# ASRS 2012 Daily Program

## November 30 (Friday)

### Location: Room 101A, 1F

#### CBTi Workshop

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>09:00-09:50</td>
<td>C1-1</td>
<td>睡眠神經生理機制&lt;br&gt;蔡宇哲(Taiwan)</td>
</tr>
<tr>
<td>10:00-10:50</td>
<td>C1-2</td>
<td>睡眠疾患與評估技術&lt;br&gt;李信謙(Taiwan)</td>
</tr>
<tr>
<td>11:00-12:30</td>
<td>C1-3</td>
<td>失眠病理機制&lt;br&gt;楊建銘(Taiwan)</td>
</tr>
<tr>
<td>13:30-14:50</td>
<td>C1-4</td>
<td>失眠評估與失眠病因概念化&lt;br&gt;吳家硕(Taiwan)</td>
</tr>
<tr>
<td>15:00-16:20</td>
<td>C1-5</td>
<td>助眠認知行為技巧&lt;br&gt;周舒翎(Taiwan)</td>
</tr>
<tr>
<td>16:30-17:50</td>
<td>C1-6</td>
<td>生理時鐘的維持與調整&lt;br&gt;詹雅雯(Taiwan)</td>
</tr>
</tbody>
</table>

### Location: Room 101B, 1F

#### Workshop[1] High Quality of Polysomnography

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<th>Time</th>
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<tbody>
<tr>
<td>09:00-09:30</td>
<td>W1-1</td>
<td>Quality control of PSG in Hong Kong&lt;br&gt;Crover Ho (Hong Kong)</td>
</tr>
<tr>
<td>09:30-10:00</td>
<td>W1-2</td>
<td>An examination of quality in the sleep laboratory&lt;br&gt;Brett Duce (Australia)</td>
</tr>
<tr>
<td>10:00-10:30</td>
<td>W1-3</td>
<td>Technical Progress for Quality Control of Polysomnography in Japan&lt;br&gt;Tomoko Yagi (Japan)</td>
</tr>
<tr>
<td>10:30-11:00</td>
<td>W1-4</td>
<td>Promotion of Polysomnography in Taiwan&lt;br&gt;Sheng-Yi Liu (Taiwan)</td>
</tr>
<tr>
<td>11:00-11:02</td>
<td></td>
<td>Discussion</td>
</tr>
</tbody>
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### Location: Room 101CD, 1F

#### The 3rd Chinese World Sleep Forum – OSA Session

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<tr>
<th>Time</th>
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<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>09:00-09:05</td>
<td>F1-1</td>
<td>Introduction&lt;br&gt;Ning-Hung Chen (Taiwan)</td>
</tr>
</tbody>
</table>
# The 3rd Chinese World Sleep Forum – OSA Session

**Moderator:** Ning-Hung Chen (Taiwan)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>09:05-09:20</td>
<td>F1-2</td>
<td>Representative Speakers give talks</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Qing-Yun Li (China)</td>
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<td></td>
<td>Albert Li (Hong Kong)</td>
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<td></td>
<td></td>
<td>Liang-Wen Hang (Taiwan)</td>
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<tr>
<td>09:20-09:50</td>
<td>F1-3</td>
<td>Other Invited Speakers express their opinions</td>
</tr>
<tr>
<td>09:50-10:00</td>
<td></td>
<td>Discussion</td>
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</table>

**Location:** Room 101CD, 1F

# The 3rd Chinese World Sleep Forum – Narcolepsy Session

**Moderator:** Fang Han (China)

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<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>10:00-10:05</td>
<td>F2-1</td>
<td>Introduction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fang Han (China)</td>
</tr>
<tr>
<td>10:05-10:20</td>
<td>F2-2</td>
<td>Representative Speakers give talks</td>
</tr>
<tr>
<td>10:20-10:50</td>
<td>F2-3</td>
<td>Other Invited Speakers express their opinions</td>
</tr>
<tr>
<td>10:50-11:00</td>
<td></td>
<td>Discussion</td>
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</table>

**Location:** Room 101CD, 1F

# The 3rd Chinese World Sleep Forum – RLS Session

**Moderator:** Yun-Kwok Wing (Hong Kong)

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<th>Session No.</th>
<th>Title/Speaker</th>
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<tbody>
<tr>
<td>11:10-11:15</td>
<td>F3-1</td>
<td>Introduction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yun-Kwok Wing (Hong Kong)</td>
</tr>
<tr>
<td>11:15-11:30</td>
<td>F3-2</td>
<td>Representative Speakers give talks</td>
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<tr>
<td></td>
<td></td>
<td>Rui Chen (China)</td>
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<tr>
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<td></td>
<td>Jihui Zhang (Hong Kong)</td>
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<tr>
<td></td>
<td></td>
<td>Scarlett Lai (Taiwan)</td>
</tr>
<tr>
<td>11:30-12:00</td>
<td>F3-3</td>
<td>Other Invited Speakers express their opinions</td>
</tr>
<tr>
<td>12:00-12:10</td>
<td></td>
<td>Discussion</td>
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</table>

