavenger receptor B3 (CD36) has been shown to mediate atherosclerosis development in other pro-atherogenic disease models. Recent reports have also suggested that epigenetic modifications may determine phenotype and activity profiles of macrophages, and more specifically the pro- or anti- atherogenic profile of macrophages in vascular areas prone to atherosclerosis, suggesting the possibility that epigenetic mechanisms may underlie aspect of macrophage M1 polarization in CIH, as well as the potential reversibility of the atherogenic process following treatment. In this symposium we will examine the contributions of IH during sleep to lipid biology and atherosclerosis (Dr. Polotsky). This will be followed by presentation of the role of CD36 macrophages in IH-induced atherosclerosis and the reversibility of the process with treatment in mice (Gozal). The effects of intermittent hypoxia on the vasculature will then be presented by Poulin and followed by results from an interventional trial in patients with OSA (Marin)

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the contribution of intermittent hypoxia (IH) during sleep to lipid homeostasis and atherosclerosis
- Define the role of specific macrophage subtypes to IH-induced atherogenesis and their epigenetic modifications
- Integrate the role of IH in humans and its effect on the vasculature
- Appraise the potential impact of sleep apnea on atherosclerosis in humans and the reversibility conferred by CPAP treatment

Target Audience
Professionals interested in sleep disordered breathing, macrophage biology, lipid biology, epigenetics, human trials, inflammation

10:30am - 10:35am	**Introduction** *D. Gozal (United States)*
10:35am - 10:55am	**Intermittent hypoxia, lipids and atherogenesis in murine models** *V. Polotsky (United States)*
10:55am - 11:15am	**Chronic intermittent hypoxia, macrophage epigenetics and atherosclerosis in mice: Is it reversible?** *D. Gozal (United States)*
11:15am - 11:35am	**Intermittent hypoxia in humans: Vascular correlates and potential mechanisms** *M. Poulin (Canada)*
11:35am - 11:55am	**Atherosclerosis in OSA: The EPIOSA study** *J.M. Marin (Spain)*
11:55am - 12:00pm	**Question and answer** *J.M. Marin (Spain)*

S15 Local sleep and local wake: From basic science to sleep arousal disorders

10:30am - 12:00pm I Meeting Hall 1B

Chairs:
R. Benca (United States)

Summary
Sleep has been traditionally considered as a global process involving the whole brain. However, it has now been established that sleep and wake are not mutually exclusive, as they can co-exist in different brain regions at the same time. "Local Sleep" has been extensively studied in non-human animals, and knowledge gained from basic research has led to extraordinary progress in our understanding of human sleep. In addition, research on human sleep disorders is shedding light on important basic sleep concepts. In particular, Disorders of Arousal (DOA) offer a unique opportunity to study sleep/wake local phenomena and represent a pathological model for local sleep and dissociation between behavior and various aspects of consciousness. This translational symposium will give an overview on the state of the art in current local sleep and local wake research, covering its cellular, neurophysiological, clinical, and methodological aspects. Vlad Vyazovskiy will start with a general introduction to the topic by summarizing findings in animals. Lino Nobili will present electrophysiological data derived from studies conducted with intracerebral recordings in epileptic patients (some of them with a DOA comorbidity); in particular, he will show physiological and pathological local phenomena and their correlation with clinical manifestations of DOA, highlighting differences and similarities between DOA and epileptic manifestations. Anna Castelnovo will cover clinical applications of local sleep research, showing that High Density EEG and applied EEG source modeling techniques might represent valuable tools for the investigation of DOA. She will show how slow wave activity is abnormally distributed in DOA and how this abnormality potentially holds true throughout different brain states, from NREM to REM and WAKE. Finally, Carlos Schenck will conclude the symposium with an overview on new clinical frontiers and promising future research areas for achieving a deeper understanding of DOA.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Summarize major findings on local sleep and wake from basic sleep research
- Integrate information from basic, neurophysiological and clinical research in the understanding of Disorders of Arousal (DOA)
- Describe Sleep Slow Wave Activity in DOA
- Identify new clinical frontiers and promising future research areas for achieving a deeper understanding of DOA

Target Audience
Sleep specialists, neurologists, neurophysiologists, residents, sleep researchers

10:30am - 10:35am	**Introduction** *R. Benca (United States)*
10:35am - 10:55am	**Local sleep and local waking: Insights from animal models** *V. Vyazovskiy (United Kingdom)*
10:55am - 11:15am	**Fluid boundaries in wake and sleep: Adaptive and maladaptive features in human sleep** *L. Nobili (Italy)*
11:15am - 11:35am	**Local changes in slow wave activity: A trait feature of sleep arousal disorders?** *A. Castelnovo (United States)*
11:35am - 11:55am	**Disorders of arousal: Future research areas** *C. Schenck (United States)*
11:55am - 12:00pm	**Question and answer** *R. Benca (United States)*

S16 Nasal obstruction and its role in sleep disordered breathing

10:30am - 12:00am I Terrace 1

Chairs:
C. Torre (United States)

Summary
Nasal obstruction is related to sleep disordered breathing (SDB) in several ways: 1) reduces airflow through the collapsible airway, therefore increasing upper airway resistance, 2) forces patients to become oral breathers during sleep, which leads to narrowing of the airway, and 3) interferes with the nasal reflexes that stimulate ventilation. Systematic evaluation of nasal obstruction remains challenging due to the high number of factors that contribute to nasal obstruction. In most settings, nasal examination is limited to the evaluation of the anterior septum, internal nasal valve angle, and inferior turbinate size. Frequently, this limited examination of the anterior nasal cavity does not correlate with patient symptoms, who may still complain of nasal obstruction despite no signs of objective anatomical abnormalities. Since structural and inflammatory problems often coexist and need to be addressed concurrently in order to reestablish normal nasal function, it is important to consider other etiologies leading nasal obstruction such as a narrow pyriform aperture, posterior septal deviation, and chronic sinusitis. Optimization of nasal breathing is particularly important in the treatment of pediatric SDB. Continuous oral breathing often leads to a transverse maxillary deficiency that deepens the palatal arch. A narrow maxilla with a high arched palate has been correlated with increased nasal airflow resistance and with increased potential of developing OSA as an adult. We will discuss the role of nonsurgical nasal cavity expansion in the treatment of pediatric SDB to reduce the potential for oral breathing and the implications this might have in the craniofacial skeletal development of the child. We formally introduce Distraction Osteogenesis Maxillary Expansion (DOME) for adults with OSA, a minimally invasive surgical-orthodontic procedure that addresses maxillary transverse deficiency with simultaneous increase in nasal cavity volume. Using maxillary expander devices secured by mini-implants into the hard palate along the mid-palatal suture, surgical osteotomies are minimized for predictable expansion of the maxilla. We report results of the first 50 patients at Stanford who underwent DOME, with emphasis on safety and efficacy assessed by objective and subjective measures. We then place the role of addressing nasal obstruction within the context of overall surgical treatment for OSA. Whether it is for improved adherence to CPAP therapy, or for patients who proceed to other airway procedures such as maxillomandibular advancement (MMA) or upper airway stimulation (UAS), we examine the contribution of intra-nasal and extra-nasal procedures to overall treatment success, and not simply surgical success, of OSA care.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Review the different elements leading to compromised nasal breathing in pediatric and adult OSA patients
- Demonstrate proper examination and identification of the factor/s leading to nasal obstruction
- Demonstrate the effects of nasal expansion and surgical methods in pediatric and adult OSA population: review of rhinomanometry and computation fluid dynamics
- Recommend the best approach to correct nasal obstruction and re-establish proper nasal breathing

Target Audience
Sleep Physicians, pediatricians, ENT surgeon, orthodontist & dentist

10:30am - 10:35am	**Introduction** *C. Torre (United States)*
10:35am - 10:55am	**Understanding nasal obstruction and compromised nasal flow dynamics** *C. Torre (United States)*
10:55am - 11:15am	**Pediatric nonsurgical nasal cavity expansion** *S. Quo (United States)*
11:15am - 11:35am	**Distraction osteogenesis maxillary expansion with palatal implants for OSA in adults** *A. Yoon (United States)*
11:35am - 11:55am	**MMA following maxillary expansion: What have we learned** *S. Liu (United States)*
11:55am - 12:00am	**Question and answer** *C. Torre (United States)*

O01 Sleep breathing disorders oral abstract presentations

10:30am – 12:00pm I Club D and E

Chairs:
A. Pack (United States), P.J. Strollo (United States)

10:30am – 10:45am	**RISK FACTORS OF OBSTRUCTIVE SLEEP APNEA SYNDROME IN CHINESE CHILDREN** *Z. Xu (China)*
10:45am – 11:00am	**AN INTEGRATED META-OMICS BASED APPROACH IN PEDIATRIC OBSTRUCTIVE SLEEP APNEA SYNDROME** *H. Xu (China)*
11:00am – 11:15am	**CIRCULATING LEVELS OF MICRORNA-210 AND MICRORNA-126 ARE INCREASED IN HYPERTENSIVE PATIENTS SUFFERING FROM OBSTRUCTIVE SLEEP APNEA: PILOT RESULTS** *J. Novak (Czech Republic)*
11:15am – 11:30am	**BIOMECHANICAL AND STRESS DISTRIBUTION EFFECTS OF MAXILLARY EXPANSION METHODS (SARPE, MARPE, DOME) USING FINITE ELEMENT MODEL** *Y.-F. Chen (United States)*
11:30am – 11:45am	**LONG TERM ORAL APPLIANCE THERAPY IMPROVES DAYTIME FUNCTION AND MOOD IN UPPER AIRWAY RESISTANCE SYNDROME PATIENTS** *L. Godoy (Brazil)*
11:45am – 12:00pm	**THE EFFECT OF EXTENDED WAKEFULNESS ON POSTURAL CONTROL IN OBSTRUCTIVE SLEEP APNEA AND HEALTHY CONTROLS** *D. Stevens (Australia)*

O02 Insomnia oral abstract presentations

10:30am – 12:00pm I Club H

Chairs:
B. Bjorvatn (Norway), K. Morgan (United Kingdom)

10:30am – 10:45am	**SHORT AND LONG TERM PROLONGED RELEASE MELATONIN TREATMENT FOR SLEEP DISORDERS IN CHILDREN WITH AUTISM SPECTRUM DISORDERS: RESULTS OF A PHASE III RANDOMIZED CLINICAL TRIAL** *P. Gringras (United Kingdom)*
10:45am – 11:00am	**YOUNG WOMEN WITH SHORT SLEEP DURATION AND INSOMNIA RUN A HIGH RISK OF DEVELOPING HYPERTENSION AND DIABETES MELLITUS. A 10-YEAR FOLLOW-UP OF THE POPULATION-BASED SHE STUDY** *J. Theorell-Haglöw (Sweden)*
11:00am – 11:15am	**HYPERACTIVITY OF THE OREXIN SYSTEM AND CHRONIC INSOMNIA IN A MOUSE MODEL OF ALCOHOL DEPENDENCE** *C. Blanco-Centurion (United States)*
11:15am – 11:30am	**DISRUPTED WHITE MATTER INTEGRITY IN INSOMNIA AND MAJOR DEPRESSIVE DISORDER: CORRELATIONS WITH SUBJECTIVE AND OBJECTIVE SLEEP PARAMETERS** *J.W. Winkelman (United States)*
11:30am – 11:45am	**THE EFFECTS OF INSOMNIA SYMPTOMS AND OBJECTIVE SHORT SLEEP DURATION ON MEMORY PERFORMANCE IN ADOLESCENTS AND YOUNG ADULTS** *J. Ling (Hong Kong)*
11:45am – 12:00pm	**ASSOCIATION BETWEEN STRESS-INDUCED AROUSAL AND NOCTURNAL SLEEP: A PRELIMINARY STUDY** *I.Y. Chen (Canada)*

K03 Racial difference in sleep disorders

Y. Inoue (Japan)

Keynote 11:15am - 12:00pm I Congress Hall

It is widely accepted that various factors including social life schedule, genetic background and bed room environment may affect nocturnal sleep. Given this, we should consider a possibility that frequency and symptomatic characteristics of sleep problems/disorders may differ depending on racial or regional difference. In this presentation, with aims of promoting sleep health and sleep medicine we would like to focus on difference in sleep habits as well as the prevalence and clinical characteristics (symptoms and treatment responses) of insomnia, narcolepsy, sleep related breathing disorders, movement disorders and parasomnias among populations in countries around the world.

TEVA Industry Satellite Symposium: Challenges of recognizing and treating excessive sleepiness

12:30pm - 2:00pm I Congress Hall

Chairs:

R. Rosenberg (United States)

Excessive sleepiness is a serious, debilitating, condition with consequences not only for the individual, but also for public health and safety. It is time to listen experts to discuss the challenges of recognizing and treatment of excessive sleepiness.

A satellite symposium brought to you by TEVA.

12:30pm – 12:45pm	**Opening Remarks** *R. Rosenberg (United States)*
12:45pm – 1:15pm	**Excessive sleepiness (ES) in OSA** *R. Grunstein (Australia)*
1:15pm – 1:45pm	**Therapeutic approaches to ES** *R. Rosenberg (United States)*
1:45pm – 2:00pm	**Panel discussion** *R. Rosenberg (United States), R. Grunstein (Australia)*

Merck Industry Satellite Symposium: New approaches to personalizing treatment of insomnia: Why and how?

12:30pm - 2:00pm I Meeting Hall IV

A satellite symposium brought to you by MSD / Merck.

12:30pm – 12:40pm	**Welcome and introduction** *C. Morin (Canada)*
12:40pm – 1:00pm	**Health consequences of insomnia: Cognition, mood and medical impact** *C. Morin (Canada)*
1:00pm – 1:20pm	**Personalization of insomnia therapy: Matching treatment mechanisms with patient needs** *A. Krystal (United States)*
1:20pm – 1:40pm	**Orexin receptor antagonists in the management of insomnia – Mechanisms and clinical implications** *T. Roth (United States)*
1:40pm – 2:00pm	**Closing remarks and question and answer** *C. Morin (Canada)*

Philips Industry Satellite Symposium: Boosting slow wave sleep to improve cognitive outcomes

12:30pm - 2:00pm I North Hall

Chairs:

T. Lee-Chiong (United States)

The Philips scientific program will explore the different mechanisms to enhance slow wave sleep and improve cognition and memory with a focus on the acoustical methods.

12:30pm – 12:35pm	**Introduction** *T. Lee-Chiong (United States)*
12:35pm – 1:05pm	**Novel ways to enhance slow wave sleep** *P. Zee (United States)*
1:05pm – 1:35pm	**Acoustical enhancement of slow wave sleep** *C. Anderson (Australia)*
1:35pm – 2:00pmq	**Question and answer**

T. Lee-Chiong (United States)

S17 New evidence on the treatment of insomnia comorbid with depression, pain, sleep apnea or circadian disorders

2:00pm - 3:30pm I Congress Hall

Chairs:
C. Morin (Canada), Y.K. Wing (Hong Kong)

Summary
Insomnia is the most common sleep-related complaint in clinical practice. It can present as a disorder of its own or, more commonly, it is associated with a comorbid psychiatric or medical disorder, or another sleep disorder. Although significant advances have been made in treating chronic insomnia, outcome is far from being optimal for many patients, particularly those with comorbidities. For instance, there is still little evidence on how best to treat insomnia that is comorbid with major depression, chronic pain, sleep-related breathing disorders, or insomnia associated with circadian disorders in teenagers. This symposium brings together international experts and investigators who will present their most recent findings from randomized clinical trials on the treatment of insomnia associated with different comorbidities. YK Wing (U. Hong Kong, China) will present new data on the added value of treating sleep disturbances in treatment-resistant depression. MR Irwin (UCLA, USA) will present findings from a randomized controlled trial looking at pain, depression, and insomnia in patients with rheumatoid arthritis. AM Sweetman (Flinders U., Australia) will present data from a recently completed clinical trial addressing the benefits of treating insomnia in patients with comorbid obstructive sleep apnea with CPAP adherence, quality of life, and sleep quality as primary end points. AG Harvey (U. California, Berkeley, USA) will address the challenging issue of treating sleep-wake disturbances in adolescents who often present circadian dysregulations. Finally, CM Morin (U. Laval, Canada) will present data from a two-site pragmatic clinical trial (n = 240 patients) investigating the effectiveness of different sequential therapies involving both CBT and medication (zolpidem, trazodone) for insomnia disorder with and without comorbid psychiatric disorders (i.e., depression and anxiety). This symposium will gather worldwide leaders on insomnia research who will present the most up-to-date evidence on how best to manage insomnia associated with different comorbidities. Because of its transdiagnostic focus and coverage of different therapeutic options (cognitive-behavioral, pharmacological and bright light therapies), this symposium is likely to attract a large audience including both sleep medicine clinicians as well as sleep research investigators who are interested in learning on the most recent advances in treating insomnia in the context of comorbid psychiatric, medical, and other sleep disorders.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Identify key diagnostic and clinical features of insomnia disorder
- Appraise the most up-to-date findings on treatment of insomnia associated with various psychiatric and medical comorbidities
- Recognize specific evidence-based therapies that can be implemented in clinical practice for treating various clinical presentations of insomnia disorders

Target Audience
Sleep medicine clinicians and researchers interested on the broad topic of insomnia therapies

2:00pm - 2:02pm	**Introduction** *C. Morin (Canada)*
2:02pm - 2:19pm	**Treating sleep disturbances in treatment resistant depression – does it help?** *Y.K. Wing (Hong Kong)*
2:19pm - 2:36pm	**Treatment of pain, depression, and sleep disturbances in rheumatoid arthritis patients** *M.R. Irwin (United States)*
2:36pm - 2:53pm	**Cognitive behavioral therapy for insomnia in patients with comorbid obstructive sleep apnea** *A.M. Sweetman (Australia)*
2:53pm - 3:10pm	**Triple vulnerability? Circadian tendency, sleep deprivation and adolescence: A randomized controlled trial** *A. Harvey (United States)*
3:10pm - 3:27pm	**Sequential therapies with CBT and medication for insomnia with and without comorbid psychiatric disorders** *C. Morin (Canada)*
3:27pm - 3:30pm	**Question and answer** *Y.K. Wing (Hong Kong)*

S18 Beyond academic walls: Society education as an essential field in sleep science

2:00pm - 3:30pm I Meeting Hall IV

Chairs:
M.L. Andersen (Brazil)

Summary

The importance of an adequate sleep for health and well-being is well known for researchers in Sleep Medicine; however, the dissemination of sleep knowledge is limited. In other words, dissemination is limited to our peers, to researcher, and to physicians who work in sleep field. One area of agreement is that sleep knowledge should be better spread in the broader society; however, questions arise when we are challenged to communicate and apply sleep knowledge in the current society: How to plan and create educational strategies? How to communicate with a diverse public? What is the impact of sleep educational strategies to health-related outcomes? The proposed symposium has the primary aim to discuss the importance of educational approaches applied in diverse contexts and target audiences to increase societal awareness about sleep. Furthermore, the symposium will explore potential gains for sleep research and clinical practice due to scientific communication beyond academic walls. We believe that scientists and physicians have an essential role in disseminate information, educate society, and influence public policies with the goal of forwarding sleep awareness. An international congress which brings together specialists in a wide range of sleep topics is a great place to discuss strategies and share effective ideas to increase society awareness about sleep.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Apply and discuss concepts of communication of sleep knowledge beyond the academic and medical boundaries
- Appraise the importance of sleep educational strategies in society, with special focus on pediatric population and education professionals
- Debate the impact of sleep awareness to improve health-related outcomes
- Plan actions to increase society awareness about sleep

Target Audience

Researchers, physicians and students

2:00pm - 2:05pm	**Introduction** *M.L. Andersen (Brazil)*
2:05pm - 2:25pm	**Scientific communication in sleep: Why, how and to whom** *P. Araujo (Brazil)*
2:25pm - 2:45pm	**Education campaigns to improve sleep of socioeconomically disadvantaged children** *J.A. Mindell (United States)*
2:45pm - 3:05pm	**Strategies to increase sleep awareness in health professionals** *L. Kim (Brazil)*
3:05pm - 3:25pm	**Sleep timing: Health consequences and social gains** *T. Roenneberg (Germany)*
3:25pm - 3:30pm	**Question and answer** *M.L. Andersen (Brazil)*

S19 Sleep disorders in the adolescent population: The missing link

2:00pm - 3:30pm I North Hall

Chairs

S. Weiss (Canada), S. Kothare (United States)

Summary

The focus of research and clinical advances in pediatric sleep medicine has been predominantly observed in neonates to children in elementary and middle school but not in teenagers. Aside from advances in understanding the biological changes in sleep/wake rhythms, and the impact of insomnia and circadian disturbances during adolescence, there has been a lack of attention to other sleep-related issues in this age group who represent the transition from childhood to adulthood.
Despite this lack of attention, there is increasing evidence that as in adults, inadequate sleep in adolescence has significant widespread ramifications across physiologic, psychological, and cognitive domains. In addition to the increasing recognition of the importance of sleep during adolescence, the understanding of how sleep disorders may manifest uniquely in this age group is also gradually evolving. The evaluation and treatment of sleep disorders in adolescents differs significantly from children and adults. Several studies suggest that the prevalence of sleep problems in adolescents is high (at least 20%) and that particular groups of adolescents, such as those with chronic medical or psychiatric problems (e.g., depression), may be at increased risk for sleep disorders. The more frequent disturbances are represented by insomnia and a delay of bedtime, with a progressive worsening often resulting in delayed sleep phase syndrome. In this perspective, the intrusion of technology into the bedroom of adolescents represents a disruptive factor for sleep quality and duration leading to chronic sleep restriction. Therefore a large number of adolescents constantly cope with "sleep debt" during school days that can lead to significant neurobehavioral disturbances, as well as negative influence on mood, vigilance, attention, memory, and school performances. The symposium will bridge the gap between management of children versus adults. This symposium brings international experts in the areas of pediatric pulmonology and neurology from five countries together to focus on sleep disorders during adolescence. The presenters are aware that many symposiums at national and international sleep meetings are focused on pediatric, adult or geriatric sleep, but few are specifically targeted on adolescent sleep disorders. Advances in research discoveries, and both investigation and clinical management of diverse sleep disorders in adolescents will be presented.
The co-chairs of the symposium have recently both published in this area; Sleep Disorders in Adolescents, A Casebook, published by Springer in 2016, co-edited by S. Kothare. Adolescent and Young Adult Health Care, A Practical Guide, published by Wolters Kluwer, 2016, chapter on Sleep Disorders by S. Weiss/A.Coloumbe.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Develop awareness of research advances in sleep medicine in adolescents
- Recognize unique sleep disorders which typically present during the adolescent years
- Recognize the missing link- how knowledge of adolescent sleep is important in the understanding of childhood disorders than can persist to become adult sleep disorders
- Summarize the unique differences in the presentation of sleep disorders in adolescents with a wide range of comorbid conditions
- Apply this knowledge to clinical practice in the evaluation and management of adolescents with sleep disorders

Target Audience

Adult and pediatric sleep physicians, fellows, nurse practitioners and research faculty

2:00pm- 2:01pm	**Introduction** *S. Weiss (Canada)*
2:01pm - 2:03pm	**Summary** *S. Kothare (United States)*
2:03pm - 2:20pm	**Sleep disordered breathing in adolescents: The missing link** *U. Katwa (United States)*
2:20pm - 2:37pm	**Sleep disorders and mental health comorbidities in adolescents** *O. Bruni (Italy)*
2:37pm - 2:54pm	**Seizures vs. nonepileptic events in sleep in adolescents** *G. Shukla (India)*
2:54pm - 3:11pm	**Sleep co-morbidity with traumatic brain injury (TBI) in adolescents** *S. Kothare (United States)*
3:11pm - 3:28pm	**Sleep disorders in adolescents with neurodevelopmental disorders** *S. Weiss (Canada)*
3:28pm - 3:30pm	**Question and answer** *S. Weiss (Canada)*

S20 Advances in obstructive sleep apnea pathogenesis and non-CPAP therapies

2:00pm - 3:30pm I Club A and B

Chairs:
R. Heinzer (Switzerland)

Summary

Obstructive sleep apnoea (OSA) is a multifactorial disorder. Current treatments are often poorly tolerated (e.g. CPAP) or only partially efficacious (e.g. oral appliances and surgical interventions). Thus, there is an urgent need for new and effective therapies to treat OSA. Improved understanding of the multiple causes of OSA offers the potential to develop and tailor novel and existing therapies according to each patient's specific pathophysiology.
Recent insights into OSA pathophysiology have identified at least four distinct phenotypic causes or traits. This session will provide a state of the art account of each of these traits derived from detailed physiological studies presented by experts in the field. Professor Alan Schwartz from John's Hopkins, USA will provide an overview of the role of upper airway anatomy and collapsibility (Pcrit), Associate Professor Danny Eckert, Neuroscience Research Australia, Sydney will cover the latest advances on the roles of upper airway muscles and the respiratory arousal and threshold, and Associate Professor Andrew Wellman, Harvard USA will discuss the role of respiratory control in OSA pathogenesis. A state of the art update on current and future pathways for non-CPAP therapies that are emerging based on improved understanding of OSA pathogenesis will be provided by Professor Malcolm Kohler, Zürich, Switzerland.
There is a pressing need to move beyond the "one-size-fits-all" approach to treatment for OSA. This session involving expert faculty from the USA, Australia and Europe will highlight the latest on disease mechanisms and potential avenues for new approaches for treatment. Thus, this session will be of major interest to World Sleep 2017 attendees.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Define the key causes of obstructive sleep apnoea
- Recognize the importance of differences in the causes of obstructive sleep apnoea between individuals
- Apply the latest knowledge in obstructive sleep apnoea pathogenesis to inform treatment decisions

Target Audience
Sleep physicians, scientists and allied health professionals with an interest in understanding the causes of obstructive sleep apnoea to improve treatment

2:00pm - 2:05pm	**Introduction** *R. Heinzer (Switzerland)*
2:05pm - 2:25pm	**The role of pharyngeal anatomy and airway collapsibility in the pathogenesis of OSA** *A. Schwartz (United States)*
2:25pm - 2:45pm	**Importance of the upper airway muscles and arousal threshold in the pathogenesis of OSA** *D. Eckert (Australia)*
2:45pm - 3:05pm	**The role of respiratory control in the pathogenesis of OSA** *A. Wellman (United States)*
3:05pm - 3:25pm	**Current and future pathways for non-CPAP therapies to treat OSA** *M. Kohler (Switzerland)*
3:25pm - 3:30pm	**Question and answer** *R. Heinzer (Switzerland)*

S21 European Narcolepsy Network (EU-NN) - Narcolepsy: From etiology to treatment

2:00pm - 3:30pm I Meeting Hall 1A

Chairs:
C. Bassetti (Switzerland)

Summary
The European Narcolepsy Network (EU-NN) narcolepsy symposium will focus on both the auto-immune etiology and current treatment options for the primary sleep disorder narcolepsy. Chairperson will be Prof. Dr. Claudio Bassetti, current vice-president and founder of the EU-NN. First, the EU-NN will be introduced by its current president, Prof. Dr. Geert Mayer. Dr. Ramin Khatami will then present the latest analyses of the EU-NN clinical database and relate these data to the current diagnostic criteria for narcolepsy type 1 and 2. The following two presentations by Dr. Ulf Kallweit and Dr. Christelle Peyron will cover the latest insights into the auto-immune hypothesis of narcolepsy. Both human and animal data will be covered. An increased incidence of narcolepsy after H1N1 infection and vaccination have triggered renewed interest in the pathology of narcolepsy. Genome-wide association studies showed a strong association with HLA DQB1*06:02 and other genes involved in immune modulation, such as T cell receptor alpha and OX40L, supporting the notion that narcolepsy is a T-cell-mediated autoimmune disease. Dr. Rolf Fronczek will end the symposium with an overview of the current methods that are used to assess narcolepsy. This will not only include well-known electrophysiological tests and questionnaires, but also tests that focus on an often neglected symptom: decreased vigilance and impaired daytime performance. Furthermore, current treatment options will be discussed as well as new options that are expected to be available in the near-future.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the existence of the European Narcolepsy Network (EU-NN) and its clinical database
- Review the new diagnostic criteria of narcolepsy type 1 and 2, but also understand the limitations of these criteria and relate this to the clinical assessment of narcolepsy
- Summarize the latest findings regarding the auto-immune hypothesis for the etiology of narcolepsy in both humans and animals
- Describe the current treatment options for narcolepsy; and gain some insight in future possible treatment options

Target Audience
Sleep specialists (physicians), basic sleep scientists, sleep technicians interested in the diagnosis and therapy of narcolepsy

2:00pm - 2:02pm	**Introduction** *C. Bassetti (Switzerland)*
2:02pm - 2:19pm	**Introduction to the EU-NN** *G. Mayer (Germany)*
2:19pm - 2:36pm	**Clinical picture of narcolepsy and its borderland (based on the EU-NN database)** *R. Khatami (Switzerland)*
2:36pm - 2:53pm	**Auto-immunity and the etiology of narcolepsy - the human story** *U. Kallweit (Switzerland)*
2:53pm - 3:10pm	**Auto-immunity and the etiology of narcolepsy - the animal story** *C. Peyron (France)*
3:10pm - 3:27pm	**Treatment for narcolepsy (and its assessment)** *R. Fronczek (The Netherlands)*
3:27pm - 3:30pm	**Question and answer** *C. Bassetti (Switzerland)*

S22 The waking, sleeping and dreaming brain: New circuits and insights

2:00pm - 3:30pm I Meeting Hall 1B

Chairs:
P.M. Fuller (United States), M. Lazarus (Japan)

Summary
Understanding the circuit basis by which the brain regulates behavioral state control, including maintenance of wake, non-rapid-eye-movement (NREM) sleep and rapid-eye-movement (REM; or paradoxical) sleep, and the transitions between these states has long been a goal of sleep neuroscientists and sleep neurologists. Over the past 5 years, and due in part to the advent of newer genetic-based tools and approaches that have permitted the unprecedented interrogation of discrete circuit elements (transmitters, pathways, cell populations) in behaving animals, we have gleaned new, and often unexpected, insights into this circuitry. In turn new cellular and molecular targets for treating sleep-based disorders have been identified, marking this work in particular as central to the research and clinical mission within the international sleep community. Additional important insights have been gleaned into the mechanisms underlying sleep/wake regulatory processes (sleep need, circadian drive, motivated behavior) and state-dependent consolidation of memory. The objective of our symposium is to highlight some of this recent work. The symposium format will allow us to cover the entire continuum of behavioral state control, rather than maintaining an exclusive focus on one behavioral state, e.g., Wake or NREM. In other words, our integrative approach will allow us to achieve a "whole" that is far greater than its individual parts. Our invited speakers have all recently published high profile papers in which they have identified new circuits controlling wake, NREM and REM sleep. For example, Dr. Luis de Lecea's group recently published a paper showing a fundamental role for midbrain dopaminergic circuitry in the maintenance of the awake state. Dr. Akihiro Yamanaka's group recently published a paper on the role of NREM sleep-dependent long-term memory consolidation in cortical structures. Dr. Antoine Adamantidis will discuss his group's work on REM-related circuits and their link to hippocampal-dependent learning. Dr. Anne Venner will discuss the discovery of a hitherto unknown population of wake-promoting lateral hypothalamic neurons. Finally, Dr. Yo Oishi will present his research on a novel sleep circuit in the basal ganglia that is under motivated behavior.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Differentiate and describe the neural circuits by which the brain regulates behavioral state control, including maintenance of wake, non-rapid-eye-movement (NREM) sleep and rapid-eye-movement (REM; or paradoxical) sleep
- Evaluate and contrast old and new concepts and models describing the regulation of sleep and wakefulness by homeostatic, circadian and motivational processes
- Describe and comprehend the relationship between state-dependent brain activity and memory and learning
- Give examples of and critically evaluate the appropriate application of cutting-edge molecular neuroscience tools, including optogenetics, chemogenetics, in vivo photometry and optical mapping

Target Audience
Sleep neuroscientists/researchers, sleep neurologists. Level: graduate students, postdoctoral fellows and faculty

2:00pm - 2:02pm	**Introduction** *P.M. Fuller (United States)*
2:02pm - 2:19pm	**Dopaminergic control of sleep and waking** *L. de Lecea (United States)*
2:19pm - 2:36pm	**A novel inhibitory wake-promoting hypothalamic circuit** *A. Venner (United States)*
2:36pm - 2:53pm	**The role of the nucleus accumbens in sleep regulation** *Y. Oishi (Japan)*
2:53pm - 3:10pm	**Regulatory mechanisms of sleep and memory by hypothalamic neurons** *A. Yamanaka (Japan)*
3:10pm - 3:27pm	**Hypothalamic control of REM sleep** *A. Adamantidis (Switzerland)*
3:27pm - 3:30pm	**Question and answer** *M. Lazarus (Japan)*

S23 In search of alternatives to dopaminergic ligands in RLS/WED: The Emerging role of glutamate and adenosine

2:00pm - 3:30pm I Club D and E

Chairs:
D. Garcia-Borreguero (Spain)

Summary
RLS/WED is a neurological disorder that causes significant sleep disturbance and sleep loss in severe patients. A previous survey showed that approx. 50% of the severe RLS patients sleep, on average, less than 5 hours per night (Hening et al., 2004). Despite the significant sleep loss, most patients do not report daytime sleepiness. Neither do multiple sleep latency test show increased daytime sleepiness, even when compared to sleep-deprived healthy controls (Gamaldo et al., 2009). Thus, it has been suggested that hyperarousal might play a role in the pathophysiology of RLS/WED and this idea has also been supported by spectral EEG analysis at sleep onset showing increased high-frequency band power, similarly to insomnia (Ferri et al., 2014). Over the years, most research on the pathophysiology of RLS/WED has focused on dopaminergic mechanisms. However, recent data suggest that glutamatergic and adenosine- related mechanisms might be playing an important role too. For example, thalamic glutamatergic activity has been demonstrated to be increased in RLS patients (Allen et al., 2013), which could produce hyperarousal associated with increased waking during sleep, mostly affecting non-REM sleep and not related to PLMS (Ferri et al., 2010, Manconi et al., 2012). And recent data on brain iron-deficient rodents suggest that adenosine A1 receptor downregulation might play a key role in PLMS and hyperarousal (Quiroz et al, 2016). The alterations in adenosine-mediated modulation of dopamine and glutamate neurotransmission may be fundamental in the elicitation of the full range of RLS symptoms, with dopamine more related to sensory symptoms and PLMS and glutamate more related to the RLS hyperarousal with sleep disruption. Furthermore, chronic sleep loss induces by itself changes in these neurotransmitters that could further worsen disease severity. Indeed, a decrease in arousal might be the mechanism by which a number of drugs improve RLS. These interactions between adenosine, glutamate and dopamine lay the ground for future treatments for RLS/WED. The Symposium will review current knowledge on the role of glutamate and adenosine in the regulation of arousal in RLS, and discuss its implications for future treatment.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize neurophysiological and neurochemical mechanisms leading to hyperarousal in RLS/WED
- Identify the role of glutamate and adenosine in RLS/WED
- Describe the relevance of hyperarousal in RLS/WED
- Identify the future treatment implications of hyperarousal in RLS

Target Audience
Basic scientists, sleep clinicians, neurologists

2:00pm - 2:02pm	**Introduction** *D. Garcia-Borreguero (Spain)*
2:02pm - 2:19pm	**The role of hyperarousal in the neurophysiology of RLS and PLMS** *R. Ferri (Italy)*
2:19pm - 2:36pm	**Adenosine neurotransmission in RLS** *S. Ferré (United States)*
2:36pm- 2:53pm	**Adenosine-dopamine receptor interactions in the spinal cord of RLS animal models** *S. Clemens (United States)*
2:53pm - 3:10pm	**Glutamate, adenosine and sleep deprivation** *H.-P. Landolt (Switzerland)*
3:10pm- 3:27pm	**Implications for future treatments** *D. Garcia-Borreguero (Spain)*
3:27pm - 3:30pm	**Question and answer** *D. Garcia-Borreguero (Spain)*

S24 Personalization of mandibular advancement devices: Digital analysis of the movements achieved and mathematical model for the study of the jaw kinematics.

2:00pm - 3:30pm I Terrace 1

Chairs:
J. Vila (Spain)

Summary
Mandibular advancement devices (MADs) have increasingly been recognized and prescribed for patients with mild to moderate obstructive sleep apnea (OSA). Furthermore, it has been shown that MADs also have beneficial effects in some cases of severe OSA. The mode of action of MADs is to increase the upper airway volume, especially at the pharyngeal level, by moving the mandible. The key to understand different degrees of success rate of MADs, is to recognize that OSA is a multifactorial disorder, and is not simply due to poor upper-airway anatomy. Recent evidence suggests that several additional non-anatomical traits also contribute to the pathogenesis of OSA including but not limited to: 1) an inability of the pharyngeal muscles to hold open or stiffen the airway, 2) an oversensitive ventilatory control system, 3) and a low respiratory arousal threshold. Investigations into how MADs alter these traits are crucial to design a customized MAD. There is a high variability in the design features of available MADs. Differences are predominantly related to the degree of customization to the patient's dentition and also to the design: one-piece (monobloc) with no option for mouth opening versus two-pieces (separate upper and lower plates). Two-pieces appliances also vary in permissible lateral jaw movement and in the coupling mechanisms which attach the two plates together. Other variations include the range of degree of mandibular advancement, amount of vertical opening, building material, and different amount of occlusal coverage. New technology makes possible to ascertain and identify the anatomical and functional features of each patient, such as mandibular length, glenoid fossa size and form and normal range of movement of the mandible. Construction of a real customized MAD for each patient should be possible. Combining the recent development of techniques to measure the underlying physiology causing an individual's OSA using routine clinical information, and the study of the personal anatomical characteristics and physiological movements, will enable progress toward individualizing MADs therapy for patients with OSA.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Review the different designs of mandibular advancement devices (MAD)
- Analyze the mandible movements both with cephalometry and a mathematical model applied to MAD design
- Define and classify the changes of the mandibular position when MAD is in situ

Target Audience
All sleep physicians and dentists who want to be updated in the treatment of Obstructive Sleep Apnea; especially those who are interested in the treatment with MAD

2:00pm - 2:05pm	**Introduction** *J. Vila (Spain)*
2:05pm - 2:25pm	**Identification of anatomical and funcional features related to the mandible as a step in the design of a customized MAD** *P. Mayoral (Spain)*
2:25pm - 2:45pm	**Different design features of available MADs** *F. Milano (Italy)*
2:45pm - 3:05pm	**Analysis of the movements of the mandible and hyoid bone in the treatment of OSA with MAD** *J. Vila (Spain)*
3:05pm - 3:25pm	**Mathematical model of the jaw kinematics and its application to MAD design** *A. Bataller (Spain)*
3:25pm - 3:30pm	**Question and answer** *P. Mayoral (Spain)*

O03 Sleep breathing disorders oral abstract presentations

2:00pm – 3:30pm I Meeting Hall V

Chairs:
R.J. Thomas (United States), O. Polo (Finland)

2:00pm – 2:15pm	**SHARED-DECISION MAKING TOOL FOR OBSTRUCTIVE SLEEP APNEA** *A. Duggins (United States)*
2:15pm – 2:30pm	**EFFECT OF UPPER AIRWAY SURGERIES ON CARDIOVASCULAR RISK PROFILES AND GLOBAL CARDIOVASCULAR RISK IN OSAHS PATIENTS** *S. Yin (China)*
2:30pm – 2:45pm	**IMPROVEMENT OF SLEEP APNEA SYNDROME AND DAYTIME SLEEPINESS AFTER SURGICALLY ASSISTED RAPID MAXILLARY EXPANSION IN ADULT PATIENTS** *P.P. Vinha (Brazil)*
2:45pm – 3:00pm	**LONGITUDINAL EFFECTS OF PERIODIC BREATHING ON CEREBRAL OXYGENATION IN TERM AND PRETERM BORN INFANTS** *R. Horne (Australia)*
3:00pm – 3:15pm	**NASAL OBSTRUCTION DECREASE AFTER TWO YEARS OF PAP TREATMENT** *M. Värendh (Sweden)*
3:15pm – 3:30pm	**IS THE RESPIRATORY STABILITY DURING SLEEP IN PATIENTS WITH SEVERE HEART FAILURE INFLUENCED BY THE NOCTURNAL OXYGEN LEVEL: A SUB-ANALYSIS OF THE PROST STUDY USING A NOVEL RESPIRATORY STABILITY INDEX** *T. Tobushi (Japan)*

T02 Cardiopulmonary resuscitation for sleep technologists part 1

2:00pm - 3:30pm I Club H

Chairs:
S. Keenan (United States), O. Ludka (Czech Republic)

The workshop is designed for the the theoretical part (first half), which will be focus on arrhythmias and sudden cardiac death (also other acute cardiovascular conditions such as acute myocardial infarction and acute heart failure will be discussed) in connection with sleep disorders, especially sleep-disordered breathing, and the second half of the session will be practical, which will focus on CPR in this context as there is a direct connection with sleep and sleep disorders.

Speakers:
I. Cundrle Jr. (Czech Republic), L. Ruzek (Czech Republic),
O. Ludka (Czech Republic)

K04 Dental Sleep Medicine

C.C.-H. Lin (Taiwan)
Keynote 3:30pm - 4:15pm I Congress Hall

Underdevelopment of craniofacial region can be accompanied by small skeletal framework, disproportion between structures, and narrowed pharyngeal airway. Segmental Maxillomandibular Rotational Advancement (SMMRA) is designed specifically for Far-East Asian OSA patients with underdeveloped maxillomandibular skeleton, featured by narrow maxilla with crowded upper dental arch, high mandibular plane angle, mandibular retrognathism, retruded chin and a generally narrowed pharyngeal airway. SMMRA advances maxilla by two segments, counterclockwisely rotates the maxillomandibular complex to improve the mandibular plane angle, advance the mandible to the optimal extent, and forward the anterior inferior mandible including chin and genioglossus tubercle. The surgery may normalize the airway, facial skeleton, occlusion, and facial aesthetics at the same time.