**Location:** Room 101B, 1F

# Debate - Conservative Treatment is Superior to Surgery in the Management of OSA

**Moderator:** Hsueh-Yu Li (Taiwan)

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<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
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</thead>
<tbody>
<tr>
<td>11:10-12:10</td>
<td>D1-1</td>
<td>Conservative Treatment is Superior to Surgery in the Management of OSA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Victor Abdullah (Hong Kong)</td>
</tr>
<tr>
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<td>Yau Hong Goh (Singapore)</td>
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</tbody>
</table>
### Location: Room 101B, 1F

**Workshop[2] Can We Prevent OSAS?**

**Moderator:** Liang-wen Hang (Taiwan)

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<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>14:00-14:30</td>
<td>W2-1</td>
<td>Pathophysiology of Upper Airway Obstruction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Shiroy Isono (Japan)</td>
</tr>
<tr>
<td>14:30-15:00</td>
<td>W2-2</td>
<td>Clinical Application to OSAS of Children Using Computational Fluid Dynamics</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tomonori Iwasaki (Japan)</td>
</tr>
<tr>
<td>15:00-15:30</td>
<td>W2-3</td>
<td>Nasal Breathing and Facial Growth in Pediatric OSA in Japan</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Shintaro Chiba (Japan)</td>
</tr>
<tr>
<td>15:30-16:00</td>
<td>W2-4</td>
<td>Can We Prevent OSAS?</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Makoto Kikuchi (Japan)</td>
</tr>
<tr>
<td>16:00-16:02</td>
<td>Discussion</td>
<td></td>
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</table>

### December 1 (Saturday)

**Location: Room 201, 2F**

**Plenary Lecture**

**Moderator:** Prof. Masako Okawa (Japan)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
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<tbody>
<tr>
<td>09:00-09:30</td>
<td>-</td>
<td>Opening Ceremony</td>
</tr>
<tr>
<td>09:30-10:30</td>
<td>KS-1</td>
<td>Sleep and Psychiatric Disorders: Cause or Effect?</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Robert Stickgold (USA)</td>
</tr>
<tr>
<td>11:00-12:00</td>
<td>KS-2</td>
<td>Phenotyping in Obstructive Sleep Apnea: A guide to Future Therapy</td>
</tr>
<tr>
<td></td>
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<td>David White (USA)</td>
</tr>
</tbody>
</table>

### Location: Room 101A, 1F

**Luncheon Seminar - GSK**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>12:00-13:00</td>
<td>L1-1</td>
<td>The role of nose in sleep – disorder breathing patients</td>
</tr>
<tr>
<td></td>
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<td>Hsueh-Yu Li (Taiwan)</td>
</tr>
</tbody>
</table>

### Location: Room 101B, 1F

**Luncheon Seminar – Apex Medical Corp.**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
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<tbody>
<tr>
<td>12:00-13:00</td>
<td>L2-1</td>
<td>Past, Present and Future of CPAP</td>
</tr>
<tr>
<td></td>
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<td>Dieter Heidmann (Germany)</td>
</tr>
</tbody>
</table>

### Location: Room 101C, 1F

**Luncheon Seminar – Philips Taiwan Ltd**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>12:00-13:00</td>
<td>L3-1</td>
<td>Central Sleep Apnea: Pathophysiology and Therapy</td>
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<tr>
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<td>David White (USA)</td>
</tr>
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</table>
### Location: Room 101A, 1F

**Symposium[1] Current Status of Research in Sleep Disorders/Sleep Deprivation**

**Moderator:**

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<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>13:00-13:22</td>
<td>S1-1</td>
<td>Functional correlates of the consequences of OSA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Seung Bong Hong (Korea)</td>
</tr>
<tr>
<td>13:22-13:44</td>
<td>S1-2</td>
<td>Neural Correlates of Reduced Processing Capacity in Sleep-Deprived Persons</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Wei-Liang Michael Chee (Singapore)</td>
</tr>
<tr>
<td>13:44-14:06</td>
<td>S1-3</td>
<td>Dysfunction of Dopamine and Serotonin System in RLS</td>
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<td>In Young Yoon (Korea)</td>
</tr>
<tr>
<td>14:06-14:28</td>
<td>S1-4</td>
<td>Partial and Total Sleep Deprivation: Effects on Performance across Cognitive</td>
</tr>
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<td>Domains, Individuals, and Circadian Phase</td>
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<td></td>
<td>June Chi-Yan Lo (Hong Kong)</td>
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<tr>
<td>14:28-14:30</td>
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<td>Discussion</td>
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### Location: Room 101B, 1F

**Symposium[2] Pediatric sleep-disordered-breathing (SDB)**

**Moderator:** Yu-Shu Huang (Taiwan), Albert Martin Li (Hong Kong)

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<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>13:00-13:22</td>
<td>S2-1</td>
<td>The Incidence and Respiratory Pattern in Pediatric Sleep Apnea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Takuro Kitamura (Japan)</td>
</tr>
<tr>
<td>13:22-13:44</td>
<td>S2-2</td>
<td>Cardiovascular Complications in Children with Sleep-Disordered Breathing</td>
</tr>
<tr>
<td></td>
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<td>Disorder</td>
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<td></td>
<td></td>
<td>Albert Martin Li (Hong Kong)</td>
</tr>
<tr>
<td>13:44-14:06</td>
<td>S2-3</td>
<td>Current Hypopnea Scoring Criteria Underscore Pediatric Sleep Disordered</td>
</tr>
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<td></td>
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<td>Breathing</td>
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<td></td>
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<td>Cheng-Hui Lin (Taiwan)</td>
</tr>
<tr>
<td>14:06-14:28</td>
<td>S2-4</td>
<td>Congenital Central Hypoventilation Syndrome: Thailand Experience</td>
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<td>Aroonwan Preutthipan (Thailand)</td>
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<td>14:28-14:30</td>
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<td>Discussion</td>
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### Location: Room 101C, 1F

**Symposium[3] Etiology of Hypersomnia and Diagnosis of Sleep Quality**

**Moderator:**

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<th>Time</th>
<th>Session No.</th>
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<tbody>
<tr>
<td>13:00-13:22</td>
<td>S3-1</td>
<td>Symptomatic Narcolepsy and Hypersomnia, Hypocretin/Orexin Involvements</td>
</tr>
<tr>
<td></td>
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<td>Takashi Kanbayashi (Japan)</td>
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<td>to Bedside</td>
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<td>Seiji Nishino (USA)</td>
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<tr>
<td>13:44-14:06</td>
<td>S3-3</td>
<td>Questions Post Hypocretin / Orexin Discovery</td>
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<td>Jean J. Askenasy (Israel)</td>
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</tbody>
</table>
### Symposium[3] Etiology of Hypersomnia and Diagnosis of Sleep Quality

**Moderator:**

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<tbody>
<tr>
<td>14:06-14:28</td>
<td>S3-4</td>
<td>Cardiopulmonary Coupling as a Sleep Quality Metrics</td>
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<td>Chang-Ho Yun (Korea)</td>
</tr>
<tr>
<td>14:28-14:30</td>
<td></td>
<td>Discussion</td>
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**Location:** Room 101D, 1F

### Symposium[4] A New Look into the Factors that Promote Sleep

**Moderator:** Hsin-Chien Lee (Taiwan)

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<th>Time</th>
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<tbody>
<tr>
<td>13:00-13:22</td>
<td>S4-1</td>
<td>Thermoregulation as Sleep Regulator</td>
</tr>
<tr>
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<td>Hruda Nanda Mallick (India)</td>
</tr>
<tr>
<td>13:22-13:44</td>
<td>S4-2</td>
<td>Importance of Yogic Practice in Promoting Sleep</td>
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<td>Bindu M Kutty (India)</td>
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<tr>
<td>13:44-14:06</td>
<td>S4-3</td>
<td>Exercise Effects on Sleep Physiology</td>
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<td>Sunao Uchida (Japan)</td>
</tr>
<tr>
<td>14:06-14:28</td>
<td>S4-4</td>
<td>Role of Sleep in Organizing Central Networks for Sexual Functions</td>
</tr>
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<td></td>
<td>Kamalesh K Gulia (India)</td>
</tr>
<tr>
<td>14:28-14:30</td>
<td></td>
<td>Discussion</td>
</tr>
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**Location:** Room 102, 1F

### Symposium[5] Basal Ganglia Regulation of Sleep-Wake Cycle

**Moderator:** Zhi-Li Huang (China), Mark R. Opp (USA)

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<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
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<tbody>
<tr>
<td>13:00-13:22</td>
<td>S5-1</td>
<td>Dissecting the Basal Ganglia Control of Sleep-Wake Behavior</td>
</tr>
<tr>
<td></td>
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<td>Jun Lu (USA)</td>
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<tr>
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<td></td>
<td>Michael Lazarus (Japan)</td>
</tr>
<tr>
<td>13:44-14:06</td>
<td>S5-3</td>
<td>Key Roles of Dopamine D2 Receptor in the Sleep-Wake Regulation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Zhi-Li Huang (China)</td>
</tr>
<tr>
<td>14:06-14:28</td>
<td>S5-4</td>
<td>The Interdependence or Independence of Arousal, Motor, and Cognition Control by the Striatal Dopamine and Adenosine Systems</td>
</tr>
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<td></td>
<td>Jiang-Fan Chen (USA)</td>
</tr>
<tr>
<td>14:28-14:30</td>
<td></td>
<td>Discussion</td>
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</table>
### Location: Room 101A, 1F

**Symposium[6] Pathogenesis and Long Term Course of REM Sleep Behavior Disorder**

**Moderator:**

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<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
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</thead>
</table>
| 15:00-15:22| S6-1        | Diagnostic Instruments of RBD - From Questionaire to Polysomnography  
Yun Kwok Wing (Hong Kong)                                                                 |
| 15:22-15:44| S6-2        | The Implication of Nigrostriatal Dopaminergic System in RBD  
In-Young Yoon (Korea)                                                                 |
| 15:44-16:06| S6-3        | Dream-Enacting Behavior is Associated with Impaired Sleep, Severe Headache-Related Disability and Depressive Symptoms in Migraine Patients  
Keisuke Suzuki (Japan)                                                                 |
| 16:06-16:28| S6-4        | Clinical Significance of REM Sleep Behavior Disorder Parkinson’s Disease  
Yuichi Inoue (Japan)                                                                 |
| 16:28-16:30|             | Discussion                                                                                                                                   |