S25 Measuring quality in the delivery of sleep medicine: Metrics and patient reported outcomes

3:30pm - 5:00pm I Meeting Hall IV

Chairs:
C. Iber (United States)

Summary
Sleep science impacts patients as it is converted into practice. The degree to which our advances impact patients depends upon not only the strength of the science, but also upon the quality with which the care is delivered. Quality in sleep medicine is multidimensional, but incorporates aspects of access, equitability, efficiency, efficacy, safety, timeliness, and patient-centeredness. Tools to measure the quality of care in sleep medicine have only begun to surface and be utilized in practice. This symposium will explore methods to measure quality in sleep medicine, including both process and outcome measures. Particular emphasis will be placed on the exploration of care models, patient reported outcomes, and measures of quality of care. Topics will include: 1. Dr. Timothy I. Morgenthaler, MD. A framework with which to measure quality of care in sleep medicine. This will serve as a general introduction to concepts of quality in healthcare, with emphasis on process measures, outcome measures, and patient reported outcomes in sleep medicine. 2. Dr. Harneet Walia, MD. Patient Reported Outcomes in Obstructive Sleep Apnea Syndrome. Most practices measure processes like CPAP compliance and sleepiness using questionnaires like the Epworth Sleepiness Scale. Although these are of importance, they are not direct measures of what matters to patients. Research that has included Patient Reported Outcomes in Obstructive Sleep Apnea will be reviewed, and the strengths, weaknesses, and new insights provided from these measures discussed. 3. Dr. Fang Han, MD. In a large practice devoted to care of patients with narcolepsy, patient reported outcomes provides unique insights into the severity of disease and the contribution of treatment to improving the patient's life. The care delivery system also plays a role. 4. Dr. Con Iber, MD. Virtual care is one tool that may help provide higher value care to patients with common sleep disorders. Experience with implementing streamlined care pathways, utilizing technology, and developing virtual care for sleep medicine will be described, along with measure of quality and patient outcomes that have accompanied the implementation.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Identify the differences, strengths, and weaknesses of quality process versus outcome measures.
- Recognize patient reported outcomes—beyond the Epworth Sleepiness Scale—and relate how they are affected by treatments of sleep disorders such as Obstructive Sleep Apnea or Narcolepsy
- Evaluate how virtual care strategies for Obstructive Sleep Apnea and insomnia and streamlined care pathways influence access, patient outcomes, and quality measures.
- Develop a quality measurement strategy that makes sense in your sleep practice

Target Audience
Sleep medicine practitioners, healthcare science researchers, policy makers

3:30pm - 3:35pm	**Introduction** *C. Iber (United States)*
3:35pm - 4:00pm	**Beyond the epworth: Patient reported outcomes in obstructive sleep apnea syndrome** *H. Walia (United States)*
4:00pm - 4:25pm	**Patient reported outcomes in narcolepsy: How to adapt treatment to the patient** *F. Han (China)*
4:25pm - 4:50pm	**Improving access and quality using virtual care** *C. Iber (United States)*
4:50pm - 5:00pm	**Question and answer** *C. Iber (United States)*

S26 Factors in night and rotating shift work associated with poor sleep and health

3:30pm - 5:00pm I Meeting Hall V

Chairs:
T. Shochat (Israel)

Summary
Night and rotating shift work create chronic misalignment between the endogenous circadian rhythms and the sleep/wake schedule. Such circadian misalignment leads to sleep deprivation, to problems at work such as impaired alertness, impaired performance, increased risks of accidents and errors, and to long-term health issues. Understanding how environmental, organizational, circadian and individual factors interact to impact functional outcomes in night and rotating shift work is crucial for the development of feasible and effective interventions. The symposium will present updated findings from both observational and interventional studies, looking at major factors associated with sleep, sleepiness, performance, health and wellbeing in night and rotating shift work. The first talk will assess individual bio-psychological factors associated with sleepiness, such as age, BMI, cognitive and somatic pre-sleep arousal and chronotype; and will evaluate how these individual differences interact with a scheduled nap during the nightshift. The second talk will evaluate the contribution of shift durations and schedules, comparing sleep and functional outcomes during both morning and night shifts in airline managers moving from 8-hour to 12-hour rotating shifts. The third talk will compare the detrimental effects of quick returns to the workplace and nightshifts on health outcomes. The forth talk will describe findings from studies looking at the effects of artificial lighting regimens on health measures of indoor workers. Finally, the last talk will provide a framework for the development of appropriate interventions targeting physiological problems in shift work, based on circadian principles. The major goal of the symposium is to evaluate conceptual models that may provide an infrastructure for creating screening criteria based on individual factors, and for implementing environmental and organizational modifications aimed at optimizing adjustment to night and rotating shift work.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Identify key individual, circadian, environmental and organizational factors that affect functional capacities including alertness, performance, health and well-being in night and rotating shift workers
- Evaluate conceptual models that integrate these key factors and their interactions on functional outcomes
- Construct screening criteria for night and rotating shift workers, based on individual factors associated with negative functional outcomes
- Design novel and feasible interventions that target environmental and organizational modifications that enhance functional outcomes, such as alertness, performance, health and well-being

Target Audience
Sleep and chronobiology researchers, clinicians

3:30pm - 3:33pm	**Introduction** *T. Shochat (Israel)*
3:33pm - 3:50pm	**The influence of bio-psychological predictors on subjective sleepiness of female nurses during the night shift with and without a short planned nap** *N. Zion (Israel)*
3:50pm - 4:07pm	**Sleep patterns and functioning during night and morning rotating shifts in the transition from 8 to 12-hour rosters among airline employees** *O. Tzischinsky (Israel)*
4:07pm - 4:24pm	**Are quick returns (<11 hours between consecutive shifts) typically seen in rotating shift work schedules more detrimental to health than night work?** *B. Bjorvatn (Norway)*
4:24pm - 4:41pm	**Artificial light and health in indoor workers** *A. Lowden (Sweden)*
4:41pm - 4:58pm	**Futuristic solutions to the physiological problems of shift work based on circadian rhythm principles** *C. Eastman (United States)*
4:58pm - 5:00pm	**Question and answer** *T. Shochat (Israel)*

S27 Functioning of the restless legs syndrome (RLS) brain: Excitability and control

3:30pm - 5:00pm I North Hall

Chairs:
R. Allen (United States)

Summary
Restless Legs Syndrome (RLS) while having a profound motor sign of periodic leg movements in sleep (PLMS) is fundamentally a sensory or sensorimotor disorder. The principal defining RLS symptom involves abnormal sensation of a need to move. RLS certainly may have a spinal and even peripheral component but at some point it must involve cortical sensory and motor systems and their interaction. Thus there is considerable interest and studies examining the functioning of sensory and motor systems in the brain. The initial studies demonstrated increased motor cortical excitability based on magnetic cortical stimulation. This has involved both hand and leg areas and some recent studies have related these to changes in RLS symptoms and status. These studies have been followed by studies evaluating the resting state in RLS. Here patterns of connections have differed for RLS compared to control subjects. The possibility of using magnetic stimulation to correct these differences has been evaluated. Finally consideration is now given not only to the brain system changes but also to issues of attentional control. These studies combined give a basic view of the functioning of the RLS brain that sets it up to respond with intent or desire to move the legs. They also open opportunities for treatments to alter these relations.
In this symposium an update is presented on excitability of the motor cortex of the hand and leg showing clearly increased excitability for the leg but less certain change for the hand. The pattern relates to clinical status of the patients. Resting state evaluations show abnormal patterns involving thalamic – cortical connections as noted in studies by Dr. Cho. Dr. Wang and his group have extended these to include consideration of effects of repetitive transcranial magnetic stimulation treatment of RLS. Finally Dr. Kassubek reports the altered connections found in RLS show patterns indicating changes in attention-related functional networks. This symposium presents each of these basic studies exploring in some detail each of these approaches and their potential interaction. This provides an opportunity to explore the best approaches to further study functioning of the RLS brain. It also offers possible new treatment approaches to RLS.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Contrast and evaluate alternate approaches to study of the functioning of the RLS brain
- Evaluate possible new treatment approaches for correcting functioning of the RLS brain
- Describe the functional and structural RLS changes insight into the brain systems producing the RLS sensory symptoms

Target Audience
Clinicians who treat sleep disorders, especially RLS, Scientists working with functional and structural evaluations of the brain, Clinicians and scientists working on new treatments for RLS

3:30pm - 3:35pm	**Introduction** *R. Allen (United States)*
3:35pm - 3:55pm	**Cortical excitability and RLS** *R. Salas (United States)*
3:55pm - 4:15pm	**Resting state connectivity** *Y.W. Cho (Republic of Korea)*
4:15pm - 4:35pm	**TMS stimulation and connectivity in RLS** *Y. Wang (China)*
4:35pm - 4:55pm	**Connectivity and attentional control** *J. Kassubek (Germany)*
4:55pm - 5:00pm	**Question and answer** *R. Allen (United States)*

S28 Arousability and loop gain: The factors that bridge insomnia and sleep-disordered breathing

3:30pm - 5:00pm I Club A and B

Chairs:
L. Parrino (Italy), D. Pevernagie (Belgium)

Summary
The symposium aims at linking arousability, sleep fragmentation and sleep disordered breathing, a connection that is regularly observed in the daily clinical practice. Obstructive sleep apnea (OSA) has traditionally been attributed only to a collapsible upper airway. However, it is increasingly recognized that additional non-anatomical mechanisms contribute to the disease. For instance, higher rates of OSA have been reported in patients with insomnia than in those without insomnia although the mechanism behind this increased prevalence remains to be investigated. Predisposition to OSA in patients with insomnia is probably due to a lower respiratory arousal threshold or a higher loop gain compared to patients without insomnia. Accordingly, sleep-disordered breathing is only partially controlled with CPAP when stressors interfere with sleep continuity and stability. On the contrary, when stressors abate, AHI tends to normalize suggesting a link between stress/anxiety, arousals and sleep-disordered breathing. To what degree CPAP (in OSA) or medications (in insomnia) modify the arousal threshold and/or the loop gain remains an open challenge. The common finding of recurrent arousals from sleep reinforces the interrelationship between OSA and insomnia. Both OSA and insomnia can induce selective independent changes in cortical or brain stem centers governing or augmenting the sympathetic neural tone. The absence of nocturnal blood pressure fall is a common finding in OSA patients. Night-time blood pressure in normotensive subjects with chronic insomnia is also characterized by a non-dipping profile of both systolic and diastolic curves. Frequent awakenings, unrefreshing sleep, fatigue, daytime sleepiness, attention, concentration and memory impairment, social and occupational dysfunction, mood disturbances, reduced energy, decreased quality of life are common symptoms associated with both OSA and insomnia. Overlap and comorbidity issues between the two sleep disorders will be inspected.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Identify the role of non-anatomical factors in the pathogenesis of OSA
- Examine, with an integrated approach, the underlying connections between different sleep disorders (insomnia and OSA) sharing common biomarkers and neurophysiological mechanisms
- Recognize the role of therapy on arousal threshold and loop gain in the daily clinical practice

Target Audience
Sleep clinicians, pulmonologists, neurologists, physiologists, internal medicine

3:30pm - 3:35pm	**Introduction** *L. Parrino (Italy)*
3:35pm - 3:55pm	**Failure to control the AHI - a clinician's perspective** *D. Pevernagie (Belgium)*
3:55pm - 4:15pm	**The role of arousability and loop gain in the pathogenesis of SDB** *M. Bosi (Italy)*
4:15pm - 4:35pm	**Sleep fragmentation in anxiety and chronic stress** *T. Akerstedt (Stockholm, Sweden)*
4:35pm - 4:55pm	**Sleep disordered breathing and insomnia: Overlap and comorbidity** *L. Parrino (Italy)*
4:55pm - 5:00pm	**Question and answer** *D. Pevernagie (Belgium)*

S29 New developments in narcolepsy diagnosis, research and therapeutics

3:30pm - 5:00pm I Meeting Hall 1A

Chairs:
T. Kilduff (United States), C. Peyron (France)

Summary
Although the sleep disorder narcolepsy is now well known to be due to loss of the hypocretin/orexin cells of the tuberal hypothalamus, the cause of this cell loss is unknown but an autoimmune mechanism has long been suspected. In addition, increased awareness of this disorder has brought into question the conventional view that the onset of the vast majority of narcolepsy cases occurs post-pubertally. In this symposium, Giuseppe Plazzi (Bologna, Italy) will discuss the increasing number of cases of childhood-onset narcolepsy and the extensive phenotypic characterization of these patients. Christelle Peyron (Lyon, France) will present an innovative mouse model to study the etiology of narcolepsy in which a neo-self-antigen is expressed specifically on the membrane of hypocretin/orexin neurons. In this model, adoptive transfer of effector neo-self-antigen-specific CD8, but not CD4, T cells leads to T-cell infiltration of the hypothalamus and specific destruction of hypocretin/orexin neurons. Akihiro Yamanaka (Nagoya, Japan) will present an alternative "Tet-off" mouse model in which the diphtheria toxin A (DTA) subunit is under control of the hypocretin promoter. This transgene is repressed as long as doxycycline is in the diet but, once it is removed, the toxic DTA protein is expressed specifically in the hypocretin neurons, which results in their rapid destruction and a disruption of sleep/wake architecture and cataplexy results. In conjunction with Dr. Plazzi's presentation on his studies in children, the presentations by Drs. Peyron and Yamanaka will emphasize symptomatology development in these animal models. Thomas Kilduff (Menlo Park, USA) will discuss use of mouse models of narcolepsy to evaluate potential new therapeutic pathways, particularly trace amine-associated receptor 1 (TAAR1) as a novel molecular target for treatment of this disorder. Together, these presentations will integrate basic and clinical studies of narcolepsy to inform the audience about the latest developments in the diagnosis of narcolepsy, research to produce animal models with greater fidelity to the human disorder, and the identification of molecular targets intended to advance potential novel therapeutics for treatments.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Compare the classical view of narcolepsy onset and diagnosis with the rapidly-evolving view that narcolepsy onset can occur much earlier than previously expected
- Give examples of the molecular, immunological and neurobiological tools that basic scientists are using to create new animal models of narcolepsy
- Describe some novel potential therapeutic pathways for treatment of this disorder.

Target Audience
Post-graduate researchers, pre- and post-doctoral students and clinicians interested in the latest developments in narcolepsy research and therapeutic development.

3:30pm - 3:35pm	**Introduction** *T. Kilduff (United States)*
3:35pm - 3:55pm	**Age-dependent narcolepsy phenotypes in patients** *G. Plazzi (Italy)*
3:55pm - 4:15pm	**Development of narcolepsy in healthy mice following an autoimmune attack** *C. Peyron (France)*
4:15pm - 4:35pm	**Generation of sleep disorder model mice by ablation of specific types of neurons** *A. Yamanaka (Japan)*
4:35pm - 4:55pm	**TAAR1 agonists as potential narcolepsy therapeutics** *T. Kilduff (United States)*
4:55pm - 5:00pm	**Question and answer** *T. Kilduff (United States)*

S30 Behavioral and neurophysiological influences of waking system on sleep

3:30pm - 5:00pm I Meeting Hall 1B

Chairs:
M.L. Andersen (Brazil), E. Garcia-Rill (United States)

Summary
Most part of our life we spend in a vigilant state. A complex brain circuitry is recruited to generate and maintenance all the behavioral repertoire present during wake time. This circuitry has a close association with sleep system, acting in a integrative manner to guarantee a perfect sleep-wake cyclicity. The neurophysiology basis of waking system have been extensive studied; however, novel ideas and mechanisms within waking system are now available. Eletrophysiological parameters, as the role of cortical oscilations, the concept of preconscious awareness, and the health impact of waking system dysregulation are some points that should be better explored. In fact, wake-sleep disturbances are common in numerous neurological and psychiatric diseases. Understanding the function and physiology of brain regions that regulate wake behavior can improve the comprehension of these disorders and improve treatment. The proposed symposium aims to look into the principles of brain activity during wakefulness and the behavioral and physiological consequences of this state. We will describe the main brain regions responsible for the vigilance state, and discuss the effects of manipulations or dysregulations on these areas. Speakers will address the neuroanatomical and neurophysiological aspects of waking generation and maintenance, highlight specific brain areas and nuclei responsible for the transitions between sleep and wake states, integrating the basic knowledge with the clinical implications of wake-sleep regulation. A firm understanding of the mechanisms behind waking will also allow a much better comprehension of the hyperarousal problems in some sleep disturbances and in a number of psychiatric and neurological disorders.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Identify the neuroanatomical and neurophysiological aspects of waking generation and maintenance
- Develop a critical view on the role of brain activity during awake state for sleep regulation
- Integrate the basic knowledge of waking system mechanisms with the clinical implications of wake-sleep dysregulation

Target Audience
Researchers and clinicians interested in neurobiology of sleep and waking behaviors

3:30pm - 3:35pm	**Introduction** *M.L. Andersen (Brazil)*
3:35pm - 3:55pm	**Neural basis of waking system** *E. Garcia-Rill (United States)*
3:55pm - 4:15pm	**Involvement of the Primate PPN in transition from wakefulness to sleep** *L. Goetz (France)*
4:15pm - 4:35pm	**Behavioral consequences of extended wakefulness** *M.L. Andersen (Brazil)*
4:35pm - 4:55pm	**Stimulation of the pedunculopontine: implications on sleep** *I. Arnulf (Paris, France)*
4:55pm - 5:00pm	**Question and answer** *E. Garcia-Rill (United States)*

O04 Neurological sleep disorders affecting sleep oral abstract presentations

3:30pm – 5:00pm I Club D and E

Chairs:
K. Sonka (Czech Republic)

3:30pm – 3:45pm	**IMPAIRED CONSCIOUSNESS STATES IN MYOTONIC DYSTROPHY-TYPE 1 MEDIATION BY γ-AMINO BUTYRIC ACID (GABA)** *D. Rye (United States)*
3:45pm – 4:00pm	**SUBJECTIVE AND OBJECTIVE FEATURES OF SLEEP DISORDERS IN PATIENTS WITH ACUTE ISCHEMIC OR HAEMORRHAGIC STROKE** *E. Pajediene (Lithuania)*
4:00pm – 4:15pm	**IMPAIRMENT OF AUTONOMIC NERVOUS SYSTEM IN AMYOTROPHIC LATERAL SCLEROSIS** *M. Puligheddu (Italy)*
4:15pm – 4:30pm	**SPECIFIC NEURONAL PROCESSES IN LATERAL HYPOTHALAMUS ACCOUNT FOR THE MAIN SLEEP AND FEEDING SYMPTOMS IN PRADER-WILLI SYNDROMES** *E. Balzani (Italy)*
4:30pm – 4:45pm	**CYCLIC ALTERNATING PATTERN AND INTERICTAL EPILEPTIFORM DISCHARGES DURING MORNING SLEEP DEPRIVED EEG IN TEMPORAL LOBE EPILEPSY** *A. Schirru (Italy)*
4:45pm – 5:00pm	**AGENESIS OF THE CORPUS CALLOSUM: EFFECT ON SLEEP ARCHITECTURE** *R. Castriotta (United States)*

O05 Psychiatric disorders affecting sleep/wake oral abstract presentations

3:30pm – 5:00pm I Terrace 1

Chairs:
P. Jennum (Denmark)

3:30pm – 3:45pm	**TROUBLED SLEEP, RESTLESS LEGS AND CATAPLEXY IN ADULTS WITH ADHD** *B. Bjorvatn (Norway)*
3:45pm – 4:00pm	**REPETITIVE TRANSCRANIAL MAGNETIC STIMULATION COMBINED WITH VENLAFAXINE FOR MAJOR DEPRESSIVE DISORDER WITH COMORBID ANXIETY AND INSOMNIA: A CONTROLLED POLYSOMNOGRAPHIC AND DIFFUSION MRI STUDY** *Z.-J. Zhang (Hong Kong)*
4:00pm – 4:15pm	**CORRELATION BETWEEN PHYSICAL ACTIVITY, ANXIETY AND SLEEP** *M. Bodnari (Republic of Moldova)*
4:15pm – 4:30pm	**INVESTIGATION OF SLEEP AND COGNITIVE FUNCTIONS ON FIRST EPISODE DRUG-NAIVE NON-AFFECTIVE PSYCHOTIC PATIENTS** *N. Yazihan (Turkey)*
4:30pm – 4:45pm	**NAPPING REDUCES ATTENTIONAL BIASES FOR NEGATIVE INTERPERSONAL STIMULI IN CLINICAL DEPRESSION** *K.N.T. Lau (Hong Kong)*
4:45pm – 5:00pm	**CIRCADIAN RHYTHM SLEEP-WAKE DISORDERS PREDICT SHORTER TIME TO RELAPSE OF MOOD EPISODES IN EUTHYMIC PATIENTS WITH BIPOLAR DISORDER: A PROSPECTIVE 48-WEEK STUDY** *Y. Takaesu (Japan)*

O06 Neural plasticity, memory, parasomnia and pharmacology oral abstract presentations

3:30pm – 5:00pm I Club H

Chairs:

3:30pm – 3:45pm	**OBSTRUCTIVE SLEEP APNEA AS A POTENTIAL EFFECT OF GABAPENTIN IN OLDER MEN** *R. Piovezan (Brazil)*
3:45pm – 4:00pm	**THE CONSEQUENCES OF SLEEP-DISORDERED BREATHING (SDB) ON THE CONSOLIDATION OF DIFFERENT MEMORY PROCESSES IN CHILDREN AND ADULTS** *E. Csábi (Hungary)*
4:00pm – 4:15pm	**THE EFFECTS OF ACUTE, SHORT-TERM VISUAL DEPRIVATION ON LOW-FREQUENCY EEG ACTIVITY DURING WAKEFULNESS AND SLEEP** *G. Bernardi (Switzerland)*
4:15pm – 4:30pm	**NEW VIDEO-POLYSOMNOGRAPHIC CRITERIA FOR THE DIAGNOSIS OF DISORDERS OF AROUSAL** *R. Lopez (France)*
4:30pm – 4:45pm	**DOES HORMONE REPLACEMENT THERAPY PROMOTE BETTER QUALITY OF SLEEP IN WOMEN? RESULTS FROM A LARGE CROSS-SECTIONAL NORWEGIAN STUDY (HUNT 3)** *M. Cvancarova Småstuen (Norway)*
4:45pm – 5:00pm	**SLEEP HOMEOSTATIC PROCESS IN CORTICAL AND CORTICOTHALAMIC CULTURES: A MODEL FOR SLEEP REGULATION** *S. Saberi Moghadam (Switzerland)*

K05 History of pediatric sleep and the contribution of sleep microstructure

O. Bruni (Italy)

Keynote 4:15pm - 5:00pm I Congress Hall

The aim of this presentation is to depict the discovery of sleep physiology and pathology in infants and the emergence of the discipline of Pediatric Sleep Medicine as a relatively autonomous entity. The gradual awareness regarding sleep disorders in infants and children began in the 19th century; children sleep had been neglected until the end of the last century with the main textbook of Pediatrics reporting none or only few paragraphs devoted to pediatric sleep, although the first observation that lead to the discovery of REM sleep was made on neonates and infants, as well as the first study on the negative behavioral consequences of sleep apnea was run in children. Researchers from different countries made important contributions for the development of the pediatric sleep medicine and actually different health providers (pediatric pulmonologists, otolaryngologists, neurologists, orthodontists and psychologists) recognize the fundamental role of sleep for the child health and development. In the last few decades, the analysis of sleep microstructure and of cyclic alternating pattern (CAP) allowed a better understanding of the neurophysiological mechanisms of sleep disturbance, especially in children. CAP can be considered as a window on pediatric sleep, allowing a new vision on how the sleeping brain is influenced by a specific pathology or how sleep protecting mechanisms try to counteract internal or external disturbing events.

S31 Parasomnias: Recent advances in etiology, assessment and treatment

5:30pm - 7:00pm I Congress Hall

Chairs:
B.A. Sharpless (United States)

Summary
There have been a number of recent advances in the scientific and clinical understandings of the parasomnias. Specifically, this symposium will focus on REM sleep behavior disorder, recurrent isolated sleep paralysis, the various NREM parasomnias, parasomnias related to medical conditions, and exploding head syndrome. After detailing their respective clinical features and etiologies, presenters will discuss what is currently known about the assessment and treatment of these conditions. The first presentation will focus on REM sleep behavior disorder (RBD). RBD is often associated with neurological conditions - most frequently the synucleinopathies (Parkinson's disease, dementia with Lewy bodies, and multiple system atrophy). Less common associations include narcolepsy and medications such as SSRIs and SNRIs. Other conditions such as sleep-related hypermotor epilepsy or obstructive sleep apnea may also masquerade as RBD. Thus, accurate assessment and differential diagnosis are crucial to effective treatment planning. Recurrent isolated sleep paralysis (RISP) was only recently added to formal diagnostic systems After briefly discussing the core features of RISP, this presentation will emphasize the known clinical impacts, discuss differential diagnosis, and review several promising treatment approaches (viz., pharmacological and cognitive behavioral). The third presentation will review the etio-pathophysiology, clinical features, differential diagnosis and treatment options of NREM sleep parasomnias (e.g., confusional arousals, sleepwalking, sleep terrors). Recent advances and insights into epidemiology, neurophysiological/neuroimaging correlates, daytime consequences, and differential diagnostic challenges (idiopathic vs secondary forms, NREM parasomnia vs sleep hypermotor epilepsy) will be discussed. Parasomnias can also arise in the context of neurological, medical, or psychiatric conditions as well as through the use of medications and substances. For example, REM behavior disorder can occur in the context of Parkinson's disease (PD), narcolepsy, and obstructive sleep apnea. In addition, complex nocturnal sleep related (hypnagogic and hypnopompic) visual hallucinations can occur with neurological disorders such as narcolepsy and PD and midbrain and diencephalic pathology. Regarding pharmacological agents, selective serotonin reuptake inhibitors, venlafaxine, tricyclic antidepressants, monoamine oxidase inhibitors, mirtazapine, bisoprolol, selegiline, or cholinergic treatment for Alzheimer disease, have been reported to be associated with acute or chronic RBD. Acute RBD can also be seen during states of withdrawal from cocaine, amphetamine, alcohol, barbiturate, and meprobamate abuse. Caffeine and chocolate abuse have been implicated in causing or unmasking RBD. β-adrenergic receptor-blocking agents can be associated with sleep related hallucinations. Sedative-hypnotics such as zolpidem and zopiclone have been associated with apparent NREM parasomnias including SRED and sleep driving. Finally, sleep-related behaviors after the ingestion of alcohol can mimic parasomnias. Exploding head syndrome is a relatively understudied sensory parasomnia. Recent findings on prevalence rates, typical symptoms, and associated features will be presented along with the available etiological hypotheses and known treatment options. Finally, important differential diagnostic considerations will be summarized.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the clinical manifestations and underlying etiologies of REM sleep behavioral disorder
- Identify the characteristic features and available treatment options for recurrent isolated sleep paralysis
- Recognize the clinical/neurophysiological features of NREM parasomnias
- Identify medical and psychiatric conditions that may be responsible for the emergence of parasomnias in clinical practice
- Identify the characteristic features and available treatment options for exploding head syndrome

Target Audience
Sleep medicine professionals

5:30pm - 5:32pm	**Introduction** *B.A. Sharpless (United States)*
5:32pm - 5:49pm	**REM sleep behavior disorder** *M. Mahowald (United States)*
5:49pm - 6:06pm	**Recurrent isolated sleep paralysis** *B.A. Sharpless (United States)*
6:06pm - 6:23pm	**NREM Parasomnias** *C. Bassetti (Switzerland)*
6:23pm - 6:40pm	**Parasomnias related to medical and psychiatric conditions** *K. Doghramji (United States)*
6:40pm - 6:57pm	**Exploding head syndrome** *B.A. Sharpless (United States)*
6:57pm - 7:00pm	**Question and answer** *B.A. Sharpless (United States)*

S32 Novel treatments for age-related sleep disruption

5:30pm - 7:00pm I Meeting Hall IV

Chairs:
J. Duffy (United States)

Summary

With advancing age, there are typical changes in sleep. These changes include reduced slow wave sleep, more frequent awakenings, and a shift of bedtimes and waketimes to earlier hours. Perhaps not surprisingly, sleep complaints also increase with age, with some surveys finding nearly half of adults over age 60 complaining of difficulty maintaining sleep, early morning awakening, and/or non-restorative sleep. While many of these individuals may have an undiagnosed sleep disorder, even very healthy older adults without sleep disorders show these characteristic changes in sleep quality and continuity. In this symposium, we will describe non-pharmacologic approaches to improving sleep in older adults: 1) While CBTi is now widely used to treat insomnia, there are special considerations for treating older patients that will be presented; 2) Light treatments have been used in a number of settings to treat sleep problems in older adults, and the use of light treatments in the home and in institutional settings will be reviewed; 3) We will review what is known about how different types and intensities of physical activity impact sleep in older adults; 4) Finally, we will present data on how cognitive training can improve sleep quality in older adults.

Learning Objectives

Upon completion of this CME activity, participants should be able to:
- Recognize typical age-related changes in sleep and how those may lead to insomnia complaints in older adults
- Define non-pharmacologic approaches to treating insomnia symptoms in older adults
- Compare non-pharmacologic treatments for insomnia in older vs. young adults
- Apply the information gained to choose the best non-pharmacologic treatment for insomnia in their older patients

Target Audience

Health care professionals who treat older patients

5:30pm - 5:35pm	**Introduction** *J. Duffy (United States)*
5:35pm - 5:55pm	**Light treatment to improve sleep quality in older adults** *K. Scheuermaier (South Africa)*
5:55pm - 6:15pm	**Cognitive training to improve sleep quality in older adults** *I. Haimov (Israel)*
6:15pm - 6:35pm	**Effects of physical activity on insomnia symptoms in later life** *K. Morgan (United Kingdom)*
6:35pm - 6:55pm	**Cognitive-behavioral treatment for insomnia in older adults: Effects on sleep, brain and body** *D. Buysse (United States)*
6:55pm - 7:00pm	**Question and answer** *J. Duffy (United States)*

S34 Pediatric OSA: Diagnostic and treatments involving a multidisciplinary team

5:30pm - 7:00pm I North Hall

Chairs:
C. Guilleminault (United States)

Summary
Pediatric OSA is still a diagnosis and treatment challenge, but important advances have been obtained with multidisciplinary collaboration. Risks factors for development of OSA may be present from birth-on, it is important to recognize them as they are open to treatment. C Guilleminault will present on how small dysfunctions leads to oral facial dysmorphoses that become risk factors for development of sleep-disordered-breathing over-time. A.Yoon will present how an abnormal lingual frenulum leads to OSA including data obtained on 1000 individuals to improve the technique of clinically recognize short lingual frenulum, a clear phenotype for OSA. Rapid Maxillary Expansion has been very much used to enlarge the oral cavity and improve the collapsibility of the upper-airway during sleep. But both RME and b-maxillary expansion have clear failures, a new approach to treat the maxillary deficiency commonly observed in OSA patients has been used in pre-pubertal and pubertal children: usage of Bollard'implants and Zygomatic traction widen the oral cavity and decrease the collapsibility of the upper-airway during sleep. Dr S Quo will present the modalities of the treatment, its indications and results.Independently of the selected treatment, the ultimate goal in OSA is 24-hours nasal breathing, reeducation to obtained nasal breathing has been applied using myofunctional therapy, beneficial results have been shown from this treatment, but failure due to non-compliance are frequent. Dr YS Huang will present the results obtained with "passive" myofunctional therapy applied during sleep both on clinical presentation, facial anatomic lend marks and polysomnograms .
Overall, the symposium involving a multidisciplinary team will demonstrate how understanding of the development of the oral-facial region that host the upper-airway, allows to perform appropriate clinical investigation of risks-factors increasing collapsibility of the upper-airway during sleep, and how new treatment avenues are able to modify these risk-factors.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize dysfunctions that can lead to development of pediatric OSA early in life
- Practice clinical evaluation guiding their diagnostic, to have notion on new treatment avenues in the field of orthodontia and the field of muscle reeducation that can be used when pediatric OSA is present. And to
- Recognize the importance of the multi-disciplinary involvement when dealing with pediatric-OSA.

Target Audience
Sleep Medicine practitioners and researchers involved in the development and treatment of obstructive sleep apnea syndrome. Sleep-Disorders Breathing is the most common sleep related medical condition, studied and addressed by many throughout the world. This symposium will be of interest to many.

5:30pm - 5:35pm	**Introduction** *C. Guilleminault (United States)*
5:35pm - 5:55pm	**From dysfunction to dysmorphoses in development of pediatric OSA** *C. Guilleminault (United States)*
5:55pm - 6:15pm	**Lingual frenulum: Role in pediatric OSA and its clinical investigation** *A. Yoon (United States)*
6:15pm - 6:35pm	**Maxillary deficiency, pediatric OSA risk and zygomatic involvement** *S. Quo (United States)*
6:35pm - 6:55pm	**Passive myofunctional therapy, long term follow-up: PSG and imaging** *Y.-s. Huang (Taiwan)*
6:55pm - 7:00pm	**Question and answer** *C. Guilleminault (United States)*

S35 Minimally invasive implantable approaches for OSA

5:30pm - 7:00pm I Club A and B

Chairs:
V. Pavelec (Czech Republic)

Summary
The program will focus on the latest medical devices, associated costs and outcomes for treatment of Obstructive Sleep Apnea. After a short introduction and general overview of surgical management of OSA, there will be four-panelist discussion on the following topics:

Vaclav Pavelec (Czech Republic): Base of tongue implants: Where We Are Now
Maria Suurna (USA): Implantable Upper Airway Neurostimulation: Current Approach
Ofer Jacobowitz: Implantable Upper Airway Neurostimulation: New Horizons
Joachim Maurer (Germany): European Experience with Different Concepts of Upper Airway Neurostimulation

The panel will address the importance to identify the level and nature of the airway obstruction during sleep in patients with OSA. The discussion will focus on use of different medical devices for treatment OSA. Patient satisfaction and compliance with different treatment modalities will be presented.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the importance of base of tongue collapse in pathophysiology of OSA and the role drug induced sleep endoscopy (DISE) in treatment selection
- Describe currently available techniques utilizing innovative technology, their advantages and limitations
- Review the future direction of medical advances in treatment of OSA
- Identify the indications for use of medical devices in treatment of OSA (This should help the participants to offer appropriate and comprehensive treatment for OSA)

Target Audience
Sleep physicians, ENT surgeons, neurologists, pneumologists, sleep lab technicians, anesthesiologists

5:30pm - 5:35pm	**Introduction** *V. Pavelec (Czech Republic)*
5:35pm - 5:55pm	**Base of tongue implants: Where we are now** *V. Pavelec (Czech Republic)*
5:55pm - 6:15pm	**Implantable upper airway neurostimulation: Current approach** *M. Suurna (United States)*
6:15pm - 6:35pm	**Implantable upper airway neurostimulation: New horizons** *O. Jacobowitz (United States)*
6:35pm - 6:55pm	**European experience with different concepts of upper airway neurostimulation** *J. Maurer (Germany)*
6:55pm - 7:00pm	**Question and answer** *V. Pavelec (Czech Republic)*

S36 Understanding the potential role for Mn in RLS etiology using novel human and animal models

5:30pm - 7:00pm I Meeting Hall 1A

Chairs:
J. Connor (United States)

Summary
Restless Legs Syndrome (RLS) is one of the most common neurological disorders; as many as 10% of the US population may have RLS, with moderate to severe RLS affecting 2-3% of adults [1]. RLS is characterized by 4 major clinical features: (1) patients have an urge to move the legs with marked leg discomfort (e.g. creeping, crawling, burning, tingling, painful or indescribable); (2) the symptoms are worse at rest; (3) there is at least partial and temporary relief with activity; and (4) the symptoms are worse later in the day or at night. RLS exhibits both familial and non-familial (idiopathic) forms, with approximately 60% of RLS cases having a family history of the disease [2-6]. Amongst non-familial US cases, MEIS1 and BTBD9 show the highest odds ratios for increased risk of RLS. RLS genetic risk factors, MEIS1 and BTBD9, are also functionally linked to Fe homeostasis. All studies to date support the concept of diminished brain Fe in RLS, even when systemic Fe stores appear to be normal [7]. However, several RLS studies implicated a broader systemic change in Fe regulation, such as alterations in the Fe management profile in lymphocytes from RLS women, despite nearly identical serum ferritin and hemoglobin levels for both RLS and control groups [8]. We have previously demonstrated that lymphocytes from RLS patients have an altered Fe management protein profile that suggests these cells lack the ability to retain Fe [9]. These exciting findings indicate a biological basis for this disorder and point to a disruption in metal homeostasis. A significant concern in the interpretation of these data, however, is whether the cell phenotype and ultimately the symptoms of RLS patients are the result of Fe deficiency or elevated concentrations of another metal that opportunistically increases when Fe levels are low. Given the established link between Fe and Mn biology, the research presented in this symposium will address whether systemic and/or neuronal alterations in Mn may contribute to the etiology of RLS. Dr. Aschner will introduce the C.elegans model, hpo-9-/- , which is a knockout of the mammalian homolog of BTBD9. He will report on survival rates, Mn uptake and HPO-9 expression levels with exposure to Mn that indicate the HPO-9 is involved in Mn homeostasis. Dr. Aschner will also discuss co-localization studies indicating that HPO-9 is localized in dopaminergic neurons indicating a potential role in dopamine signaling. Dr. Walters will report on serum iron and Mn levels in patients with RLS and will correlate the findings to the presence of two well know genetic variants that are present in RLS, BTBD9 and MEIS1. Drs. Connor and Patton will summarize alterations in manganese/iron homeostasis by reporting on the proteins alterations involved in the divalent metal homeostatic pathways such as DMT1, ferritin (H and L), transferrin receptor and ferroportin in RLS peripheral lymphocytes. They will also discuss the role of hypoxia pathway activation in RLS lymphocytes.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Identify a novel role for BTBD9 in regulating manganese (Mn) homeostasis and possibly dopamine signaling in C. elegans. • Participants should also be able to describe high throughput models such as C. elegans and their utility in addressing molecular underpinnings of RLS
- State whether RLS patients exhibit elevated brain or serum Mn levels and interpret whether they were influenced by Fe-status or inheritance of genetic risk factors (e.g. common variants of MEIS1 and BTBD9)
- Determine the role of Mn dyshomeostasis in the etiology of RLS using human lymphocytes as a model

Target Audience
Clinicians and researchers with an interest in Restless Legs Syndrome, understanding the underlying disease mechanism for RLS and novel models for studying RLS.

5:30pm - 5:35pm	**Introduction** *J. Connor (United States)*
5:35pm - 6:00pm	**Identification of a novel role for BTBD9 in regulating Mn signaling in C. elegans** *M. Aschner (United States)*
6:00pm - 6:25pm	**Brain and serum Mn levels and their relationship to iron status and genetic risk factors (BTBD9 and MEIS1)** *A. Walters (United States)*
6:25pm - 6:50pm	**Understanding the role of Mn dyshomeostasis in RLS etiology using peripheral lymphocytes as a model** *S. Patton (United States)*
6:50pm - 7:00pm	**Question and answer** *J. Connor (United States)*

S37 Basic research & new treatment approaches in sleeprelated breathing disorders

5:30pm - 7:00pm I Meeting Hall 1B

Chairs:
A. Büttner-Teleaga (Germany)

Summary
Basic Sleep Research (especially in the field of Sleep-related Breathing Disorders) includes diverse areas of Sleep Research such as Neuroscience, Genetics, Cell biology, Endocrinology and Physiology. From these perspectives, animal and/or human research is aimed at understanding the molecular, neurophysiological or neuronal mechanisms controlling the sleep-wake cycle and states. Research also comprises the Phylogeny, Ontogeny and General physiology of sleep. The most important aim of it is to find alternative and better treatment approaches for the most important sleep disorders, but until now the standard therapy for Obstructive Sleep Apnoea (OSA) is Continuous Positive Airway Pressure (CPAP) therapy. However, long-term adherence remains at ~50% despite improvements in behavioural and educational interventions.
In our symposium firstly we try to give the audience more knowledge about how Sleep-related Breathing Disorders work and causes of these kind of disorders. On the end we connecting the news of basic research with new treatment approaches and we combine it with continuous monitoring from clinical settings.
Many sleep disorders (e.g. Sleep-related Breathing Disorders) and/or neurodegenerative disorders are associated with rapid eye movement sleep (REMS) loss; however, the mechanism was unknown. As REMS loss elevates noradrenaline (NA) level in the brain as well as induces neuronal apoptosis and degeneration.
Studies of Mallick and co-workers delineated the intracellular molecular pathway, which is involved in REMS deprivation (REMSD)-associated NA-induced neuronal apoptosis. Furthermore they studied the role of NA firstly by intraperitoneal (i.p.) injection of NA-ergic alpha1 adrenoceptor antagonist prazosin (PRZ) and secondly by downregulation of NA synthesis in locus coeruleus (LC) neurons by local microinjection of tyrosine hydroxylase siRNA (TH-siRNA).
In Sleep-related Breathing Disorders brainstem apolipoprotein AII (apoa2) mRNA expression correlates with apnoeas in breathing, which are present in the adult C57Bl/6J (B6) sleep apnea model: Apoa2 WT do, but KO and heterozygote (+/-) mice do not exhibit apnoeas during post-hypoxic breathing, measured in vivo. In the in situ model, pauses and instability in fictive phrenic bursting are substantially reduced in KO vs. WT preparations. In 24 RISs, apnoea number in vivo was higher in strains with B6 apoa2 than with DBA apoa2 alleles. The B6 apoa2 polymorphism is directly involved in breath production, and it's identification suggests a novel pathway influencing risk for adult sleep apnoea.
Based on this prior work, Strohl and his group, explored whether regularity of breathing during wakefulness could be a physiologic predictor of CPAP adherence.
Our symposium shall be included new basic research findings of Sleep-related Breathing Disorders on one hand, and new treatment strategies and their clinical importance on the other hand.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the associations of breathing pauses with genes (animal model)
- Identify REM loss as indicator of neuronal apoptosis and degeneration
- Review new treatment strategies and their clinical importance's

Target Audience
Sleep Scientists/Researchers/Doctors; Neurologists; Psychiatrists; Doctors of Internal Medicine; Biologists

5:30pm - 5:35pm	**Introduction** *A. Büttner-Teleaga (Germany)*
5:35pm - 5:55pm	**REMS loss as indicator of neuronal apoptosis and degeneration** *B. Mallick (India)*
5:55pm - 6:15pm	**Association of breathing pauses with genes (animal model)** *K. Strohl (United States)*
6:15pm - 6:35pm	**New treatment strategy, nasal airway stent (NAS) in SBD** *M. Satoh (Japan)*
6:35pm - 6:55pm	**Clinical importance of alternative treatments in SBD** *A. Büttner-Teleaga (Germany)*
6:55pm - 7:00pm	**Question and answer** *A. Büttner-Teleaga (Germany)*

S38 Young Investigator: Sleep research in neurodegeneration

5:30pm - 7:00pm I Club D and E

Chairs:
C. Trenkwalder (Germany), C. Schenck (United States)

5:30pm - 5:45pm	**DIFFERENT MARKERS IN IDIOPATHIC RAPID EYE MOVEMENT (REM) SLEEP BEHAVIOR DISORDER (RBD), POSSIBLE PREDICTORS OF CONVERSION TO DIFFERENT TYPES OF ALPHA-SINUCLEINOPATHIES** *C. Gutierrez Muñoz (Spain)*
5:45pm – 6:00pm	**ENVELOPE ANALYSIS OF ELECTROMYOGRAM IN REM SLEEP BEHAVIOR DISORDER PATIENTS** *D. Espinoza (Chile)*
6:00pm – 6:15pm	**STRIATAL DYSFUNCTION AND DIMINISHED FUNCTIONAL CONNECTIVITY IN IDIOPATHIC REM SLEEP BEHAVIOR DISORDER WITH SUBTLE MOTOR ALTERATION** *G. Yamada (Japan)*
6:15pm – 6:30pm	**INCREASED SERUM CYSTATIN C IN PARKINSON'S DISEASE WITH OBJECTIVE SLEEP DISTURBANCE** *K.-P. Xiong (China)*
6:30pm – 6:45pm	**DIAGNOSING REM SLEEP BEHAVIOUR DISORDER IN PARKINSON DISEASE WITHOUT A GOLD STANDARD: A LATENT CLASSES MODELS STUDY** *M. Figorilli (France)*
6:45pm – 7:00pm	**FAMILIAL AGGREGATION OF REM SLEEP BEHAVIOR DISORDER AND NEURODEGENERATIVE BIOMARKERS: A CASE-CONTROL FAMILY STUDY** *Y. Liu (Hong Kong)*

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S39 Obstructive sleep apnea severity and the role of oral appliances

5:30pm - 7:00pm I Terrace 1

Chairs:
M. Braem (Belgium)

Summary
Obstructive sleep apnea (OSA) is highly prevalent in the population according to recent epidemiological research. Eighty-four percent of men and 61% of women have an apnea-hypopnea index (AHI) of ≥5. Milder forms of the disease are most common, particularly in the younger age groups. These high numbers of potential patients have raised questions about treatment needs and more exact indications for various treatment options in subjects with OSA of various severity. CPAP has been considered as the gold standard treatment, but increasing evidence support the use of oral appliances (OAs) in the treatment of OSA. OAs have been primarily recommended for patients with mild to moderate disease. Recent research has, however, discovered similar health outcomes from OA and CPAP in patients with more severe disease; those with moderate to severe OSA. These results were suggested to be explained by the higher adherence to OA-therapy, that might modify the health benefits from the highly effective CPAP machine. Subsequently, the indications for various OSA-treatments based on AHI have been questioned. The pathophysiology differs between OSA patients. Those with milder OSA more often have a normal upper airway morphology and a less collapsible airway, which is supposed to suit the mechanism of action of OAs. But, additional factors that are associated with OSA, such as a hypersensitive ventilatory control system (high loop gain) and a lower arousal threshold might have an impact on disease severity and treatment outcomes. In the first study evaluating physiological factors in the prediction of treatment success with OAs, patients who at baseline had a less collapsible airway and a low/normal loop gain showed the greatest reduction in their AHI. Disease severity based on AHI was a weaker predictor of success. In line with these results, good prediction possibilities have been detected by a verified widening of pharynx visualized by nasendoscopy or a sufficient reduction of AHI during an overnight test with a remotely controlled device. For patients who are not completely successful with an OA, combinations of treatments might be introduced. All these recent research challenge current practice parameters that recommend that OA treatment should only be considered in patients with milder OSA or in those who have failed or refuse CPAP treatment. More research about sophisticated prediction methods of success and knowledge about adherence and longer term health outcomes will be needed for the future. In conclusion, oral appliances have become a common alternative for the many patients with OSA, which strengthens the need for continuous updating of knowledge about various aspects of this therapy. This symposium will provide the most recent knowledge about OAs in the treatment of OSA of various severity.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Evaluate the clinical relevance and approaches to the management of mild obstructive sleep apnea.
- Identify physiological traits that are related to obstructive sleep apnea and may be used to predict treatment response to an oral appliance.
- Demonstrate other clinical ways to identify predictors of success and analyze combination treatments and their role in the treatment with OAs.
- Compare OA therapy with CPAP in terms of indications and health outcomes

Target Audience
Professionals with an interest in the treatment of sleep apnea with oral appliances

5:30pm - 5:35pm	**Introduction** *M. Braem (Belgium)*
5:35pm - 5:55pm	**Mild obstructive sleep apnoea: Clinical relevance and approaches to management** *W. McNicholas (Ireland)*
5:55pm - 6:15pm	**Physiological traits related to obstructive sleep apnea and prediction of treatment response to an oral appliance** *B. Edwards (Australia)*
6:15pm - 6:35pm	**Other clinical ways to identify predictors of success and analyze combination treatments and their role in the treatment with OAS** *M. Braem (Belgium)*
6:35pm - 6:55pm	**OA vs CPAP: comparative effectiveness** P. Cistulli (Australia)
6:55pm - 7:00pm	**Question and answer** *M. Braem (Belgium)*

O07 Narcolepsy Oral Abstract Presentations

5:30pm – 7:00pm I Meeting Hall V

Chairs:
S.-C. Hong (Republic of Korea)

5:30pm – 5:45pm	**EVIDENCE FOR A NARCOLEPSY SPECTRUM DISORDER IN FAMILY MEMBERS OF PATIENTS WITH TYPE 1 NARCOLEPSY** *P. Wang (China)*
5:45pm – 6:00pm	**MEASUREMENT OF NARCOLEPSY SYMPTOMS: THE NARCOLEPSY SEVERITY SCALE** *I. Jaussent (France)*
6:00pm – 6:15pm	**A DOUBLE-BLIND, PLACEBO-CONTROLLED, RANDOMIZED-WITHDRAWAL, MULTICENTER STUDY OF THE EFFICACY AND SAFETY OF SODIUM OXYBATE IN PEDIATRIC SUBJECTS WITH NARCOLEPSY WITH CATAPLEXY** *G. Plazzi (Italy)*
6:15pm – 6:30pm	**NOCTURNAL REM WITHOUT ATONIA, A DIAGNOSTIC BIOMARKER FOR PEDIATRIC NARCOLEPSY** *S. Bin-Hasan (Canada)*
6:30pm – 6:45pm	**WIDESPREAD WHITE MATTER CONNECTIVITY ABNORMALITIES IN NARCOLEPSY TYPE 1 PATIENTS: A DIFFUSION TENSOR IMAGING STUDY** *J.K. Gool (The Netherlands)*
6:45pm – 7:00pm	**A STANDARDIZED TEST TO DOCUMENT CATAPLEXY** *F. Pizza (Italy)*

T03 Cardiopulmonary resuscitation for sleep technologists part 2

5:30pm - 7:00pm I Club H

Chairs:
S. Keenan (United States), O. Ludka (Czech Republic)

The workshop is designed for the the theoretical part (first half), which will be focus on arrhythmias and sudden cardiac death (also other acute cardiovascular conditions such as acute myocardial infarction and acute heart failure will be discussed) in connection with sleep disorders, especially sleep-disordered breathing, and the second half of the session will be practical, which will focus on CPR in this context as there is a direct connection with sleep and sleep disorders.