### Location: Room 101B, 1F

**Symposium[7] Sleep Surgery in Asia**

**Moderator:** Hsueh-Yu Li (Taiwan)

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<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
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</table>
| 15:00-15:22| S7-1        | Multilevel surgery for OSA  
Prasit Mahakit (Thailand)                                                                                                                               |
| 15:22-15:44| S7-2        | Multiple Factors Predicting the Outcome of Surgery for Obstructive Sleep Apnea  
Jing-Ying Ye (China)                                                                                                                                     |
| 15:44-16:06| S7-3        | Advancement of Sleep Surgery in Asia  
Sung Wan Kim (Korea)                                                                                                                                       |
| 16:06-16:28| S7-4        | The Concept of Sleep Surgery in Japan: Indication of UPPP and Postoperative Management in Japan  
Shintaro Chiba (Japan)                                                                                                                                     |
| 16:28-16:30|             | Discussion                                                                                                                                             |

### Location: Room 101C, 1F

**Symposium[8] Sleep Consciousness Interaction**

**Moderator:**

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<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
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</table>
| 15:00-15:22| S8-1        | Conscious Awareness in Dreams: Proof-of-Principle  
Ursula Voss (Germany)                                                                                                                                   |
| 15:22-15:44| S8-2        | Sleep, Dreams and Psychotic Disorders  
Armando d’Agostino (Italy)                                                                                                                                |
| 15:44-16:06| S8-3        | Changes in Anterior-Posterior Feedback Connectivity during Anesthesia and Sleep in Rat  
Dinesh Pal (USA)                                                                                                                                             |
Symposium[8] Sleep Consciousness Interaction

Moderator: Jean J. Askenasy (Israel)

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<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
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<tbody>
<tr>
<td>16:06-16:28</td>
<td>S8-4</td>
<td>Dreams versus Hallucinations</td>
</tr>
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<td>16:28-16:30</td>
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<td>Discussion</td>
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Location: Room 101D, 1F

Symposium[9] Respiratory Regulation in Sleep Apnea: from Basic Research to Clinical Practice

Moderator: Fang Han (China)

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<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
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</thead>
<tbody>
<tr>
<td>15:00-15:22</td>
<td>S9-1</td>
<td>Respiratory Control in Sleep Apnea : from Phenotype to Genotype</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fang Han (China)</td>
</tr>
<tr>
<td>15:22-15:44</td>
<td>S9-2</td>
<td>Principle and Efficacy of Transvenous Phrenic Nerve Stimulation for Cheyne-Stokes Respiration in Patient with Chronic Heart Failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Xi-Long Zhang (China)</td>
</tr>
<tr>
<td>15:44-16:06</td>
<td>S9-3</td>
<td>Neural Drive of Diaphragm and Upper Airway Muscles in Sleep Apnea</td>
</tr>
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<td></td>
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<td>Yuan-Ming Luo (China)</td>
</tr>
<tr>
<td>16:06-16:28</td>
<td>S9-4</td>
<td>OSAS Phenotypes and Breathing Irregularity -Lessons from Animal Studies</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Motoo Yamauchi (Japan)</td>
</tr>
<tr>
<td>16:28-16:30</td>
<td></td>
<td>Discussion</td>
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</table>

December 2 (Sunday)

Location: Room 201, 2F

Plenary Lecture

Moderator: Ken-ichi Honma (Japan), Ning-Hung Chen (Taiwan)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>09:00-10:00</td>
<td>KS-3</td>
<td>Sleep Disordered Breathing and Cardiovascular Disease?</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sau-May Mary Ip (Hong Kong)</td>
</tr>
</tbody>
</table>

Location: Room 101A, 1F

Symposium[10] Sleep Movements and Dopaminergic System

Moderator: Ronit Gilad (Israel)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>10:30-10:52</td>
<td>S10-1</td>
<td>From Restless Legs Syndrome to Willis-Ekbom Disease: a Diagnostic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>and Treatment Conundrum</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Denise Sharon (USA)</td>
</tr>
<tr>
<td>10:52-11:14</td>
<td>S10-2</td>
<td>RLS and PLMS, Two Phenotypes of the Sleep Leg Movement Disorder (SLMD)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Jean J. Askenasy (Israel)</td>
</tr>
<tr>
<td>11:14-11:36</td>
<td>S10-3</td>
<td>Epilepsy and Sleep</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ronit Gilad (Israel)</td>
</tr>
</tbody>
</table>
### Symposium[10] Sleep Movements and Dopaminergic System

**Moderator:**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>11:36-11:58</td>
<td>S10-4</td>
<td>Sleep Movements and ADHD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Marieta Anca-Herschkovitsch (Israel)</td>
</tr>
<tr>
<td>11:58-12:00</td>
<td>Discussion</td>
<td></td>
</tr>
</tbody>
</table>

**Location: Room 101B, 1F**


**Moderator:** Pon Poh Hsu (Singapore), Hsin-Ching Lin (Taiwan)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>10:30-10:52</td>
<td>S11-1</td>
<td>Application of Acoustic Analysis of Snoring Sounds for Dynamic Evaluation of Upper Airway Morphology of OSAS Patients</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hirotaka Hara (Japan)</td>
</tr>
<tr>
<td>10:52-11:14</td>
<td>S11-2</td>
<td>Changes of Snore Sound after Sleep Surgery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Li-Ang Lee (Taiwan)</td>
</tr>
<tr>
<td>11:14-11:36</td>
<td>S11-3</td>
<td>Midazolam sedation sleep endoscopy: What can we see now after 20 years</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Victor Abdullah (Hong Kong)</td>
</tr>
<tr>
<td>11:36-11:58</td>
<td>S11-4</td>
<td>Computer-Assisted Quantitative Upper Airway Analysis Following Modified Uvulopalatal Flap And Lateral Pharyngoplasty For Obstructive Sleep Apnoea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pon Poh Hsu (Singapore)</td>
</tr>
<tr>
<td>11:58-12:00</td>
<td>Discussion</td>
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**Location: Room 101C, 1F**

### Symposium[12] Molecular and Cellular Mechanisms of Circadian Clock and Sleep

**Moderator:** Ken-ichi Honma (Japan), Chung-Yao Hsu (Taiwan)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>10:30-10:52</td>
<td>S12-1</td>
<td>Multi-oscillator System of Mammalian Circadian Clock</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sato Honma (Japan)</td>
</tr>
<tr>
<td>10:52-11:14</td>
<td>S12-2</td>
<td>Dissection of Neural Mechanisms Underlying Circadian Pacemakers Using Brain Region/Cell-Specific Bmal1 Deficient Mice</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Michihiro Mieda (Japan)</td>
</tr>
<tr>
<td>11:14-11:36</td>
<td>S12-3</td>
<td>Circadian Phase Waves and Long-range Networks in Suprachiasmatic Nucleus</td>
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<td></td>
<td>Kyoung Jin Lee (Korea)</td>
</tr>
<tr>
<td>11:36-11:58</td>
<td>S12-4</td>
<td>Clock Genes and Human Sleep Behavior Phenotypes</td>
</tr>
<tr>
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<td></td>
<td>Ying-Hui Fu (USA)</td>
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<tr>
<td>11:58-12:00</td>
<td>Discussion</td>
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</table>
**Location: Room 101D, 1F**

**Symposium[13] Promotion of Sleep Education in Asia**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
</table>
| 10:30-10:48| S13-1       | Taiwan Status of Sleep Education  
Chia-Mo Lin (Taiwan)                                                          |
| 10:48-11:06| S13-2       | Korean Sleep Education  
Ji Ho Choi (Korea)                                                            |
| 11:06-11:24| S13-3       | Sleep Education in USA  
Kingman Strohl (Korea)                                                        |
| 11:24-11:42| S13-4       | Sleep Education in Japan  
Soichiro Miyazaki (Japan)                                                      |
| 11:42-12:00| S13-5       | Sleep Education in Hong Kong - From Healthcare Professionals to General Population  
Yun Kwok Wing (Hong Kong)                                                      |
| 12:00-12:02|             | Discussion                                                                    |

**Location: Room 101A, 1F**

**Luncheon Seminar – Medtronic (Taiwan) Ltd**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
</table>
| 12:00-13:00| L4-1        | Minimally Invasive OSA Surgery: Rationale, Pitfalls and Outcomes  
Hsin-Ching Lin (Taiwan)                                                        |

**Location: Room 101B, 1F**

**Luncheon Seminar – BROJAW INC**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
</table>
| 12:00-13:00| L5-1        | Heart Disease with SDB  
Glenn Richards (New Zealand)         |

**Location: Room 101A, 1F**

**Symposium[14] Sleep, its Regulation and Function: Approach from fMRI And EEG**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
</table>
| 13:00-13:22| S14-1       | Seeing A Dreaming Brain -Brain Activation Accompanying Rapid Eye Movements During REM Sleep-  
Satoru Miyauchi (Japan)                                                      |
| 13:22-13:44| S14-2       | What Happens to Our Brain Upon Awakening?  
Chang-Wei W. Wu (Taiwan)                                                      |
| 13:44-14:06| S14-3       | Alterations in Decision Making Induced by Sleep Deprivation  
Wei-Liang Michael Chee (Singapore)                                            |
### Symposium[14] Sleep, its Regulation and Function: Approach from fMRI And EEG

**Moderator:** Tong-Ping Su (Taiwan)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>14:06-14:28</td>
<td>S14-4</td>
<td>Local, Experience-Dependent Sleep/Wake Regulation and Brain Plasticity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ching-Sui Hung (Taiwan)</td>
</tr>
<tr>
<td>14:28-14:30</td>
<td></td>
<td>Discussion</td>
</tr>
</tbody>
</table>

**Location:** Room 101B, 1F

### Symposium[15] Surgical Outcomes of Obstructive Sleep Apnea

**Moderator:** Shih-An Liu (Taiwan), Cheng-Hui Lin (Taiwan)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>13:00-13:22</td>
<td>S15-1</td>
<td>CPAP Compliance after Nasal surgery for CPAP failure</td>
</tr>
<tr>
<td></td>
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<td>Seiichi Nakata (Japan)</td>
</tr>
<tr>
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<td>Chairat Neruntarat (Thailand)</td>
</tr>
<tr>
<td>13:44-14:06</td>
<td>S15-3</td>
<td>Tongue Base Surgery for Moderate to Severe OSA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prasit Mahakit (Thailand)</td>
</tr>
<tr>
<td>14:06-14:28</td>
<td>S15-4</td>
<td>Limitations and Complications of Skeletal Surgery for OSA</td>
</tr>
<tr>
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<td></td>
<td>Sung Wan Kim (Korea)</td>
</tr>
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<td>14:28-14:30</td>
<td></td>
<td>Discussion</td>
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**Location:** Room 101C, 1F