Speakers:
I. Cundrle Jr. (Czech Republic), L. Ruzek (Czech Republic), O. Ludka (Czech Republic)

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K06 Sleep and Synaptic homeostasis

C. Cirelli (United States)
Keynote 8:00am - 9:00am I Congress Hall

Sleep is universal, tightly regulated, and many cognitive functions are impaired if we do not sleep. But why? Why do our brains need to disconnect from the environment for hours every day? The synaptic homeostasis hypothesis (SHY) states that sleep is the price we pay for brain plasticity and predicts that synaptic connections throughout the brain undergo net potentiation during wakefulness, while we learn new facts and regularities about the environment. Synaptic renormalization during sleep restores the homeostasis of energy and cellular supplies, with beneficial effects at the cellular and systems level, including memory acquisition, consolidation, integration, and smart forgetting. I will discuss the rationale underlying this hypothesis and summarize previous electrophysiological, molecular and genetic studies in flies, rodents and humans that confirmed SHY's main predictions. Synaptic size correlates with synaptic strength and most excitatory synapses in the cortex occur on spines. Thus, a strong prediction of SHY is that cortical spines should grow after wake and shrink after sleep, independent of circadian time. I will present new ultrastructural results obtained in mice using serial block face scanning electron microscopy that confirm this prediction, supporting the hypothesis that a core function of sleep is to renormalize overall synaptic strength increased by wake.

S41 Sleep and sex: What can go wrong?

9:00am - 10:30am I Meeting Hall IV

Chairs:

C. Schenck (United States)

Summary

This symposium intends to address the various problems when two basic instinctual behaviors become pathologically intertwined, viz. sleep and sex. Sleep related abnormal sexual behaviors (sexsomnia; sleepsex) are officially recognized in the ICSD-3 to be a variant of Non-REM sleep parasomnias, predominantly confusional arousals, but also sleepwalking. Obstructive sleep apnea is another recognized trigger for abnormal arousals with sexual behaviors. The full range of sexuality can inappropriately emerge with sexsomnia, and can result in injuries to self (from aggressive masturbation) and to the bed partner from sexual assaults. Marital and relationship problems from sexsomnia are not uncommon. There is growing awareness of the forensic consequences from sexsomnia with adults and with minors. Knowledge of the basic science of physiological genital events during sleep and associated sexual autonomic nervous system arousal is critical for understanding the biological substrates for the emergence of sexsomnia in the context of recognized clinical risk factors. Sleep related sexual seizures have also been documented, and can be compared and contrasted with sexsomnia as a parasomnia. This symposium will first focus on the basic science underpinning of sexsomnia, then present an update and synthesis of the world literature on sexsomnia and ictal sexsomnia, followed by a presentation of the optimal methods for clinical evaluation and management of patients. As with most parasomnias, therapy of sexsomnia is often effective, as is the therapy of epileptic sexsomnia. The forensic aspects of sexsomnia will be presented, along with a critical review of published legal cases. In addition, the association of recurrent abnormal wakeful sexual behaviors emerging in tandem with periodic hypersomnia (Kleine-Levin syndrome) will be presented and discussed. There is a growing literature on these topics and an increasing awareness by sleep clinicians of the existence of these problems, which underlines the need for and timeliness of this symposium to assist clinicians in expanding their knowledge of the clinical profiles, optimal assessment and management approaches, and the forensic issues. All the speakers in the symposium have published peer-reviewed papers on the topics they will be discussing, as listed (in part) below. Andersen ML, et al. Sexsomnia: abnormal sexual behavior during sleep. Brain Res Reviews 2007;56:271-82. (Reviewed the physiological genital events during sleep and associated autonomic arousal).
Schenck CH, Arnulf I, Mahowald MW. Sleep and sex: what can go wrong? A review of the literature on sleep related disorders and abnormal sexual behaviors and experiences. Sleep 2007;30:683-702. (Formulated the first classification of sleep related disorders and abnormal sexual behaviors and experiences). Schenck CH. Update on sexsomnia, sleep related sexual seizures, and forensic implications. NeuroQuantology 2015; 4:518-41. Dubessy AL, Leu-Semenescu S, Attali V, Maranci J-B, Arnulf I. Sexsomnia: a specialized non-REM parasomnia? Sleep 2016 (in press). (A group of patients with sexsomnia had their clinical and sleep measures compared and contrasted with those of sleepwalkers and healthy controls). Arnulf I, et al. Kleine-Levin syndrome: a systematic review of 186 cases in the literature. Brain 2005; 128:2763-76.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Recognize the basic science of physiological genital events during sleep and associated sexual autonomic arousal
- Define the typical clinical profile of patients with sexsomnia, and the recommended evaluation and management
- Identify the forensic consequences of sexsomnia with sexual assault during sleep
- Recognize the association of recurrent abnormal wakeful sexual behavior with episodes of recurrent hypersomnia in Kleine-Levin syndrome.

Target Audience

Sleep medicine clinicians and basic scientists interested in instinctual behaviors related to sleep.

9:00am - 9:05am	**Introduction** *C. Schenck (United States)*
9:05am - 9:25am	**The basic science of sex physiology and how it can pathologically relate to sleep** *M.L. Andersen (Brazil)*
9:25am - 9:45am	**Update and synthesis of the world literature on sexsomnia and ictal sexsomnia** *C. Schenck (United States)*
9:45am - 10:05am	**Abnormal sexual behaviors associated with Kleine-Levin Syndrome** *I. Arnulf (France)*
10:05am - 10:25am	**Forensics aspects of sexsomnia and review of legal cases** *F. Ingravallo (Italy)*
10:25am - 10:30am	**Question and answer** *C. Schenck (United States)*

S42 Sleep dependent brain oscillations as early markers of neurodegeneration

9:00am - 10:30am I Meeting Hall V

Chairs:

J.-F. Gagnon (Canada)

Summary

Neurodegenerative disorders such as Alzheimer's disease, Parkinson's disease, and Huntington's disease are incurable and devastating medical conditions characterized by a progressive loss of normal cognitive and motor skills. This gradual loss of functions leads to an early deficit in self-autonomy and a drastic drop in quality of life for both patients and caregivers. With the extension of life expectancy in modern societies, the physiological age dependent cognitive decline as well as the prevalence of dementia steeply increase and put the health care system under enormous financial burden. An early detection of pathophysiological changes would allow early interventions that could delay or prevent the disease onset. Although the neurodegenerative process often precedes the clinical manifestation of the diseases by several years, its detection is not yet possible due the lack of feasible quantitative biomarkers. Disrupted sleep and abnormal brain oscillations appear long time before the clinical onset of the diseases suggesting their possible mechanistic involvement in the neurodegenerative process. This is supported by the observation that normal sleep and brain oscillatory activity are essential for brain restoration as well as for neural plasticity underlying learning and memory formation. Furthermore, the complex pattern of sleep dependent brain oscillations emerges at the intersection of the circadian and homeostatic sleep-wake regulatory systems and reflect intrinsic features of functional neuro-architecture within the brain. Therefore, novel quantitative measures of sleep EEG are strong candidate biomarkers of the neurodegenerative process allowing early detection of disease onset and the monitoring of disease progression in both research and clinical settings, as well as in clinical trials. However, their widespread acceptance requires a better understanding of the presumed mechanistic relationship between sleep and neurodegeneration. This may help to identify novel therapeutic targets and methods for treating neurodegenerative disorders expectedly at an earlier stage of the disease. This symposium will provide an overview of some of the recent development in patients with neurodegenerative disorders (Jean-Francois Gagnon, Alpar Lazar) and in animal disease models (Bettina Platt, Sandor Kantor). Professor Gagnon from the Université du Québec à Montréal, Canada will present on sleep EEG features as prodromal markers of Parkinson's disease dementia and Alzheimer's disease. Professor Platt from the University of Aberdeen, UK will present data from amyloid and tau knock-in mice as well as novel computational approaches for qEEG analyses. Dr Lazar from the University of East Anglia, UK and Cambridge University, UK is going to present on the contribution of age, sex, genetic and sleep regulatory factors modulating the earliest sleep EEG biomarkers of neurodegeneration in Huntington's disease. Dr Kantor from Cambridge University, UK will demonstrate how drugs, often used for symptomatic treatment of Huntington's disease, can normalize the early sleep and EEG abnormalities in a transgenic mouse model of the disease. The speakers will not only summarize results from own research but will discuss methodological aspects and implications for future research and clinical practice. The symposium is anticipated to be attractive to a broad audience and have outstanding educational value.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Identify new sleep-related biomarkers of cognitive decline in Parkinson's disease, Alzheimer's disease and their preclinical stages
- Characterise sleep/wake changes and corresponding EEG profiles in amyloid- vs tau-expressing mouse models of dementia
- Identify early sleep EEG biomarkers of neurodegeneration in Huntington's disease and discriminate between the contributions of age, sex, genetic defect and sleep-wake regulatory components to the modulation of those biomarkers
- Identify and describe the drug induced changes in EEG oscillations, which may also predict their therapeutic efficacy in neurodegenerative disorders

Target Audience

Graduate and postdoctoral students, researchers and clinicians interested by the association between sleep, EEG and the development of neurodegenerative diseases.

9:00am - 9:05am	**Introduction** *J.-F. Gagnon (Canada)*
9:05am - 9:25am	**Sleep EEG features as prodromal markers of Parkinson's disease dementia and alzheimer's disease** *J.-F. Gagnon (Canada)*
9:25am - 9:45am	**Sleep and EEG profiles of amyloid- vs tau-expressing mouse models of dementia** *B. Crouch (United Kingdom)*
9:45am - 10:05am	**Sleep EEG biomarkers of huntington's disease: Contributions of age, sex, huntingtin (htt) gene and sleep-wake regulation** *A. Lazar (United Kingdom)*
10:05am - 10:25am	**Changes in abnormal brain oscillations can predict the efficacy of therapeutics in huntington's disease mice** *S. Kantor (United Kingdom)*
10:25am - 10:30am	**Question and answer** *J.-F. Gagnon (Canada)*

S43 Sleep related learning and behavioural functioning in children with developmental disorders

9:00am - 10:30am I North Hall

Chairs:
D. Dimitriou (United Kingdom)

Summary
The role of sleep in the developing brains is vital. Yet our understanding about the role of sleep on cognitive, emotional and behavioural functioning in children with developmental disorders is still in its infancy. For typically developing healthy individuals with IQ within norms, cognitive reserve provides some degree of protection against the potential insults of sleep restriction or impaired sleep quality/quantity. However, children with developmental disorders such as ADHD, Down Syndrome and Williams syndrome often suffer from sleep problems and learning difficulties. In line with previous studies, an increased prevalence of sleep problems and atypical cognitive and behavioural profiles are now showing several associative links with sleep disorders. Despite this double jeopardy, developmental disorders have remained an area of little attention in sleep research possibly due to many challenges when testing children with these disorders. The current studies in the symposium aim to address these issues and examine sleep specific learning and behavioural patterns.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Examine important recent work in the study of sleep in developmental disorders across different ages
- Describe the association between sleep problems and atypical cognitive and behavioural profiles
- Recognize which developmental disorders often correlate with sleep problems

Target Audience
Physicians treating developmental disorders; sleep health professionals; Pediatricians

9:00am - 9:05am	**Introduction** *D. Dimitriou (United Kingdom)*
9:05am - 9:25am	**Sleep and cognition in children with down syndrome** *G. Spanò (United Kingdom)*
9:25am - 9:45am	**Poor sleep in childhood ADHD and its impact on daytime functioning** *F. Knight (United Kingdom)*
9:45am - 10:05am	**Poor sleep and atypical learning trajectories in Williams syndrome** *J. Hayton (United Kingdom)*
10:05am - 10:25am	**Sleep related learning patterns are different in different developmental disorders** *D. Dimitriou (United Kingdom)*
10:25am - 10:30am	**Question and answer** *D. Dimitriou (United Kingdom)*

S44 Myofunctional therapy as an adjunct treatment for sleep disordered breathing: Validation of screening tools and objective measurements of progress for an emerging standard of care

9:00am - 10:30am I Club A and B

Chairs:
M. Moeller (United States), O. Bruni (Italy)

Summary
Orofacial Myofunctional Therapy (OMT) has been gaining attention as an adjunct treatment for snoring and OSA as research has emergied around the world. The Brazilian Sleep Society (ABSONO), the Asian Pediatric Pulmonary Society (APSS), the French Society of Sleep Medicine, and the Italian Ministry of Health have all recently made statements and issued guidance on the efficacy or importance of OMT as a standard of care as an adjunct treatment of sleep disordered breathing. Research (Camacho et al 2015, Ieto et al 2015, Villa et al 2013, Guilleminault et al 2013, Guimares et al 2009) has shown great promise, but much more is still yet to be done to clarify and define orofacial myofunctional disorder (OMD) pathology, their association to SDB, and their prevalence in the general population. This symposium will survey the current state of OMT research around the world and will present the latest OMD screening tools undergoing validation, objective measurements of progress in OMT for SDB patients, and RCTs evaluating the evidence for its efficacy.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize orofacial myofunctional disorders (OMDs) and their relationship to OSA and snoring
- Evaluate the potential value for orofacial myofunctional therapy (OMT) in a snoring or OSAS patient.
- Measure objective outcomes and progress of OMT as an adjunct treatment for snoring and OSA.
- Utilize screening tools to identify potential OMDs in a clinical setting

Target Audience
Sleep Specialists, sleep researchers, dentists, sleep technologists, sleep medicine instructors, allied health professionals, myofunctional therapists, public health specialists

9:00am - 9:02am	**Introduction** *M. Moeller (United States)*
9:02am - 9:19am	**Evaluation and measurements of a 12 week orofacial myofunctional therapy program for sleep disordered children** *M. Moeller (United States)*
9:19am - 9:36am	**Universal screening tools for speech pathologists: Orofacial myofunctional disorders as clinical markers for OSA** *O. Bruni (Italy), M. Moeller (United States)*
9:36am - 9:53am	**Myofunctional therapy for adult snoring and OSA: Changes in tongue fat and airway in a RCT** *E. Bianchini (Brazil)*
9:53am - 10:10am	**Myofunctional therapy and tongue tone in chilrdren with sleep disordered breathing** *M. Pia Villa (Italy)*
10:10am- 10:27am	**Standardizing myofunctional exercises for chilrdren with residual OSAS** *M. Evangelisti (Italy)*
10:27am - 10:30am	**Question and answer** *O. Bruni (Italy)*

S46 New approaches to studies of genetics of sleep and its disorders

9:00am - 10:30am I Meeting Hall 1B

Chairs:
A. Pack (United States)

Summary
There is considerable evidence that most aspects of sleep, response to sleep deprivation and common sleep disorders are heritable. However, apart from restless legs syndrome and narcolepsy, there has been limited progress in identifying gene variants. This symposium brings together investigators from different institutions to describe new approaches to this issue. Novel concepts and approaches are emerging and they are being employed to determine novel gene variants. These new concepts include: new high-throughput phenotyping strategies; new methods to recruit subjects for genetic research; new mouse resources; new approaches to identifying genes with pleiotrophic effects on multiple phenotypes; new analytical approaches based on machine learning. The goals of this symposium are to present these new approaches and to give up-to-date state-of-the-art presentations.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize new approaches to genetic studies of sleep and its disorders
- Review new approaches that have been developed to identify gene variants using mouse models
- Describe new analytical strategies that are being employed to address the genetic heterogeneity of sleep disorders.

Target Audience
Investigators in study of sleep and its disorders; trainees, clinicians with an interest in genetic studies

9:00am - 9:02am	**Introduction** *A. Pack (United States)*
9:02am - 9:19am	**New strategies based on clinical sleep centers** *E. Mignot (United States)*
9:19am - 9:36am	**Use of new approaches to mice** *A. Pack (United States)*
9:36am - 9:53am	**Characterizing pleiotropic effects of genetic variation** *O. Veatch (United States)*
9:53am - 10:10am	**Phenotypic approaches to sleep disorders** *P. Cistulli (Australia)*
10:10am- 10:27am	**Understanding complex genetic interactions using machine learning** *D. Mazzotti (United States)*
10:27am - 10:30am	**Question and answer** *A. Pack (United States)*

S47 Insomnia phenotypes: Identification and treatment response

9:00am - 10:30am I Club D and E

Chairs:
W. Wohlgemuth (United States)

Summary
Recent evidence shows that sleep specialists did not consistently agree on which insomnia disorders apply to a particular individual. Since the various diagnoses were unreliably applied to those with insomnia, the latest iteration of the International Classification of Sleep Disorders (ICSD) has simplified the diagnosis of insomnia into one disorder. An underlying assumption of having multiple insomnia diagnoses is that many insomnia phenotypes exist. The matching of a diagnosis with an observed phenotype may allow the proper course of treatment to be prescribed. However, the elimination of several insomnia diagnoses from the nosology does not imply that various insomnia phenotypes do not exist. It may simply mean that a better strategy to identify insomnia phenotypes is necessary. Strategies to identify insomnia phenotypes include statistical methods to identify distinct sets of patients (i.e., latent profile analysis), rationally derived subsets of patients (i.e., insomnia patients who sleep less than six hours), or response to treatment by the patient (i.e., responders vs. non-responders to therapy). Each of these approaches is different and provide unique information. Historically, the identification of insomnia phenotypes has been based on observation and description of the patient in the clinic. This latter approach has, as described above, led to unreliable categories of insomnia. Some of the strategies to identify insomnia phenotypes have begun to be applied. For example, with regard to insomnia and sleep apnea, investigators have reported that some patients present with insomnia, some with daytime sleepiness, and others with neither. These various symptom combinations may occur even for patients with equally severe sleep apnea. Furthermore, numerous studies have identified a form of insomnia in which the complaint of insomnia occurs in the context of objectively short sleep (as determined by polysomnography). These objectively short sleeping insomniacs are at increased risk of medical morbidity, particularly cardiometabolic dysfunction. Gaining a better understanding of the variety of symptom presentations for insomnia may help guide treatment or identify those for whom current treatments may be ineffective. We intend in this symposium to demonstrate the usefulness of a variety of methods to identify insomnia phenotypes. We propose to present a set of studies which investigate a variety of types of insomnia in 1) a clinical sample of insomnia patients, and 2) a population-based sample. In one study, we report different types of sleep architecture profiles in insomnia patients. A large population study used latent profile and transition analysis for a data-driven identification of robust insomnia subtypes characterized by non-sleep-related traits and life history. A third study uses a unique profile analysis to determine the simultaneous reliability of multiple insomnia diagnoses. This allows the reliability of one nosology (i.e., ICSD) to be compared to another (i.e., DSM). Finally, using objectively short sleeping insomniacs, we will report the increased risk of medical conditions as well as attenuated treatment response. The set of presentations in this symposium use a variety of study designs and data sources to help begin to improve our understanding of insomnia typologies.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the usefulness of a variety of methods that can be used to detect insomnia phenotypes
- Characterize several possible insomnia phenotypes
- Describe the influence of insomnia phenotypes on health and treatment outcome

Target Audience
Sleep specialists including psychologists, psychiatrists and neurologists

9:00am - 9:05am	**Introduction** *W. Wohlgemuth (United States)*
9:05am - 9:25am	**Sleep architecture profiles in insomnia patients** *W. Wohlgemuth (United States)*
9:25am - 9:45am	**Trait and life history profiles reveal stable insomnia subtypes** *E. Van Someren (The Netherlands)*
9:45am - 10:05am	**Predictors of Insomnia severity index profiles among veterans with obstructive sleep apnea** *D. Wallace (United States)*
10:05am - 10:25am	**Insomnia with objective short sleep duration: Impact on hypertension risk and insomnia treatment response** *J. Edinger (United States)*
10:25am - 10:30am	**Question and answer** *W. Wohlgemuth (United States)*

S48 NON-PAP treatment of obstructive-sleep apnea in late teenagers and early adulthood: What can be done, orthodontia and surgery

9:00am - 10:30am I Terrace 1

Chairs:
C. Guilleminault (United States), P. Pirelli (Italy)

Summary
Late teen-agers and young adults are non-compliant with nasal CPAP, except for subjects with enlarged adenotonsils or important septum deviation, soft tissue surgery has not been a successful long-term treatment approach. What can be offered to this age-group of patients? There have been efforts to find solutions to this issue with the knowledge that there are known facts: OSA worsen with aging and inappropriately treated OSA will worsen faster and will lead to co-morbidities and women with OSA will always get worse at menopause and OSA may be a problem during pregnancy. Dental devices have been a suggested solution but such devices are not for everybody and where do we stand as far as long term usage when given to this age group? What are the problems, compliance data, and real gain? Maxillo-mandibular surgery-MMA- is usually performed on 35 to 50 years old subjects. Would it be better to perform such surgery earlier? What are the data supporting such early surgical approach? What are the long term results, gain and down-sides? A combination of surgery and orthodontic treatments have been proposed in the recent past; what do we know about them, what are the risk, complication? These approaches involved the 'bon-bone" palatal expansion and the recently reported expansion with placement of palatal plate. Results of bone-bone palatal expansion have been prospectively study; combination of bone-bone palatal expansion with associated surgery has been performed. Where do we stand with these new approaches?

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the positive and negative results of usage of dental devices in a late teen-age and early adulthood patients group with OSA
- Assess the recommendation to perform maxilla-mandibular-advancement in young individuals, its risks and long-term results in this young adult group
- Describe the usage of bone-bone expansion in this age group with a closed intermaxillary suture and gain and risks of having a plate placed in the palate
- Review the results of combination of bone-bone palatal expansion and Lefort-1 surgery

Target Audience
Surgeons involved in sleep surgery; Orthodontists involved in treatment of young adults with OSA; Sleep specialists dealing with OSA particularly late teen-agers and early adulthood that have been shown to be the least compliant to PAP treatment

9:00am - 9:02am	**Introduction** *C. Guilleminault (United States)*
9:02am - 9:19am	**Role of dental devices, negative and positive responses –including long term-in young adults with OSA** *M. Marklund (Sweden)*
9:19am - 9:36am	**Maxillo-mandibular advancement in young OSA adult, including long term results, positive and negative findings** *C. C.-H. Lin (Taiwan)*
9:36am - 9:53am	**Bone to bone palatal expansion orthodontia and surgery in late teenagers and early adulthood: problems, complication, indications and results** *B. Vande Vannet (Belgium)*
9:53am - 10:10am	**Transverse maxillary deficiency in obstructive sleep apnea syndrome (OSAS): Benefits of utilizing a palatine plate** *T. Loncle (France)*
10:10am - 10:27am	**Distraction Osteogenesis Maxillary Expansion (DOME)** *S. Liu (United States)*
10:27am - 10:30am	**Question and answer** *P. Pirelli (Italy)*

T04 Group scoring discussion: Respiratory related breathing disorders

9:00am - 10:30am I Club H

Chairs:
T. Penzel (Germany)

Summary
In this course there are the most common and frequently requested topics to be covered in order to get familiar with the latest and most updated guidelines as per the AASM. The topics will discuss very interesting practical and clinical examples where most scorers find these very confusing. Each participant will have the opportunity to do hands on under the supervision of the experienced trainer, whom is certified by the American Board of Sleep Medicine and the Board of Sleep Technologists.

Participants will be using computer software within the course.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Summarize the updated AASM guidelines for scoring sleep stages and arousals
- Summarize the updated AASM guideliness for scoring respiratory events
- Identify the sleep related motor disorders
- Recognize sleep related events and score them accourding to the AASM guidelines

Target Audience
Health professionals interested in getting updated about the latest AASM scoring rules and to increase their confidence in scoring by understanding the methodology of applying these rules on real demo traces.

9:00am - 9:10am	**Introduction** *T. Penzel (Germany)*
9:10am - 9:50am	**Recommended and alternative sensors in PSG** *A. Obeidat (United States)*
9:50am - 10:30am	**AASM guidelines for scoring sleep stages in adults** *A. Obeidat (United States)*

K07 Sleep: from single neuron to behavior

M. Tafti (Switzerland)
Keynote 10:30am - 11:15am I Congress Hall

Sleep is conserved throughout the evolution independent of the organization of the nervous system. This suggests that mechanisms regulating this complex behavior must also be conserved at the very basic molecular and cellular levels. We have shown that cortical cultures show robust similarities in terms of electrophysiology, transcription, and metabolism to the intact cortex of living animals. That sleep can occur in in vitro models is now reported by several groups. Such a simple model is very powerful for discovering the molecular and cellular bases of sleep. We recently showed that if cortical cultures are stimulated with waking neuromodulators, they show surprising homeostatic adaptations, very similar to the effects of sleep deprivation in animals. We also used this model to dissect the signaling pathways leading to the accumulation of plasticity-related transcripts during wakefulness. A major and highly evolutionary conserved pathway (Erk) was identified and we showed that Erk phosphorylation during wakefulness regulates sleep-related genes, sleep duration and consolidation in mice. Whether sleep is a self-sustained, cell autonomous, or a neural network property is under investigation.

S49 Daytime sleepiness: Newest research results and experts' opinions

10:30am - 12:00pm I Meeting Hall IV

Chairs:
A. Büttner-Teleaga (Germany)

Summary
Excessive Daytime Sleepiness with partly imperative sleeping attacks as well as deficits in vigilance and attention is a symptom of various sleep-related diseases as well as diverse neurological diseases. In sleep disorders, especially in Sleep-related Breathing Disorders (SBD), Narcolepsy and / or Insomnia, it leads to reduced sleep quality, a strongly reduced recovery function because of deep sleep or REM suppression, increased nocturnal arousal reactions and / or extended awaking phases. The resulting impairment of mental and physical performance (due to daytime sleepiness, drowsiness and / or accident risks) plays a significant role in professional and social life.
Patients with Excessive Daytime Sleepiness (EDS) often have problems in periods of physical rest and / or in long-lasting, monotonous concentration tasks. Performance limitations are seen usually in the area of concentration, attention, vigilance, figural memory, frontal brain function and affective behaviour.
Excessive Daytime Sleepiness or the second sleep at the wheel is the most frequent cause of serious traffic accidents in Germany and one of the most frequent worldwide. Sleepiness-/Drowsiness-related traffic accidents and their triggering factors must be analyzed and preventive steps should be discussed.
In our symposium, the newest research results of Excessive Daytime Sleepiness shall be presented on one hand. On the other hand, expert opinions about vigilance and Excessive Daytime Sleepiness shall be given.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Describe Excessive Daytime Sleepiness
- Review the recent research results of this topic
- Summarize the guidelines of experts' opinions of this field

Target Audience
Sleep Scientists/Researchers/Doctors; Neurologists; Psychiatrists; Doctors of Internal Medicine; Family doctors

10:30am - 10:35am	**Introduction** *A. Büttner-Teleaga (Germany)*
10:35am - 10:55am	**Sleep disorders and excessive sleepiness: Who needs treatment for safety critical tasks** *M. Howard (Australia)*
10:55am - 11:15am	**EDS in kleine-levin syndrome – an update about the new research** *F. Han (China)*
11:15am - 11:35am	**Experts' opinions: EDS and its effects hypersomnia and neurological diseases** *S. Kotterba (Germany)*
11:35am - 11:55am	**Experts' opinions: EDS and its effects in sleep breathing disorders (e.g. OSA/CSA)** *M. Orth (Germany)*
11:55am - 12:00pm	**Question and answer** *A. Büttner-Teleaga (Germany)*

S50 Multiple sclerosis, sleep and sleep disorders

10:30am - 12:00pm I Meeting Hall V

Chairs:

L. Ferini-Strambi (Italy)

Summary

Multiple sclerosis (MS) is a chronic disabling disease of the central nervous system that mainly affects young subjects. Some recent studies suggest that MS patients are at increased risk for sleep disturbances and that sleep disturbances contribute to fatigue and other chronic symptoms in MS. For example, the prevalence of RLS in MS patients is higher than the prevalence of RLS in people of the same age in the general population. Moreover, MS patients with RLS have higher scores in the Expanded Disability Status Scale compared to MS patients without RLS. The possible association between SM and narcolepsy is an interesting topic: may the two different diseases share pathophysiologic mechanisms? Evidence suggests that autoimmune diseases tend to co-occur so that patients with an autoimmune disorder are at higher risk of a second autoimmune disease. RBD is rarely associated with non-synuclein structural lesions affecting the pons, medulla, or limbic system. Pons and medulla are responsible for the generation and maintenance of REM sleep muscle atonia, while more rostral structures in mesiotemporal limbic may facilitate dream enactment behavior. The spectrum of lesional RBD comprises demyelinating brain lesions, and in these cases neuroimaging findings are very important. Patients presenting with RBD along with focal neurologic deficits, such as in MS patients, are recommended to undergo brain imaging study. In RBD patients with a normal neurologic exam, MRI is generally not warranted. Few studies have specifically addressed the impact of drugs for MS on sleep. Moreover, even when sleep is considered, quantitative assessment by standardized questionnaires or PSG is lacking. Interferon-beta and some symptomatic medications may affect sleep, thus contributing to fatigue, depression, and poor quality of life; conversely, natalizumab and cannabinoids may improve sleep. Consideration of the impact on sleep should be part of the design of trials of new therapies for MS. The objectives of this symposium are to show the latest relevant data on sleep disorders in MS and offer a helpful approach to the identification and workup of the most common sleep problems in this population. Unexplored research avenues and opportunities to address important questions at the interface of sleep and MS will be also discussed. Clinicians need to remember to ask about sleep in all MS patients and intervene when appropriate. A systematic approach that takes sleep into account should be recommended to enhance recognition and appropriate management of sleep disruption, including disorders related to medications for MS.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Identify the most common sleep disorders in MS patients
- Treat sleep disorders in MS patients
- Recognize the possible effects of drugs for MS on sleep

Target Audience

Sleep medicine experts, neurologists, psychiatrists, psychologists, technicians

10:30am - 10:32am	**Introduction** *L. Ferini-Strambi (Italy)*
10:32am - 10:49am	**Sleep-related motor disorders in MS** *M. Manconi (Switzerland)*
10:49am - 11:06am	**EDS and narcolepsy-like phenotypes in MS and related disorders** *C. Bassetti (Switzerland)*
11:06am - 11:23am	**Parasomnias and MS** *G. Mayer (Germany)*
11:23am - 11:40am	**Imaging explaining sleep-disorders and fatigue in MS** *G. Rizzo (Italy)*
11:40am - 11:57am	**The impact of drugs for MS on sleep** *L. Ferini-Strambi (Italy)*
11:57am - 12:00pm	**Question and answer** *L. Ferini-Strambi (Italy)*

S51 Diagnosis, morbidity and treatment of pediatric OSA: What's new?

10:30am - 12:00pm I North Hall

Chairs:

D. Gozal (United States), L. Kheirandish-Gozal (United States)

Summary

Pediatric obstructive sleep apnea syndrome (OSAS) has become a major public health diagnostic entity not only because of its relatively high prevalence, but particularly because of the increased risk for cognitive and behavioral deficits associated with OSAS. Furthermore, evidence for cardiovascular involvement manifesting as endothelial dysfunction, systemic and pulmonary hypertension, and alterations in left ventricular geometry and contractility have been reported, along with heightened risk for metabolic perturbations such as insulin resistance, and dyslipidemias, nocturnal enuresis, and excessive daytime sleepiness (EDS). As a result of OSAS, increases in healthcare utilization and costs have been reported, while effective treatment exerts obvious quality of life improvements. Current clinical practice heavily and justifiably relies on clinical presentation and physical examination with overnight polysomnography (PSG) constituting the definitive diagnostic tool. In the usual clinical settings, evaluation for the presence of any of the aforementioned morbidities is seldom if ever performed. However, PSG-derived measurements are marginally predictive of any of OSAS-associated morbidities, such that two patients with similar PSG findings may exhibit markedly divergent phenotypes: i.e., presence or absence of end-organ OSAS-related morbidities. – The extent and "hidden" phenotypes of OSAS-associated morbidities will be presented in this symposium by Professor David Gozal. Furthermore, in most countries the availability of pediatric sleep laboratories is too limited to enable generalized implementation of PSG to match clinical needs. Moreover, several factors decrease PSG effectiveness and utility as the gold standard for young children and infants, particularly due to the inconvenience it imposes for both parents and children who need to spend the whole night in a sleep laboratory, and the high aversion manifested by a proportion of children when multiple attached sensors are placed. Therefore, novel home-based approaches have been explored in recent years and will be summarized in this symposium by Dr. Hui-Leng Tan. Treatment of OSAS has also evolved and the "one size fits all" approach is clearly inadequate. Dr. Maria Luz Alonso-Alvarez will present her recent findings from prospective cohorts in non-obese and obese children with OSAS and summarize the literature on adenotonsillectomy. Then, Professor Leila Kheirandish-Gozal will tackle the non-surgical options that have recently emerged including anti-inflammatory therapy, intraoral appliances and orthodontics and myofunctional interventions.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Recognize the limitations of polysomnography in the detection of the end-organ morbidities associated with pediatric OSAS
- Describe various home-based approaches and options for the diagnosis of high pre-test probability of OSAS in children
- Identify the risk/benefit ratio of adenotonsillectomy for treatment of OSAS in chidren
- Review the state-of-the art results from non-surgical treatment options for pediatric OSAS

Target Audience

Professionals with an interest in pediatric sleep disorders

10:30am - 10:35am	**Introduction** *D. Gozal (United States)*
10:35am - 10:55am	**The morbidity of pediatric OSAS: The hidden iceberg** *D. Gozal (United States)*
10:55am - 11:15am	**OSAS diagnosis: When PSG is relatively unavailable** *H.-L. Tan (United Kingdom)*
11:15am - 11:35am	**Adenotonsillectomy for pediatric OSAS: The good, the bad and the ugly** *M.L. Alonso-Alvarez (Spain)*
11:35am - 11:55am	**Non-surgical options for pediatric OSAS** *L. Kheirandish-Gozal (United States)*
11:55am - 12:00pm	**Question and answer** *L. Kheirandish-Gozal (United States)*

S52 Dynamic circuit connecting the circadian clock and sleep/wakefulness

10:30am - 12:00pm I Club A and B

Chairs:
K.-i. Honma (Japan)

Summary
Circadian rhythms in behaviors including sleep and wakefulness in mammals are primarily regulated by the pacemaker located in the suprachiasmatic nucleus (SCN). The circadian pacemaker receives photic information such as daily light-dark cycles and photoperiods to regulate the timing as well as the length of sleep. On the other hand, behaviors are known to feedback onto the circadian pacemaker which in turn modifies the circadian regulation of behaviors. Thus, there is a circuit between the circadian pacemaker and behavior rhythms. However, little is known about output mechanisms of the circadian pacemaker onto behaviors and of behavioral feedback onto the circadian pacemaker. Recently, taking advantage of a variety of clock gene reporter systems and in vivo monitoring of clock gene expression in the SCN of freely moving mice (Nature Comm, 2016), we are able to examine the SCN circadian clockworks and behaviors simultaneously in the same animals. This and other technical advances enable us to identify the missing links of the clock-behavior circuit. In the present symposium, four speakers will talk their brand new findings related with the circuit including unpublished results and integrate these findings. Dr. Sato Honma will discuss the relationship between the circadian rhythms in clock gene expression in the SCN and two behavioral phase markers (PNAS, 2016 under 2nd review), the onset and end of an activity band, which is known to decouple under different photoperiods. She will also talk about the distinct locations of oscillators regulating either of the two behavioral phases (Nat Neurosci, 2016 in submission). Dr. Ying Xu will report a novel molecule (ZBTB20) which regulates one of the two major circadian phases of behavioral rhythms, suggesting different molecular mechanisms underlying the two (Elife, 2016). With these two papers, we are now able to access to the molecular and cellular mechanisms of the so-called Evening and Morning Oscillators in mammals proposed ca. 50 years ago. Dr. Alena Sumová will talk about the ontogeny and aging of circadian clock in the SCN in reference to environmental impacts (PLoS One, 2014, Chronobiol Int, 2016). Environmental periodicities affect the circadian clock and behavior rhythms not only as time cues but also as masking factors. Masking effects of environmental periodicities are important to identify the feedback effects of behaviors onto the circadian pacemaker especially in the early and latest stages of life. Finally, Dr. Ken-ichi Honma (Chairperson) will integrate these three papers, revealing the dynamics of circuits connecting the circadian clock and behaviors in environmental impacts. To this end, he will propose the system regulating the circadian pacemaker in the SCN and the extra-SCN oscillators in the brain DA system which is deeply involved in sleep and wakefulness.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Measure molecular and cellular events in the mammalian brain in vivo as well as in ex vivo and to integrate apparently complex phenomenon such as behaviors into a sequence of events based on a simple principle
- Recognize the oscillatory nature of sleep-wake cycle and its relationship to the circadian clock
- Differentiate the internal and external (masking) oscillation, both of which express similar circadian rhythms
- Predict the changes of sleep-wakefulness in various impacts of environment
- Generalize the interaction between two rhythmic functions

Target Audience
Graduate students, researchers in brain science especially circadian rhythms, behavior and ontogeny (aging), clinicians who are interested in circadian rhythm disorders, seasonal sleep disorders and social jet-lag

10:30am - 10:35am	**Introduction** *K.-i. Honma (Japan)*
10:35am - 10:55am	**Oscillators in the suprachiasmatic nucleus regulating seasonality in sleepwake rhythm** *S. Honma (Japan)*
10:55am - 11:15am	**Responsiveness of the circadian system to environmental challenge during the lifespan** *A. Sumova (Czech Republic)*
11:15am - 11:35am	**ZBTB20 acts as a circadian output regulator of activity rhythms** *Y. Xu (China)*
11:35am - 11:55am	**Integration: dynamic circuits connecting the circadian clock and sleep/wakefulness** *K.-i. Honma (Japan)*
11:55am - 12:00pm	**Question and answer** *K.-i. Honma (Japan)*

S53 How the reticular activating system (RAS) modulates perception and movement

10:30am - 12:00pm I Meeting Hall 1A

Chairs:

S. Datta (United States)

Summary

Gamma rhythms have been proposed to promote the feed forward or "bottom-up" flow of information from lower to higher regions in the brain during perception, movement, and consciousness. On the other hand, beta rhythms have been proposed to represent feedback or "top-down" influence from higher regions to lower. This symposium is concerned with questions about bottom-up processes, specifically, where does the gamma activity arise? Is the gamma band activity generated only at the level of the cortex, or does it arise from lower centers to interact with ongoing cortical activity? The discovery of gamma band activity in the RAS suggests that at least some of it modulates higher centers. What mechanisms generate activity at such frequencies? The findings showing high threshold calcium channels are responsible for generating gamma band activity in the RAS identifies at least one mechanism. Is gamma band activity different during waking vs REM sleep? Results showing that waking is mediated by CaMKII modulation of P/Q-type channels and REM sleep is modulated by cAMP/PK modulation of N-type channels points to different intracellular pathways influencing each state. Which lower centers generate gamma band activity, and is it coherent with cortical gamma band activity? Pathways from the RAS through the intralaminar thalamus manifest the same intracellular mechanisms to help drive gamma band activity in the cortex. Recent studies show that cortical gamma band coherence is present during waking but not during REM sleep. Breakthrough studies show that the same primate RAS neurons exhibiting activity in relation to arousal are also involved in voluntary movement. Moreover, activation of this region using deep brain stimulation (DBS) of the pedunculopontine nucleus (PPN) in humans has salutary effects on movement, sleep, and cognition. These novel findings demonstrate that gamma band activity is generated in the RAS, that it projects at least through the intralaminar thalamus to the cortex, and that RAS gamma activity determines the presence of gamma band coherence in the cortex during waking but not REM sleep. In addition, these cells help modulate voluntary movement. Gamma oscillations appear to participate in sensory perception, problem solving, and memory, and coherence at these frequencies may occur at cortical or thalamocortical levels. Rather than participating in the temporal binding of sensory events, gamma band activity generated in the RAS may help stabilize coherence related to arousal, providing a stable activation state during waking, and relay such activation to the cortex, which thus participates in "non-specific" thalamocortical processing. Continuous sensory input will thus induce gamma band activity in the RAS to participate in the processes of preconscious awareness, and provide the essential stream of information for the formulation of many of our actions. Most of our thoughts and actions are driven by preconscious processes. This unique and comprehensive symposium will reveal how RAS output modulates our perceptions and movements on a continuous basis.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Recognize, at the cellular level, the latest discoveries on calcium channel and intracellular pathways involved in the function of RAS neurons that generate gamma band activity, a field that promises to explode with novel therapeutic advances
- Recognize, at the behavioral level, how RAS neurons modulate perception and action, especially in mammals and primates, a concept that expands our view of the role of the RAS
- Describe, at the clinical level, how pioneering studies using PPN DBS, have advanced our therapeutic approaches and opened new avenues for addressing disorders of arousal and movement

Target Audience

Basic scientists and clinicians interested in startling advances on the role of the RAS that expand its purview from sleep/wake control to new areas including its role in preconscious awareness, its modulation of context during perception, and its shaping of voluntary movement. These advances open novel avenues for research and treatment of diseases beyond those involving sleep disorders.

10:30am - 10:32am	**Introduction** *S. Datta (United States)*
10:32am - 10:49am	**Mechanisms modulating the control of waking vs REM sleep** *S. Datta (United States)*
10:49am - 11:06am	**RAS interactions with cerebellar motor control** *E. Scamati (Italy)*
11:06am - 11:23am	**Gamma band coherence during waking and REM sleep** *P. Torterolo (Uruguay)*
11:23am - 11:40am	**Effects of deep brain stimulation of the RAS in parkinson's disease** *P. Mazzone (Italy)*
11:40am - 11:57am	**Gamma activity and its modulation of arousal and movement** *E. Garcia-Rill (United States)*
11:57am - 12:00pm	**Question and answer** *S. Datta (United States)*

S54 Menopause matters: Hormones, hot flashes and sleep disorders

10:30am - 12:00pm I Meeting Hall 1B

Chairs:
F. Baker (United States)

Summary
The proposed symposium addresses a need for furthering awareness and understanding of sleep health in women. Menopause represents one of the pivotal time points in a woman's life when sleep disturbances emerge, or worsen, and sleep disturbance is a leading symptom that causes women to seek treatment. Over 40% of women transitioning to menopause report poor sleep quality and more than a quarter of midlife women have severe sleep disturbances that qualify them for an insomnia disorder. Multiple factors contribute to this increase in trouble sleeping, ranging from the presence of hot flashes, the development of primary sleep disorders such as sleep-disordered breathing, changes in mood state, demands in the home and at work, as well as the changing hormone profile. The proposed symposium takes a translational and multifactorial view of the causes and treatment of disturbed sleep in women around menopause. The symposium will be relevant to a broad audience, including researchers, trainees, and clinicians. Presenters include expert researchers and clinicians in the field of women's sleep and health. They will present new research uncovering mechanisms of action of sex steroids on sleep-regulatory centers in the rodent brain, as well as data about the factors that contribute to disturbed sleep in menopausal women, including aging processes, depression, and work-related stress. New data will also be presented showing the contributions of hot flashes and changing sex steroid hormone levels to poor polysomnographic and subjective sleep quality in perimenopausal women with insomnia disorder, which have implications for treatment. There is a rise in the prevalence of sleep-disordered breathing following menopause; the etiology, recognition and treatment of sleep-disordered breathing in this context will be discussed.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize mechanisms of action of female sex steroid modulation of sleep through their actions on sleep centers in the brain
- Demonstrate knowledge about physiological, psychological, and social causes of sleep disruption in midlife women
- Distinguish how hot flashes and sex steroids contribute to the development of insomnia in the menopausal transition
- Identify options for treatment of sleep complaints that arise in the context of the menopausal transition.
- Recognize the development of sleep-disordered breathing in midlife women and how best to treat it

Target Audience
Clinicians, researchers, and trainees in the field of sleep medicine, particularly those with an interest in women's health

10:30am - 10:35am	**Introduction** *F. Baker (United States)*
10:35am - 10:55am	**Mechanisms underlying the influence of female sex steroids on sleepwake regulatory systems** *J. Mong (United States)*
10:55am - 11:15am	**Why does sleep quality decrease during the menopausal transition?** *P. Polo-Kantola (Finland)*
11:15am - 11:35am	**Impact of hot flashes and hormone fluctuations on sleep in perimenopausal women with insomnia** *F. Baker (United States)*
11:35am - 11:55am	**The role of menopause in the development of sleep-disordered breathing in women** *E. Lindberg (Sweden)*
11:55am - 12:00pm	**Question and answer** *F. Baker (United States)*

S55 OSA treatment in adult [men and women] non compliant with nasal PAP, indication and potential treatment: Surgical approaches and nerve implantation

10:30am - 12:00pm I Terrace 1

Chairs:
C.C.-H. Lin (Taiwan), S.-W. Kim (Republic of Korea)

Summary
Middle aged men and pre and post menopausal women may not be compliant with PAP treatment and may look for other treatment options. Surgery and XII nerve implantation have been two avenues that have been followed. However results have been variables and long term data are often. missing. Evaluation of patients and their selection will be considered with presentation of new techniques to assess patients before surgery. Surgical implantation of hypoglossal nerve is very much considered in different places and fairly short term results are available; but transcutaneous stimulation, a less invasive technique that has been rule-out before is again a potential option.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Review alternative treatments for adults 40 years and older with OSA that cannot tolerate nasal CPAP
- Recognice the role of computer aided surgical stimulation when performing maxilla-mandibular advancement
- Describe the role of counter-clockwise rotation when performing maxilla-mandibular advancement
- Recognize the role of upper airway stimulation, surgical approach and results obtained both in Europe and North America
- Recognize a new stimulation of upper airway dilators through transcutaneous stimulation

Target Audience
Specialists considering alternative treatment in OSA patients' refractory to CPAP; Surgeons and Radiologists involved in upper-airway surgery; Specialists involved in nerve stimulation of upper-airway

10:30am - 10:32am	**Introduction** *C.C.-H. Lin (Taiwan)*
10:32am - 10:49am	**Computer aided surgical simulation for maxillomandibular advancement** *S.S. Hsu (Taiwan)*
10:49am - 11:06am	**Maxillomandibular advance with CCW rotation for surgical treatment of OSA** *J. Cifuentes (Chile)*
11:06am - 11:23am	**European experience with different concepts of upper airway neurostimulation** *J. Maurer (Germany)*
11:23 - 11:40	**Patient screening, selection and surgical device implantation** *M. Boon (United States)*
11:40am - 11:57am	**Transcutaneous electrical stimulation of the upper airway dilator muscles in patients with obstructive sleep apnoea** *J. Steier (United Kingdom)*
11:57am - 12:00pm	**Question and answer** *S.-W. Kim (Republic of Korea)*

O08 REM behavior disorders oral abstract presentations

10:30am – 12:00pm I Club D and E

Chairs:
C. Schenck (United States), A. Iranzo (Spain)

10:30am – 10:45am	**INCREASED EEG DESYNCHRONIZATION DURING PHASIC REM SLEEP IN IDIOPATHIC REM SLEEP BEHAVIOR DISORDER** *J.-S. Sunwoo (Republic of Korea)*
10:45am – 11:00am	**SEVERITY OF REM SLEEP MUSCLE ATONIA LOSS IN IDIOPATHIC REM SLEEP BEHAVIOUR DISORDER CORRELATES WITH THE DEGREE OF ABNORMALITIES IN THE VESTIBULAR EVOKED MYOGENIC POTENTIALS** *M. Puligheddu (Italy)*
11:00am – 11:15am	**INVESTIGATING BIOMARKERS FOR PREDICTING THE CONVERSION TO ALPHA-SYNUCLEINOPATHIES IN PATIENTS AFFECTED BY REM SLEEP BEHAVIOR DISORDER: A COMPREHENSIVE ANALYSIS OF CLINICAL, NEUROPSYCHOLOGICAL, NEUROIMAGING, AND CEREBROSPINAL-FLUID DATA** *C. Liguori (Italy)*
11:15am – 11:30am	**ABNORMAL ACTIVITY IN THE REWARD SYSTEM IN PARKINSON'S DISEASE PATIENTS WITH RAPID EYE MOVEMENTS SLEEP BEHAVIOR DISORDER** *C. Beal (France)*
11:30am – 11:45am	**PREVALENCE AND ASSOCIATED FACTORS FOR REM SLEEP BEHAVIOUR DISORDER: A NATION-WIDE POPULATION-BASED STUDY OF 30,097 CANADIAN ADULTS** *C. Yao (Canada)*
11:45am – 12:00pm	**REM SLEEP AS A PREDICTOR OF SEIZURE RECURRENCE IN GENETIC GENERALIZED EPILEPSY: A PROSPECTIVE STUDY** *J. Mekky (Egypt)*

O09 Sleep breathing disorders oral abstract presentations

10:30am – 12:00pm I Club H

Chairs:
G. Plazzi (Italy), R. Heinzer (Switzerland)

10:30am – 10:45am	**LIMITATIONS OF THE APNEA-HYPOPNEA INDEX FOR ASSESSING THE CLINICAL SEVERITY OF OBSTRUCTIVE SLEEP APNEA IN CHILDREN** *Y.-S. Chang (Taiwan)*
10:45am – 11:00am	**UPPER AIRWAY STIMULATION EFFECTIVELY TREATS REM OBSTRUCTIVE SLEEP APNEA** *F. Luyster (United States)*
11:00am – 11:15am	**CPAP WITHDRAWAL ON 24-HOUR BLOOD PRESSURE AND ARTERIAL STIFFNESS IN WOMEN AND MEN WITH OBSTRUCTIVE SLEEP APNEA. A RANDOMIZED CONTROLLED TRIAL** *C. Sahlin (Sweden)*
11:15am – 11:30am	**A MULTICENTER PILOT STUDY ON THE INDICATIONS OF THE NEGATIVE PRESSURE SLEEP THERAPY SYSTEM FOR THE TREATMENT OF OBSTRUCTIVE SLEEP APNEA** *C.M. Lin (Taiwan)*
11:30am – 11:45am	**PREOPERATIVE APNEA-HYPOPNEA INDEX PREDICTS INCREASED POSTOPERATIVE INTRATHORACIC PRESSURE DURING SLEEP IN PATIENTS WHO UNDERWENT ENDOSCOPIC SINUS SURGERY** *M. Suzuki (Japan)*
11:45am – 12:00pm	**REM-ASSOCIATED SLEEP DISORDERED BREATHING: PREVALENCE AND CLINICAL SIGNIFICANCE IN THE HYPNOLAUS COHORT** *P. Acosta-Castro (Spain)*

K08 Restless legs syndrome: Towards a new concept of the disease

J. Winkelmann (Germany)
Keynote 11:15am - 12:00pm I Congress Hall

Restless legs syndrome: towards a new concept of the disease. Genome-wide association studies led to the identification of first genetic risk variants for RLS and ongoing meta-analysis of large consortia yealed to many new risk loci. Variants detected by this approach are common genetic variants, which individually confer only a minor increase in risk of the disease. Large scale sequencing studies complemented the picture and showed that RLS is a complex genetic disorders where common and rare genetic variants contribute to the phenotype. This knowledge changed our idea about our pathophysiological concept. Furthermore, we are gaining new ideas towards the mode of action of drugs and potential new drugs.

Bioprojet Industry Satellite Symposium: Cataplexy: From fundamentals to the clinic

12:30pm - 2:00pm I Congress Hall

Chairs:
G.J. Lammers (The Netherlands)

A satellite symposium brought to you by Bioprojet.

12:30pm – 12:36pm	**Cataplexy: From fundamentals to the clinic** *G.J. Lammers (The Netherlands)*
12:36pm – 1:04pm	**Neurobiology of REM sleep and Cataplexy: a short update** *C. Peyron (France)*
1:04pm – 1:32pm	**Histamine neurones, REM sleep and cataplexy** *J.-C. Schwartz (France)*
1:32pm – 2:00pm	**Cataplexy data: HARMONY CTP and HARMONY III clinical trials data** *Z. Szakacs, Budapest (Hungary)*

NOX Medical Industry Workshop: Evolution of sleep medicine – challenging the status quo

12:30pm – 1:15pm I North Hall

The industry workshop is intended to address some of the main challenges in sleep medicine today. Current sleep diagnostic tools and measurements show room for improvements using new technology and approaches. Knowing who to treat and how technology can be used to maximize clinician impact on more patients' lives are topics which will be addressed. Those interested in new approaches in diagnostics and how we can continue to evolve sleep medicine should find this symposium of interest. Dr. Allan Pack has chosen to donate his honorarium to the Helgi Kristbjarnarson Memorial Fund, to support promising students and researchers within the fields of sleep research and sleep technology.

An Industry Workshop made available by NOX Medical.

12:30pm – 12:45pm	**Opening Remarks** *L. Parrino (Italy)*
12:45pm – 1:00pm	**Evolution of Sleep medicine part 1** *A. Pack (United States)*
1:00pm – 1:15pm	**Evolution of Sleep medicine part 2** *E. Sif Arnardottir (Iceland)*

S56 What can we learn from recent large negative clinical trials in sleep-disordered breathing?

2:00pm - 3:30pm I Congress Hall

Chairs:
A. Pack (United States)

Summary
We have reached the stage in our field when we are conducting large phase 3 randomized clinical trials. Unfortunately, and surprisingly, these studies have been negative, i.e. , treatment of sleep-disordered breathing did not produce the expected benefits. They did not replicate the findings of observational studies and/or smaller randomized trials. This may reflect issues related to selection of relevant subjects, bias in recruitment selecting less impacted individuals, poor adherence to therapy, etc. The goal of this symposium is not to simply hear the results of these large studies, but rather to have leading investigators involved in these efforts explain what they learned from the conduct of the study and what they would do differently if they had to do this again. It is only by self-criticism that we can improve and do better studies in the future. The symposium has speakers who will discuss each of the four major negative studies. SERVE-HF – Assessment of ASV in treatment of central sleep apnea in patients with heart failure (Cowie MR, et al, New England Journal of Medicine 373:1095-1105, 2015). APPLES Study – Assessment of longer term (6 months) benefit of treatment by CPAP on neurophysiological factors (Kushida C, et al, Journal of Sleep Research 2016 May 30. doi: 10.1111/jsr.12430). CHAT Study – Assessment of efficacy of adenotonsillectomy in children with obstructive sleep apnea (Marcus CL, et al, New England Journal of Medicine 368:2366-2376, 2013). SAVE Study – Study to assess whether CPAP reduces cardiovascular events in patients with known cardiovascular and/or cerebrovascular disease (McEvoy RD, et al, New England Journal of Medicine 375:919-931, 2016). We are fortunate that Principal Investigator or leader in each of the areas have agreed to speak at this symposium.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Review recent major clinical trials in sleep-disordered breathing and their results
- Recognize the importance of careful selection of patient populations to be studied in clinical trials and of primary end-points for assessment
- Describe the changes that need to be introduced to conduct future large phase 3 clinical trials

Target Audience
Clinicians, clinical trials experts, investigators in sleep-disordered breathing

2:00pm - 2:05pm	**Introduction** *A. Pack (United States)*
2:05pm - 2:25pm	**Critique of SERVE-HF study** *D. Bradley (Canada)*
2:25pm - 2:45pm	**Commentary on APPLES study** *C. Kushida (United States)*
2:45pm - 3:05pm	**The CHAT study** *D. Gozal (United States)*
3:05pm - 3:25pm	**Why was the SAVE study negative?** *D. McEvoy (Australia)*
3:25pm - 3:30pm	**Question and answer** *A. Pack (United States)*

S57 Ascertainments of RBD and its clinical implications for neurodegeneration

2:00pm - 3:30pm I Meeting Hall IV

Chairs:
C. Trenkwalder (Germany)

Summary
This symposium appraises the current research on REM sleep behavior disorder (RBD) in relation to neurodegenerative diseases. Research of RBD in the last 10 years has given rise to several questions: Is RBD a disease entity by itself, a rare sleep disorder? Is it only a prodromal state announcing a future neurodegenerative disease, or concomitant to other conditions? If RBD converts into neurodegeneration, how reliably can we diagnose RBD, what is the percentage of converters to Parkinson syndromes and how honestly can we counsel patients about their future disease perspectives?
In this symposium, RBD experts will provide insight into their recent research areas and bridge the questions on basic and clinical research and counseling patients in sleep practice. Yun Kwok Wing presents the possibilities of assessing RBD using various available questionnaires and scales. The validity and reliability of these clinical instruments will be critically appraised. The reliable diagnosis by polysomnography can even be determined earlier when assessing REM without atonia and using video PSG. Rigorous real-time video review of REM phases in de novo Parkinson's disease patients has revealed prodromal RBD with mostly mild dream-associated motor behaviors and/or vocalizations without excessive loss of physiologic REM muscle atonia. This poses the question whether the ability to sustain muscle atonia and suppress dream enactments reflects on the extent and pathways of neurodegeneration. Recent research of synucleinopathies benefits from RBD as a biomarker and uses this approach in clinical cohorts of prodromal Parkinson's disease, when combining RBD and other markers such as hyposmia, autonomic nervous system disorders or cognitive decline. Recent cohorts such as the PARS study, TREND study or P-PPMI have integrated RBD as a major factor, but the percentage of RBD in relation to other markers varies considerably within current cohorts and the role of RBD should be re-evaluated. It is noteworthy and important, however, to know the limits of RBD diagnosis and the variability of the disease course. The "non-converters" and the likelihood of conversion is discussed by Birgit Högl, who will try to characterize phenotypes and symptoms which increase the likelihood of conversion and demonstrate examples of those that obviously never converted into a neurodegenerative disease. Erik St. Louis will discuss the ethical implications and current approaches toward the prognostic counseling of RBD patients. While it is known that the risk for phenoconversion to a synucleinopathy neurodegenerative disorder is high, defining the timeframe and precise prognostic risk for individuals remains uncertain, and when and how to deliver prognostic information to the patient remains a difficult and imprecise art. Some recent data concerning delivery of prognostic information to RBD patients, and discussion of possible approaches will be outlined.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Evaluate frequently used scales and questionnaires for assessment of REM Sleep Behavior Disorder (RBD)
- Recognize the features of prodromal RBD in video-polysomnography and differentiate them from clinical RBD
- Recognize RBD as a biomarker from discussion of known cohorts which have applied RBD as a key feature to measure conversion from idiopathic RBD to neurodegenerative disease.
- Recall the variable forms of RBD and the possible determinants of so-called "non-converters"

Target Audience
Sleep clinicians, neurologists, psychologists, basic science sleep researchers, students

2:00pm - 2:02pm	**Introduction** *C. Trenkwalder (Germany)*
2:02pm - 2:19pm	**Assessment of RBD with questionnaires and scales in the population: How valid reliable are they?** *Y.K. Wing (Hong Kong)*
2:19pm - 2:36pm	**Assessment of prodromal RBD using vPSG– earlier rather than early?** *F. Sixel-Döring (Germany)*
2:36pm- 2:53pm	**Worldwide cohorts address RBD for prodromal parkinson – is it worth the effort?** *C. Trenkwalder (Germany)*
2:53pm - 3:10pm	**RBD – the non-converters: What do they tell us?** *B. Högl (Austria)*
3:10pm- 3:27pm	**Ethical aspects of RBD prognostic counseling – what do patients want and need to know?** *E. St. Louis (United States)*
3:27pm - 3:30pm	**Question and answer** *C. Trenkwalder (Germany)*

S58 Environmental challenges: The impact of artificial light on sleep and circadian biology

2:00pm - 3:30pm I Meeting Hall V

Chairs:
A. Skeldon (United Kingdom)

Summary
The ability to produce artificially bright light has had a profound effect on our light environment, giving us much greater flexibility over when we work and socialise. But with what impact? Light pollution affects the habits of animals and humans, with light at night disrupting rhythms and behaviours of wildlife and correlated with poor sleep quality and reduced sleep duration in humans. Not only is there light pollution at night, our lifestyles mean that we spend the majority of time indoors and expose ourselves to artificial light long after sunset. The net effect is that we have a greatly reduced exposure to the natural cycle of light and dark. The reduced contrast between light intensity experienced during the day versus the night results in delayed circadian rhythms, later sleep timing, social jet-lag and difficulties maintaining 24 hour rhythmicity. All of which are further exacerbated by the increased use of lighting in the evening with an enhanced-blue component. This is a problem since circadian rhythm disorders are linked with poor health outcomes. Yet artificial light also presents an opportunity: we have control over our lighting environment and intelligent lighting design can enhance circadian rhythmicity with corresponding positive health benefits. This session will provide an overview of problems associated with our modern lighting environment and some of the possible solutions. The talks will consider: (i) Measuring and specifying lighting for the circadian system. (Speaker: Mariana Figuero, USA). This talk will focus on how to measure and specify circadian effective light, discuss some of the light at night studies that assessed light exposures experienced in several different populations. It will also review recent studies measuring the impact of self-luminous displays on acute melatonin suppression in the evening, with special emphasis on adolescents. (ii) Assessing the impact of light-at-night on circadian function and mood. (Speaker: Andrew Coogan, Ireland). This talk will discuss the impact of light-at-night on circadian function in both animal models and people and explore the effect that light-at-night exerts on mood. The links between artificial light, circadian rhythms, sleep and mood status will be examined. (iii) The modern versus natural lighting environment and its impact on sleep and circadian biology. (Speaker: Kenneth P Wright Jr USA). Effects of modern versus natural lighting environments on sleep and circadian timing in humans will be discussed. Topics will include the circadian timing of sleep and awakening, changes in circadian rhythms across seasons, and effects of the weekend. (iv) The interaction of light and social constraints with sleep and circadian biology. (Speaker: Anne Skeldon, UK). Reduced light during the day and self-selected light in the evening delays sleep, exacerbating mismatches between preferred sleep timing and the time we need to get up for work/school and resulting in `social jetlag'. Using a modelling approach, the effect of changing social constraints (e.g starting school later) will be contrasted with the effect of changes to our light environment.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Record circadian effective light and identify its role in determining circadian timing
- Recognize how light affects mood, alertness and sleep consolidation
- Identify the role of light and social constraints in determining sleep timing

Target Audience
Basic sleep and circadian rhythms researchers interested in the biological effects of light; Clinicians who use light as a therapy for circadian rhythm disorders or who are interested in using light in a clinical setting to promote sleep; Those interested in optimising interventions to ameliorate social jetlag.

2:00pm - 2:05pm	**Introduction** *A. Skeldon (United Kingdom)*
2:05pm - 2:25pm	**Measuring and specifying lighting for the circadian system** *M. Figuero (United States)*
2:25pm - 2:45pm	**Assessing the impact of light-at-night on circadian function and mood** *A. Coogan (Ireland)*
2:45pm - 3:05pm	**The modern versus natural lighting environment and its impact on sleep and circadian biology** *K. Wright (United States)*
3:05pm - 3:25pm	**The interaction of light and social constraints with sleep and circadian biology** *A. Skeldon (United Kingdom)*
3:25pm - 3:30pm	**Question and answer** *A. Skeldon (United Kingdom)*

S59 Developmental aspects of sleep's influence on memory and general cognitive abilities

2:00pm - 3:30pm I North Hall

Chairs:

K. Hoedlmoser (Austria), O. Bruni (Italy)

Summary

There is now extensive evidence from studies of adults supporting the idea that sleep strongly benefits the consolidation of newly acquired memories (declarative but also procedural) thereby enhancing performance at a later recall. Besides this sleepdependent memory consolidation aspect there is growing evidence that sleep - particularly sleep spindles – serves as a powerful marker of individual differences in cognitive ability. The aim of this symposium is to discuss the current state of knowledge on developmental aspects of the role of sleep in memory consolidation as well as in general cognitive abilities and learning efficiency. We will present recent findings in infants (9-16 months), pre-school aged children (3-5 yrs), elementary school aged children (8-10yrs) and adolescents (12-16 yrs) on the impact of sleep on quantitative and qualitative changes in memory but also on general cognitive abilities. More specifically, we will demonstrate that i) sleep benefits the generalization of semantic knowledge in infants (Ines Wihlelm), ii) Napping habitually enhances the cognitive benefit of naps in preschool-aged children (Rebecca Spencer), iii) sleep EEG during adolescence (12-14yrs) is highly heritable as shown by comparing high-density sleep recordings between monozygotic and dizygotic twin pairs (Leila Tarokh) and iv) developmental enhancement of thalamo-cortical sleep spindle activity from pre- to post-pubertal age, reflects an increase of general cognitive abilities (Kerstin Hoedlmoser). Development is a time of tremendous brain plasticity suggesting that sleep might more efficiently benefit processes of memory consolidation at that time. By understanding the impact of the maturation processes on the threefold relationship between sleep, memory and cognitive abilities, we can open up new avenues for sleep and developmental research.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Report developmental changes of normal human sleep on the macro-(e.g. sleep architecture) but also the microscopic level (e.g. sleep spindles, slow oscillations)
- Recall and classify theories on sleep, memory and general cognitive abilities from infancy up to adolescence
- Recognize the distinction between declarative and procedural sleep-dependent memory consolidation
- Evaluate the quality of different study designs used in the sleep and memory literature (longitudinal vs. cross-sectional; sleep deprivation; wake vs. sleep; night vs. diurnal sleep etc.)
- State that the variability in the sleep EEG spectrum is largely due to genes and shows a high heritability, the degree of which is dependent on sleep state and frequency

Target Audience

Sleep Research/Medicine specialists (psychologists, physicians, pediatricians as well as other healthcare professionals and specialists)

2:00pm - 2:05pm	**Introduction** *K. Hoedlmoser (Austria)*
2:05pm - 2:25pm	**The impact of sleep on the consolidation of newly acquired word meanings in infants (9-16 months)** *I. Wilhem (Switzerland)*
2:25pm - 2:45pm	**Sleep and cognition in preschool-age (3-5 yrs) children – a nap study** *R. Spencer (United States)*
2:45pm - 3:05pm	**Sleep and cognition from elementary school (8-10yrs) up to adolescence (14-16yrs): A longitudinal approach** *K. Hoedlmoser (Austria)*
3:05pm - 3:25pm	**Sleep and cognition in early adolescence (12-14 yrs): Results from a twin study** *L. Tarokh (Switzerland)*
3:25pm - 3:30pm	**Question and answer** *O. Bruni (Italy)*

S60 Sleep loss and socio-emotional functioning

2:00pm - 3:30pm I Club A and B

Chairs:
J. Axelsson (Sweden)

Summary

The past few years have seen an increase in research on sleep loss and emotional functioning as it pertains to social settings. Recognizing others' emotions, understanding others, being empathetic, and acting appropriately are all important factors in positive social interactions. Studying the role of sleep in these abilities is imperative for understanding the effects of sleep loss in all facets of life. The speakers in the proposed symposia will review the presently known links of how sleep affects central aspects of our social life as well as show own recent data. The first presenter, Dr John Axelsson will swiftly introduce the symposia, the speakers and the aims. The second speaker, Dr Peter Franzen will present data on sleep and socio-emotional functioning in youth. Dr Franzén will also present data from brain imaging studies illustrating the underlying mechanisms of how sleep affects central cognitive functions for social functioning. The third presenter, Dr Tina Sundelin, will review how sleep loss affects social perception and social abilities, which includes recent experimental studies determining how sleep deprivation affects self-reported sociability, emotional expressiveness, moral awareness, leadership ability, and empathic accuracy. The fourth presenter, Louise Bettie will review how clinical sleep disturbances affects social performance, and present recent and ongoing data on how insomnia affects social functioning. All speakers are at the forefront of this relatively new line of research within sleep, and the proposed symposium will contribute with knowledge about how sleep deprived individuals interact with people around them both in natural and experimental settings. Our objective within this symposium is to present and discuss recent advances of the role of sleep for social functioning. The included speakers will, with their expertise from different research areas, contribute to a fruitful discussion about how sleep affects social life, the involved mechanisms, and the clinical relevance of the presented findings. We believe that the novel symposia will inspire and stimulate junior and senior researchers so that they will contribute to the development of research in the present area.

Learning Objectives

Upon completion of this CME activity, participants should be able to:
- Identify relevant aspects of social functioning that are affected by sleep loss
- Recognize the extent to which these influence behavior
- Give examples from both experimental and natural settings
- Recognize the involved mechanisms of how sleep affect social life

Target Audience

Researchers in the fields of social functioning after sleep, including practitioners working with people who suffer from sleep related issues and anyone interested in basic research on the effects of sleep loss

2:00pm - 2:05pm	**Introduction** *J. Axelsson (Sweden)*
2:05pm - 2:30pm	**Sleep in relation to neural systems: implications for emotion regulation and psychopathology** *P. Franzén (United States)*
2:30pm - 2:55pm	**The effects of sleep loss on social perception and interpersonal interactions** *T. Sundelin (Sweden)*
2:55pm - 3:20pm	**Emotion and face recognition in insomnia** *L. Beattie (United Kingdom)*
2:20pm - 3:30pm	**Question and answer** *J. Axelsson (Sweden)*

S61 Idiopathic hypersomnia: A neglected disorder

2:00pm - 3:30pm I Meeting Hall 1A

Chairs:

I. Arnulf (France)

Summary

Bedrich Roth in Prague first comprehensively described Idiopathic hypersomnia (IH), a devastatating but little studied central disorder of hypersomnolence. Most research into this family of disorders has focused on narcolepsy type 1, which is a relatively homogeneous, immuno-genetically mediated disorder and Kleine-Levin syndrome. In contrast, IH is often considered a heterogeneous disorder that is also rare, and that includes clinical phenotypes ressembling narcolepsy without SOREMPs or cataplexy (with repeated, short restorative naps), and a phenotype with an unambiguous excess of sleep, sleep drunkeness, continuously impaired alertness, and infrequent long, non restorative naps. Conventional diagnostic sleep tests reflect this heterogeneity. In contrast with narcolepsy, MSLTs are greater than 8 min in 2/3 of the cases, poorly repeatable, and when they are abnormal, clinicans remain fearful of having missed an alternate 'cause' for hypersomnolence such as upper airway resistance syndrome, depression or chronic sleep restriction in a long sleeper. That being said, patients with IH are not that rare. They are young, and there is a clear female predominance which has only recently been studied per se. Sleep excess and impaired alertness are inadequately captured by a single night polysomnography followed by MSLT, whereas extended monitoring identifies 11-16 h of sleep per 24 h. In the absence of long term monitoring, the diagnosis may remain elusive and delayed, with many women being considered as depressed or fatigued. We focus this symposium on several new aspects in idiopathic hypersomnia: 1) cluster analysis of central disorders of hypersomnolence, identifies a form ressembling narcolepsy type 2 without SOREMPs and a form with a clear sleep excess; 2) the female phenotype of IH (as 70% of IH cases are women) will be compared to the male phenotype in a large multicenter study; 3) possible mechanisms contributing to idiopathic hypersomnia has progressed over the recent years from considerations of deficiencies in arousal systems,to inappropriate stimulation of sleep-inducing systems, as recently identified with GABA-inducing factors; and 4) treatments. New putative mechanisms pointing to excess in GABA related brain circuits is a major novelty in the hypersomnia field, and requires further development as it carries many consequences in terms of understanding and treating this disorder and probably others. Treatments have also been more studied, with double-blind placebo controlled studies of modafinil and clarithromycin in IH, as well as naturalistic studies of modafinil, pitolisant, sodium oxybate, and flumazenil in large patient series. This symposium will comprehensively review the latest advances in understanding, diagnosing and treating idiopathic hypersomnia, derived from large series of patients studied in tertiary referral centers.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Identify the various clinical phenotypes of idiopathic hypersomnia (female vs. male, clustering of signs and symptoms)
- Distinguish these phenotypes from other central disorders of hypersomnolence
- Recognize the values and limitations of the MSLT and long term sleep monitoring, as well as procedures to diagnose idiopathic hypersomnia
- Identify arousal systems and GABA sleep inducting factors and their putative contributions to IH
- Recognize the effects of wake promoting agents, psychostimulants and sodium oxybate, and GABA-A receptor antagonists such as flumazenil, clarithromycin, and novel drugs in development in IH, and in which IH cases their use may be most efficacious.

Target Audience

Sleep clinicians ; scientists working on mechanisms of hypersomnia; pharmacists interested in the effects of various drugs including those with novel mechanisms of action in treating idiopathic hypersomnia

2:00pm - 2:05pm	**Introduction** *I. Arnulf (France)*
2:05pm - 2:25pm	**Phenotypes of idiopathic hypersomnia** *K. Sonka (Czech Republic)*
2:25pm - 2:45pm	**Idiopathic hypersomnia: A female disorder?** *I. Arnulf (France)*
2:45pm - 3:05pm	**New insights into the mechanisms of hypersomnia** *D. Rye (United States)*
3:05pm - 3:25pm	**Treatments of idiopathic hypersomnia** *G. Mayer (Germany)*
3:25pm - 3:30pm	**Question and answer** *I. Arnulf (France)*

S62 Sleep slow waves: From cells to consciousness

2:00pm - 3:30pm I Meeting Hall 1B

Chairs:
R. Huber (Switzerland)

Summary
Electroencephalographic (EEG) slow waves (< 4 Hz) are the major characteristic of deep non-rapid eye movement sleep. Their importance for brain functioning is best exemplified by their causal relationship with the recovery function of sleep. The objective of the proposed symposium is to highlight how fundamental slow waves are for our brain across various domains. To approach this, findings from cells to system level, from multi-unit activity to consciousness and from healthy to pathological functioning are discussed. VV will start by describing the activity pattern at the cellular level and how these patterns are related to behavior in animals. YN extends these observations into humans, bridging the gap between the cellular and EEG levels, and describes how intracranial and scalp recordings of slow waves and ongoing sleep activities influence sensory processing and mediate disconnection from the external environment during sleep. RH will then provide a look from the outside, i.e. the surface high-density EEG, focusing on how slow waves are locally regulated and how they might relate to plastic changes. Finally, MM will show how slow waves may disrupt complex interactions within the brain, leading to loss of consciousness in sleep as well as in pathological conditions. Finally, RB will bridge to clinical sleep research by discussing local slow wave sleep changes in various sleep and psychiatric disorders. In summary, the presentations included in the proposed symposium are highly integrated, cover a key topic of sleep research on multiple levels, translate these findings to clinical populations and should therefore appeal to a large basic science and clinical audience.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize detailed brain activity at the cellular and circuit level that underlies the observed slow waves recorded non-invasively from the scalp
- Develop knowledge of basic neurophysiologic substrates underlying the dynamics of cortical oscillations during wakefulness and sleep in laboratory animals and humans
- Describe the extent to which the sleeping brain processes sensory information, and how slow waves relate to disconnection from the external environment
- Recognize how mapping of slow wave activity during sleep by means of high-density EEG provides insights into pathophysiological processes related to brain plasticity (e.g. in psychiatric disorders)

Target Audience
Basic and clinical sleep researchers

2:00pm - 2:02pm	**Introduction** *R. Huber (Switzerland)*
2:02pm - 2:19pm	**Local and global dynamics of cortical slow waves in mice** *V. Vyazovskiy (United Kingdom)*
2:19pm - 2:36pm	**How do slow waves affect sensory responses? Insights from intracranial electrophysiology and scalp EEG** *Y. Nir (Israel)*
2:36pm- 2:53pm	**Mapping of EEG slow waves in health and disease** *R. Huber (Switzerland)*
2:53pm - 3:10pm	**Slow-waves, brain complexity and consciousness** *M. Massimini (Italy)*
3:10pm- 3:27pm	**Local sleep changes in sleep and psychiatric disorders** *R. Benca (United States)*
3:27pm - 3:30pm	**Question and answer** *R. Huber (Switzerland)*

S63 OSA, upper-airway surgery and MAD: Techniques to select location, surgical techniques to be used, personalization of mandibular-advancement-device

2:00pm - 3:30pm I Terrace 1

Chairs:
N. Montesdeoca (Spain), C.C.-H. Lin (Taiwan)

Summary
Upper-airway surgery and usage of mandibular advancement devices are approaches very much considered in treatment of OSA. Adenotonsils even if not always curative, improve very significantly the sleep-related obstructive breathing of children, but surgical techniques are important issues. Also, techniques trying to locate the location of the upper airway collapse in adults have been very much investigated from drug-induced-endoscopy, to sleep MRI, to drug induced CT-scan and different protocols have been used, but little comparative information have been presented. Different surgical techniques have been used to address palatal and pharyngeal obstruction with variable success. The goals are enlargement of the upper-airway during sleep, with selection of the best location for the surgical intervention. Mandibular advancement devices have a similar goal, but can the study of the personal anatomical characteristics and physiological movements of jaws with such device, enable progress toward individualizing MADs therapy for patients with OSA.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the techniques that may give information on the best anatomic location to perform upper-airway surgeries in OSA
- Review surgical techniques aiming at best approaches for treatment of upper-airway in children and adult OSA patients
- Use anatomical characteristics and physiological movements of the jaws to personalize MAD therapy

Target Audience
Sleep Surgery and Medicine specialists, ENT, Orthodontists and dentists

2:00pm - 2:02pm	**Introduction** *N. Montesdeoca (Spain)*
2:02pm - 2:19pm	**Advance in surgical technique for adenotonsillectomy** *S.-W. Kim (Republic of Korea)*
2:19pm - 2:36pm	**Protocol for identifying level of obstruction: DISE vs. sleep MRI** *P.V. Krishnan (India)*
2:36pm - 2:53pm	**Virtual surgery and simulation analyses on upper airway of obstructive sleep apnea patients** *I.H. Lo (Taiwan)*
2:53pm - 3:10pm	**Selected surgical techniques for palatal and pharyngeal obstruction** *P. Baptista (Spain)*
3:10pm- 3:27pm	**Analysis of the movements of the mandible and hyoid bone in the treatment of OSA with MAD** *J. Vila (Spain)*
3:27pm - 3:30pm	**Question and answer** *C.C.-H. Lin (Taiwan)*

O10 Restless legs syndrome (RLS/WED) oral abstract presentations

2:00pm – 3:30pm I Club D and E

Chairs:
R. Silvestri (Italy), D. Kemlink (Czech Republic)

2:00pm – 2:15pm	**VALIDATION OF AN AUTOMATIC SCORING ALGORITHM FOR THE ANALYSIS OF PERIODIC LIMB MOVEMENTS ACCORDING TO THE WASM2016 GUIDELINES** *D. Alvarez-Estevez (The Netherlands)*
2:15pm – 2:30pm	**ASSOCIATION OF BTBD9 AND MAP2K5/SKOR1 WITH RESTLESS LEGS SYNDROME IN CHINESE POPULATION** *G. Li (China)*
2:30pm – 2:45pm	**COMORBIDITIES TO RESTLESS LEGS SYNDROME - RESULTS FROM THE DANISH BLOOD DONOR STUDY** *M. Didriksen (Denmark)*
2:45pm – 3:00pm	**CIRCADIAN VARIATION OF FLEXOR WITHDRAWAL AND CROSSED EXTENSOR REFLEXES IN RESTLESS LEGS SYNDROME** *C. Dafkin (South Africa)*
3:00pm – 3:15pm	**POLYSOMNOGRAPHIC FINDINGS IN RESTLESS LEGS SYNDROME (RLS) PATIENTS WITH SEVERE AUGMENTATION** *M.-L. Muntean (Germany)*
3:15pm – 3:30pm	**USING THE BEHAVIORS INDICATORS TEST-RESTLESS LEGS (BIT-RL) TO DIAGNOSE RESTLESS LEGS SYNDROME (RLS) IN ALZHEIMER'S PATIENTS WITH DEMENTIA AND NOCTURNAL AGITATED BEHAVIORS (ADNA)** *K. Richards (United States)*

T05 Breathing re-education to achieve nasal and normal minute volume for sleep

2:00pm - 3:30pm I Club H

Chairs:
S. Keenan (United States), O. Ludka (Czech Republic)

Summary
The shift from nasal to oral breathing is physiologically disadvantageous leading to an unstable breathing pattern during sleep. Breathing re-education to help restore nasal breathing and normalise breathing volume may have therapeutic applications in sleep disordered breathing. During this workshop, Patrick McKeown will detail breathing exercises to decongest the nose, switch to nasal breathing and improve breathing patterns. Practice each exercise and explore the relationship between rhinitis, asthma and obstructive sleep apnoea.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Discuss basic cognitive and behavioral aspects of breathing
- Understand these principles sufficiently to integrate concepts into patient support
- Appreciate the role of breathing re education as a powerful adjunctive treatment to CPAP and Surgery

Speakers:
P. McKeown (United Kingdom)

K09 Chronic insomnia and the immune response

M.R. Irwin (United States)
Keynote 3:30pm - 4:15pm I Congress Hall

Insomnia is considered a public health epidemic, which contributes to increased risk of inflammatory disorders and all-cause mortality. Dr. Irwin will provide an integrated understanding of the reciprocal relationships between sleep and inflammation; present innovative findings on impact of sleep on the regulation of genomic, cellular, and systemic markers of inflammation, as well as molecular processes of cellular aging; and demonstrate the robust efficacy of insomnia treatment to reverse inflammatory activation in humans.

S64 Understanding the pathophysiology of RBD and REM sleep

3:30pm - 5:00pm I Meeting Hall IV

Chairs:
R. Ferri (Italy)

Summary

The session will cover a wide range of aspects of REM sleep mechanisms and their changes in RBD. Starting from animal models, recent advancements in the understanding of these mechanisms are now being confirmed in humans by means of advanced imaging and neurophysiological techniques. This enhanced knowledge might lead to a better understanding of the mechanisms of action of the current therapy of RBD and provide hints for developments in this field. Dr. Peever will present "REM sleep circuitry abnormalities in RBD", covering basic research outlining how forced degeneration of REM sleep circuits in the brainstem lead to RBD symptoms in animal models. His presentation not only underscores basic mechanisms of REM sleep control but will also relate to RBD pathogenesis. Dr. Arnaldi will present "Brain neuroimaging abnormalities in patients with RBD"; these techniques allow in vivo investigation of the neuronal system involved in RBD. Several structural and functional brain neuroimaging studies have been conducted in RBD patients that, in particular, have investigated the relationship between RBD and neurodegenerative disease. Understanding the most recent findings on brain neuroimaging in RBD lead to a better knowledge of human pathophysiology of RBD and its relationship with neurodegenerative disease. Dr. Jennum will present "Electromyographic correlates in RBD" describing data about the relationship between EMG activity during REM sleep and the nigrostriatal dopamine system, evaluated by SPECT, in RBD and dopamine therapy in PD. Finally, Dr. Ferri will present "Spectral EEG changes during REM sleep in RBD and their modifications by clonazepam", reporting on recently found subtle but significant alterations in REM sleep EEG structure and stability, partially reverted by clonazepam therapy, that might represent the early expression of neurodegenerative processes and be the target of better and effective therapeutic strategies. Taken all together, the data presented in the session constitute a complex of knowledge for a better understanding of the pathophysiology of RBD and might indicate the target of the treatment of RBD symptoms, in the absence of an effective neuroprotection. Taken all together, the data presented in the session constitute a complex of knowledge for a better understanding of the pathophysiology of RBD and might be the target of the treatment of RBD symptoms, in the absence of an effective neuroprotection. The Session will provide the most recent understanding of the pathophysiology of RBD and of the mechanisms involved in its disordered REM sleep. The session will be of major interest for both sleep clinicians and for the basic researchers because it will try to understand the link between the current treatment and its effects on REM sleep, providing hints for the development of new strategies.