### Symposium[16] Epidemiology of Sleep among the Working Population

**Moderator:**

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>13:00-13:22</td>
<td>S16-1</td>
<td>An Overview of Epidemiology of Sleep Among the Working Population</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sadik Ardiç (Turkey)</td>
</tr>
<tr>
<td>13:22-13:44</td>
<td>S16-2</td>
<td>Impact of Sleep Disorders on Occupational Health</td>
</tr>
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<td></td>
<td></td>
<td>Murat Aksu (Turkey)</td>
</tr>
<tr>
<td>13:44-14:06</td>
<td>S16-3</td>
<td>Impact of Work Related Conditions on Sleep</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hikmet Yılmaz (Turkey)</td>
</tr>
<tr>
<td>14:06-14:28</td>
<td>S16-4</td>
<td>Epidemiology of OSA in Working Population – Taiwan Chapter</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pa-Chun Wang (Taiwan)</td>
</tr>
<tr>
<td>14:28-14:30</td>
<td></td>
<td>Discussion</td>
</tr>
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</table>

**Location:** Room 101D, 1F

### Symposium[17] Neuroendocrines on Sleep

**Moderator:** Testuo Shimizu (Japan) Fang-Chia Chang (Taiwan)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
</tr>
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<tbody>
<tr>
<td>13:00-13:22</td>
<td>S17-1</td>
<td>Optogenetic Manipulation of Orexin Neuronal Activity Affects Sleep/</td>
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<tr>
<td></td>
<td></td>
<td>Wakefulness State in Mice</td>
</tr>
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<td>Tomomi Tsunematsu (Japan)</td>
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</tbody>
</table>
### Symposium[17] Neuroendocrines on Sleep

**Moderator:** Testuo Shimizu (Japan) Fang-Chia Chang (Taiwan)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/Speaker</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>Mark R. Opp (USA)</td>
</tr>
<tr>
<td>13:44-14:06</td>
<td>S17-3</td>
<td>Neural Circuit of Orexin-producing Neurons</td>
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<tr>
<td></td>
<td></td>
<td>Takeshi Sakurai (Japan)</td>
</tr>
<tr>
<td>14:06-14:28</td>
<td>S17-4</td>
<td>Neuroendocrines on Epilepsy-Induced Sleep Disruptions</td>
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<tr>
<td></td>
<td></td>
<td>Fang-Chia Chang (Taiwan)</td>
</tr>
<tr>
<td>14:28-14:30</td>
<td></td>
<td>Discussion</td>
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</tbody>
</table>

**Location:** Room 101A, 1F

### Symposium[18] Active Brain in Sleep – New Approaches to the Brain Functions -

**Moderator:** Rou Shayn Chen (Taiwan)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/ Speaker</th>
</tr>
</thead>
<tbody>
<tr>
<td>15:00-15:22</td>
<td>S18-1</td>
<td>Responsiveness of the brain to external stimulations via new methodological applications</td>
</tr>
<tr>
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<td></td>
<td>Murat Ozgoren (Turkey)</td>
</tr>
<tr>
<td>15:22-15:44</td>
<td>S18-2</td>
<td>Emotion processing in sleeping brain</td>
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<td></td>
<td></td>
<td>Ya-Wei Cheng (Taiwan)</td>
</tr>
<tr>
<td>15:44-16:06</td>
<td>S18-3</td>
<td>How to realize a causality of sequential events of bodily functions with sleep</td>
</tr>
<tr>
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<td>Ken-Ichi Honma (Japan)</td>
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<tr>
<td>16:06-16:28</td>
<td>S18-4</td>
<td>Implicit memory and tactile stimulations in sleep</td>
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<td></td>
<td>Adile Oniz (Turkey)</td>
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<tr>
<td>16:28-16:30</td>
<td></td>
<td>Discussion</td>
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**Location:** Room 101B, 1F

### Symposium[19] Sleep Disordered Breathing and Cardiovascular Disease

**Moderator:** Shin-ichi Ando (Japan), Pei-Lin Lee (Taiwan)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session No.</th>
<th>Title/ Speaker</th>
</tr>
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<tbody>
<tr>
<td>15:00-15:18</td>
<td>S19-1</td>
<td>Association between Obstructive Sleep Apnea and Coronary Artery Disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Akira Tamura (Japan)</td>
</tr>
<tr>
<td>15:18-15:36</td>
<td>S19-2</td>
<td>Long-Term Effects of CPAP on Carotid Artery Intima-Media Thickness</td>
</tr>
<tr>
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<td></td>
<td>David Shu Cheong Hui (Hong Kong)</td>
</tr>
<tr>
<td>15:36-15:54</td>
<td>S19-3</td>
<td>Obstructive Sleep Apnea and Hypertension – A Multicenter Study in China</td>
</tr>
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<td>Bao Yuan Chen (China)</td>
</tr>
<tr>
<td>15:54-16:12</td>
<td>S19-4</td>
<td>Undetermined</td>
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<td>Dai Yumino (Japan)</td>
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<tr>
<td>16:12-16:30</td>
<td>S19-5</td>
<td>Sleep apnea and ischemic stroke</td>
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<td></td>
<td>Jee Hyun Kim (Korea)</td>
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<tr>
<td>17:30-17:32</td>
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<td>Discussion</td>
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### Location: Room 101C, 1F