Learning Objectives

Upon completion of this CME activity, participants should be able to:
- Review the neurophysiological and neurochemical mechanisms of REM sleep and its changes in RBD
- Appraise recent studies on brain imaging and advanced clinical neurophysiology
- Evaluate the current and future treatment implications of RBD symptoms

Target Audience

Basic scientists, Sleep clinicians, Neurologists

3:30pm - 3:35pm	**Introduction** *R. Ferri (Italy)*
3:35pm - 3:55pm	**REM sleep circuitry abnormalities in RBD** *J. Peever (Canada)*
3:55pm - 4:15pm	**Brain neuroimaging abnormalities in patients with RBD** *D. Arnaldi (Italy)*
4:15pm - 4:35pm	**Electromyographic correlates in RBD** *P. Jennum (Denmark*
4:35pm - 4:55pm	**Spectral EEG changes during REM sleep in RBD and their modifications by clonazepam** *R. Ferri (Italy)*
4:55pm - 5:00pm	**Question and answer** *R. Ferri (Italy)*

S65 Sleep-related hypermotor epilepsy (SHE): From the basic mechanisms to the differential diagnosis

3:30pm - 5:00pm I Meeting Hall V

Chairs:
L. Nobili (Italy), F. Provini (Italy)

Summary
Nocturnal frontal lobe epilepsy is of considerable interest to practicing neurologists because of complexity in differential diagnosis from more common, benign sleep disorders such as parasomnias. To improve the definition of the disorder and establish diagnostic criteria with levels of certainty, a consensus conference using formal recommended methodology was held in Bologna in September 2014. It was recommended that the name be changed to sleep-related hypermotor epilepsy (SHE), reflecting evidence that the attacks are associated with sleep rather than time of day, the seizures may arise from extrafrontal sites, and the motor aspects of the seizures are characteristic. The etiology may be genetic or due to structural pathology, but in most cases remains unknown. Diagnostic criteria were developed with 3 levels of certainty: witnessed (possible) SHE, video-documented (clinical) SHE, and video-EEG-documented (confirmed) SHE.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Diagnose SHE
- Integrate electroclinical data and the spectrum of etiologies involved
- Differentiate SHE from NREM Parasomnias

3:30pm - 3:35pm	**Introduction** *F. Provini (Italy)*
3:35pm - 3:55pm	**Physiopathological mechanisms of sleep-related seizures** *L. Nobili (Italy)*
3:55pm - 4:15pm	**From nocturnal paroxysmal dystonia to SHE** *F. Provini (Italy)*
4:15pm - 4:35pm	**Semeiological and anatomoclinical correlation of sleep-related hypermotor seizures** *P. Kahane (France)*
4:35pm - 4:55pm	**SHE and nrem parasomnias: The key features for the differential diagnosis** *C. Derry (United Kingdom)*
4:55pm - 5:00pm	**Question and answer** *L. Nobili (Italy)*

S66 Deficient sleep in children and adolescents: Generating solutions for a global epidemic

3:30pm - 5:00pm I North Hall

Chairs:
J. Owens (United States)

Summary
Deficient sleep in children and adolescents, defined as both chronic sleep loss and misalignment of circadian rhythms with societally-determined sleep-wake patterns, has become a global epidemic and a public health issue. Insufficient sleep is associated with an array of negative impacts on physical and mental health, behavior, safety, and performance including long-term cardiovascular and metabolic sequelae such as obesity and type 2 diabetes, increased risk of depression and suicidal ideation, higher rates of risk-taking behaviors such as substance and alcohol use, increased risk of drowsy driving and sports-related injuries, academic failure and decreased quality of life. There is a myriad of internal and external factors contributing to deficient sleep among young people, including biological and environmental factors such as the circadian-based shift in sleep onset-offset times occurring in conjunction with puberty, the melatonin-suppressing effects of evening electronic media screen exposure, academic demands, extracurricular activities, and early school start times which conflict with circadian biology. This dilemma has led to increased efforts to both identify modifiable factors contributing to the global epidemic of deficient sleep in children and adolescents and to design the most effective ways to address it. This symposium will include an overview of the genesis of insufficient sleep in the pediatric population and then will specifically focus on some of the more innovative strategies currently being developed that are potentially applicable across a wide variety of audiences and cultures. These include school-based interventions for elementary grade students, which are based on the concept that both knowledge acquisition regarding healthy sleep habits and support and motivation for behavioral change are critical to success; individual-level solutions that incorporate new technologies to encourage adoption of healthy sleep behaviors in adolescents; and "operational" strategies such as school start times which allow for appropriate amount and timing of sleep in middle and high school students.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Define contributing factors to the global epidemic of deficient sleep in children and adolescents
- Describe key components of school-based sleep health programs for elementary and middle school students
- Outline innovative technologies to deliver sleep health messages to adolescents
- Review the evidence supporting healthy school start times as a strategy to improve sleep in middle and high school students

Target Audience
Sleep researchers interested in developing and/or implementing evidence-based strategies to improve sleep health in children and adolescents, sleep health advocates and educators and policy makers interested in sleep and health

3:30pm - 3:33pm	**Introduction** *J. Owens (United States)*
3:33pm - 3:50pm	**Overview of a global epidemic** *Y.K. Wing (Hong Kong)*
3:50pm - 4:07pm	**School-based sleep education in Canada** *R. Gruber (Canada)*
4:07pm - 4:24pm	**Applied technology for delivering sleep health messages** *M. Quante (Germany)*
4:24pm - 4:41pm	**School-based educational interventions in the UK** *G. Illingworth (United Kingdom)*
4:41pm - 4:58pm	**Healthy school start times: A US perspective** *J. Owens (United States)*
4:58pm - 5:00pm	**Question and answer** *J. Owens (United States)*

S67 Respiratory muscle function and intervention of upper airway in patients with sleep disordered breathing

3:30pm - 5:00pm I Club A and B

Chairs:
Y.-M. Luo (China)

Summary
Obstructive sleep apnea is characterized with repeated upper airway collapse which is related to weakness of upper airway dilator muscle or imbalance between upper airway dilator muscle activity and collapsing forces generated by inspiratory muscle activity. Assessment of respiratory muscle function including recording neural respiratory drive help us further understand the physiological mechanism of sleep disordered breathing. This symposium will begin with an introduction of assessment of respiratory muscle including upper airway dilating muscle function. Neural respiratory drive in normal subjects, patients with OSA and overlap syndrome will then be discussed by Professor Yuan Ming Luo based on his recent publication (Thorax. 2016 Nov 2. pii: thoraxjnl-2016-208467 [Epub ahead of print]). An intervention of respiratory muscle function both by invasive transvenous nerve stimulation and noinvasive transcutaneous electrical stimulation would then be demonstrated by Professor Xilong Zhang and Joerg Steier. Finally, predicting the need for noinvasive ventilation in motor neuron disease would be introduced by professor MI Polkey based on his paper recently published in Am J Respir Crit Care Med. 2016. All the speakers listed in this symposium have published many papers in this area and are expert in respiratory muscle and OSA.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the interaction between respiratory pump muscles and upper airway muscles in patients with sleep disordered breathing
- Review treatment effect of electrical stimulation of upper airway muscle on sleep disordered breathing
- Identify sleep disordered breathing in motor neuron disease

Target Audience
Doctors and scientist who are interested in sleep medicine and respiratory medicine

3:30pm - 3:33pm	**Introduction** *Y.-M. Luo (China)*
3:33pm - 3:50pm	**Respiratory muscle reflex responses to the onset of flow limitation in obstructive sleep apnoea** *P. Catcheside (Australia)*
3:50pm - 4:07pm	**Neural respiratory drive in patients with COPD, OSA and overlap syndrome** *Y.-M. Luo (China)*
4:07pm - 4:24pm	**Principle, feasibility and efficacy of transvenous nerve stimulation for treatment of sleep apnea** *X. Zhang (China)*
4:24pm - 4:41pm	**Transcutaneous electrical stimulation of the upper airway dilator muscles in patients with obstructive sleep apnoea** *J. Steier (United Kingdom)*
4:41pm - 4:58pm	**Predicting the need for NIV in ALS** *M. Polkey (United Kingdom)*
4:58pm - 5:00pm	**Question and answer** *Y.-M. Luo (China)*

S68 Sleep disorders in post-menopausal women: The impact on health

3:30pm - 5:00pm I Meeting Hall 1A

Chairs:
R. Silvestri (Italy)

Summary
Aging and hormonal changes during menopause deeply affect metabolism, cardiovascular and mental health of middle age elderly women. Hormonal replacement therapy, adequate life and exercise schedules, a correct diet with necessary supplementations and weight restriction measures may prevent or help treatment of secondary sleep problems, improving life quality in women of this age group. The hormonal metabolic effects of this age changes on sleep apnea and cardiovascular health will be addressed in term of diagnostic and therapeutic strategies. The impact of sleep deprivation due to insomnia, organic disease or altered circadian rhythms on cognition and mood in elderly women will be illustrated and discussed in terms of the most recent evidence linking Alzheimer disease and neurodegeneration to an impaired function of sleep. RLS/WED in elderly women may often be underdiagnosed. Specific clues and clinical interview suggestions will be proposed for symptoms recognition in postmenopausal RLS. In addition, treatment options and cardiovascular prevention in this age group will be addressed.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the importance of good sleep in the post-menopausal condition to allow an adequate quality of life and prevent physical and mental deterioration
- Review preventive strategies to prevent post-menopausal changes leading to sleep disorders and chronic sleep deprivation
- Recognize diagnose and adequately treat post-menopausal sleep disorders affecting life quality in women

Target Audience
Sleep specialists, gynecologists, endocrinologists, pneumologists, cardiologists, movement disorders specialists, and all practitioners with interests in gender specific sleep disorders

3:30pm - 3:35pm	**Introduction** *R. Silvestri (Italy)*
3:35pm - 3:55pm	**Treatment of menopausal insomnia** *P. Polo-Kantola (Finland)*
3:55pm - 4:15pm	**Effects of the postmenopausal cardio-respiratory changes on woman health and metabolism** *M. Bonsignore (Italy)*
4:15pm - 4:35pm	**Sleep rhythms alterations in elderly women and their impact on cognitive decline** *B.M. Guarneri (Italy)*
4:35pm - 4:55pm	**The impact of RLS on women cardiovascular health and quality of life** *M. Manconi (Switzerland)*
4:55pm - 5:00pm	**Question and answer** *R. Silvestri (Italy)*

S69 Effects of sleep deprivation: Novel agents and mechanisms

3:30pm - 5:00pm I Meeting Hall 1B

Chairs:
T. Porkka-Heiskanen (Finland)

Summary
Both epidemiological and experimental research has shown that loss of sleep has substantial adverse effects on human health. Efforts to clarify the potential mechanisms by which insufficient sleep have included research both on animal models and human subjects. Although considerable advances have been made in understanding the complexity of body's response to sleep loss, the key mechanisms, and as recently has turned out, also the potential key players in this response, remain unclear. At brain level, in addition to neurons, both astrocytes and microglia respond to sleep and loss of sleep. At the level of the rest of the body, metabolic changes have been characterized using new and efficient analysis methods and very recently, also the role of the gut flora, the microbiome, has been shown to react depending on the sleep/wake state. The present symposium will introduce four previously either completely unknown or only rudimentary characterized new players in response to loss of sleep. The contents of the symposium consists of a substantial amount of previously unpublished data, and has been collected from both animal and human studies. Dr. Michele Bellesi will talk about the role of astrocytes in shaping cortical synapses when exposed to sleep deprivation. Recent studies showed that astrocytes, as well as microglia, display phagocytic activity, being capable of engulfing synapses. Using a combination of molecular biology and tri-dimensional electron microscopy, Bellesi and colleagues show that a few hours of sleep deprivation trigger astrocytic phagocytosis of synaptic elements through the activation of the Mertk receptor on astrocytes. This process affects ~ 8% of cortical synapses (layer II/III) and mainly large synapses, and is accompanied by microglia activation, if sleep loss is further prolonged. Thus, sleep loss may promote structural remodelling of synapses via astrocytic phagocytosis. Dr. Henna-Kaisa Wigren will clarify the role of microglia in response to sleep deprivation. Recent technological advances have changed the view of microglia as a mere immune response cell of the brain. In addition to immune surveillance, highly motile, "resting", microglia participate in many physiological maintenance functions. In order to clarify the role of microglias response to sleep deprivation, mice with genetically marked microglial cells has been sleep deprived for 3-9 h. The measurements consist of polysomnography, in vivo microdialysis, in vivo 2-photon fluorescent microscopy, immunohistochemistry, fluorescence associated cell sorting, quantitative RT-PCR and 3D-electron microscopy. Dr. Kenneth Wright will discuss how sleep loss influences the microbiome in humans. Dr. Debra Skene will introduce the effects of sleep loss on the human metabolome. Using controlled sleep and circadian protocols, the effect of sleep deprivation on human plasma metabolite profiles has been characterised using untargeted and targeted liquid chromatography/mass spectrometry metabolomics. Metabolic profiling during sleep and sleep deprivation and characterization of the diurnal and circadian metabolite rhythms provides a novel view of human sleep/wake regulation.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the latest progress in experimentation on sleep deprivation
- Evaluate the latest results from sleep deprivation experiments
- Use the information when constructing new experiments with novel goals and methods
- Evaluate the potential use of the new results in clinical work

Target Audience
Sleep researchers, students, clinicians with interest in novel findings in sleep research

3:30pm - 3:35pm	**Introduction** *T. Porkka-Heiskanen (Finland)*
3:35pm - 3:55pm	**Astrocytes mediate the reshaping of cortical synapses in response to sleep loss** *M. Bellesi (Italy)*
3:55pm - 4:15pm	**Microglia and sleep: The effects of sleep deprivation on microglial function** *H.-K. Wigren (Finland)*
4:15pm - 4:35pm	**Sleep loss and the microbiome** *K. Wright (United States)*
4:35pm - 4:55pm	**Sleep and circadian metabolomics: Effect of sleep deprivation** *D. Skene (United Kingdom)*
4:55pm - 5:00pm	**Question and answer** *T. Porkka-Heiskanen (Finland)*

O11 Sleep breathing disorders oral abstract presentations

3:30pm – 5:00pm I Club D and E

Chairs:
L. Pham (United States), R. Riha (France)

3:30pm – 3:45pm	**REAL-TIME IDENTIFICATION OF UPPER AIRWAY OCCLUSION USING ELECTRICAL IMPEDANCE TOMOGRAPHY IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA** *S.-W. Kim (Republic of Korea)*
3:45pm – 4:00pm	**VALIDATION OF THREE-DIMENSIONAL AIRWAY IMAGING FOR SCREENING FOR SLEEP APNEA IN PEDIATRIC PATIENTS** *A. Masoud (Saudi Arabia)*
4:00pm – 4:15pm	**DIFFERENCES IN THE DURATION OF OBSTRUCTIVE SLEEP APNEA EVENTS AMONG HIGHLAND TIBETANS AND HANS AND LOWLAND HANS AT LOW ALTITUDE** *L. Tan (China)*
4:15pm – 4:30pm	**PREDICTING RESPONSE TO OXYGEN THERAPY IN OBSTRUCTIVE SLEEP APNEA PATIENTS USING VENTILATORY CHEMOREFLEX TEST DURING WAKEFULNESS** *D. Wang (Australia)*
4:30pm – 4:45pm	**CLINICAL VALIDATION OF A DIAGNOSTIC PATCH FOR THE DETECTION OF SLEEP APNEA** *M. Merchant (United States)*
4:45pm – 5:00pm	**RISK FACTORS FOR RESPIRATORY COMPLICATIONS AFTER ADENOTONSILLECTOMY IN OSA CHILDREN** *S. Weber (Brazil)*

O12 Behavior, cognition and dreaming and neurological sleep disorders affecting sleep oral abstract presentations

3:30pm – 5:00pm I Terrace 1

Chairs:
R. Salas (United States)

3:30pm – 3:45pm	**THE EFFECT OF CHRONIC SLEEP RESTRICTION AND PRIOR SLEEP DURATION ON SLEEP INERTIA MEASURED USING COGNITIVE PERFORMANCE** *E. Klerman (United States)*
3:45pm – 4:00pm	**SIMULTANEOUS EEG-FMRI REVEALS SPINDLE-RELATED NEURAL CORRELATES OF HUMAN INTELLECTUAL ABILITIES DURING NREM SLEEP** *S. Fogel (Canada)*
4:00pm – 4:15pm	**SLEEP SPINDLE ACTIVITY SIGNIFICANTLY CORRELATES WITH IMPLICIT STATISTICAL LEARNING CONSOLIDATION IN OBSTRUCTIVE SLEEP APNEA PATIENTS** *D. Stevens (Australia)*
4:15pm – 4:30pm	**AGE-DEPENDENT EFFECTS OF SLEEP DEPRIVATION ON TASK PERFORMANCE AND MIND WANDERING** *J. Schwarz (Sweden)*
4:30pm – 4:45pm	**EMOTIONAL WORKING MEMORY IN OLDER ADULTS AFTER TOTAL SLEEP DEPRIVATION** *A. Gerhardsson (Sweden)*
4:45pm – 5:00pm	**IMPACT OF MILD COGNITIVE IMPAIRMENT ON DREAM MENTATION IN PARKINSON'S DISEASE** *M. Roussel (France)*

O13 Basic research oral abstract presentations

3:30pm – 5:00pm I Club H

Chairs:
P.-H. Luppi (France), H. Mallick (India)

3:30pm – 3:45pm	**ON THE EFFECTS OF TWO VERSIONS OF SLOW WAVE SLEEP DEPRIVATION IN THE RELATION TO REM SLEEP** *L. Maisuradze (Georgia)*
3:45pm – 4:00pm	**EEG SIGNATURES OF BRAIN MATURATION IN CHILDREN: AGE-RELATED AND ACROSS-NIGHT DYNAMICS IN SPATIAL PROPAGATION OF SLOW OSCILLATIONS** *S. Kurth (Switzerland)*
4:00pm – 4:15pm	**QUANTIFICATION OF CHANGES IN GLUTAMATE LEVELS IN HEALTHY YOUNG ADULTS ACROSS THE SLEEP WAKE CYCLE USING PROTON MAGNETIC RESONANCE SPECTROSCOPY** *C. Volk (Switzerland)*
4:15pm – 4:30pm	**THE EFFECTS OF ENERGY SUBSTITUTION DURING SLEEP DEPRIVATION ON THE FOLLOWING REBOUND SLEEP** *Z. Lelkes (Hungary)*
4:30pm – 4:45pm	**MOLECULAR PROFILING OF THE LATERAL HYPOTHALAMIC NEURONS IDENTIFIED MOLECULES FOR DISEASE AND DEVELOPMENT** *A. Seifinejad (Switzerland)*
4:45pm – 5:00pm	**SLEEP RESTRICTION INCREASES TELOMERE LENGTH IN SKIN OF RATS** *R. Gimenes Albuquerque (Brazil)*

K10 Treating sleep and circadian problems: A transdiagnostic approach

A. Harvey (United States)
Keynote 4:15pm - 5:00pm I Congress Hall

Past treatments for sleep and circadian disturbances have been disorder-focused—they have treated a specific sleep problem (e.g., insomnia) in a specific diagnostic group (e.g., depression). However, real life sleep and circadian problems are often not so neatly categorized: Features of insomnia commonly overlap with features of hypersomnia, delayed sleep phase and irregular sleep-wake schedules. To address this complexity, the process of developing and testing the Transdiagnostic Intervention for Sleep and Circadian Dysfunction (TranS-C) will be described. TranS-C is transdiagnostic in two ways; it treats a range of the most common sleep and circadian problems across mental disorders.

S71 Sleep, clocks and neurodegeneration

5:30pm - 7:00pm I Meeting Hall IV

Chairs:
A. Videnovic (United States)

Summary
The regulation of sleep and wake states is a major function of the nervous system and has a profound influence on its activity in health and in disease. The propensity for sleep and wake are regulated by a complex interaction of the sleep homeostatic and circadian clock systems. Sleep and circadian timing are essential in modulating neural function. Recent landmark advances in our understanding of the neurocircuitry and molecular mechanisms underlying the generation of sleep and circadian rhythms have led to improved understanding of their role in the expression and treatment of neurological disorders. This program will focus on the bi-directional relationship between circadian and sleep dysregulation and neurodegenerative disorders including Alzheimer's, Parkinson's and Huntington's Diseases.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the impact of sleep and circadian homeostasis on overall neurological health.
- Describe the role of sleep and circadian rhythmicity in the risk and clinical course of Alzheimer's disease
- Recall how circadian dysregulation and impaired sleep-wake cycle impact Parkinson's and Huntington's diseases
- Recognize strategies to improve circadian function in neurodegenerative disorders

Target Audience
Practitioners, academicians, residents, postdoctoral fellow, allied health professionals

5:30pm - 5:35pm	**Introduction** *A. Videnovic (United States)*
5:35pm - 5:55pm	**Sleep and circadian homeostasis – implications for brain health** *P. Zee (United States)*
5:55pm - 6:15pm	**Sleep, circadian clocks and alzheimer's disease** *E. Musiek (United States)*
6:15pm - 6:35pm	**Circadian and sleep dysregulation in Parkinson's disease** *A. Videnovic (United States)*
6:35pm - 6:55pm	**Disruption of sleep and circadian timing in huntington's disease** *J. Morton (United Kingdom)*
6:55pm - 7:00pm	**Question and answer** *A. Videnovic (United States)*

S72 The duality of sleep movement

5:30pm - 7:00pm I Meeting Hall V

Chairs:
J.J. Askenasy (Israel)

Summary
Sleep has a strange dual effect on muscle activity. Dual effect means that movement may be inhibited or generated. There is still no explanation for this dual effect of sleep. The inhibition of a permanent Parkinsonian tremor, or of permanent choreo-athetotic movements, is an almost classic well-known phenomenon. Its clinical and experimental facets in Parkinson's disease will be analyzed. The classic sleep generated movements include Periodic Leg Movement and Restless Leg Syndrome. Bruxism will be briefly reviewed. The duality phenomenon has clinical, anatomo-histologic, epidemiologic, age distribution, and molecular aspects which will be emphasized by the symposium. Motor control and dyscontrol triggering jerks and shakes during sleep will be also reviewed. Attention will be directed to "sleep-related muscle contractions with pain," which is beginning to be framed as a new entity. Arousal, excitation and inhibition related to movement during sleep will be presented as a concept that may explain the dual effect of sleep on muscle movement.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Interpret the dual effect of sleep on movement
- Recobnize a strategic frame, the dual effect of sleep on movement
- Integrate in a strategic frame, the dual effect of sleep on movement

Target Audience
Sleep medicine researchers, neurologists and psychiatrists

5:30pm - 5:32pm	**Introduction** *J.J. Askenasy (Israel)*
5:32pm - 5:49pm	**The dual phenomenon** *J.J. Askenasy (Israel)*
5:49pm - 6:06pm	**Motor control and dyscontrol in sleep** *S. Chokroverty (United States)*
6:06pm - 6:23pm	**Sleep in Parkinson's disease: Updates from animal experiments** *S. Nishino (United States)*
6:23pm - 6:40pm	**Sleep-related muscle contractions with pain** *T. Mano (Japan)*
6:40pm - 6:57pm	**Arousal, excitation and inhibition in sleep, related to movements a new concept** *R. Allen (United States)*
6:57pm - 7:00pm	**Question and answer** *J.J. Askenasy (Israel)*

S73 Practical aspect of pediatric sleep medicine

5:30pm - 7:00pm I North Hall

Chairs:
N. Simakajornboon (United States)

Summary
The significant progress that has been achieved worldwide in the past 50 years with respect to the understanding of the pathogenic mechanisms and consequences of sleep disorders, especially obstructive sleep apnea syndrome (OSAS) in adults, subsequently led to the development of pediatric sleep medicine. This has been made by opening sleep labs dedicated to children, providing additional specialization to physicians, as well as by conducting studies whose results have been published as a part of the effort to understand the characteristics of different age groups. Obstructive sleep apnea syndrome (OSAS) in children and adults are different entities and therefore need a different approach. Since children and adolescents are "developing" individuals requiring more sleep than adults, OSA events may not only adversely affect daytime performance, but also may have long-term consequences. The same holds true for other sleep disorders. Despite of recent international advances in research and clinical practice, there are countries where pediatric sleep medicine is still underprivileged and underrepresented. Recent prevalence data for these countries, however, reflect the importance of this issue. They are justifying the efforts to increase public awareness and training of physicians, as well as the need of financial support to establish a network for proper diagnosis, monitoring and treatment of pediatric sleep apnea syndrome and other sleep-related disorders. This symposium will provide an updated view of some key features of sleep disordered breathing in children and adolescents. Novel findings related to obesity in children and the influence on SRDB are equally presented as the challenge of CPAP treatment and compliance. Decision-making for GH therapy in children with Prader-Willi syndrome with respect to the respiratory risk and the efforts of an Eastern European country to introduce pediatric sleep medicine and to achieve a modern international standard are other topics of this symposium. Altogether, the symposium tries to demonstrate how important pediatric sleep medicine is for the outcome of affected patients.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Summarize the underlying pathophysiological mechanisms of obstructive sleep apnea in children and assimilate the increasing role of obesity coupled with improved approaches towards diagnosis and management
- Examine the role of CPAP therapy and compliance/adherence in pediatric patients
- Analyze and develop a systematic approach to the evaluation of GH treatment in children with Prader Willi syndrome with respect to polysomnografic evaluation
- Develop a plan for set-up a pediatric sleep lab and increase awareness about pediatric sleep disorders breathing in health professionals and in the community
- Recognize the unique aspects of pediatric sleep pathology and the importance of proper diagnosis and therapeutic multidisciplinary approach as a preventive measure for developing individuals

Target Audience
Primary care physicians, pediatricians, psychologists, respiratory therapists, sleep medicine technicians and nurses; Practicing Specialists in sleep medicine, pulmonary/critical care medicine, otolaryngology, neurology, psychiatry, cardiology, oral & maxillofacial surgery and dentistry

5:30pm - 5:32pm	**Introduction** *N. Simakajornboon (United States)*
5:32pm - 5:49pm	**Sleep apnea and GH therapy in Prader Willi syndrome** *A. Salvatoni (Italy)*
5:49pm - 6:06pm	**Obesity and OSA in children** *H. Sawnani (United States)*
6:06pm - 6:23pm	**CPAP initiation and adherence in children with OSA** *N. Simakajornboon (United States)*
6:23pm - 6:40pm	**The challenge of opening a pediatric sleep clinic in an Eastern European country** *M. Oros (Romania)*
6:40pm - 6:57pm	**Headache related to sleep disordered breathing in children with neuromuscular disease (case report)** *A. Lupusor (Republic of Moldova)*
6:57pm - 7:00pm	**Question and answer** *N. Simakajornboon (United States)*

S74 Extracting sleep breathing phenotypes from lab and home data

5:30pm - 7:00pm I Club A and B

Chairs:

R.J. Thomas (United States)

Summary

This symposium has a focus on an integrated multi-component approach to phenotyping sleep apnea and associated sleep alterations beyond traditional polysomnographic methods. The apnea-hypopnea index is the result of distinct interacting biological processes, all of which can contribute to the severity of clinical sleep apnea individually and collectively. These are high loop gain, low arousal threshold, air way collapsibility, and reduced negative pressure reflex response. It would be useful in clinical practice if there were features on the conventional PSG or metrics computed from PSG signals which differentiated phenotypes that could guide therapy. Specifically, a high loop gain phenotype may benefit from supplemental oxygen, acetazolamide, or hypocapnia minimization strategies, while sedatives could be an option in those who have low arousal thresholds in NREM sleep. Clinical and biomarker data are able to categorize sleep apnea phenotypes beyond that of the obese male. Clinical, laboratory and home data provide signal and visual data rich environments for sophisticated analysis. The classic multiple morphological categories include obstructive, central, and periodic breathing/Cheyne-Stokes respiration types. Respiratory events may be dominant in NREM or REM sleep, and at times be equally severe in both states. These events can be short, as at high altitude (25 seconds or less), or long, as in congestive heart failure (often over 60 seconds). NREM stages are further characterized into grades, N1 through N3 but alternate methods of characterizing sleep include using autonomic and vascular reactivity analysis, cyclic alternating pattern (CAP) of NREM sleep, and cardiopulmonary coupling (high, low and very low frequency coupling of autonomic and respiratory drives, modulated by cortical delta power). Sleep type and stage interact with apnea physiology to generate phenotypes. Most importantly, multiple phenotypes open the avenue for pharmacological targeting of sleep apnea. The data presented will integrate advanced analysis of signals across multiple dimensions across methods (including wearable devices), therapy types (continuous and adaptive positive pressure, home ventilation), phenotypes (obstructive, central, hypoventilation) and environments (laboratory, ambulatory), enabling articulation of a comprehensive integrated approach to clinically useful phenotypes in sleep apnea. Experts in sleep apnea phenotypes will present a synthesis of relevant data including previously unpublished current work.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Describe clinical and biomarker phenotypes in sleep apnea beyond the caricature of the obese sleepy male
- Distinguish sleep apnea phenotypes beyond the apnea-hypopnea index
- Evaluate pharmacological targeting of sleep apnea phenotypes
- Diagnose high loop gain and patient-ventilator desynchrony from home therapy positive pressure device signals
- Integrate a comprehensive set of signals across multiple dimensions to establish clinically useful and pathophysiologically distinct sleep apnea phenotypes

Target Audience

Sleep medicine clinicians, researchers, and developers of sleep medicine technology

5:30pm - 5:35pm	**Introduction** *R.J. Thomas (United States)*
5:35pm - 5:55pm	**More than airway closure: Pathophysiological, polysomnographic and clinical phenotypes of OSA** *W. Randerath (Germany)*
5:55pm - 6:15pm	**Pharmacotherapeutic options in relation to various proposed phenotypes** *J. Hedner (Sweden)*
6:15pm - 6:35pm	**Beyond the male obese sleepy patient: Clinical phenotypes and biomarkers in obstructive sleep apnoea** *D. Pevernagie (Belgium)*
6:35pm - 6:55pm	**Success and failure biomarkers during home apnea therapy** *R.J. Thomas (United States)*
6:55pm - 7:00pm	**Question and answer** *R.J. Thomas (United States)*

S75 Sleep and mental health in a changing society

5:30pm - 7:00pm I Meeting Hall 1A

Chairs:

N. Glozier (Australia)

Summary

This symposium provides up to date evidence about the how changing trends in our environment, specifically the workplace, neighborhood and technology affect our sleep and mental health integrating evidence from different contexts Sleep is a vital component of our physical and mental wellbeing. Rapid technological and social changes have been implicated in changing sleep patterns and their sequelae. We will present new data arising from prospective studies demonstrating the patterns of association between experimental typologies of sleep disturbance and mental illness and the, impact of modern workplaces and environment on sleep. In particular the four proposed talks will sum current evidence on the topic, highlighting the use of different study designs and novel methodology, such as latent class analyses, and analyzing observational data as non-randomized pseudo-trials. This will provide innovate and potentially challenging ways to improve population sleep health. First talk covers how sleep disturbances in the Swiss general population are tied to mental health problems. Based on different epidemiological samples, empirical patterns of sleep problems were explored and associations with clinical variables were examined. Then, based on a prospective study design the temporal development of sleep problems over a 30 year period as well as time-varying covariates were explored. The comparison between cross-sectional and longitudinal data aims to address whether sleep problems and related factors vary over the individual's life course (i.e. a cohort effect) or are an effect of changing demands and environmental conditions over the decades. Second talk sums key findings on the associations between working conditions and poor sleep. Main emphasis is on new studies using observational data as pseudo-trials, showing how changes in psychosocial working conditions can have both favorable and unfavorable effects on sleep. Third and the fourth talks present the evidence linking noise to sleep disturbances. The third talk will address the environmental impact of noise on sleep. The presentation will provide an overview of the public health approach and burden of illness estimations related to noise and sleep, in addition to recent data from research on the topic. In the fourth talk, we will discuss the social neighborhood determinants of insufficient sleep and poor sleep quality. In the past ten years, associations between adverse neighborhood exposures and poor health outcomes have been widely documented. Sleep disturbance may represent an important pathway by which neighborhoods influence health. Main emphasis will be looking at the association between the social neighborhood environment and objective measures of sleep in large representative samples. The final talk will explore the changing patterns of technology use and compare the results of ecological temporal trend associations and those from cross sectional and experimental analyses over the past decades.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Recognize how latent class analyses might help elucidate the association of sleep and mental health problems
- Interpret the results from pseudo-trials and distinguish between the favorable and unfavorable changes in psychosocial working conditions and their contribution to sleep
- Appraise the effects of environmental noise on sleep in modern societies
- Appraise the current evidence supporting the impact of changing technology on temporal changes in sleep duration quality and timing

Target Audience

The target audience would be all those interested in the interplay of societal changes and sleep. This would include psychologists, epidemiologists, physicians and policy makers. An additional point of interest is the use of novel methods and study designs to better judge causality and produce more compelling evidence, integrating science from various disciplines and cultural contexts.

5:30pm - 5:32pm	**Introduction** *N. Glozier (Australia)*
5:32pm - 5:49pm	**Sleep disturbance and mental health in the community: Symptom patterns and trajectories** *M. Müller (Switzerland)*
5:49pm - 6:06pm	**Working conditions and sleep - not all bad news** *T. Lallukka (Finland)*
6:06pm - 6:23pm	**Neighborhood and sleep: How bad for you is noise?** *S. Øverland (Norway)*
6:23pm - 6:40pm	**Where we live matters for our sleep: Neighborhood social environment and objective sleep measures** *G. Simonelli (United States)*
6:40pm - 6:57pm	**How has technology affected our sleep over the past few decades?** *N. Glozier (Australia)*
6:57pm - 7:00pm	**Question and answer** *N. Glozier (Australia)*

S76 Sleep, slow waves and brain temperature: Insights from hibernators

5:30pm - 7:00pm I Meeting Hall 1B

Chairs:
R. Amici (Italy)

Summary
Research into the relationship between sleep and hypothermic states like hibernation and daily torpor has a long history. All three are characterized by a reversible reduction in metabolic rate that produces a decrease in body temperature. Research investigating the similarities and differences between hibernation/torpor and sleep showed that deep hibernation and daily torpor are probably not similar to sleep as they are followed by a period of deep sleep with increased EEG slow-wave activity, as if the animals were sleep deprived during torpor. This is extra remarkable as sleep is probably the permissive state that allows torpor and hibernation to be induced. The nature of such sleep bout is still unknown, since it could be interpreted as either a homeostatic effect induced by the lack of sleep during the torpor/hibernation period, or a non-homeostatic reactivation of the cerebral cortex following the hypometabolic period. In the present symposium, in which we managed to bring together the few research groups that are active in this field, we will try to gain insight into this matter by discussing newly obtained data and analysis within this context. First a historical overview on the relationship hypothermic states and slow-wave sleep will be given (De Boer). This will be followed by a presentation and discussion of a new detailed analysis of EEG slow waves during the initial sleep after a bout of daily torpor in comparison to recovery sleep after sleep deprivationin the Djungarian hamster, concentrating on slow-wave morphology (Vyazovskiy). Subsequently data will be presented about the relationship between adenosinergic mechanisms and hypothermia and hibernation (Drew). Finally results will be presented of sleep and EEG data obtained from a non-hibernating rodent brought in hypothermia by pharmacological means (Cerri). In virtue of new advancements in our understanding of how sleep and hypometabolic states are related, and current theories addressing the generation and meaning of slow waves and slow wave sleep, this symposium aims at re-evaluating the data in the scientific literature under the light of these new findings The hypothesis of a reciprocal relationship between sleep and torpor/hibernation that could help unraveling the dynamics and the meaning of both will be explored.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Summarize differences and similarities between hypothermic states (torpor/hibernation) and sleep
- Recognize the mechanisms behind the hibernation, torpor and sleep
- Relate these mechanisms to non-hibernaters
- Question the dichotomy between the hibernation/torpor and sleep

Target Audience
Fundamental sleep researchers interested in sleep mechanisms and function; Physicians interested in hypometabolic states and sleep

5:30pm - 5:35pm	**Introduction** *R. Amici (Italy)*
5:35pm - 5:55pm	**Hypothermic states and their relation to slow wave sleep** *T. de Boer (The Netherlands)*
5:55pm - 6:15pm	**Slow wave morphology during sleep after daily torpor** *V. Vyazovskiy (United Kingdom)*
6:15pm - 6:35pm	**Seasonal rhythm in adenosine A1 receptor signaling in hibernation** *K. Drew (United States)*
6:35pm - 6:55pm	**Slow wave sleep and torpor; insights from a non-hibernator** *M. Cerri (Italy)*
6:55pm - 7:00pm	**Question and answer** *R. Amici (Italy)*

S77 Young Investigator: Sleep research in respiratory sleep medicine

5:30pm - 7:00pm I Club D and E

Chairs:
T. Penzel (Germany), J. Puertas (Spain)

5:30pm – 5:45pm	**WHY IS OBSTRUCTIVE SLEEP APNEA IN PEOPLE WITH RESISTANT HYPERTENSION MISSED SO OFTEN - A CLINICAL AND POLYSOMNOGRAPHIC CASE-CONTROLLED STUDY** *A. Gupta (India)*
5:45pm – 6:00pm	**CARDIAC BIOMARKERS IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA SYNDROME AND HEART FAILURE WITH PRESERVED EJECTION FRACTION** *I. Andreieva (Ukraine)*
6:00pm – 6:15pm	**HYPOCAPNIA HAS MINIMAL INFLUENCE ON GENIOGLOSSUS MUSCLE AFTER-DISCHARGE ELICITED BY AROUSAL FROM SLEEP IN HEALTHY INDIVIDUALS** *J. Cori (Australia)*
6:15pm – 6:30pm	**IMPACT OF INTERMITTENT HYPOXIA ON CARDIOVASCULAR REMODELING IN A MURINE MODEL OF SLEEP APNEA: EFFECT OF AGE** *A.L. Castro-Grattoni (Spain)*
6:30pm – 6:45pm	**DOES MATERNAL SLEEP APNOEA AFFECT CHILDHOOD HEALTH AND EDUCATIONAL OUTCOMES? A LONGITUDINAL STUDY USING POPULATION RECORD LINKAGE** *Y.S. Bin (Australia)*
6:45pm – 7:00pm	**EFFECTS OF MORPHINE ON THE PHENOTYPIC CAUSES OF OBSTRUCTIVE SLEEP APNEA** *R. Tomazini Martins (Australia)*

O14 Aging and excessive daytime sleepiness oral abstract presentations

5:30pm – 7:00pm I Terrace 1

Chairs:
K. Richards (United States)

5:30pm – 5:45pm	**THE EFFECTS OF CANNABIS ON VIGILANCE AND SIMULATED DRIVING** *S. Hartley (France)*
5:45pm – 6:00pm	**REVISED DIAGNOSTIC CRITERIA FOR IDIOPATHIC HYPERSOMNIA: A 32-HOUR BED-REST PROTOCOL** *E. Evangelista (France)*
6:00pm – 6:15pm	**RAPID EYE MOVEMENTS IN REM SLEEP FEATURES AS BIOMARKER OF MATURITY IN HEALTHY INFANTS** *M. Merino-Andreu (Spain)*
6:15pm – 6:30pm	**LESS SWS, REM SLEEP AND MORE WASO ARE ASSOCIATED WITH GREATER DEPRESSION AND POORER TIME-BASED PROSPECTIVE MEMORY IN COMMUNITY-DWELLING OLDER ADULTS** *E. Hodgson (Australia)*
6:30pm – 6:45pm	**ASSOCIATION BETWEEN SLEEP SLOW WAVE ACTIVITY AND BRAIN STRUCTURE DURING ADOLESCENCE** *A. Goldstone (United States)*
6:45pm – 7:00pm	**SLEEP DEPRIVATION MODIFIES THE USUAL BEHAVIOR OF ALPHA OSCILLATORY ACTIVITY DURING COGNITIVE TASKS** *S. Montamat (Switzerland)*

T06 The 3 C's: Credentialing, certification, CECs

5:30pm - 7:00pm I Club H

Chairs:

S. Keenan (United States), O. Ludka (Czech Republic)

Summary

In this workshop, the presenters will discuss the advantages of obtaining various Board of Registered Polysomnographic Technologists credentials and the eligibility requirements for each. The process for recertification will be covered, as well. Lastly, a summary of the various benefits of American Association of Sleep Technologists membership will be delivered to attendees.

Learning Objectives

- Describe the advantages of obtaining the Certified Polysomnographic Technician (CPSGT), Registered Polysomnographic Technologist (RPSGT), and Certification in Clinical Sleep Health (CCSH) credentials through the Board of Registered Polysomnographic Technologists (BRPT).
- Identify the various benefits of being a member of the American Association of Sleep Technologists (AAST).

Speakers:

D. Lane (United States), D. Wolfe (United States)

RS1 Society Symposium ASRS and IASSA: Sleep medicine in Asia: Across the discipline

7:00pm - 9:00pm I Meeting Hall IV

Chairs:

H. Mallick (India), P.V. Krishnan (India)

Summary

This is a regional symposium jointly conducted by Asian Sleep Research Society (ASRS) and Indian Association of Surgeons for Sleep Apnoea (IASSA). The sympsoium covers: (i) Latest translational research in insomnia using Yoga Nidra (Yogic Sleep) as a therapeutic model, which will be the highlight of the symposium presented by a basic young researcher in sleep. (ii) Manjari Tripathi will give an insight view of alternative practices in treating insomnia which will be of general interest. (iii) Hiroshi Kadotoni will speak about the certification system in sleep medicine in Japan. This will be an important example for developing certification programme in sleep medicine in many Asian countries. (iv) As health insurance system in sleep medicine practice is unheard of in many Asian countries. Shintaro Chiba will enlighten the audience on 'Insurance System in Sleep medicine practice in Japan. (v) Vijaya Krishnan as an ENT sleep surgeon will bridge the gap between physicians and surgeons by highlighting his experiences in management of obstructive sleep apnoea in India.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Define Yoga Nidra which has been practiced for thousands of years to produce local sleep
- Recognize the role of Yoga Nidra as an alternate modality in treating insomnia
- Review the certification and insurance system in sleep medicine practice in Japan
- Recognize and debate the role of upper airways surgery in treating obstructive sleep apnoea

Target Audience

Basic researchers, sleep physicians, ENT surgeons, dentists and residents in sleep medicine

7:00pm - 7:10pm	**Introduction** *H. Mallick (India)*
7:10pm - 7:25pm	**Translational research in insomnia using Yoga Nidra: Therapeutic model and results** *K. Datta (India)*
7:25pm - 7:40pm	**Alternative therapies in Insomnia: An Insight** *M. Tripathi (India)*
7:40pm - 7:55pm	**Sleep medicine in Japanese health care insurance system** *S. Chiba (Japan)*
7:55pm - 8:10pm	**Certification system in Japanese sleep medicine** *H. Kadotani (Japan)*
8:10pm - 8:30pm	**Protocol and surgical guidelines formulated by IASSA for OSA patients in Indian scenario** *P.V. Krishnan (India)*
8:30pm - 9:00pm	**Panel discussion**

RS2 Society Symposium ASA: Biomarkers for sleep disordered breathing: Clinical, physiological, neurocognitive and genetic

7:00 - 9:00 I Terrace 1

Chairs:
M. Barnes (Australia)

Summary
Andrew Vakulin will summarise what we know about the clinical and novel biomarkers of alertness failure and driving impairment in OSA. He will discuss research that is being carried out currently that is developing and refining fitness to drive assessments for patients with OSA, in particular the clinical accuracy of roadside testing and implications for occupational health and safety. Danny Eckert will summarise the current research on the pathophysiolgical causes of sleep disordered breathing. Recent developments in the translation of more complex physiological phenotyping approaches to those that can be used in the clinic to inform treatment decisions according to a precision and targetted medicine approach will also be highlighted. Romola Bucks will summarise what we know about the nature and mechanisms of cognitive dysfunction in OSA and the evidence for the impact of treatments. Sutapa Mukherjee will review the role that genes play in OSA with particular emphasis on the latest gene discovery efforts for OSA. Clinicians will become familiar with tools that were previously available only to researchers, understanding that these can now be used to assist in the clinical assessment and management of patients. This enables targetted, personalised therapies. Researchers will learn about the latest developments in the field and also gain valuable insights into the questions and concerns that are of clinical importance in this area.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognise biomarkers for alertness failure and driving impairment in patients
- Assess the differential physiological contribution to sleep disordered breathing inindividual patients.
- Develop personalised treatment approaches for patients with OSA, according to physiological measures.
- Evaluate the neurocognitive impairment in patients with OSA and be able to give patients a realistic expectation of their response to treatment.
- Understand the genetic contribution to OSA and the application in the clinical setting

Target Audience
Clinicians and researchers in the field of respiratory sleep disorders

7:00pm - 7:30pm	**Society Networking: Light food and drinks will be provided**
7:30pm - 7:35pm	**Introduction** *M. Barnes (Australia)*
7:35pm - 7:55pm	**Clinical biomarkers for the assessment of alertness failure and driving performance in patients with OSA** *A. Vakulin (Australia)*
7:55pm - 8:15pm	**Physiological biomarkers of OSA** *D. Eckert (Australia)*
8:15pm - 8:35pm	**Neurocognitive biomarkers in OSA** *R. Bucks (Australia)*
8:35pm - 8:55pm	**Genetic biomarkers in OSA** *S. Mukherjee (Australia)*
8:55pm - 9:00pm	**Question and answer**

RS3 Society Symposium ESRS and WSS: Sleepiness and accidents

7:00pm - 9:00pm I North Hall

Chairs:
C. Kushida (United States), L. Nobili (Italy)

Summary
Lack of alertness in private and commercial transportation is increasingly responsible for a large proportion of fatal accidents. This session will provide attendees with an understanding of how and when sleep-related accidents are likely to occur and strategies to prevent them as effectively as possible.

Learning objectives
Upon completion of this CME activity, attendees should be able to:
- Recognize the association between physiologically-based sleepiness/fatigue and human error-related accidents
- Identify the increased risk of sleepiness related-accidents to OSA patients
- Explain the role of regulations regarding driving for patients with OSA

Target audience
Clinicians and researchers in sleep medicine

7:00pm - 7:30pm	**Society Networking: Light food and drinks will be provided**
7:30pm - 7:35pm	**Introduction** *C. Kushida (United States)*
7:35pm - 7:50pm	**Awareness of sleepiness on the road, in the air, and at sea: Links to physiology and behavior** *T. Akerstedt (Sweden)*
7:50pm - 8:05pm	**Sleep apnea and sleepiness: Do mechanisms matter?** *A. Malhotra (United States)*
8:05pm - 8:20pm	**More than just nodding off: The relationship between sleepiness and accidents** *K. Honn (United States)*
8:20pm - 8:35pm	**OSA and driving risk: The role of regulation** *W. McNicholas (Ireland)*
8:35pm - 8:50pm	**OSA in commercial drivers: An evolving story** *A. Pack (United States)*
8:50pm - 9:00pm	**Question and answer** *L. Nobili (Italy)*

World Sleep Society Membership

Individual Membership is open to public health officials, government representatives, health organization administrators, patient support group members, and the general public as affiliated non-voting members.

Student Membership and special dispensation for individuals from economically underprivileged countries are also available. The membership committee will establish membership categories and fees.

Associate Society Membership is open to Clinical and Scientific Societies with relevance to sleep. To learn more or become a member, go to www.worldsleepsociety.org/join.

Current Associate Society Members of World Sleep Society

* Founding Member | Listed in alphabetical order

Asian Sleep Research Society *
www.asrsonline.org

Australasian Sleep Association *
www.sleep.org.au

Australasian Sleep Technologist Association
sleeptechnologists.org

British Sleep Society
www.sleepsociety.org.uk

Bulgarian Association of Obstructive Sleep Apnea & Snoring
www.sleepapnea.bg

Canadian Sleep Society *
www.css-scs.ca

Czech Sleep Research and Sleep Medicine Society
www.sleep-society.cz

European Sleep Research Society *
www.esrs.eu

Federation of Latin American Sleep Societies *

Finnish Sleep Research Society
www.sus.fi

French Society for Sleep Research and Sleep Medicine
www.sfrms.org

German Sleep Society
www.dgsm.de

Indian Association of Surgeons for Sleep Apnoea
www.iassa.in

Indian Society for Sleep Research
www.issr.in

Integrated Sleep Medicine Society Japan
www.ismsj.org

International Restless Legs Syndrome Study Group
www.irlssg.org

Israel Sleep Society

Japanese Society of Sleep Research
jssr.jp

Peruvian Association of Sleep Medicine (Apemes)
www.apemesperu.com

Romanian Association for Pediatric Sleep Disorders
www.somn-copii.ro

Russian Society of Somnologists
www.sleep.ru

Serbian Sleep Society
www.sss.rs

Sleep and Wakefulness Medicine Moroccan Federation
www.fmmsv.com

Sleep Research Society *
www.sleepresearchsociety.org

Taiwan Society of Sleep Medicine
www.tssm.org.tw

Turkish Sleep Medicine Society
www.tutd.org.tr

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RS4 Society Symposium SRS: Circadian rhythm sleep-wake disorders: Looking to the future

7:00pm - 9:00pm I Meeting Hall V

Chairs:

S. Abbott (United States)

Summary

This session will aim to provide attendees with an understanding of the impacts of circadian and sleep disruption on health. Speakers will present new research on the treatment of circadian rhythm disorders and discuss novel measures recently developed to assess circadian and sleep disruption.

Learning Objectives

Upon completion of this CME activity, particpants should be able to:

- Recognize the basics of circadian rhythm disruption
- Describe the treatment of sleep-wake disorder with melatonin
- Identify circadian disrupton in neurodegenerative disorders
- Recognize meausres and marker of circadian and sleep timing

Target Audience

Clinicians and basic and clinical researchers

7:00pm - 7:30pm	**Society Networking: Light food and drinks will be provided**
7:30pm - 7:40pm	**Introduction** *S. Abbott (United States)*
7:40pm - 8:00pm	**Delayed sleep-wake phase disorder: Functional consequences and management with melatonin** *S. Rajaratnam (Australia)*
8:00pm - 8:20pm	**In search of novel circadian biomarkers** *S. Abbott (United States)*
8:20pm - 8:40pm	**Sleep and circadian dysregulation in neurodegeneration** *A. Videnovic (United States)*
8:40pm - 9:00pm	**Is it the time or the timing: The effect of sleep regularity/irregularity on mood, circadian phase and performance in college students** *E. Klerman (United States)*

K11 H1N1, seasonality and childhood narcolepsy

F. Han (China)

Keynote 8:00am - 9:00am I Congress Hall

Narcolepsy is recognized different across ethnic groups in many aspects including prevalence, predisposing factors and clinical presentations. In a series of 2500 narcolepsy cataplexy patients received over 20 years in a sleep lab in China, narcolepsy symptoms onset is highly correlated with seasonal and annual patterns of upper airway infections. More specially, a large rise in childhood onset cases associated with the pH1N1 outbreak, but independent of vaccination, was noted in China, and the increased incidence returned to previous levels in 2011 till to the end of 2016. This allows further cross-ethnic comparisons, and facilitate our understanding of the neurologic autoimmune mechanisms of narcolepsy.

S78 Sleep at high altitude

9:00am - 10:30am I Meeting Hall IV

Chairs:
J. Ulfberg (Sweden)

Summary
A significant portion of the world's population sleeps at high altitudes and many more travel for short stays at the high altitudes. High altitude appears to affect sleep at least from acute exposure but also, although there is some uncertainty, for those living at high altitudes. There are several significant issues about the effects of hypobaric hypoxia on sleep. The relatively few scientific studies provide important but somewhat conflicting information about status of sleep for those living at higher altitudes. Sleep fragmentation is reported but it is unclear if this results from increased sleep-disordered breathing or from increased problems with restless legs syndrome and periodic limb movements in sleep. All of these effects relate to other significant health issues. The affects with rapid change in altitude appear to be fairly pronounced on breathing in sleep, but also produce increased leg movements and sleep fragmentation that are not clearly related to changes in respiration. The hypobaric hypoxia effects may alter basic sleep regulation producing less stable sleep patterns. There are also indications of gender, age and cultural differences in the effects of both acute/short-term and chronic sleep at higher altitudes. The hypobaric hypoxia effects on sleep have obvious clinical significance both for travelers and for those living at high altitudes. Sleep disruption at high altitudes may increase vulnerability to other major diseases, may disrupt wake time performance and may impact development. The mechanisms producing effects of hypobaric-hypoxia on sleep also inform about the basic biology of major sleep disorders. Sleep-disordered breathing characteristics of sleep at higher altitudes provide insight into the more general issues of biological regulation of breathing during sleep and effects on major health issues, particularly diabetes. Similarly the putative increase in periodic leg movements and in restless legs syndrome at higher altitude indicate biological features producing these. Particularly interesting is the relation between iron management adjustments, hypoxia pathway activation and brain iron status. These factors have been related to development of RLS and presumably also PLMS. This symposium first presents some new work by Dr. Pham on sleep disordered breathing at high altitudes in South America. This interesting work explores the altitude effects on both sleep –disordered breathing and also the related issue of glucose metabolism. This talk is followed by Dr. Hill addressing the important question of children's sleep at higher altitudes. Her data raise questions about defining the breathing patterns in these children during these developmental years noting also issues of age and genetic ancestry. Dr. Gupta presents in the next talk new data on restless legs syndrome in the higher altitudes of the Indian Himalayas. His study explores major factors contributing to RLS, particularly iron status. Finally the symposium will end with Dr. Stefani presenting experimental data on effects of sleeping in a chamber with reduced oxygen. Increased periodic leg movements are noted. These presentations provide the basis for general discussion about sleep at higher altitudes providing both interesting insights into sleep mechanisms and also possible treatment considerations.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Describe the various types of high altitude effects on sleep
- Identify the high altitude effects on breathing during sleep
- Identify the high altitude effects on restless legs syndrome and PLMS
- Recognize impact on health of altitude effects on sleep

Target Audience
Sleep clinicians who treat or advise those living/traveling to high altitudes; Sleep researchers who have interest in hypoxia or iron effects on sleep; Those living at or planning trips to high altitudes

9:00am - 9:05am	**Introduction** *J. Ulfberg (Sweden)*
9:05am - 9:25am	**Sleep, high altitude and glucose metabolism** *L. Pham (United States)*
9:25am - 9:45am	**Sleep and breathing in children resident at high altitude** *C. Hill (United Kingdom)*
9:45am - 10:05am	**Restless legs syndrome and iron status at higher altitudes** *R. Gupta (India)*
10:05am - 10:25am	**Chamber hypoxia effects on sleep and PLMS** *A. Stefani (Austria)*
10:25am - 10:30am	**Question and answer** *J. Ulfberg (Sweden)*

S79 Cerebral networks during sleep and after sleep deprivation

9:00am - 10:30am I Meeting Hall V

Chairs:

J. Carrier (Canada), J.-M. Lina (Canada)

Summary

Over the last decades, the whole-brain modeling in neuroscience studies emphasized the crucial role of functional networks in behavior and cognitive processes. This global perspective of the brain activity, which accounts for the brain mechanisms underlying the segregated activities regrouped in an integrated functional networking, has been recently extended to the sleep state. In the past decade, studies of the changes in cerebral functional connectivity (FC) during Non-Rapid-Eye-Movement (NREM) sleep, combining electroencephalogram (EEG) and functional magnetic resonance imaging (fMRI), provided important insights on sleep mechanisms and functions in humans. For instance, studies demonstrated significant FC modifications during NREM sleep characterized by greater local cortical FC but breakdown of long range cortico-cortical FC with the descent from wakefulness to slow-wave sleep. More recently, innovative methodological approaches (electrophysiology, electrocorticography, fMRI, magnetoencephalography, transcranial magnetic stimulation-EEG) have been used to study the fundamental bases of FC during sleep and after sleep loss. The aim of this symposium is to discuss new advancements in our understanding of how cerebral networks are modulated by sleep and after sleep loss. NREM sleep is commonly considered a global brain process, characterized by a generalized decrease in effective connectivity at a macroscopic level. U. Olcese will present recent findings on how communication between single neurons is instead heterogeneously modulated based on several factors: spatio-temporal scale, neuronal type, distance between neurons, and functional specialization. At the scale of the electrocorticography, G. Piantoni will demonstrate that sleep spindles are regulated at a local level, but the degree to which they are local depends on the underlying cortical regions. He will also discuss that the interaction of local characteristics and global organization of the spindle provides a flexible framework to support the many functions ascribed to sleep. Using dynamic fMRI FC, A. Bagshaw will discuss how thalamic and thalamocortical networks change with sleep onset, and how this might interact with a pathology of the thalamocortical system, namely generalised epilepsy. Finally, G. Vandewalle will present recent results about neural mass modeling of local neural subpopulation interplay during normal waking and sleep loss in young and older adults. He will further show how local variations translate into large scale effective connectivity among the neural population.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Interpret the usual metrics used in functional connectivity applied to sleep research
- Recognize the cerebral integration processes during sleep and sleep loss from cellular scale activities to macroscopic functional connectivity networks
- Recognize the contributions of functional connectivity studies to our understanding of the functions of sleep

Target Audience

Confirmed researchers, doctoral and post-doctoral fellows involved in neuroimaging and fundamental sleep research

9:00am - 9:02am	**Introduction** *J. Carrier (Canada)*
9:02am - 9:19am	**A brief review on functional connectivity metrics in sleep research** *J.-M. Lina (Canada)*
9:19am - 9:36am	**Neuron-level functional and effective connectivity during NREM sleep: A highly heterogeneous picture of a global brain state** *U. Olcese (The Netherlands)*
9:36am - 9:53am	**Human electrocorticography reveals cortical variability of spindle characteristics** *G. Piantoni (United States)*
9:53am - 10:10am	**Thalamic functional connectivity during sleep in control subjects and patients with generalized epilepsy** *A. Bagshaw (United Kingdom)*
10:10am- 10:27am	**Local and global connectivity changes during sleep loss in aging** *G. Vandewalle (Belgium)*
10:27am - 10:30am	**Question and answer** *J.-M. Lina (Canada)*

S80 Sleep during early stage of life affects long-term outcomes

9:00am - 10:30am I North Hall

Chairs:

J. Kohyama (Japan)

Summary

Insufficient sleep duration affects various brain functions, and is associated with every aspect of children's and adolescents' well-being and daytime functioning. These effects range from decreased cognitive functioning, to poor academic performance, decreased emotional regulation, and increased behavior problems and psychopathology. Short sleep duration has been shown to significantly increase the risk of obesity in children, especially in young children. Obese children are at increased risk for metabolic disorders as well as sleep disorders, particularly obstructive sleep apnea. Physical, somatic and mental disturbances are also associated with circadian rhythm disturbances, such as shift work, jet lag and social jet lag.

From developmental point of view, short sleep duration during childhood has been reported to be associated with obesity in adults. Although poor daily habits of modern society (loss of physical contact, decreased physical activity, and short sleep duration with late bed and waking times) during the early stages of life are reported to be associated with truancy or dropping out of school, quitting employment, and committing suicide during subsequent years, the long-term effects of unfavorable early daily habits (insufficient sleep duration and disrupted circadian rhythms during the early stages of life) on brain functions are not fully understood. Interestingly, children who were less able to delay gratification at 4-year-old showed lower self-control in their forties with less activation in the prefrontal cortex (Marshmallow test). It should be noted that shortage of sleep is known to decrease activity of the prefrontal cortex, and sleep deprivation is also known to be linked to decreased metabolic activity in the ventromedial prefrontal cortex. Thus, it could be hypothesized that sleep during early stages of life affects long-term outcomes via prefrontal cortex. This symposium is intended to provide clinical evidence needed to prove a part of this hypothesis The current symposium will introduce findings from recent cohort studies on the long-term effects of sleep on cognitive, physical, and developmental issues. Jun Kohyama from Japan, chairperson, will overview the symposium with an introduction of Marshmallow test, and review several cohort studies that had sequential data on sleep. Prof. Kelly from the UK will present findings on sleep and developmental outcomes using data from Millennium Cohort Study. Prof Sekine from Japan will show the results of the Toyama cohort study focusing on the developmental aspect of sleep parameters. Prof. Mindell will provide us data from several cross-sectional studies with different age groups, and show us relationship of sleep with social-emotional and developmental outcomes in young children. This symposium is a clinical portion to prove a part of hypothesis that sleep during early stages of life affects long-term outcomes via prefrontal cortex, and participants can deepen their understanding of sleep from aspects of child, development, social viewpoints and also from an aspect of basic brain science on sleep.

General active discussion is expected with many audience of pediatricians, policy makers, school teachers, public health practitioners, students and also basic sleep scientists.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Recognize the importance of sleep especially during early stage of life from the aspect of long-term outcomes
- Describe the cognitive, behavioral and health problems among adults could be of roots during early stage of life
- Review the developmental aspect of brain function involved in sleep
- Recognize the importance of social activity to save child sleep

Target Audience

Pediatricians, policy makers, school teachers, public health practitioners, students and also basic sleep scientists

9:00am - 9:05am	**Introduction** *J. Kohyama (Japan)*
9:05am - 9:25am	**Overview of the symposium and introduction of several cohort studies focusing on sleep** *J. Kohyama (Japan)*
9:25am - 9:45am	**Sleep and child development – findings from the UK MCS** *Y. Kelly (United Kingdom)*
9:45am - 10:05am	**Sleep problems among Japanese children and their outcomes: Results from the Toyama study** *M. Sekine (Japan)*
10:05am - 10:25am	**Relationship of sleep with social-emotional and developmental outcomes in young children** *J.A. Mindell (United States)*
10:25am - 10:30am	**Question and answer** *J. Kohyama (Japan)*

S81 Sleep, brain-heart relationships and sudden death risk

9:00am - 10:30am I Club A and B

Chairs:
V. Somers (United States), E. St. Louis (United States)

Summary
The course will provide a comprehensive update on the mechanistic interaction of sleep and sleep apnea with cardiovascular and brain health and disease and sudden death risk, including myocardial ischemia, stroke, heart failure, sudden cardiac death, sudden unexpected death in epilepsy, and sudden death in infants (SIDS). It will examine the role of obstructive sleep apnea in coronary artery dysfunction and disease. The contribution of hypoxemia and apnea to nocturnal chest pain and objective evidence of cardiac ischemia will be discussed as will the implications of OSA for the risk of nocturnal MI and sudden cardiac death. There is evidence that OSA is a risk factor for stroke, yet strategies to identify and treat pre-existing and event-related sleep disordered breathing are not common place at most centers, and remains a developing therapeutic potential and active research area. OSA is more common in refractory epilepsy patients, and sudden unexpected death in epilepsy (SUDEP) is common during nocturnal hours, suggesting possible overlapping pathophysiology between sleep, OSA, and SUDEP risk. Last, the "Back to Sleep" campaign has led to a significant decline in incidence of sudden infant death syndrome (SIDS), but the disorder still persists, and potassium channel and metabolic dysfunction, as well as contributions of the hypocretin system and the medullary serotonergic network may be related to the pathogenesis of SIDS.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Review the complex interrelationships of sleep and sleep apnea with cardiovascular health, including the risk for myocardial ischemia, stroke, and heart failure
- Identify risk factors and the relationship of sleep and sleep apnea to sudden cardiac death
- Recall possible mechanisms and the role of sleep and sleep disorders in sudden unexpected death in epilepsy
- Recognize risk factors and preventative strategies for sudden infant death syndrome

Target Audience
Sleep medicine physicians and allied health personnel, neurologists, cardiologists, pulmonologists, psychiatrists, nurses, physician assistants, and student/resident/fellow trainees in these disciplines

9:00am - 9:05am	**Introduction** *V. Somers (United States)*
9:05am - 9:25am	**From obstructed pharyngal airway to obstructed coronary artery – mechanisms and clinical consequences** *V. Somers (United States)*
9:25am - 9:45am	**Sleep and sudden cardiac death risk** *A. Chahal (United Kingdom)*
9:45am - 10:05am	**Sleep, sleep apnea and sudden unexpected death risk in epilepsy** *E. St. Louis (United States)*
10:05am - 10:25am	**An update on infant apnea and SIDS** *S. Kotagal (United States)*
10:25am - 10:30am	**Question and answer** *E. St. Louis (United States)*

S82 Animal models for restless legs syndrome: New developments and future challenges

9:00am - 10:30am I Club D and E

Chairs:
A. Salminen (Germany)

Summary
Restless legs syndrome (RLS) is a common movement disorder, capable of having a severe impact on the sleep quality and quality of life in patients suffering from it. RLS is a hereditary disorder, with several risk factors identified at genetic loci including genes such as MEIS1, BTBD9 and PTPRD. The hallmark symptoms of RLS, such as an urge to move the legs, are subjective and are diagnosed in a patient interview. The lack of validated biological markers of the disease makes the development of mouse models challenging. In this symposium, the existing potential animal models for RLS are introduced and compared, providing a state-of-the-art overview at the development of RLS animal models. MEIS1 is the RLS gene with the highest effect size in most GWAS, harboring both rare and common genetic variants linked with RLS. Meis1 haploinsufficiency and point mutation mouse models have been investigated for motor and sleep phenotypes, and the results will be reviewed in the first part of the symposium. Future perspectives regarding MEIS1-based approaches will be discussed. BTBD9 is the second highest-effect RLS GWAS hit, and the highest-effect genetic risk factor for periodic leg movements during sleep. Btbd9-based models have been investigated in mice and in fruit flies with reported hyperactivity and sleep disturbance. These findings are discussed in the second part of the symposium in relation to the RLS phenotype in the clinic. PTPRD is a cell-adhesion molecule associated with RLS in GWAS. Ptprd knock-out models are another potential RLS animal model, showing motor restlessness and specific sleep phenotypes. In the third part of the symposium, the data from this model is reviewed and discussed with focus on validation of sleep disruption measures, validation of mice with constitutive reductions in expression based on data from human postmortem brains, and use of mouse data as a springboard for novel drug discovery.
The fourth part of the symposium will introduce an alternative approach: a forward genetic mouse model generated by looking for inbred mouse strains that show one biological RLS-related marker under iron deprivation. This mouse strain was then screened for other RLS-related phenotypes. To conclude the symposium, the four different potential RLS animal models are compared in relation to the signs we associate with human RLS. At the same time, the future perspectives of the field are discussed: are the currently available readouts sufficient for validating an RLS mouse model? Would the monitoring of tibialis anterior EMG in mice be a potential new RLS readout? The symposium aims to cover the most recent developments in modeling RLS in animals, while providing basic knowledge of the topic to clinicians as well as scientists. The audience will gain an up-to-date understanding of the current status in the race towards a validated RLS animal model.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Identify the different strategies to model a genetic human disease in animals
- Evaluate and compare the existing animal models for restless legs syndrome based on their reported phenotypes
- Describe the current and future challenges in the development of an animal model for restless legs syndrome

Target Audience
Neurologists, pulmonologists, trainees, RLS specialists, scientists interested in modeling human disease in animals

9:00am - 9:02am	**Introduction** *A. Salminen (Germany)*
9:02am - 9:19am	**MEIS1: Mouse models based on the highest-confidence RLS gene** *A. Salminen (Germany)*
9:19am - 9:36am	**BTBD9: Modeling RLS in mouse and fly** *Y. Li (United States)*
9:36am - 9:53am	**PTPRD: Knocking out a cell adhesion molecule to model RLS in mouse** *G. Uhl (United States)*
9:53am - 10:10am	**Forward genetics: BDX 40 as an RLS animal model** *R. Allen (United States)*
10:10am - 10:27am	**Comparison of current animal models and future perspectives** *M. Manconi (Switzerland)*
10:27am - 10:30am	**Question and answer** *A. Salminen (Germany)*

S94 Circadian rhythm sleep-wake disorders and insomnia: What are the consequences and how do we optimize treatment?

9:00am - 10:30am I Terrace 1

Chairs:
Y.K. Wing (Hong Kong)

Summary
Research indicates that individuals with strong circadian preferences, especially those with eveningness tendencies, have more health problems, poor mental health, and experience higher irregularity in sleep schedules. Additionally, these individuals are also more prone to insomnia symptoms, with highly reported comorbidity between insomnia disorder and delayed sleep-phase sleep disorder. However, there has been little research on the effect of both sleep disorders compared to having a single sleep disorder, and how to optimize treatment for these individuals when comorbid conditions exist.
In this symposium, we aim to present research on the negative effects of circadian rhythm sleep disorders comorbid with insomnia disorder in various affective, cognitive, and health domains. Additionally, we also look at different clinical populations that face challenges in keeping a regular sleep schedule, within circadian typology and in exogenous circadian-rhythm sleep wake disorders such as shift work disorder. Finally, we aim to provide guidelines that will help the clinician make treatment decisions based on pharmacological and non-pharmacological treatment modalities for individuals who have comorbid circadian-rhythm sleep-wake disorder and insomnia disorder.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Demonstrate understanding of the negative effects of circadian rhythm sleep disorders (CRSD) and insomnia disorder (ID)
- Idenitfy the challenges of individuals who keep irregular sleep schedules in affective, cognitive, and health domains
- Cite guidelines for optimal pharmacological and non-pharmacological treatments for individuals with CRSD comorbid with ID

Target Audience:
Students, researchers, and clinicians

9:00am - 9:02am	**Introduction** *Y.K. Wing (Hong Kong)*
9:02am - 9:19am	**Independent or joint effects of eveningness and insomnia on mental health** *S.X. Li (Hong Kong)*
9:19am - 9:36am	**Differential effects of circadian typology on sleep, fatigue, mood and quality of life** *I.-Y. Yoon (Republic of Korea)*
9:36am - 9:53am	**Insomnia and cognitive function in shift-working police officers** *S. Suh (Republic of Korea)*
9:53am - 10:10am	**Group cognitive-behavioral therapy for college students with insomnia: Effects on sleep and predisposing factors** *T. Shochat (Israel)*
10:10am- 10:27am	**Optimization of prescription of prolonged-release melatonin based on sleep-wake schedules** *S. Chung (Republic of Korea)*
10:27am - 10:30am	**Question and answer** *Y.K. Wing (Hong Kong)*

T07 Group scoring discussion: PLMD

9:00am - 10:30am I Club H

Chairs:
T. Penzel (Germany)

Summary
In this course there are the most common and frequently requested topics to be covered in order to get familiar with the latest and most updated guidelines as per the AASM. The topics will discuss very interesting practical and clinical examples where most scorers find these very confusing. Each participant will have the opportunity to do hands on under the supervision of the experienced trainer, whom is certified by the American Board of Sleep Medicine and the Board of Sleep Technologists.

Participants will be using computer software within the course.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Summarize the updated AASM guidelines for scoring sleep stages and arousals
- Summarize the updated AASM guideliness for scoring respiratory events
- Identify the sleep related motor disorders
- Recognize sleep related events and score them accourding to the AASM guidelines

Target Audience
Health professionals interested in getting updated about the latest AASM scoring rules and to increase their confidence in scoring by understanding the methodology of applying these rules on real demo traces

9:00am - 9:40am	**AASM guidelines for scoring respiratory events in adults** *A. Obeidat (United States)*
9:40am - 10:20am	**AASM guidelines for motor related events in adults** *A. Obeidat (United States)*
10:20am - 10:30am	**Question and answer** *A. Obeidat (United States)*

K12 Parasomnias: A window into dream

I. Arnulf (France)
Keynote 9:45am - 10:30am I Congress Hall

Parasomnias in REM sleep (RBD) are newly identified behaviors strongly associated with neurodegeneration. Researches are now focused on following other early signs of neurodegeneration and developing neuroprotective therapy. In contrast, NREM parasomnias (sleepwalking, sleep terrors) have been identified for a century, but somehow neglected and considered as benign or childish behaviors. We would like to raise attention on the fact that these two adult parasomnias correspond to enacted dreams. In addition to be treatable medical disorders, they open a brief but exceptional window into the dreaming cognitive and motor activity. The ethology of all behaviors during sleep is an open field for investigation, including sleep-associated speeches, facial expressions, and movements. These dreaming mental images are made visible for external investigators. This is a plea for giving as much importance to video and audio at night as to functional brain imaging, in order to access to the complex brain functioning during sleep.

K13 Sleep restriction in adolescents: Cognitive effects and remedies'

M. Chee (Singapore)
Keynote 10:30am - 11:15am I Congress Hall

Adolescents in the developed world are sleeping less compared to previous generations. Reduced sleep is most prominent in East Asian countries where scholastic achievement is highly venerated. A snapshot of current sleep behaviours and factors underlying short sleep in a representative country (Singapore) will be presented. Even in high performing adolescents, sleep restriction to 5h/night over a simulated school week causes degradation in vigilance, working memory, processing speed and mood. Such changes are exacerbated by a second cycle of sleep restriction. Topographical and declarative memory encoding are affected but problem solving skills are inconsistently affected. Napping for one hour in the afternoon can be of help for some cognitive operations. Starting school later has a positive effect on mood. Providing tools for evaluating time use can help shift behaviour and modifying study methods might also benefit sleep restricted students.

S83 The relationship between sleep, pain and fatigue following traumatic brain injury: From bench to bedside

10:30am - 12:00pm I Meeting Hall IV

Chairs:

D. Zalai (Canada)

Summary

Sleep alterations and the development of sleep disturbance are common sequeale following traumatic brain injury (TBI) across the continuum of recovery and occur in all levels of severity from concussion to moderate-severe injuries. Disturbed sleep compromises the recovery process; exacerbates pain, behavioural and mood symptoms; negatively impacts cognitive outcomes; and is a risk factor for re-injury. Despite its significance, the specific causes and maintaining factors of post TBI sleep-wake disturbances remain largely unknown and there is dearth of evidence-based assessment guidelines and treatment options tailored specifically for post TBI sleep pathology. This symposium brings together an inter-disciplinary team from three continents to present cutting-edge, translational research concerning the relationship between sleep, pain, fatigue and cognitive impairment following TBI. The presenters connect molecular, histological, electro-physiological, and clinical findings and discuss the implications of these on post -TBI sleep assessment and treatment. To generate a tool for the study of sleep in the context of TBI, Dr. Noain characterized a TBI model compatible with electro-physiological recordings in vigilance states in terms of sleep-wake disturbances, cognition, and histological trauma outcome. This model has led to a novel TBI treatment strategy involving sleep modulation in the acute phase of TBI resulting in reduced diffuse axonal injury and post-trauma cognitive decline. These findings suggest an alleviating role of slow wave sleep-induction on trauma-associated behavioural and histopathological outcomes. Dr. Noaim's findings obtained from animal models are consistent with Dr. Wiseman-Hakes' clinical research results. Specifically, Dr. Wiseman-Hakes will present self-report and objective data to demonstrate alterations in sleep quality, need for day-time sleep, and sleep architecture, and show the relationship between poor sleep and cognitive recovery. Furthermore, she will show that optimizing sleep can improve cognitive outcomes. She will also discuss the clinical implications and future research directions for optimizing sleep right from the early acute stage of injury. Pain is a common consequence of TBI that may impact sleep and hinder effective sleep interventions. Dr. Khoury will elucidate the effect of pain on sleep macro and micro-architecture after brain trauma and will discuss how pain is associated with greater sleep need in the early stages of mild TBI. She also will present recent advances in genetics and genomics and will show that we now have the necessary tools and technology to study molecular mechanisms behind the deleterious interaction between poor sleep and pain. In addition to pain, post -TBI fatigue is another debilitating condition associated with poor sleep. Dr. Rajaratnam will present a model concerning the inter-relationship between vigilance performance, fatigue, depression, anxiety, and excessive daytime sleepiness. He will also show the results of a clinical trial demonstrating the beneficial effect of blue light therapy on post -TBI fatigue. Finally, Dr. Zalai will discuss electro-physiological, sleep, pain, fatigue, and cognitive predictors of non-restorative sleep among patients with post-concussion insomnia. She will also demonstrate that there is an exceptionally high rate of sleep and circadian rhythm disorders among patients seeking treatment for insomnia following mild TBI and will discuss the implications of these findings on post-concussion insomnia assessment and management.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Identify differences in sleep quality and sleep architecture in the acute and chronic stage of traumatic brain injury (TBI)
- Recognize that in mild TBI patients with pain, sleep recuperative function seems to be disturbed by persistent wake EEG activity, corroborating patients' complaints such as feeling awake when asleep
- Identify the predictors and consequences of non-restorative sleep and fatigue following TBI
- Demonstrate awareness of the impact of disturbed sleep on the recovery process and describe methods for the evaluation and treatment of post-TBI sleep-wake disorders and fatigue

Target Audience

Physicians of different specialties (e.g. neurology, neurosurgery, psychiatry, sport) with interest or specialization in sleep medicine; nurses, psychologists, occupational therapists, researchers

10:30am - 10:32am	**Introduction** *D. Zalai (Canada)*
10:32am - 10:49am	**Traumatic brain injury and sleep: A back and forth relationship with therapeutic potential** *D. Noain (Switzerland)*
10:49am - 11:06am	**The interaction between pain and sleep in mild traumatic brain injury** *S. Khoury (Canada)*
11:06am - 11:23am	**The clinical implications of sleep disturbance following brain injury** *C. Wiseman-Hakes (Canada)*
11:23am - 11:40am	**Fatigue and its treatment following traumatic brain injury** *S. Rajaratnam (Australia)*
11:40am - 11:57am	**Post-concussion insomnia: Contributing factors and assessment strategies** *D. Zalai (Canada)*
11:57am - 12:00pm	**Question and answer** *D. Zalai (Canada)*

S84 Cortical nNOS neurons: A nexus between homeostatic sleep drive and EEG slow wave activity?

10:30am - 12:00pm I Meeting Hall V

Chairs:
T. Kilduff (United States)

Summary
In contrast to vast majority of cortical neurons, a rare population of GABAergic cortical interneurons has been found to be sleep-active in three rodent species. This neuronal population, characterized by co-expression of somatostatin (SST), Neuropeptide Y (NPY) and neuronal nitric oxide synthase (nNOS), is currently the rarest known cell type in the cerebral cortex. Subsequent studies have shown that these cells express the transcription factor c-FOS in proportion to homeostatic sleep drive, i.e., the longer animals are kept awake, the greater the proportion of cortical nNOS neurons are activated. These cells also have an unusual characteristic for a cortical GABAergic neuron: they have long distance intracortical axonal projections. Collectively, these observations have led to the hypothesis that cortical nNOS neurons integrate homeostatic sleep drive originating in subcortical structures and effect cortical slow wave activity through intracortical release of GABA, SST, NPY and/or NO. After a brief introductory overview of cortical neuron taxonomy based on molecular markers by the symposium Chair, Dr. Dmitry Gerashchenko (Boston) will present the studies that initially led to the discovery that cortical nNOS neurons were sleep-active and more recent studies directed toward targeting these cells for experimental manipulation. Dr. Lars Dittrich (Bonn) will then present the unusual sleep/wake phenotype of nNOS knockout mice, studies which showed that sleep-active cortical nNOS neurons also express NK1, and that activation of these cells is proportional to the degree of homeostatic sleep pressure that accumulates during wakefulness. Dr. Rhiannan Williams (Menlo Park) will then present in vitro physiological studies of cortical nNOS neurons that establish their response to neurotransmitters and neuromodulators such as acetylcholine, adenosine and hypocretin/orexin. Dr. Capogna (Aarhus) will then highlight the relationship between amygdala circuits and the sleep-wake cycle and present novel data showing that the activity of a population of nNOS neurons in the rodent amygdala are also modulated by the sleep/wake cycle, suggesting a novel cellular mechanism that links anxiety and emotion with sleep deprivation. Together, these presentations will present the latest results on this unusual neuronal population and their potential role as a link to help understand sleep homeostasis and Borbely's Process S.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Describe the diversity of cortical interneuron subtypes based on molecular taxonomy
- Compare the relative activation of principal neurons vs. interneurons of the cerebral cortex across the sleep/wake cycle
- Cite evidence that cortical nNOS neurons may integrate homeostatic sleep drive and EEG slow wave activity
- Give examples of how the activity and phenotype of Type I nNOS neurons are similar between the cerebral cortex and the amygdala

Target Audience
Post-graduate researchers, pre- and post-doctoral students and clinicians interested in neural control of sleep/wake and cortical activity

10:30am - 10:40am	**Introduction** *T. Kilduff (United States)*
10:40am - 11:05am	**Coupling homeostatic sleep pressure to deep, consolidated sleep - a role for cortical nNOS/NK1 neurons** *L. Dittrich (Germany)*
11:05am - 11:30am	**Neurotransmitter control of cortical nNOS neuron excitability** *R. Williams (United States)*
11:30am - 11:55am	**Modulation of nitric oxide synthase expressing neurons of the amygdala by sleep** *M. Capogna (Denmark)*
11:55am - 12:00pm	**Question and answer** *T. Kilduff (United States)*

S85 Suicide, sleep and circadian rhythms in adolescents

10:30am - 12:00pm I North Hall

Chairs:

C.M. Shapiro (Canada)

Summary

Sleep and psychiatric disorders are inextricably linked. The vast majority of clinically depressed individuals struggle with sleep, and insomnia is a well-proven risk factor for suicide across different cultures and age groups. Recent media coverage has underscored the issue of increased suicide in youth. This symposium aims to focus on recent discoveries and advances in this area and how these relate to sleep, mood and suicide. Growing evidence supports the bidirectional linkages among sleep, depression and circadian rhythm disruption, all of which are characterized by negative cognitive biases. Given that recent studies point to a direct effect of sleep on affective processing in healthy populations, it is conceivable that disrupted sleep can play a role in affective biases in depression and suicide. The link between sleep and suicide is particularly relevant in adolescents where this relationship has been shown to be independent of symptoms of depression. The literature points to a role of circadian rhythm disorders in the development of depression and suicidal behaviours. Depression is poorly recognized in adolescents and a crucial preventative role exists for all health professionals, particularly sleep specialists, in the management of sleep disorders that can serve to trigger depression and suicidal behaviour.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Recognize and Associate the link among sleep disturbance, depression and the risk of suicide across all age groups
- Integrate knowledge from recent scientific publications in the field of sleep, depression and suicide
- Demonstrate competence in the recognition of depressive symptoms and increased suicide risk due to the presence of an untreated sleep disorder
- Appraise and critique the scientific literature in the field of sleep, depression and suicide

Target Audience

All sleep and mental health professionals that work with adolescent patient populations

10:30am - 10:32am	**Introduction** *C.M. Shapiro (Canada)*
10:32am - 10:49am	**The links between sleep and circadian rhythms with depression, suicidal thinking and behaviour** *C.M. Shapiro (Canada)*
10:49am - 11:06am	**Insufficient Sleep And suicidality in Korean adolescents** *Y.J. Lee (Republic of Korea)*
11:06am - 11:23am	**Effects of short sleep and school start time on mood in adolescents** *J.J. Gooley (Singapore)*
11:23am - 11:40am	**Effects of Sleep on affective cognition in individuals with depression** *E.Y.Y. Lau (Hong Kong)*
11:40am - 11:57am	**Depression, sleep difficulties and suicide risk in adolescents** *A. Shahid (Canada)*
11:57am - 12:00pm	**Question and answer** *C.M. Shapiro (Canada)*

S86 Vitamin D and sleep

10:30am - 12:00pm I Club A and B

Chairs:
R. Silvestri (Italy)

Summary

According to basic and clinical recent reports and reviews, vit D deficiency plays a pivotal role in the causative mechanisms of many human sleep disorders. Via the activation of pro-inflammatory and hypoxic pathways. Most recent evidence of these mechanisms in different sleep disorders will be the object of this proposal. Vitamin D deficiency activates pro-inflammatory pathways, hypoxia cascade, and plays a crucial role on early dopaminergic receptors development. Recent proteomic analysis of the cerebrospinal fluid of patients with RLS have identified vit D binding protein as a candidate marker for early onset RLS. Inflammation and altered immune modulation may play an important role in human hypersomnia and pose a specific risk for the occurrence of specific syndrome. CRP increase excessive daytime sleepiness, cardiovascular risk and insulin resistance susceptibility may be mediated by vit D deficiency in OSA subpopulations according to ethnicity, geographic and cultural/environmental challenges. Vit D is associated to muscle-skeletal pain, relevant to growing pains. Vit D deficiency contributes to RLS via the hypoxic inflammatory pathway activation and the altered development of dopaminergic receptors. Vit D supplementation may represent a relevant therapeutic option for specific patient populations at risk for deficiency.
So far, the impact of vit D deficiency in the development and clinical course of different sleep disorders has not been consistently addressed. The possibility of a strong impact on disease by early supplemental therapy should be of relevant interest to most clinicians and researchers.