#### Symposium[20] Sleep and Autonomic Functioning

<table>
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<th>Time</th>
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<th>Title/Speaker</th>
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<tbody>
<tr>
<td>15:00-15:22</td>
<td>S20-1</td>
<td>Performance of the Frequency Domain Indices with Respect to Sleep Staging</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Terry Bo-Jau Kuo (Taiwan)</td>
</tr>
<tr>
<td>15:22-15:44</td>
<td>S20-2</td>
<td>Estimation of Sleep Level Transition using Lorenz Plot of ECG RR Intervals</td>
</tr>
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<td></td>
<td></td>
<td>Hiroshi Hagiwara (Japan)</td>
</tr>
<tr>
<td>15:44-16:06</td>
<td>S20-3</td>
<td>The Relationship of Sleep And Autonomic Activities in Hypertension</td>
</tr>
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<td></td>
<td></td>
<td>Yu-Che Tsai (Taiwan)</td>
</tr>
<tr>
<td>16:06-16:28</td>
<td>S20-4</td>
<td>The relationship between depressive symptoms, sleep arousal, and autonomic</td>
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<td></td>
<td>activities in Major Depressive Disorder</td>
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<td></td>
<td>I-Mei Lin (Taiwan)</td>
</tr>
<tr>
<td>16:28-16:30</td>
<td>Discussion</td>
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### Location: Room 101D, 1F

#### Symposium[21] Non-pharmacological treatments for insomnias

<table>
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<th>Time</th>
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<th>Title/Speaker</th>
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<tbody>
<tr>
<td>15:00-15:22</td>
<td>S21-1</td>
<td>Comparisons of Short Term Efficacy between Individual and Group</td>
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<tr>
<td></td>
<td></td>
<td>Cognitive - Behavioral Therapy for Primary Insomnia</td>
</tr>
<tr>
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<td></td>
<td>Wataru Yamadera (Japan)</td>
</tr>
<tr>
<td>15:22-15:44</td>
<td>S21-2</td>
<td>The Effect of Cognitive Behavioral Therapy for Insomnia on Vulnerability to</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Development and Relapse of Insomnia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Isa Okajima (Japan)</td>
</tr>
<tr>
<td>15:44-16:06</td>
<td>S21-3</td>
<td>Association Between Treatment Outcome and Changes in Cognitive and Behavioral</td>
</tr>
<tr>
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<td></td>
<td>Variables Following CBT for Insomnia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chien-Ming Yang (Taiwan)</td>
</tr>
<tr>
<td>16:06-16:28</td>
<td>S21-4</td>
<td>Cognitive Behavior Therapy for Secondary Insomnia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Yuichi Kamei (Japan)</td>
</tr>
<tr>
<td>16:28-16:30</td>
<td>Discussion</td>
<td></td>
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</table>
## ASRS 2012 Speaker List

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<thead>
<tr>
<th>Country</th>
<th>Name</th>
<th>Topic</th>
</tr>
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<tbody>
<tr>
<td>Australia</td>
<td>Brett Duce</td>
<td>Quality Control of PSG in Australasian Sleep Technologists Association</td>
</tr>
<tr>
<td>China</td>
<td>Bao-Yuan Chen</td>
<td>Obstructive Sleep Apnea and Hypertension – a Multicenter Study in China</td>
</tr>
<tr>
<td></td>
<td>Fang Han</td>
<td>Respiratory Control in Sleep Apnea: from Phenotype to Genotype</td>
</tr>
<tr>
<td></td>
<td>Zhi-Li Huang</td>
<td>Key Roles of Dopamine D2 Receptor in the Sleep-Wake Regulation</td>
</tr>
<tr>
<td></td>
<td>Yuan-Ming Luo</td>
<td>Neural Drive of Diaphragm and Upper Airway Muscles in Sleep Apnea</td>
</tr>
<tr>
<td></td>
<td>Jing-Ying Ye</td>
<td>Multiple Factors Predicting the Outcome of Surgery for Obstructive Sleep Apnea</td>
</tr>
<tr>
<td></td>
<td>Xi-Long Zhang</td>
<td>Principle and Efficacy of Transvenous Phrenic Nerve Stimulation for Cheyne-Stokes Respiration in Patient with Chronic Heart Failure</td>
</tr>
<tr>
<td>Germany</td>
<td>Ursula Voss</td>
<td>Conscious Awareness in Dreams: Proof-Of-Principle</td>
</tr>
</tbody>
</table>
| Hong Kong| Victor Abdullah            | 1. Midazolam Sedation Sleep Endoscopy: What Can We See Now after 20 Years  
2. Debate: Conservative Treatment is Superior to Surgery in the Management of OSA |
|          | Crover Ho                  | Quality Control of PSG in Hong Kong                                  |
|          | Albert Martin Li           | Cardiovascular Complications in Children with Obstructive Sleep Apnoea |
|          | David S.C. Hui             | 1. Long-Term Effects of CPAP on Carotid Artery Intima-Media Thickness  
2. Sleep Education in Hong Kong                                           |
|          | Yun Kwok Wing              | Longitudinal Outcome of RBD, RBD Variants or Questionnaire Measurement of RBD |
| India    | Hruda N Mallick            | Thermoregulation as Sleep Regulator                                    |
|          | Bindu M Kutty              | Importance of Yogic Practice in Promoting Sleep                       |
|          | Kamlesh K Gulla            | Role of Sleep in Organizing Central Networks for Sexual Functions     |
| Israel   | Jean Askenasy              | 1. Questions Post Hypocretin / Orexin Discovery                      
2. Dreams versus Hallucinations                                           
3. RLS and PLMS, Two Phenotypes of the Sleep Leg Movement Disorder (SLMD) |
|          | Ronit Gilad                | Seizures and Sleep                                                   |
|          | Marieta Anca-Herschkovitsch| Sleep Movements and ADHD                                              |
| Italy    | Armando d'Agostino         | Sleep, Dreams and Psychotic Disorders                                 |
| Japan    | Shintaro Chiba             | 1. The Concept of Sleep Surgery                                       
2. Role of Nasal Breathing during Sleep                                   |
|          | Hiroshi Hagiwarra          | Estimation of Sleep Level Transition using Lorenz Plot of ECG RR Intervals |
|          | Hirotaka Hara              | Application of Caustic Analysis of Snoring Sounds for Dynamic Evaluation of Upper Airway Morphology of OSAS Patients |
|          | Ken-Ichi Honma             | Zeitgeber Effects on Active Brain                                    |
|          | Sato Honma                 | Multi-oscillation System of Mammalian Circadian Clock                  |
|          | Yuichi Inoue               | Clinical Significance of RBD in Parkinson Disease                     |
|          | Shiroh Isono               | Pathophysiology of Upper Airway Obstruction                           |
|          | Tomonori Iwasaki           | Clinical Application to OSAS of Children Using Computational Fluid Dynamics |
|          | Yuichi Kamei               | Cognitive Behavior Therapy for Secondary Insomnia                     |
|          | Takashi Kanbayashi         | Symptomatic Narcolepsy and Hypersomnia, Hypocretin/Orexin Involvements |
|          | Makoto Kikuchi             | Can we prevent OSAS?                                                 |
|          | Takuro Kitamura            | The Incidence and Respiratory Pattern in Pediatric Sleep Apnea         |
|          | Michael Lazarus            | Role of adenosine A2A receptors in the nucleus accumbens for sleep-wake regulation |