Learning Objectives

Upon completion of this CME activity, participants should be able to:
- Recognize the role of vit D deficiency in sleep disorders and its potential therapeutic effects
- Diagnose and appropriately address replacement of vit D deficiency within a wide spectrum of sleep disorders
- Predict populations at risk for vit D deficiency according to suggestive sleep phenotypes and influence sleep disorders prognosis

Target Audience

Sleep specialists, basic scientists, pneumologists, pediatricians, internists, movement disorders specialists

10:30am - 10:35am	**Introduction** *R. Silvestri (Italy)*
10:35am - 10:55am	**Vitamin D: Muscle-skeletal, cardio-vascular and immune role in health and disease (recent evidence for a modulating effect in sleep disorders)** *A. Stefani (Austria)*
10:55am - 11:15am	**Vitamin D deficiency in central hypersomnias** *Y. Dauvilliers (France)*
11:15am - 11:35am	**Vitamin D and OSA: New evidence of a possible role in EDS** *F. Placidi (Italy)*
11:35am - 11:55am	**Vitamin D deficiency in RLS /WED and growing pains** *R. Silvestri (Italy)*
11:55am - 12:00pm	**Question and answer** *R. Silvestri (Italy)*

O15 Chronobiology/circadian disorders oral abstract presentations

10:30am – 12:00pm I Club D and E

Chairs:
A. Sumova (Czech Republic), K. Lushington (Australia), S. Abbott (United States)

10:30am – 10:45am	**DISRUPTING DIURNAL CYCLING OF NAD+-DEPENDENT SIRTUIN 1 DEACETYLASE ACTIVITY WITH GENOTOXIC STRESS OR CHANGING LIGHT/DARK CYCLES INCREASES SUSCEPTIBILITY TO MAMMARY CARCINOGENESIS BY UNCOUPLING DNA DAMAGE RESPONSE AND REPAIR GENES FROM CIRCADIAN CONTROL** *H. Zarbl (United States)*
10:45am – 11:00am	**NEUROBEHAVIORAL ALTERATIONS IN MOUSE MODEL OF SHIFT WORK DISORDER** *S. Arora (India)*
11:00am – 11:15am	**EVENING LIGHT EXPOSURE FROM COMPUTER SCREENS DISRUPTS SLEEP, BIOLOGICAL RHYTHMS AND ATTENTION ABILITIES** *A. Green (Israel)*
11:15am – 11:30am	**THE DIFFERENTIAL EFFECTS OF REGULAR SHIFT WORK AND OBSTRUCTIVE SLEEP APNEA ON SLEEPINESS, MOOD, VIGILANCE AND NEUROCOGNITIVE FUNCTION** *J. Cori (Australia)*
11:30am – 11:45am	**MELATONIN SECRETION AND POOR SLEEP QUALITY IN PATIENTS WITH TETRAPLEGIA : A PILOT STUDY** *R. Davillé-Blicq (France)*
11:45am – 12:00pm	**A NEURAL NETWORK MODEL TO PREDICT CIRCADIAN PHASE IN NORMAL LIVING CONDITIONS** *J.E. Stone (Australia)*

O16 Sleep breathing disorders oral abstract presentations

10:30am – 12:00pm I Terrace 1

Chairs:
O. Ludka (Czech Republic), J. Puertas (Spain)

10:30am – 10:45am	**INTERACTION BETWEEN SEVERITY OF OBSTRUCTIVE SLEEP APNEA AND GENDER ON THE LEVEL OF HEMOGLOBIN** *T. Li (China)*
10:45am – 11:00am	**NEUROMUSCULAR INJURIES IN THE SOFT PALATE CORRELATES WITH PHARYNGEAL DYSFUNCTION IN SLEEP APNEA SUBJECTS** *F. Shah (Sweden)*
11:00am – 11:15am	**RESPIRATORY SYMPTOMS ARE MORE COMMON AMONG SHORT SLEEPERS INDEPENDENT OF OBESITY** *E. Björnsdóttir (Iceland)*
11:15am – 11:30am	**EFFECT OF SLEEP APNEA AND INSOMNIA ON THE ASSOCIATION OF DEPRESSION WITH QUANTITATIVE ELECTROENCEPHALOGRAM MEASURES (QEEG) IN ADULT MEN DURING SLEEP - THE MAILES STUDY** *R. Adams (Australia)*
11:30am – 11:45am	**WHOLE GENOME SEQUENCE ASSOCIATION ANALYSIS OF SLEEP-DISORDERED BREATHING TRAITS IN TRANS-OMICS FOR PRECISION MEDICINE (TOPMED)** *B. Cade (United States)*
11:45am – 12:00pm	**CORRECTION OF SLEEP DISORDERS BY THE SENSOMOTOR CONTROL METHOD OF RESPIRATION IN PATIENTS WITH HYPERVENTILATION SYNDROME** *A. Barulin (Russian Federation)*

O17 Sleep health and other issues oral abstract presentations

10:30am – 12:00pm I Club H

Chairs:
M. Didriksen (Denmark), Y.K. Wing (Hong Kong)

10:30am – 10:45am	**SCHOOL START TIME CHANGE, SLEEP DURATION AND CAR CRASHES IN HIGH SCHOOL STUDENTS** *S. Bin-Hasan (Canada)*
10:45am – 11:00am	**ACTIGRAPHIC SLEEP PATTERNS AND HYPERTENSION IN THE HISPANIC COMMUNITY HEALTH STUDY/STUDY OF LATINOS** *A. Ramos (United States)*
11:00am – 11:15am	**EVALUATION OF SLEEP AND FACTORS AFFECTING IT IN PATIENTS RECOVERING IN INTENSIVE CARE UNITS (ICU) AND STEP DOWN UNITS (SDU)** *B. Prajapat (India)*
11:15am – 11:30am	**QUALITY OF LIFE AND MOOD IN CHILDREN AND ADOLESCENTS WITH CYSTIC FIBROSIS; ASSOCIATIONS WITH SLEEP QUALITY** *M. Vandeleur (Australia)*
11:30am – 11:45am	**SLEEP DURATION AS AN INDEPENDENT FACTOR ASSOCIATED WITH VITAMIN D LEVELS** *D.L. de Oliveira (Brazil)*
11:45am – 12:00pm	**SLEEP HABITS IN INFANTS: THE ROLE OF MATERNAL EDUCATION** *R. Ferreira (Portugal)*

K14 Circadian rhythm sleep disorders: Challenges in diagnosis and treatment

D. Skene (United Kingdom)
Keynote 11:15am - 12:00pm I Congress Hall

The mismatch between the circadian timing system and behavioral rhythms in sleep/wake and feeding/fasting has both acute and chronic adverse effects on many physiological systems. Elucidation of the molecular clockwork, the melanopsin-mediated photic pathways and discovery of peripheral clocks throughout the body has provided not only new opportunities but also challenges in the diagnosis and treatment of circadian rhythm disorders. Accurate diagnosis of both central and peripheral clock timing in humans and the role of photic and nonphotic time cues (meals, melatonin) in synchronizing/resetting these rhythms will be discussed.

K15 Control of sleep-related breathing

M. Morrell (United Kingdom)
Keynote 12:30pm - 1:15pm I Congress Hall

This keynote lecture will explore the interactions between sleep and respiratory control that lead to sleep-related breathing disorders. In particular how measurement of physiological parameters such as upper-airway collapsibility, arousal threshold and loop gain may predict the development of sleep-related breathing disorders and responses to treatment. Such a personalised approach my improve adherence with therapy and help to target those who will benefit most from new treatments. Specifically in vulnerable groups such older people and patients with heart failure, in whom changes in sleep and respiratory control predispose to high rates of sleep-related breathing disorders.

K16 Cardiovascular risk, OSA and CPAP (SAVE study)

D. McEvoy (Australia)
Keynote 1:15pm - 2:00pm I Congress Hall

S87 Phenotyping and genotyping sleep apnea

2:00pm - 3:30pm I Congress Hall

Chairs:
T. Penzel (Germany)

Summary
Sleep disordered breathing has shown to have a very high prevalence in the population. Today we understand, that sleep disordered breathing is found in different phenotypes: Obstructive sleep apnea presents a major risk factor for cardiovascular disorders. Obesity, carniofacial characteristics, impaired chemoreceptor regulation may be important traits. Some patients show central sleep apnea combined with obstructive sleep apnea together with heart failure or other cardiac problems. Sleep disordered breathing has proven to show an increased prevalence in the elderly population and therefore sleep disordered breathing may be associated with an aging of respiratory regulation in some patients. Only a comprehensive assessment of symptoms, combined with clinical investigation and detailed diagnosis of sleep with cardiorespiratory polysomnography will allow to distinguish phenotypes and then, later, may allow to genotype these phenotypes. A large multi-national study covering different continents (SAGIC - sleep apnea global interdisciplinary consortium) is devoted to this problem. In this symposium the group will present latest genetic concepts, craniofacial morphologies across severity of sleep apnea and ethnicities, sleep and apnea scoring across continents, analysis of sleep recordings, and the development of practical questionnaires for the assessment of symptoms to be used in clinical practice as well. The symposium covers theoretical background, modern analysis for phenotyping, and practical proposals useful for clinical practice.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Prepare diagnostic procedures for sleep disordered breathing
- Predict phenotypes of sleep apnea
- Summarize possible genetic variants responsible for sleep apnea
- Differentiate patients with sleep apnea with dedicated comprehensive questionnaires

Target Audience
Clinicians with an interest in understanding mechanisms of sleep disordered breathing and Basic scientists interested in the pathophysiology of sleep disordered breathing

2:00pm - 2:02pm	**Introduction** *T. Penzel (Germany)*
2:02pm - 2:19pm	**Phenotyping sleep apnea and the pathway to genotyping** *A. Pack (United States)*
2:19pm - 2:36pm	**Quantify craniofacial differences** *R. Schwab (United States)*
2:36pm- 2:53pm	**Scoring of sleep recordings for sleep apnea and hypopnea** *U. Magalang (United States)*
2:53pm - 3:10pm	**Analysis of physiological sleep signals for phenotyping** *P. de Chazal (Australia)*
3:10pm- 3:27pm	**Is there an optimized questionnaire for assessing sleep disordered breathing?** *T. Gislason (Iceland)*
3:27pm - 3:30pm	**Question and answer** *T. Penzel (Germany)*

S88 Novel biomarkers for sleep insufficiency and sleep disorders

2:00pm - 3:30pm I Meeting Hall IV

Chairs:
D.-J. Dijk (United Kingdom)

Summary
Aim: To highlight recent advances and novel approaches for identifying biomarkers of sleep insufficiency. Sleep insufficiency and sleep disorders are highly prevalent worldwide and are associated with significant negative health consequences and economic costs. Accurate and cost effective assessment, diagnosis and treatment of sleep insufficiency and sleep disorders could be significantly enhanced by easily accessible point of contact biomarkers from blood or other bodily fluids. Such biomarkers are currently not available. Recently, several novel approaches for the identification of biomarkers have been applied in laboratory and field studies of insufficient sleep and in patient populations. Novel approaches include omics such as transcriptomics and lipidomics of blood samples. These omics data have been analyzed with the aim to discover univariate or multivariate (i.e., signatures) biomarkers. This symposium will provide an overview of recently published and unpublished results from four leading researchers from three continents and covering both laboratory and field studies as well as healthy participants and sleep disorder patients. Derk-Jan Dijk, PhD (University of Surrey, UK) and his team have pioneered analyses of the effects of repeated partial sleep deprivation, total sleep deprivation and circadian rhythmicity on the human blood transcriptome. In his presentation (Human blood transcriptome based biomarkers for insufficient sleep and circadian abnormalities) he will review these studies. He will also discuss ongoing efforts to identify biomarkers and classifiers for insufficient sleep, cognitive vulnerability to sleep insufficiency and circadian disruption. Joshua Gooley, PhD (Duke-NUS Medical School, Singapore) and his team have investigated approaches for monitoring and predicting cognitive responses to sleep deprivation. In his presentation (Biomarkers of sleep insufficiency and cognitive vulnerability to sleep deprivation) he will discuss behavioural, physiological and lipidomics based biomarkers of sleep deprivation, and their associations with vigilance and drowsy driving. Tarja Porkka-Heiskanen, PhD (University of Helsinki, Finland) and her team have applied blood transcriptomics and metabolomics to study effects of sleep restriction in the laboratory and in two epidemiological cohorts. In her presentation (Metabolomics as a biomarker for sleep debt) she will discuss pathways in cholesterol metabolism and inflammatory responses affected by insufficient sleep that may serve as potential markers. Leila Kheirandish-Gozal, MD (University of Chicago, USA) and her team have focused on identifying biomarkers of morbidities in pediatric obstructive sleep apnea syndrome (OSAS) based on blood transcriptomics and circulating exosomal miRNAs. In her presentation (Morbidity-related biomarkers in pediatric obstructive sleep apnea) she will discuss current evidence for the presence of morbidity-related biomarkers among children with OSAS. The symposium will be chaired by Derk-Jan Dijk, an advocate of the development of biomarkers for sleep and circadian rhythm research and medicine. He has previously attended the workshops on this topic organised by SRS and ESRS at the NIH, Sleep 2016 and the ESRS-Bologna meeting in 2016. It is anticipated that this symposium will be of interest to basic and clinical sleep scientists.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the potential of novel biomarker discovery approaches in sleep research and sleep medicine
- Recognize the potential of biomarker approaches for refined phenotyping in sleep disorders
- Summarize the advantages and disadvantages of the various omics approaches used in biomarker discovery
- Evaluate the strengths and weaknesses of protocols used in biomarker discovery for insufficient sleep and sleep disorders

Target Audience
Basic and clinical sleep scientists who are interested in the use of novel biomarkers in research and clinical practice. These will include: Basic scientists, clinical scientists, Sleep scientists, Sleep epidemiologists

2:00pm - 2:05pm	**Introduction** *D.-J. Dijk (United Kingdom)*
2:05pm - 2:25pm	**Human blood transcriptome based biomarkers for insufficient sleep and circadian abnormalities** *D.-J. Dijk (United Kingdom)*
2:25pm - 2:45pm	**Biomarkers for cognitive vulnerability to sleep deprivation and sleep insufficieny** *J.J. Gooley (Singapore)*
2:45pm - 3:05pm	**Metabolomics as a biomarker for sleep debt** *T. Porkka-Heiskanen (Finland)*
3:05pm - 3:25pm	**Morbidity-related biomarkers in pediatric obstructive sleep apnea** *L. Kheirandish-Gozal (United States)*
3:25pm - 3:30pm	**Question and answer** *D.-J. Dijk (United Kingdom)*

S89 Restless legs syndrome, augmentation and dopamine treatment - clinical data and emerging new models

2:00pm - 3:30pm I Meeting Hall V

Chairs:
S. Clemens (United States)

Summary
Restless Legs Syndrome (RLS) is a sensorimotor disorder that heavily affects sleep and sleep quality. While the underlying causes are not fully understood, evidence suggests that genetic disposition may play a role, as well as alterations in iron homeostasis that in turn may alter the dopamine system. RLS is commonly, and very successfully initially, treated with dopamine receptor agonists that target the inhibitory subtypes of the dopamine receptor family (D2-like: D2, D3, and D4). However, long-term treatment with these compounds often leads over time to the development of augmentation, the worsening of the symptoms as a function of the treatment.
In this symposium we will first provide a clinical overview of the efficacy of the dopamine-based treatment in RLS, from initial and fast relief to the development of augmentation over time, and the emergence of alternate drug therapies. We will then present evidence how alterations of the dopamine D3R system at the spinal cord level alone can induce changes in sleep pattern and locomotor activity in a rodent model. This will be followed by a report on the switch of the therapeutically active D3 receptor compounds from analgesic to hyperalgesic effects over time, and the resolution of this inversion by concomitant block of the excitatory dopamine receptor, D1. Lastly, we will present evidence of how interactions of the different dopamine receptor subtypes between themselves and other G protein-coupled receptors forming receptor heteromers can shape cellular excitability and behavioral outcome, and how targeting the molecular switch between inhibitory (D2-like) and excitatory (D1-like) dopamine receptor subtypes might provide a new pharmacological target that will help maintain D3 mimetic efficacy without inducing augmentation.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize RLS patients and distinguish them form other sleep or movement disorders
- Identify parameters that point to the emergence of augmentation in RLS patients
- Develop patient-centered treatments options that minimize the risk for augmentation
- Integrate pre-clinical data gained from the off-label use of clinically available alternate compounds for the use of in RLS and augmentation

Target Audience
Neurologists, sleep disorder specialists, movement disorder specialists, restless legs syndrome specialists

2:00pm - 2:05pm	**Introduction** *S. Clemens (United States)*
2:05pm - 2:25pm	**Augmentation: Caused by D1, D3 or D5 receptors?** *W. Paulus (Germany)*
2:25pm - 2:45pm	**The validity of EMG activity during sleep in rodents model** *M. Manconi (Switzerland)*
2:45pm - 3:05pm	**Long-term exposure to inhibitory D3 receptor agonists modifies excitatory D1 receptor properties** *S. Clemens (United States)*
3:05pm - 3:25pm	**Dopamine receptor heteromers in restless legs syndrome** *S. Ferré (United States)*
3:25pm - 3:30pm	**Question and answer** *S. Clemens (United States)*

S90 Sleep across cultures in young children from around the world

2:00pm - 3:30pm I North Hall

Chairs:
D. Goh (Singapore)

Summary
Sleep practices and habits differ across the globe and they are greatly influenced by cultural backgrounds, traditions, environmental conditions and the sleeping location (surfaces used for sleeping). These aspects do not only influence bedtimes, but also have a bearing on the duration and regularity of sleep. The changing lifestyles in modern society has also significantly influenced our sleep timings and patterns. It has been described that sleep habits and practices vary across the world and amongst different groups in each region. It is important to understand the varying sleep practices across cultures to better appreciate the cultural and other factors that impact on sleep habits. Health and performance are highly dependent upon sleep and rest, especially in children. This symposium is aimed at evaluating the sleep cultures in young children across the world. We will be delving on how these different practices influence a child's growth, development and behavior. The symposium will also capture the differences and similarities that exist in sleep behaviors in Asian children and Caucasian children. Finally, the symposium will touch upon the aspects of snoring and other sleep-related disorders in children.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the cultural influences on sleep patterns and practices in young children
- Assess the impact of sleep practices on the child's growth and development
- Apply the knowledge in providing sleep advice for young children to promote health for the child, while keeping in mind the cultural factors

Target Audience
General practitioners, sleep researchers, sleep medicine physicians, pediatricians, sleep technicians, pediatric neurologists, child and adolescent psychiatrists and pediatric nurses

2:00pm - 2:05pm	**Introduction** *D. Goh (Singapore)*
2:05pm - 2:30pm	**Sleep outcomes in young children from around the world** *J.A. Mindell United States)*
2:30pm - 2:55pm	**Sleep habits and practices across Asian and Caucasian young children** *D. Goh (Singapore)*
2:55pm - 3:20pm	**Snoring and sleep-related breathing disorders across cultures** *A. Li (Hong Kong)*
3:20pm - 3:30pm	**Question and answer** *D. Goh (Singapore)*

S91 Improving insomnia treatments: Less pain, more gain?

2:00pm - 3:30pm I Club D and E

Chairs:
C. Morin (Canada)

Summary
There is no longer any doubt that Cognitive Behavioral Therapy for insomnia, CBT-i, is a very effective treatment when delivered individually, in group or as therapist guided self-help via e.g. a book or the Internet. Several studies also show large treatment effects for completely un-guided Internet treatments. Recent studies have, however, started to show what clinicians have always known - that the most effective treatment technique, Sleep Restriction Therapy, can be very hard to execute and is associated with adverse effects, such as poorer cognitive performance, excessive sleepiness and headaches. Also, while time again presenting very large treatments effects, there is still a substantial proportion of study participants who do not respond to the treatment. The presentations target the following questions: why might treatment need to be refined/reconsidered mechanistically; can you make refinements without compromising efficacy; can we integrate sleep and circadian therapies to better treat the constellation of sleep-wake disturbances that typically characterize those with mental illness; and can you predict for whom treatment might not be 'working', prompting early intervention with enhanced therapeutic support.
The presentations will be followed by a 30 minutes panel discussion, also involving the audience, about future directions in the improvement of insomnia treatments. Dr. Charles Morin is chair and discussant. Dr. Simon D. Kyle presents results of a new, randomized controlled trial (RCT) with n = 1600, assessing adverse effects in digital CBT-i compared to Sleep Hygiene. Previous work by Dr. Kyle's group reveals that Sleep Restriction Therapy, while effective, engenders subjective and objective daytime impairment during acute implementation. His work has important implications for patient safety and suggests there may be an element of "no pain, no gain" in the behavioral treatment of insomnia. Dr. Susanna Jernelöv presents new data from an RCT, n = 180, comparing Sleep Restriction Therapy to Sleep Compression. Sleep Compression is considered to produce less adverse effects than Sleep Restriction Therapy. This study explores whether this is true, and whether the treatments' positive effects on insomnia may be equivalent. If so, Sleep Compression could be put forward as a less difficult alternative for treatment of insomnia. Dr. Allison Harvey presents a new transdiagnostic treatment protocol. The goal of the Transdiagnostic Intervention for Sleep and Circadian Dysfunction (TranS-C) is to improve functional impairment, disorder-focused symptoms, and sleep and circadian functioning. This talk will present preliminary data and discuss the unique challenges of developing and implementing TranS-C in different populations. Dr. Kerstin Blom presents new results from an RCT, n = 280, of Therapist-guided Internet-delivered CBT-i. Previous studies have claimed that the nature of therapist support in Internet treatments is of no or little importance for treatment outcomes, and this study challenges that claim. We predict participants' success or failure 4 weeks into treatment, using a structured algorithm. Those predicted to fail are randomized between continued treatment as usual (with standard therapist guidance) or increased, more flexible support aiming at turning predicted failures into successes. Panel and audience discussion: improvement of current psychological insomnia treatments.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the importance of measuring and addressing adverse events in research and treatment of insomnia
- Give examples of new insomnia treatment initiatives addressing adverse events and/or increased effects
- Formulate and/or debate clinical and/or research questions developing this theme further

Target Audience
Insomnia treatment researchers and Insomnia treatment clinicians

2:00pm - 2:05pm	**Introduction** *C. Morin (Canada)*
2:05pm - 2:20pm	**Adverse effects of CBT for insomnia** *S.D. Kyle (United Kingdom)*
2:20pm - 2:35pm	**A comparison between sleep compression and sleep restriction** *S. Jernelöv (Sweden)*
2:35pm - 2:50pm	**Transdiagnostic approaches to treating sleep and circadian problems** *A. Harvey (United States)*
2:50pm - 3:05pm	**An adaptive treatment strategy for Internet-CBT: Predicting and preventing failures** *K. Blom (Sweden)*
3:05pm - 3:30pm	**Question and answer** *C. Morin (Canada)*

O18 Technology and technical oral abstract presentations

2:00pm – 3:30pm I Club A and B

Chairs:
R. Ferri (Italy), R. Chiang (Taiwan)

2:00pm – 2:15pm	**PREDICTION OF LEVEL OF DROWSINESS USING AN ADAPTIVE GEOMETRIC BROWNIAN MOTION MODEL, WITH APPLICATION TO DROWSY DRIVING ACCIDENT PREVENTION** *P. Ebrahimbabaie Varnosfaderani (Belgium)*
2:15pm – 2:30pm	**AUTOMATIC SLEEP CLASSIFICATION USING ADAPTIVE SEGMENTATION REVEALS INCREASED NUMBER OF SLEEP STAGE TRANSITIONS** *J.A.E. Christensen (Denmark)*
2:30pm – 2:45pm	**ASSESSMENT OF SLEEP PARAMETERS FROM RAW ACCELEROMETRY DATA** *V.T. van Hees (The Netherlands)*
2:45pm – 3:00pm	**ADVANCED ANALYSIS OF SLEEP SPINDLES: FROM HEALTHY TO DAMAGED BRAINS** *A. Mensen (Switzerland)*
3:00pm – 3:15pm	**A MACHINE LEARNING APPROACH TO DETECTING SLEEP AND SLEEP DISORDERS IN ACCELERATION SENSOR DATA** *R. Leenings (Germany)*

O19 Sleep breathing disorders oral abstract presentations

2:00pm – 3:30pm I Terrace 1

Chairs:
M. Zucconi (Italy)

2:00pm – 2:15pm	**THE ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNEA AND ALZHEIMER'S DISEASE: A META-ANALYSIS PERSPECTIVE** *H. Khazaie (Islamic Republic of Iran)*
2:15pm – 2:30pm	**WHETHER TONSILLECTOMY IS NECESSARY FOR PEDIATRIC OSA WITH SMALL TONSILS? DRUG INDUCED SLEEP ENDOSCOPY CAN TELL** *S. He (China)*
2:30pm – 2:45pm	**SPONTANEOUS IMPROVEMENT IN BOTH OBSTRUCTIVE SLEEP APNEA AND COGNITIVE IMPAIRMENT AFTER STROKE** *J. Slonkova (Czech Republic)*
2:45pm – 3:00pm	**THE PACE (PHARMACOTHERAPY OF APNEA BY CANNABIMIMETIC ENHANCEMENT) CLINICAL TRIAL: CHARACTERISTICS OF CLINICAL RESPONDERS TO DRONABINOL TREATMENT OF OBSTRUCTIVE SLEEP APNEA** *D.W. Carley (United States)*
3:00pm – 3:15pm	**SAFETY AND EFFICACY OF 6-MONTH USE OF SHAM-CPAP IN OBSTRUCTIVE SLEEP APNEA PATIENTS** *L. Mello-Fujita (Brazil)*
3:15pm – 3:30pm	**IMPROVING QUESTIONNAIRE SCREENING FOR OSA IN CHILDREN** *G. Nixon (Australia)*

T08 Oximetry interpretation

2:00pm - 3:30pm I Club H

Chairs:
S. Keenan (United States), O. Ludka (Czech Republic)

2:00pm - 3:30pm	**Oximetry interpretation** *C. Navin, United Kingdom*

S92 Creating a framework for analyses of movement patterns of challenging/disruptive sleep and wake behaviours

3:30pm - 5:00pm I Congress Hall

Chairs:
O. Ipsiroglu (Canada)

Summary
Analyses of behavioural and movement patterns have always been of interest in the domain of sleep and wake behaviours. The introduction of video technology into the field has catalyzed widespread research concepts and supported new clinical understandings, particularly in sleep-related rhythmic movement disorders and epilepsy. Currently, the use of video technology is booming thanks to the widespread use of user-friendly smartphones and tablet; parents often approach clinicians with various clips. In this session, we will present a framework for the use of video recordings of challenging/disruptive sleep/wake behaviours of children/adolescents/adults with neurodevelopmental conditions in clinical assessments and provide a guideline for reviewing and analyzing recordings. The session will be conducted in a round table format: five international sleep research teams with multidisciplinary backgrounds (neuropsychiatry, neurology, developmental pediatrics and mental health, engineering) will present their suggestions for analyzing video recordings of movement and behavioural patterns in various states of wakefulness and sleep. Dr. Andrea Guzzetta (University of Pisa, Italy) will present on 'Spontaneous Movement Patterns in Infants'; Dr. Isabelle Arnulf Hôpital Pitié-Salpêtrière, Paris, France) will present on 'REM Sleep Behaviour Disorders'; Dr. Federica Provini (University of Bologna, Italy) will present 'nREM Sleep Behaviour Disorders'; Dr. Rosalia Silvestri (University of Messina, Italy) will present on 'Sleep-related Rhythmic Movement Disorders and Epileptic Movement Patterns'; Mr. Gerhard Kloesch (Medical University of Vienna, Austria) will present 'Assessment of Vigilance'; Dr. Osman Ipsiroglu (Sleep/Wake Behaviour Clinic Research Lab, BC Children's Hospital, University of British Columbia, Canada) will present on 'Hyperarousability-Insomnia and Hypermotor-Restlessness in Children with Neurodevelopmental Condtions'; and, lastly, Dr. Heinrich Garn (Austrian Institute of Technology, Austria) will present on 'State-of-the-Art Technologies to Capture and Analyze Movement Patterns'. Attendees will have the opportunity to submit video-recordings and combine them with clinical questions; software for annotation of clips will be provided.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Cite a framework for where and how patient narratives and associated video observations of sleep and wake behaviours can and should be used.
- Review guidelines for reviewing and analyzing challenging/disruptive sleep and wake behaviours of children/adolescents/adults with neurodevelopmental conditions

Target Audience
Medical practitioners, allied health (including pharmacists) interested in and/or working with individuals with neurodevelopmental conditions

3:30pm - 3:33pm	**Introduction** *O. Ipsiroglu (Canada)*
3:33pm - 3:45pm	**Spontaneous movement patterns in infants** *A. Guzzetta (Italy)*
3:45pm - 3:57pm	**REM sleep behaviour disorders** *I. Arnulf (France)*
3:57pm - 4:09pm	**nREM sleep behaviour disorders** *F. Provini (Italy)*
4:09pm- 4:21pm	**Sleep-related rhythmic movement disorders and epileptic movement patterns** *R. Silvestri (Italy)*
4:21pm - 4:33pm	**Assessment of vigilance** *G. Kloesch (Austria)*
4:33pm - 4:45pm	**Hyperarousability-insomnia and hypermotor-restlessness (H-behaviours) in children with neurodevelopmental condtions** *O. Ipsiroglu (Canada)*
4:45pm - 4:57pm	**State-of-the-art technologies to capture and analyze movement patterns** *H. Garn (Austria)*
4:57pm - 5:00pm	**Question and answer** *O. Ipsiroglu (Canada)*

S93 Infra-slow (< 0.1 Hz) oscillations: From the cell to the clinic

3:30pm - 5:00pm I Meeting Hall IV

Chairs:
S. Fulda (Switzerland)

Summary

Infra-slow oscillations (ISOs) refer to periodic alterations in electrophysiological signals reoccurring in cycles that range from 10 to 100 s (corresponding frequencies, 0.01 to 0.1 Hz) and are generated spontaneously in the brain during both waking and sleep states. ISOs are of particular importance for sleep, during which sleep stage-specific oscillatory background activity and phasic events can periodically interchange over tens of seconds. ISOs have been observed in both humans and animals across brain structures and spatial scales, including, but not limited to, the dynamics of single unit firing rates, the fluctuations in the direct electrical potential, amplitude coupling across frequencies and also hemodynamic activities. Infra-slow periodicities are also prominently apparent in leg movement activity during sleep and in slow cardiovascular rhythms, thus making ISOs interesting candidates for translational approaches in the field concerning sleep disorders. The aim of the present symposium is to provide a broad overview over recent developments in this field. We will trace the phenomenology of ISOs from cell preparations in vitro to full-band EEG in sleep-disordered patients, including local field potentials and EEG studies in mice and power-band fluctuations in healthy sleepers. The symposium brings together experts from both basic and clinical research to aim for a differentiated introduction to the field that is accessible and relevant to both basic and clinical sleep researchers. The first presentation will focus on ISOs in thalamic neurons that generate such rhythms at the level of the local field potential and single neuron recordings, thereby grouping faster rhythms and possibly controlling seizures occurrence. Similar ISOs are also present in acute thalamic slice preparations where they also group faster network activity and paroxysmal discharges. Mechanistic investigations suggest an intriguing role for adenosine signaling and astrocytes in the genesis of thalamic ISOs and indicate that infra-slow fluctuations in the intact brain may involve a complex interaction between neuronal and non-neuronal cells. Second, based on EEG, local field potentials, autonomic nervous system activity, and behavioral responsivity in sleeping mice, the possible functions of ISOs for sleep in mammals will be addressed. Among the highlights is the demonstration of a prominent 40-50-s long ISO in sleep spindle recurrence, hippocampal ripples, and variations in heart rate that point to a repetitive cycling of the brain between offline periods and enhanced environmental alertness. The third presentation will translate these intriguing results to human sleep with a focus on the infra-slow, spontaneous fluctuations of sleep spindle activity and delineate how they prevail in different sleep stages, their specificity to fast spindles, their pattern of predominance over cortical areas and their putative role in the organization of sleep-mediated memory consolidation processes. The last presentation will present new data on ISOs during sleep assessed with full band EEG which offers the possibility to record infra-slow oscillations directly on the level of the surface EEG. The presentation will focus on new data systematically characterizing ISO across all stages of sleep relating it to microstructural events of sleep in both healthy subjects and subjects with RLS/PLMS.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Recognize the phenomenology of infra-slow oscillations in human and rodent sleep
- Identify the characteristics and mechanisms of infra-slow oscillations that coordinate faster EEG rhythms such as alpha waves and their potential relationship to seizure recurrence
- Describe how sleep architecture is organized between the micro- and macroscale
- Integrate new knowledge about infra-slow oscillations with established clinical observations such as the cyclic alternating pattern
- Recognize the potential of infra-slow oscillations as a translational parameter

Target Audience

Basic and clinical sleep researchers

3:30pm - 3:35pm	**Introduction** *S. Fulda (Switzerland)*
3:35pm - 3:55pm	**Infra-slow oscillations in the thalamus: Mechanisms and significance** *S. Hughes (United Kingdom)*
3:55pm - 4:15pm	**Wake-up or sleep-through the noise: Infra-slow timing of arousal** *A. Lüthi (Switzerland)*
4:15pm - 4:35pm	**Human sleep spindle activity — ultra-powerful at infra-slow timing?** *F. Weber (Germany)*
4:35pm - 4:55pm	**Sleep infra-slow oscillations in full band EEG recordings** *S. Fulda (Switzerland)*
4:55pm - 5:00pm	**Question and answer** *S. Fulda (Switzerland)*

S95 Hypertrophic cardiomyopathy and sleep disordered breathing: Implications for atrial arrhythmias and sudden cardiac death

3:30pm - 5:00pm I North Hall

Chairs:
V. Somers (United States)

Summary
This course will provide a contemporary and comprehensive update on the role of sleep apnea in patients with hypertrophic cardiomyopathy (HCM). HCM is the most common genetic heart muscle disease, associated with refractory symptoms, atrial fibrillation, heart failure, syncope, stroke, ventricular arrhythmias and sudden cardiac death. Greater awareness and improved screening with cardiac imaging has led to an increase in diagnosis and suggests a prevalence as high as 1 in 200. Leaders in the field will discuss exciting new findings of the prevalence of sleep apnea and how this may contribute to the known symptoms, arrhythmias and heart failure burden. Sleep experts will be increasingly asked to assess patients with HCM for sleep disordered breathing which is a fascinating but highly challenging condition to manage. The speakers will present the latest research in (1) sleep and Hypertrophic cardiomyopathy; (2) how this impacts screening and risk stratification for atrial arrhythmias, stroke and sudden cardiac death; (3) therapeutic implications; and (4) anticipated changes to clinical guidelines.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize and describe hypertrophic cardiomyopathy (HCM) phenotypes, clinical burden and family screening
- Identify the prevalence, severity and types of sleep disordered breathing seen in HCM
- Review the pathophysiological mechanisms of untreated sleep apnea in HCM
- Describe risk factors and preventative strategies targeting sleep disordered breathing for preventing arrhythmia and sudden death in HCM

Target Audience
Sleep medicine physicians and allied health personnel, cardiologists, pulmonologists, nurses, physician assistants, and student/resident/fellow trainees in these disciplines

3:30pm - 3:35pm	**Introduction** *V. Somers (United States)*
3:35pm - 3:55pm	**Hypertrophic cardiomyopathy – one gene, many effects and difficult to manage** *A. Chahal (United Kingdom)*
3:55pm - 4:15pm	**Sleep apnea: Pathophysiology and CV consequences – implications for HCM** *V. Somers (United States)*
4:15pm - 4:35pm	**Atrial arrhythmias in hypertrophic cardiomyopathy – sleep apnea may be the missing link** *T. Konecny (United States)*
4:35pm - 4:55pm	**Prevalence of sleep apnea in hypertrophic cardiomyopathy – what our preliminary data shows us** *T. Kara (United States)*
4:55pm - 5:00pm	**Question and answer** *V. Somers (United States)*

S96 Autonomic disorders in sleep medicine

3:30pm - 5:00pm I Club A and B

Chairs:
M. Miglis (United States)

Summary
Sleep is a complex homeostatic function, regulated in part by control nuclei of the autonomic nervous system. When sleep is disrupted, symptoms of autonomic impairment may emerge. In addition, many primary disorders of autonomic dysfunction can disrupt sleep. This symposium will review the anatomy of the autonomic and sleep systems, the physiology of autonomic function during normal sleep, and some of the techniques used to measure autonomic function. Autonomic and neurological disorders with prominent sleep impairment will be reviewed, including fatal familial insomnia, familial dysautonomia, and neurodegenerative disorders associated with REM sleep behavior disorder. Autonomic impairment in obstructive and central sleep apnea will also be discussed.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize the importance of healthy sleep in homeostasis and autonomic balance
- Recognize the signs of autonomic dysfunction in patients with sleep disorders
- Identify the mechanisms of autonomic dysfunction in obstructive and central sleep apnea
- Identify the pathophysiology of fatal familial insomnia and the implications for other sleep disorders
- Describe the connection between REM sleep behavior disorder and autonomic function

Target Audience
Sleep clinicians, sleep researchers, neurologists and physiologists

3:30pm - 3:33pm	**Introduction** *M. Miglis (United States)*
3:33pm - 3:50pm	**Sleep and autonomic impairment: Why does it matter?** *P. Cortelli (Italy)*
3:50pm - 4:07pm	**Sleep apnea and cardiovascular autonomic dysfunction** *C. Lombardi (Italy)*
4:07pm - 4:24pm	**Fatal familial insomnia: A model of sleep and autonomic dysfunction** *G. Calandra Buonaura (Italy)*
4:24pm - 4:41pm	**Sleep disordered breathing in familial dysautonomia: Implications for sudden death during sleep** *J.A. Palma (United States)*
4:41pm - 4:58pm	**Autonomic impairment in REM sleep behavior disorder** *M. Miglis (United States)*
4:58pm - 5:00pm	**Question and answer** *M. Miglis (United States)*

S33 Sleep and the kidney

3:30pm - 5:00pm I Club H

Chairs:
P. Hanly (Canada)

Summary
It has been recognized for some time that individuals with end-stage renal disease (ESRD) have a high prevalence of sleep apnea and non-respiratory sleep disorders. More recently, there has been growing interest and evidence that intermittent hypoxia associated with sleep apnea can injure the kidney. Consequently, there is a bidirectional relationship between sleep and renal function that has many clinical implications for patients with pre-dialysis dependent chronic kidney disease (CKD) and those who have reached ESRD and are dialysis-dependent. Experimental animal data will be presented that show how hypoxia can damage the kidney (Dr Ayas). The clinical implications of this phenomenon for patients with sleep apnea will be reviewed (Dr Hanly). The pathogenesis and management of sleep apnea in patients who have ESRD will be discussed (Dr Lyons). Finally, the role of non-respiratory sleep disorders and how sleep can be improved in patients with ESRD will be addressed (Dr Novak). DETAILED DESCRIPTION: The prevalence of chronic kidney disease (CKD) is increasing and is currently found in more than 10% of adults. Although this has been largely attributed to diabetes, hypertension and obesity, these chronic medical disorders do not fully explain the growth in prevalence of CKD; furthermore, many CKD patients progress to end-stage renal disease (ESRD) despite optimal management of these conditions. This raises the possibility that other untreated co-morbidities play a role. Obstructive sleep apnea occurs in up to 40% of CKD patients who are not dialysis dependent and the accompanying hypoxia is associated with accelerated deterioration in kidney function. There are multiple potential mechanisms for this association, which have been explored in experimental animal models and clinical studies. They include both direct effects of hypoxia on the kidney and indirect mechanisms through oxidative stress, endothelial dysfunction, inflammation, sympathetic nervous system activation and hypertension, and increased renin angiotensin system (RAS) activity within the kidney. Once patients reach ESRD, there are many potential consequences for sleep and breathing. Sleep apnea is found in over 50% of patients with ESRD and develops through a variety of mechanisms that are specific to this patient population. These mechanisms include alteration in the central control of breathing and mechanical changes in the upper airway such as rostral fluid shift. This provides the opportunity for alternative therapies such as modification of how dialysis is administered. Co-existing sleep apnea has the potential to impact important clinical outcomes such as cardiovascular morbidity and mortality which is the single greatest complication in this patient population. ESRD also increases the prevalence and severity of non-respiratory sleep disorders, such as restless legs syndrome, periodic limb movement disorder and insomnia. Once again, the underlying mechanisms are varied and include the metabolic consequences of kidney failure and the type of renal function replacement that is used to treat it. The challenging combination of ESRD and poor sleep is an increasingly common problem for sleep medicine practitioners to deal with. The aims of this symposium are to address "both sides of the coin" ie the potential impact of sleep apnea on the kidney and the consequences of kidney failure for sleep and breathing. Presentations will include both basic science and clinical data. All 4 speakers have published research on this topic and have clinical experience in the management of sleep and breathing in this patient population.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Recognize how hypoxia can injure the kidney
- Assess the potential relevance of OSA to patients with chronic kidney disease
- Recognize the pathogenesis and management of sleep apnea in patients with end-stage renal disease
- Describe how sleep can be improved in patients with end-stage renal disease

Target Audience
Investigators interested in pathogenesis of sleep disorders, clinicians of patients who have impaired sleep and kidney function

3:30pm - 3:35pm	**Introduction** *P. Hanly (Canada)*
3:35pm - 3:55pm	**Impact of intermittent hypoxia on kidney histology: Evidence from animal models** *N. Ayas (Canada)*
3:55pm - 4:15pm	**Effect of sleep apnea on kidney function: Evidence from human studies** *P. Hanly (Canada)*
4:15pm - 4:35pm	**Sleep-disordered breathing in End-Stage Renal Disease (ESRD)** *O. Lyons (Canada)*
4:35pm - 4:55pm	**Non-respiratory sleep disorders in End-Stage Renal Disease (ESRD)** *M. Novak (Canada)*
4:55pm - 5:00pm	**Question and answer** *P. Hanly (Canada)*

O20 Basic research oral abstract presentations

3:30pm – 5:00pm I Club D and E

Chairs:

3:30pm – 3:45pm	**URINARY PROTEINS AS POTENTIAL BIOMARKERS FOR ADULT PATIENTS WITH OBSTRUCTIVE SLEEP APNEA** *M. Kohli (India)*
3:45pm – 4:00pm	**HEARTBEAT-RELATED ACTIVITY OF CORTICAL NEURONS IN THE SLEEP-WAKE CYCLE IN CATS** *V. Lavrova (Russian Federation)*
4:00pm – 4:15pm	**ULTRADIAN RHYTHMICITY IN SLEEP-WAKEFULNESS IS COLOUR-RELATED IN NESTLING BARN OWLS** *M.F. Scriba (Switzerland)*
4:15pm – 4:30pm	**HYPOCRETIN (OREXIN) SIGNALING IN DISTINCT NEURONAL GROUPS DIFFERENTIALLY REGULATES OSCILLATORY COMPONENTS OF WAKEFULNESS AND SLOW-WAVE-SLEEP** *A. Vassalli (Switzerland)*
4:30pm – 4:45pm	**MULTITASKING NETWORKS IN THE LATERAL HYPOTHALAMUS: THE ROLE INHIBITORY NEURONS IN SLEEP AND METABOLISM** *C. Gutierrez Herrera (Switzerland)*
4:45pm – 5:00pm	**SLOW-WAVE ENERGY ENHANCEMENT ASSOCIATED WITH REDUCED SYNUCLEINOPATHY IN MURINE MODEL OF PARKINSON'S DISEASE** *M.M. Morawska (Switzerland)*

O21 Insomnia oral abstract presentations

3:30pm – 5:00pm I Meeting Hall V

Chairs:
Y. Inoue (Japan)

3:30pm – 3:45pm	**TREATMENT OF INSOMNIA WITH MELATONIN IN PATIENTS AGED 45-60 YEARS OLD: A RANDOMIZED DOUBLE BLIND PLACEBO-CONTROLLED STUDY** *Y. Qian (China)*
3:45pm – 4:00pm	**MARKERS FOR HYPNOTIC ABUSE LIABILITY: CORTISOL IN INSOMNIA** *T. Roehrs (United States)*
4:00pm – 4:15pm	**IS THE INSOMNIA SEVERITY INDEX CUTOFF FOR REMISSION CORROBORATED BY SLEEP DIARY DATA AND PATIENT'S PERCEPTION?** *S. Beaulieu-Bonneau (Canada)*
4:15pm – 4:30pm	**FALSE FEEDBACK ABOUT SLEEP DELIVERED VIA ACTIGRAPHY BIASES DAYTIME SYMPTOM REPORTS: IMPLICATIONS FOR INSOMNIA DISORDER AND WEARABLE DEVICES** *D. Gavriloff (United Kingdom)*
4:30pm – 4:45pm	**A POTENTIAL PHARMACOLOGICAL TARGET OF INSOMNIA: THE MOLECULES INVOLVED IN THE CA2+-DEPENDENT HYPERPOLARIZATION PATHWAYS PLAY A PIVOTAL ROLE IN THE REGULATION OF SLEEP HOMEOSTASIS** *S. Shi (Japan)*
4:45pm – 5:00pm	**DATA-DRIVEN TOPIC ANALYSIS OF HIGH DENSITY EEG REVEALS CONCOMITANT SUPERFICIAL SLEEP DURING DEEP SLEEP IN INSOMNIA** *J.A.E. Christensen (Denmark)*

S97 Sleep and sexual dysfunction

5:30pm - 7:00pm I Meeting Hall V

Chairs:
H.-W. Shin (Republic of Korea)

Summary

Sleep disturbance and obstructive sleep apnea (OSA) are known to be related with sexual and reproductive dysfunction. Moreover, recent prospective findings have suggested those two sleep-related problems as an independent risk factor for the sexual dysfunction. Several observational studies suggested the significant association between them, but the exact mechanism for this association is difficult to clarify, since multiple factors such as obesity, sleep fragmentation, intermittent hypoxia, and psychological distress act as confounding factors in human. Animal studies mimicking OSA have showed that intermittent hypoxia contributes to erectile dysfunction, reduced fertility, and decreased libido. It has been shown that the effects on erectile dysfunction or sexual function are reversible by treatment of OSA including continuous positive airway pressure (CPAP) and oral appliances, but it remained inconclusive. In this symposium, the epidemiological findings on the association between sexual/reproductive function and OSA will be reviewed. The symposium will also discuss the influence of sleep disturbance and OSA on sexual and reproductive function, based on the data from prospective and animal studies. Finally, the symposium will discuss the beneficial effect of CPAP treatment for OSA patients on erectile or sexual function.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Summarize the existing literature on the epidemiology of sexual function and OSA
- Review the epidemiologic and experimental findings on the effect of sleep disturbance and OSA on sexual and reproductive function
- Describe the possible mechanisms by which sleep disturbance and OSA result in sexual dysfunction
- Describe the importance of OSA treatment to improve sexual function

Target Audience

Sleep clinicians caring for patients with increased risk of sexual dysfunction; Sleep clinicians and researchers in Sleep-disordered breathing

5:30pm - 5:35pm	**Introduction** *H.-W. Shin (Republic of Korea)*
5:35pm - 5:55pm	**Epidemiology of sexual function and OSA** *K. Melehan (Australia)*
5:55pm - 6:15pm	**Sleep and Sexual function: Evidences from animal models** *M. L. Andersen (Brazil)*
6:15pm - 6:35pm	**Effect of Sleep disturbance and OSA on sexual and reproductive function evidence from humans** *C. Hoyos (Australia)*
6:35pm - 6:55pm	**Does OSA treatment improve sexual function?** *H.-W. Shin (Republic of Korea)*
6:55pm - 7:00pm	**Question and answer** *H.-W. Shin (Republic of Korea)*

S98 Sleep and interventions in children and young people

5:30pm - 7:00pm I North Hall

Chairs:

L. McLay (New Zealand)

Summary

Sleep problems are highly prevalent among children and young people with autism spectrum disorder (ASD). Such problems can include delayed sleep onset latency, frequent and/or prolonged night-time awakenings, and co-sleeping. If untreated, sleep problems in children and young people with ASD are likely to persist. However, little is known about the prevalence of sleep problems in young adults with ASD, and its predictors. In the first presentation Associate Professor Amanda Richdale will examine the role of arousal in 75 young people with ASD aged 15-25 years. Results of this study indicated that those with sleep problems differed significantly from those without sleep problems. The findings suggest that arousal, particularly somatic arousal, underlies insomnia symptoms in ASD. Improving our understanding of those factors that contribute to poor sleep can lead to more targeted behavioral and pharmacological interventions. The presentation of sleep problems in children with ASD is complex. In order to identify possible treatments, a comprehensive assessment must be undertaken. In the treatment of sleep disturbance, where multiple, individual variables influence the behavior strong connections between assessment and treatment are necessary. One approach to forging such connections is Functional Behavioural Assessment (FBA). FBA is an evidence-based method used to specify a particular challenging behaviour and to identify the antecedents (environmental context) and the consequences affecting the behaviour. To date, there is little research investigating the use of FBA in the treatment of sleep disturbance. Identifying efficacious, durable, and socially acceptable interventions to promote the health and development of young children is a shared goal among parents, pediatricians, behavior analysts, and other child-care professionals. In the second presentation, Assistant Professor Sandy Jin will focus on critical features of empirically supported and comprehensive behavioral interventions for sleep problems. Discussion will include (a) an assessment process (including FBA) that allows caregivers and clinicians to identify the likely causes of common sleep problems, including delayed sleep onset, night and early awakenings, nighttime routine noncompliance, and sleep interfering behavior, and (b) a comparative evaluation of the efficacy of and consumer preference for function-based treatments designed to reduce problem behavior that interferes with sleep onset. In the third presentation, Dr Laurie McLay will build on the themes introduced by Assistant Professor Jin, by presenting data from a series of single-case studies that used FBA to develop individualized, parent-implemented, behaviourally-based interventions for unwanted co-sleeping in children with ASD. Data will be presented on the effect of interventions for unwanted co-sleeping; and the maintenance of treatment effects over time. In the final presentation, Associate Professor Karyn France will present the result of a qualitative study that investigated the successes and challenges experienced by beginning clinicians who were involved in a research project investigating the use of FBA to treat sleep problems in children with ASD. Content analysis from this study revealed a number of important themes, that may represent important developmental areas for clinicians preparing to enter the field, as well as having important implications for the assessment and treatment of sleep problems.

Learning Objectives

Upon completion of this CME activity, participants should be able to:

- Recognize the complexity of sleep and its phenomenology in autism
- Distinguish the components of functional behavioral assessment and its application in the treatment of behavioral sleep presentation in autism
- Recognize the challenges in doing functional behavioral assessment as a clinician and plan appropriate learning measures and support prior to implementing these

Target audience

Clinicians and academics who are working with, or interested in, children and young people with autism spectrum disorder including: pediatricians; general practitioners; psychologists; behavior analysts; and child care professionals

5:30pm - 5:35pm	**Introduction** *L. McLay (New Zealand)*
5:35pm - 5:55pm	**Exploring the role of arousal in predicting sleep problems in youth and young adults with ASD** *A. Richdale (Australia)*
5:55pm - 6:15pm	**Through the lens of a contingency: Designing individualized, functionbased and consumer-friendly interventions for sleep problems of children diagnosed with autism spectrum disorder** *S. Jin (United States)*
6:15pm - 6:35pm	**Using functional behavioral assessment to inform treatments for unwanted co-sleeping in children with autism** *L. McLay (New Zealand)*
6:35pm - 6:55pm	**FBA for sleep with clinically complex children with autism: Lessons from experienced and new clinicians** *K. France (New Zealand)*
6:55pm - 7:00pm	**Question and answer**

S99 Young Investigator: Oral presentation

5:30pm - 7:00pm I Club D and E

Chairs:
F. Sixel-Döring (Germany), K. Sonka (Czech Republic)

5:30pm – 5:45pm	**SEEKING A NEW STANDARD: A NOVEL CHARACTERIZATION OF SLEEP SPINDLES THROUGH TIME-FREQUENCY PEAK ANALYSIS** *M. Prerau (United States)*
5:45pm – 6:00pm	**ULTRA-SLOW (0.0002 HZ) FLUCTUATIONS IN HUMAN INTRACRANIAL RECORDINGS CORRELATE WITH SLEEP CYCLES** *G. Piantoni (United States)*
6:00pm – 6:15pm	**THE EVOLUTIONARY CONSERVED MICRORNA MIR-137 REGULATES GENE EXPRESSION AND DIURNAL RHYTHM OF THE WAKE-PROMOTING HYPOCRETIN NEUROPEPTIDES** *A. Holm (Denmark)*
6:15pm – 6:30pm	**EXPERIENCING FEAR IN DREAMS RELATES TO BRAIN RESPONSES TO AVERSIVE STIMULI DURING WAKEFULNESS** *L. Perogamvros (Switzerland)*
6:30pm – 6:45pm	**TRAJECTORIES OF USE OF OVER-THE-COUNTER AND NATURAL PRODUCTS FOR SLEEP: A FIVE YEAR FOLLOW-UP** *J.M.Y. Cheung (Canada)*
6:45pm – 7:00pm	**EXPLORATION OF CARDIAC AUTONOMIC FUNCTION BY MYOCARDIAL 123-I-MIBG SCINTIGRAPHY IN NARCOLEPSY TYPE 1** *L. Barateau (France)*

S100 The characteristics of type 2 narcolepsy in Asian patients

5:30pm - 7:00pm I Congress Hall

Chairs:
S.-C. Hong (Republic of Korea)

Summary
The onset of narcolepsy is known to be related with autoimmune process. There would be genetic differences between Caucasian and Asian narcolepsy patients, which could lead to unique clinical presentation, functional impairment, or study findings. This symposium includes the metabolic characteristics of Asian type 2 narcolepsy patients through the study of multiple sleep latency test variables, functional imaging studies of children and adolescent narcolepsy patients, and HLA typing, hypocretin levels in the CSF studies. As long as 10 year-interval follow up of MLST variables in type 2 patients will be demonstrated, enabling a deeper understanding in this specific group of narcolepsy patients. Moreover, specific MSLT characteristics and PSG variables in type 2 narcolepsy patients will be demonstrated. Furthermore, specific functional brain imaging results on this group will be demonstrated, with detailed comparisons with the results from type 1 narcolepsy patients and controls. Compared to normal controls, type 2 narcoleptics showed differential PET findings when compared with controls of type 1 narcoleptics. Overview of type 2 narcolepsy cases will demonstrate some interesting clinical features, comorbidities, polysomnography, multiple sleep latency test variables noted that harbor clinically meaningful implications. Valuable data on children and adolescent narcolepsy patients in China will be presented, including HLA typing, hypocretin level in the CSF results, during the diagnosis and follow-up for patients suffering from Children narcolepsy. Comparisons will be made among type 1 narcoleptics, type 2 narcoleptics and idiopathic hypersomnia.

Learning Objectives
Upon completion of this CME activity, participants should be able to:
- Identify specific objective sleep parameters involved in the diagnosis of type 2 narcolepsy patients
- Identify how the functional imaging (PET) elucidate the brain function in children and adolescent type 2 narcolepsy patients
- Identify the clinical characteristics and pathophysiology of type 2 narcolepsy patients
- Identify the autoimmune processes involved in type 2 narcolepsy patients in children and adolescents

Target Audience
Sleep physicians, professors, clinical and basic researchers, students

5:30pm - 5:32pm	**Introduction** *S.-C. Hong (Republic of Korea)*
5:32pm - 5:49pm	**Long-term follow up of MSLT variables in type 2 narcolepsy patients in Korea** *S.-C. Hong (Republic of Korea)*
5:49pm - 6:06pm	**Characteristics of MSLT and PSG variables in type 2 narcolepsy patients** *Y. Inoue (Japan)*
6:06pm - 6:23pm	**PET studies and lab finding in type 2 narcolepsy patients** *Y.-s. Huang (Taiwan)*
6:23pm - 6:40pm	**Overview of type 2 narcolepsy cases in Korea** *Y.H. Um (Republic of Korea)*
6:40pm - 6:57pm	**Type 2 narcolepsy in children and adolescents** *F. Han (China)*
6:57pm - 7:00pm	**Question and answer** *S.-C. Hong (Republic of Korea)*

O23 Sleep breathing disorders and research oral abstract presentations

5:30pm – 7:00pm I Terrace 1

Chairs:

5:30pm – 5:45pm	**ASSOCIATION BETWEEN DESATURATION INDICES AND COMORBIDITIES IN PATIENTS WITH OSA- A CROSS SECTIONAL STUDY** *A. Choudhury (India)*
5:45pm – 6:00pm	**EVOLUTION OF SLEEP ARCHITECTURE AND LEVEL OF ALERTNESS MEASURED BY MWT IN APNEIC PATIENTS TREATED BY GENIOGLOSSUS STIMULATION (INSPIRE ® THERAPY)** *P. Philip (France)*
6:00pm – 6:15pm	**TREATMENT OF SLEEP APNEA SYNDROME BY ELECTRICAL AURICLE STIMULATION USING MINIATURIZED SYSTEM OF SECOND GENERATION** *V. Donic (Slovakia)*
6:15pm – 6:30pm	**MICROSTRUCTURE OF RESPIRATORY AROUSALS IN PATIENTS WITH SLEEP-DISORDERED BREATHING** *H. Gouveris (Germany)*
6:30pm – 6:45pm	**INVESTIGATION OF NON-PAINFUL TACTILE STIMULI VIA BISPECTRAL INDEX SYSTEM DURING SLEEP** *M. Ozgoren (Turkey)*
6:45pm - 7:00pm	**ROLE OF HYPO PHARYNGEAL COLLAPSE IN OBSTRUCTIVE SLEEP APNEA - HOW TO ADDRESS IT** *R. Anand (India)*

T09 Experiences in sleep medicine around the world

5:30pm - 7:00pm I Club H

Chairs:
S. Keenan (United States), O. Ludka (Czech Republic)

5:30pm - 5:32pm	**Introduction** *S. Keenan (United States)*
5:32pm - 5:44pm	**Clinical practice in Canada** *M. Eden (Canada)*
5:44pm - 5:56pm	**Clinical practice in Japan** *N. Tachibana (Japan), H. Muraki (Japan),*
5:56pm - 6:08pm	**Clinical practice in the United States** *A. Robinson (United States)*
6:08pm - 6:20pm	**Clinical practice in the Czech Republic** *M. Pretl (Czech Republic)*
6:20pm - 6:32pm	**Clinical practice in Thailand** *M. Veeravigrom (Thailand)*
6:32pm - 6:44pm	**Clinical practice in Iceland** *E. Sif Arnardottir (Iceland)*
6:44pm - 7:00pm	**Question and answer**

Closing Ceremony

7:00 - 7:30 I Congress Hall

Chairs:
C. Morin (Canada)

7:00 - 7:30	**Closing remarks** *C. Samuels (Canada), R. Gruber (Canada), C. Bastien (Canada)*

Poster Abstracts

Poster abstracts should be posted on the boards per session. For Poster Sessions 1, 3 and 5, poster abstracts should be posted on the boards by 10:00am and removed between 1:30pm and 2:00pm. Authors need to be present by posters during the scheduled time of Noon to 12:30PM. For Poster Sessions 2, 4 and 6, poster abstracts should be posted on the boards between 2:00pm and 3:00pm and removed by 8:00pm. Authors need to be present by posters during the scheduled time of 5:00pm to 5:30pm.

Panorama Hall

Poster Sessions

Monday, October 9

Poster 1	12:00pm - 12:30pm
Poster 2	5:00pm – 5:30pm

Tuesday, October 10

Poster 3	12:00pm - 12:30pm
Poster 4	5:00pm – 5:30pm

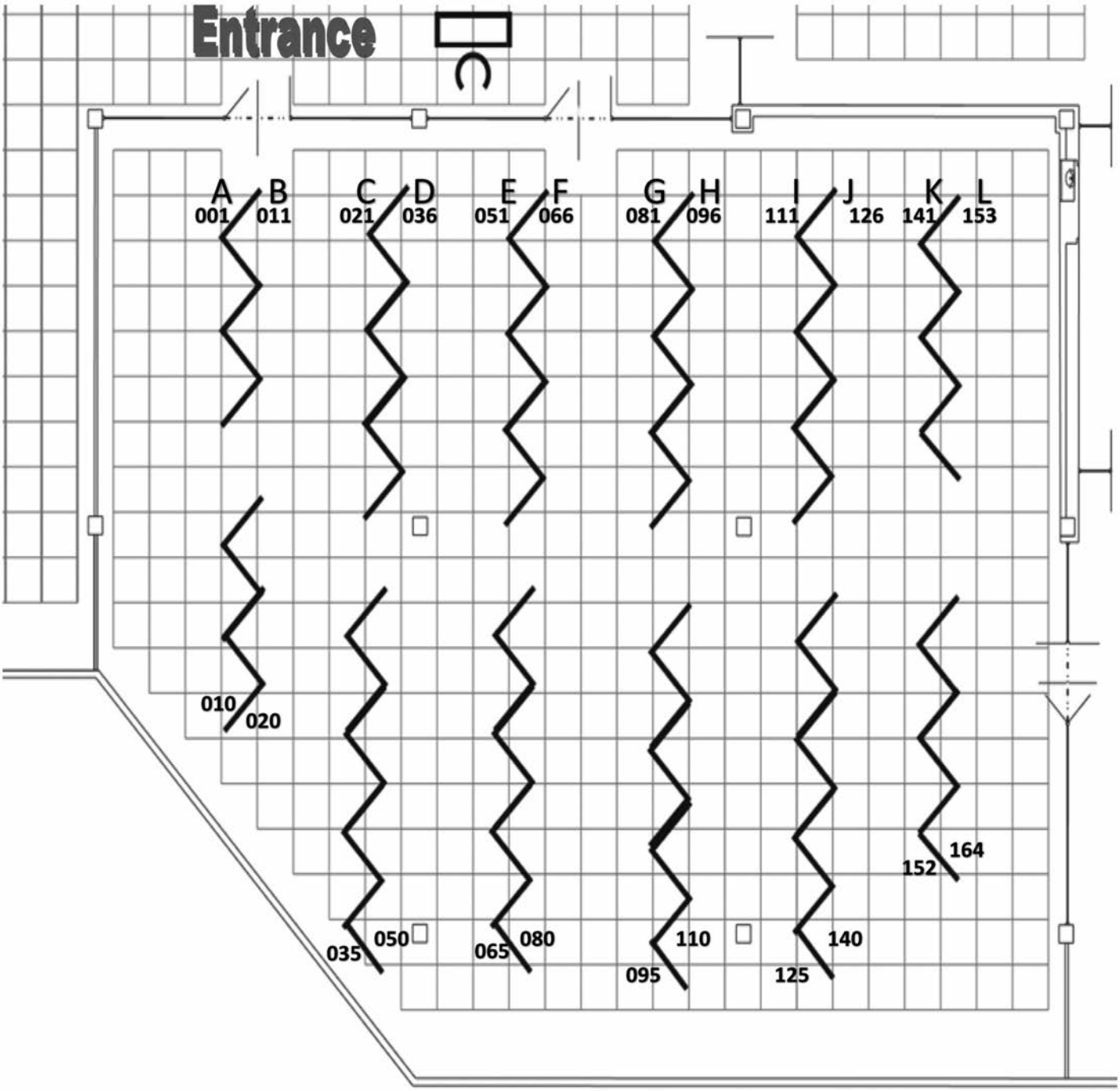
Wednesday, October 11

Poster 5	12:00pm - 12:30pm
Poster 6	5:00pm – 5:30pm

Poster Board Numbers

Row A:	001–010
Row B:	011-020
Row C:	021-035
Row D:	036-050
Row E:	051-065
Row F:	066-080
Row G:	081-095
Row H:	096-110
Row I:	111-125
Row J:	126-140
Row K:	141-152
Row L:	153-164

Poster Abstract Board Floor Layout - Panorama Hall



CATAPLEXY: FROM FUNDAMENTALS TO THE CLINIC

INVITATION

TUESDAY 10TH OCTOBER 2017, 12:30-2:00 PM
CONGRESS HALL

Satellite Symposium of the World Sleep Congress

bioprojet

Sponsors/Exhibitors

World Sleep 2017 Partners

World Sleep 2017 is pleased to announce this program was made possible, in part, by an Independent Medical Education Grant from Jazz Pharmaceuticals, Inc., as well as sponsorships from Bioprojet, Merck, Natus, Philps and Teva. We appreciate their support of our mission to provide the best in sleep medicine.

Booth 265 **aamsinfo.org**

The Academy of Applied Myofunctional Sciences (AAMS) is a non-profit (501c3 USA based) scientific society engaged in advancing research, scientific standards, education, and public health related to myofunctional therapy (MFT) around the world. The AAMS has helped start 11 regional non-profit scientific societies in the area of MFT around the world (Brazil, Japan, Scandinavia et al) and currently is helping in the formation of 14 new societies (Hong Kong/Taiwan/China, Australia, United Kingdom, et al). Join us as we help advance this important emerging field.

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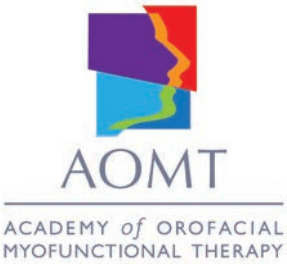
aastweb.org

The American Association of Sleep Technologists (AAST) is the premier allied health membership association for professionals dedicated to improving the quality of sleep and wakefulness in all people. The AAST is committed to promoting and advancing the sleep technologist profession while meeting the professional and educational needs of more than 4,200 members.



Booth 265 **aomtinfo.org**

The Academy of Orofacial Myofunctional Therapy (AOMT) is the leading post-graduate institution for allied health professional training in the area of orofacial myofunctional therapy. Additionally the AOMT helps facilitate research, curriculum development, and public health initiatives and has relationships with more than 40 universities and hospital groups around the world.

Booth 310 **advancedbrainmonitoring.com**

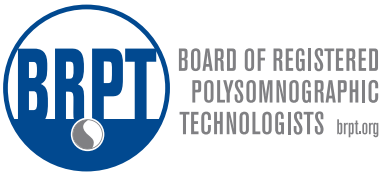
Advanced Brain Monitoring, Inc. is an industry leader in the development of novel diagnostic and treatment technologies for the sleep medicine field. The Sleep Profiler is clinically-validated system for sleep and sleep-disordered-breathing in the home or ICU. Night Shift and Apnea Guard deliver therapy to those suffering from Sleep Apnea.

Booth 610 **ardeninnovations.com**

Arden Innovations is the sole worldwide distributor for the CPAP Hose Lift and the BedSide CPAP Table – both innovative products that are essential elements to any and every successful CPAP ~ Sleep Apnea treatment System. Both products were designed for dual purpose use – at Home, and during Travel.

Booth 450 **bioprojet.fr/bioprojet-pharma.html**

Bioprojet Created in 1982 by Dr Jeanne-Marie Lecomte and Pr Jean-Charles Schwartz, Bioprojet is a medium size, independent Research and Development pharmaceutical company. Bioprojet's purpose is to design, select and develop innovative drugs, mostly in the CNS field, acting on novel biological targets discovered in collaboration with basic science institutes like French INSERM.



Booth 620 **brpt.org**

Board of Registered Polysomnographic Technologists (BRPT) The mission of The Board of Registered Polysomnographic Technologists is to build upon its history as the global leader in sleep technologist credentialing and certification; provide high quality sleep technology products and services that inspire professional excellence, recognition, and lifelong learning; and create long-term value for credential and certificate holders.

Booth 605 **camntech.com**

CamNtech Ltd has over 20 years of experience with wearable technology for sleep monitoring. The MotionWatch is one of the smallest, lightest Actigraphy devices currently available with recording for up to 120 days. Manufactured to the highest standards in our ISO13485 facility, our products are CE marked, FDA cleared medical devices.

Booth 470 **cerevast.com**

Cerevast Medical, Inc. is a neuroscience company specializing in treatment of neurological disorders. Neuros™ Sleep utilizes pulsed Transdermal Electrical Stimulation (pTES) to modulate nerves in the face and neck which form pathways to regions of the brain involved in regulation of sleep and mood. Cerevast is also developing transcranial ultrasound (TUS) to enhance thrombolytic effect after ischemic stroke and improve clinical outcomes during post-stroke rehabilitation.

Booth 615 **cidelec.net**

CIDELEC is a French company manufacturing diagnostic devices for sleep breathing disorders. Our polygraph and polysomnograph use our unique PneaVoX technology based on tracheal sound analysis. CIDELEC systems offer a complete range of portable ambulatory devices and sleep laboratory system.

Booth 345 **compumedics.com.au**

Compumedics Ltd, founded in 1987, is a global leader in innovative medical technology solutions for sleep diagnostics, neuro-diagnostics and brain research. Compumedics offers a comprehensive range of innovative solutions for in-lab, ambulatory, home and research applications. Compumedics has produced many world's-first such as the High-Definition GraelHD system and the Bluetooth-enabled, PSG-Anywhere SomtePSG. Compumedics leads the sleep software market with their ProFusion Sleep Suite and neXus Lab management products.

Booth 435 **condorinst.com.br/en**

Condor Instruments offers high quality wrist actimeters (actigraphs) for medical and research applications, specially tailored for sleep and circadian disorders monitoring.

Booth 315 **dymedix.com**

Dymedix Diagnostics, Inc., (DDI) is a United States based medical device company that manufacturers and globally markets innovative sleep diagnostic (PVDF) reusable and disposable airflow, snoring, and respiratory effort sensors in adult and pediatric versions. DDI is committed to developing technology to advance the science of sleep diagnostics medicine.

Booth 500 **emfit.com**

Team Emfit consists of 20 highly motivated, skilled and experienced professionals. Together the team has over 150 years of experience at Emfit. Many of the members are owners at the company, living in Finland, Germany, USA, Canada and China. The whole team has a complementary set of skills including science, engineering, sales experience and entrepreneurship.

Booth 630 **earls.eu**

European Alliance for Restless Legs Syndrome (EARLS) aims to increase awareness of restless legs syndrome. EARLS is an umbrella organization of national patient advocacy groups. At World Sleep 2017, EARLS will present the results of a major study on the cost of mis- and late diagnoses and treatment of RLS, done in collaboration with the European Brain Council and the London School of Economics.

Booth 475 **esrs.eu**

European Sleep and Research Society (ESRS) is an international scientific non-profit organization and promotes all aspects of sleep research and sleep medicine. This includes the publication of the Journal of Sleep Research(JSR), the organization of scientific meetings, and the promotion of training and education, the dissemination of information, and the establishment of fellowships and awards.

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Elsevier is a world-leading provider of information solutions that enhance the performance of science, health, and technology professionals, empowering them to make better decisions, and deliver better care. Visit our booth to find out about our Sleep Medicine journals, how to publish (open access), and how to enrich your journal article with exciting innovation.

Booth 210 **clinicaltrial.avadel.com**

Flamel Ireland Limited is a subsidiary of Avadel Pharmaceuticals plc and is responsible for the Company's clinical development programs. Flamel is currently conducting a Phase III clinical trial with its new extended-release oral suspension formulation of sodium oxybate. The study is currently recruiting patients in the USA, Canada and Europe.

Booth 465 **hypersomniafoundation.org**

Hypersomnia Foundation is a US public 501(c)3 nonprofit with a mission to improve the lives of people with idiopathic hypersomnia and related disorders by advocating on their behalf, providing support, educating the public and healthcare professionals, raising awareness, and funding research into effective treatments, better diagnostic tools, and, ultimately, a cure for these debilitating conditions.

Booth 300 **ihypnus.com**

Hypnus is a new series of high performance CPAPs in OSA therapy devices, several technologies are ahead of the international leading level. Through accurate detection and following of advanced respiratory events, it can adaptively adjust the treatment pressure and flow, with super low noise, providing user a quiet, natural and comfortable sleeping environment, and significantly improving treatment compliance.

Booth 625 **ifa3d.com**

IFA3D Medical Solutions GmbH was founded 2016, as an outsourcing of the Institute of Anaplastology Velten & Hering. We have experience in individual Breathing Masks over 20 years. Now we use the 3D Technology, to produce light and best fit Individual Masks for Patients with Sleep Disorders

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Booth 525 **inspiresleep.com**

Inspire Medical Systems, Inc. is the leading developer of innovative, implantable neurostimulation systems to treat Obstructive Sleep Apnea. Utilizing technologies from the fields of cardiac pacing and neurostimulation, Inspire has developed an Upper Airway Stimulation therapy, designed to improve sleep and enrich the lives of people suffering from sleep apnea.

Booth 245 **iopimedical.com**

IOPI Medical LLC manufactures the Iowa Oral Performance Instruments (IOPI), products that objectively measures tongue and lip strength and can be used for biofeedback for oral motor exercises. It is typically used by speech pathologists and otolaryngologists with patients that have a variety of diseases, such as stroke, head and neck cancer, traumatic brain injury, Parkinson's disease, Bell's palsy and ALS.

Booth 303 **loewensteinmedical.de/en**

Löwenstein Medical GmbH + Co. KG has been a company in the Löwenstein Group since 2013. The development, production and marketing of diagnostic and therapeutic solutions for sleep medicine and homecare ventilation are among the core competencies of the firm. The success of products "Made in Germany" is based on good ideas, years of experience, foresight and innovations.

Booth 545 **myluminette.com**

Lucimed is a Belgian company specialized in the conception and manufacturing of wearable light therapy solutions to treat depressives disorders and sleep disorders.

Booth 420 **sprachwahl.thera-mon.com**

MC Technology GmbH is focussing on smart technical solutions by means of implementing state of the art technology and sophisticated but easy to use software approaches. TheraMon® is a dedicated Micro chip system for reporting objective patient compliance of dental appliances like orthodontic braces or MRA´s for patients suffering from sleep apnea. Knowing objective patient compliance is essential for effective and fast treatment considerations.

merck.com

Merck is a global health care leader working to help the world be well. Merck is known as MSD outside the United States and Canada. Through our prescription medicines, vaccines, biologic therapies, and animal health products, we work with customers and operate in more than 140 countries to deliver innovative health solutions.

Booth 330 **natus.com**

Natus is a leading provider of healthcare products used for the diagnosis and monitoring of neurological disorders, such as epilepsy, sleep disorders, stroke, neuropathies, neuromuscular diseases, myopathies and neurosurgical procedures. Product offerings include computerized neurodiagnostic systems for neurology, polysomnography, software systems and a complete range of supplies and accessories

Booth 485 **noxmedical.com**

Nox Medical builds medical devices for sleep diagnostics. Our mission is to advance sleep diagnostics through simplification, increased efficiency and comfort in all patient groups. Nox Medical's products include a full range of sleep diagnostic solutions, such as the Nox T3, Nox A1 PSG System, and the Noxturnal Software.

Booth 455 **orthoapnea.com**

OrthoApnea Investigation and innovation are the main cornerstones of OrthoApnea, providing revolutionary solutions for Mandibular Advancement Devices (MAD). The implementation of the newest technologies, untiring clinical research and scientific collaboration with top Sleep Experts and major Hospitals and Universities turned OrthoApnea into a leading global company into the Dental Sleep Medicine.

Booth 445 **oventus.com.au**

Oventus Medical is an Australian medical device company commercializing a revolutionary treatment platform for snoring and obstructive sleep apnoea (OSA) via a suite of oral appliances. Our O2VentTM customized oral devices incorporate Oventus Airway Technology – a unique airway built into its patented design, allowing for breathing through the device to bypass nasal resistance and velopharyngeal obstruction.

Booth 425 **phasya.com**

Phasya offers solutions for measuring drowsiness levels and ocular movements (i.e. blinks, saccades, pupil dilation) from eye images. Our product "Drowsimeter R100" enables researchers to do easily these measurements. Phasya also develops solutions for detecting other physiological and cognitive states such as stress, cognitive load, consciousness, and mind wandering.

Booth 405 **philips.com**

Philips seeks to transform how healthcare is delivered. As a global leader with 40 + years of leadership in sleep apnea management, oxygen therapy, noninvasive ventilation and respiratory drug delivery, we are committed to developing novel solutions to help people sleep and breathe better.

Booth 635 **quantactions.com**

QuantActions technology transforms behavioural patterns from smartphone taps into actionable insights, while preserving user privacy. Benefits include improved patient compliance, simplified clinical trial management and access to rich data sets for research into multiple disorders.

Booth 410 **resmed.com**

ResMed (NYSE: RMD) changes lives with award-winning medical devices and cutting-edge cloud-based software applications that better diagnose, treat and manage sleep apnea, chronic obstructive pulmonary disease (COPD) and other chronic diseases. ResMed is a global leader in connected care, with more than 3 million patients remotely monitored every day.

Booth 205 **timmyzzz.com**

Sedona Wellness LPP Franz Zach of Sedona has been manufacturing, distributing PEMF (Pulsed Electro Magnetic Field) therapy devices for more than 25 years. Just recently, Franz Zach launched his latest product "TimmyZzz," a pillow with integrated PEMF technology which not only helps with sleeping problems, but also with many more health issues.

Booth 335 **sefam-medical.com**

Since it was founded in 1982, the Nancy-based French company SEFAM has been designing, manufacturing, and commercializing diagnostic and therapy devices for Sleep disordered breathing.

Booth 305 **nastent.sevendreamers.com**

Seven Dreamers Laboratories, Inc. is a company where a group of technologists create and develop things that the world has never seen. We created laundroid, a fully automatic laundry folding robot, as well as nastent, a device to reduce snoring in sleep apnea. Moreover, we develop carbon golf shafts which are fully made-to-order with high quality and design.

Booth 350 **sleepmultimedia.com**

Sleep Multimedia Inc. SleepMultiMedia v. 10.0 is a computerized textbook of sleep medicine with text, sound, graphics, animation, & video. Updated annually with 140 CME credits, the program covers clinical sleep medicine, dental sleep medicine, sleep physiology, polysomnography, sleep research and sleep practice management. NEW: Online real-time access to references through Medline. Available on a USB Flash Drive.

SOMNICS

Sleep well. Wherever you are.

Booth 520 **somnics.com**

Somnics Inc. is a research-based and needs-driven medical device company engaging in the development of innovative treatment for sleep-disordered breathing. iNAP® Sleep Therapy System is designed to provide OSA patients a treatment which is more comfortable, travel-friendly and easier to use, and allow patients with OSA to breath naturally wherever they are.

SOMNO medics

Booth 360 **somnomedics.eu**

SOMNOmedics designs, manufactures, markets, distributes and services products dedicated to sleep diagnostics. Our products are utilized for a variety of sleep related tests and comply with the AASM standards. SOMNOmedics devices are small, lightweight and worn by the patient. We are compatible with in lab diagnostics as well as home sleep testing. SOMNOmedics wireless solution allows patients video, audio and data to be observed from any environment.

TerniMed

Wir verbinden Medizin mit Technik

Booth 480 **ternimed.de**

TerniMed UG specializes in high-quality medical accessories and consumable materials for neurological and sleep medical diagnostic. Based on many years of professional experience in the field, we have an elevated level of technical competency in dealing with issues relating to neurology and sleep medicine. Sound consultation and excellent support are key points of our activities to meet customer needs

TEVA

tevapharm.com

TEVA specialty medicines business is focused on delivering innovative solutions to patients and providers via medicines, devices and services, based on the core therapeutic areas of the central nervous system including pain, migraine, movement and neurodegenerative disorders and respiratory illnesses. We rank among the leading pharmaceutical companies in the world and are active in 80 countries.

WESTIN®

HOTELS & RESORTS

starwoodhotels.com/westin

Westin Hotels & Resorts, a leader in wellness and hospitality for more than a decade, empowers guests and associates to embrace well-being and be their best selves through the brand's six pillars of Well-Being: Sleep Well, Eat Well, Move Well, Feel Well, Work Well, and Play Well. At more than 200 hotels and resorts in nearly 40 countries and territories, guests can experience offerings that include the iconic Heavenly Bed, RunWESTIN and Westin Gear Lending with New Balance, delicious and nutritious SuperFoodsRx™ and more.

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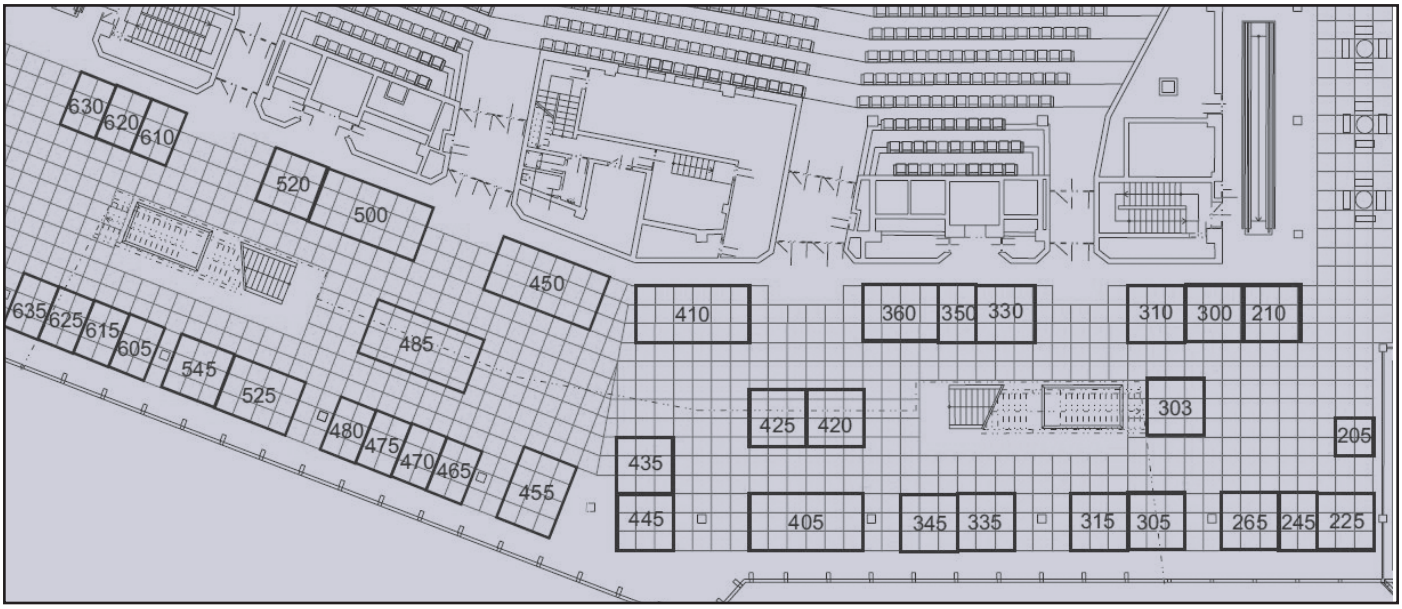

Exhibitor Floor Plan

205 – Sedona TimmyZzz
210 – Flamel Ireland Limited (Avadel)
225 – Wisepress Ltd.
245 – IOPI Medical
265 – Academy of Applied Myofunctional Sciences & Academy of Orofcial Myofunctional Therapy
300 – Guangzhou Hypnus Healthcare
303 – Lowenstein
305 – Seven Dreamers
310 – Advanced Brain Monitoring
315 – DyMedix Diagnostics
330 – Natus
335 – SEFAM Health Tech
345 – Compumedics
350 – Sleep Multimedia
360 – Somnomedics
360 – Somnomedics
405 – Philips
410 – ResMed
420 – MC Technology GmbH
425 – Phasya
435 – Condor
445 – Oventus Medical
450 – Bioproject
455 – Ortho Apnea
465 – Hypersomnia Foundation
470 – Cerevast Medical
475 – ESRS
480 – TerniMed UG
485 – Nox Medical
500 – Emfit Ltd
525 – Inspire
545 – Lucimed
605 – CamNtech Limited
610 – Arden Innovations
615 – Cidelec
620 – Board of Registered Polysomnographic Technologists (BRPT)
625 – Institut für Anaplastologie Berlin
630 – European Alliance for RLS
635 – QuantActions
American Association of Sleep Technologists
Elsevier

Sleep Medicine Research Foundation
KICK-OFF PARTY

During WORLD SLEEP 2017

DRESS: CASUAL

Tuesday October 10, 2017 | 20:00-22:30
Prague, Czech Republic

THE GREAT HALL- NEW TOWN HALL PRAGUE

Karlovo namesti 1/23 I Charles Square,
Prague 120 00, Czech Republic

SMRF
SLEEP MEDICINE
RESEARCH FOUNDATION

Join the Sleep Medicine and Research Foundation for our Inaugural Reception and help support the work & collaborations of Dr. Christian Guilleminault and advance the state of sleep health worldwide.

The Sleep Medicine and Research Foundation invites you to a Kick-Off Party on Tuesday October 10th, 2017 during World Sleep 2017, in celebration of the 40th Anniversary of Dr. Christian Guilleminault's first description of pediatric sleep apnea and the 45th Anniversary of his first description of central sleep apnea and insomnia syndrome. Please join us for hors d'oeuvres, and unlimited wine, beer and drinks from 20:00 - 22:30++ ***- Program starts at 21:00***

Speakers in Tribute

Jed Black, Oliviero Bruni, David Gozal, Meir Kryger, Atul Malhotra, Marie Marklund, Joy Lea Moeller, Pierre Jean Monteyrol, Daniel Ng, Judy Owens, Paola Pirelli

Online RSVP address:

www.sleepmedicineresearchfoundation.org/register

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WORLD ASSOCIATION OF SLEEP MEDICINE
& WORLD SLEEP FEDERATION

www.worldsleepcongress.com

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University of Rome, Tor Vergata

World Sleep 2017
7 – 10 October 2017 • Prague, Czech Republic

MSD

New Approaches to Personalizing Treatment of Insomnia: Why? and How?

Monday, 9 October 2017
12:30 – 14:00

Satellite Symposium
Sponsored by MSD

Conference Center
Meeting Hall IV
Prague, Czech Republic

Chair: Charles Morin

CHAIR

Charles Morin, PhD

Full Professor, Department of Psychology
Laval University
Director, Centre d'étude des troubles du sommeil
Canada Research Chair in Sleeping Disorders
Quebec City, Canada

FACULTY

Andrew Krystal, MD

Professor of Psychiatry
UCSF Department of Psychiatry
Executive Vice Chair for the Langley Porter Psychiatric Institute (LPPI)
San Francisco, California, USA

Thomas Roth, PhD

Chief, Division Head
Sleep Disorders and Research Center
Henry Ford Hospital
Detroit, Michigan, USA

AGENDA

12:30 – 12:40	Welcome and Introduction	Charles Morin
12:40 – 13:00	Health Consequences of Insomnia: Cognition, Mood, and Medical Impact	Charles Morin
13:00 – 13:20	Personalization of Insomnia Therapy: Matching Treatment Mechanisms With Patient Needs	Andrew Krystal
13:20 – 13:40	Orexin Receptor Antagonists in the Management of Insomnia – Mechanisms and Clinical Implications	Thomas Roth
13:40 – 14:00	Closing Remarks and Q&A	Charles Morin

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BEL-195774-IN

TEVA Growth Markets Symposium at the World Sleep Congress 2017
Prague | Czech Republic

Challenges of Recognizing and Treating Excessive Sleepiness

Excessive sleepiness is a serious, debilitating, condition with consequences not only for the individual, but also for public health and safety.

It is time to listen experts to discuss the challenges of recognizing and treatment of excessive sleepiness.

Monday, October 9th, 2017 | 12.30 – 14.00 pm
Prague Congress Centre – Congress Hall Auditorium

Schedule:

12:30 -12:45 pm
Opening Remarks - R. Rosenberg, PhD,USA

12:45 -13:15 pm
Excessive sleepiness (ES) in OSA - R. Grunstein, MD, Australia

13:15-13:45 pm
Therapeutic Approaches to ES - R. Rosenberg, PhD,USA

13:45 -14:00 pm
Panel Discussion - R. Rosenberg, PhD,USA and R. Grunstein, MD, Australia

nox medical

ADVANCING SLEEP DIAGNOSTICS

Satellite Symposium

Evolution of Sleep Medicine – Challenging the Status Quo
Tuesday, October 10th 2017
12:30 – 13:15, Prague Convention Centre - North Hall

Speakers

Allan I. Pack, M.B.Ch.B., Ph.D., FRCP
John Miclot Professor of Medicine
Director, Center for Sleep and Circadian Neurobiology
Perelman School of Medicine at the University of Pennsylvania

Erna Sif Arnardóttir, PhD
Director of Sleep Measurements
Landspitali - The National University Hospital of Iceland
Postdoctoral Researcher and Adjunct Professor, University of Iceland

Chair

Prof. Liborio Parrino
Associate Professor of Neurology
Director School of Specialization in Neurology
Director, Sleep Disorders Center
University of Parma, Italy

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