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**2012 ASRS Secretariat**  
**Ying Han Gan** Tel: +886-2-8226-1010 ext.107   Fax:+886-2-8226-2785 E-mail: 2012asrs@gmail.com  
**Address:** 6F.-9., No.2, Jian 8th Rd., Jhonghe City, Taipei 235, Taiwan
<table>
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<tr>
<th>Country</th>
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<tr>
<td>Michihiro Mieda</td>
<td>Dissection of Neural Mechanisms Underlying Circadian Pacemakers Using Brain Region/Cell-Specific Bmal1 Deficient Mice</td>
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<tr>
<td>Satoru Miyauchi</td>
<td>Seeing A Dreaming Brain - Brain Activation Accompanying Rapid Eye Movements During REM Sleep</td>
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<td>Soichiro Miyazaki</td>
<td>Sleep Education in Japan</td>
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<td>Seiichi Nakata</td>
<td>CPAP Compliance after Nasal Surgery for CPAP Failure</td>
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<td>Isa Okajima</td>
<td>The Effect of Cognitive Behavioral Therapy for Insomnia on Vulnerability to Development and Relapse of Insomnia</td>
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<td>Takeshi Sakurai</td>
<td>Neural Circuit of Orexin-Producing Neurons</td>
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<td>Keisuke Suzuki</td>
<td>Dream-Enacting Behavior is Associated with Impaired Sleep, Severe Headache-Related Disability and Depressive Symptoms in Migraine Patients</td>
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<td>Akira Tamura</td>
<td>Association between Obstructive Sleep Apnea and Coronary Artery Disease in the Symposium Entitled Sleep Disordered Breathing and Cardiovascular Disease</td>
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<td>Tomomi Tsunematsu</td>
<td>Optogenetic Manipulation of Orexin Neuronal Activity Controls Sleep/Wakefulness State in Mice</td>
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<td>Sunao Uchida</td>
<td>Exercise Effects on Sleep Physiology</td>
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<td>Tomoko Yagi</td>
<td>Quality Control of PSG in Japanese Society of Sleep Research</td>
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<td>Wataru Yamadera</td>
<td>Comparisons of Short Term Efficacy between Individual and Group Cognitive - Behavioral Therapy for Primary Insomnia</td>
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<td>Motoo Yamauchi</td>
<td>OSAS Phenotypes and Breathing Irregularity - Lessons from Animal Studies</td>
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<td>Dai Yumino</td>
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<td>Korea</td>
<td>Ji-Ho Choi</td>
<td>Korean Sleep Education</td>
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<td>Seung-Bong Hong</td>
<td>Functional Correlates of the Consequences of OSA</td>
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<td>Jee-Hyun Kim</td>
<td>Sleep Apnea and Ischemic Stroke</td>
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</table>
| Sung-Wan Kim | 1. My Experience of Sleep Surgery  
2. Limitations and complications of skeletal surgery for OSA |
| Kyoung-Jin Lee | Cytosolic Calcium Concentration and Long-Range Network Connections in the Suprachiasmatic Nucleus |
| In-Young Yoon | 1. Neuroimaging Studies for Pathogenesis of RLS  
2. REM Sleep Behavior Disorder and Nigrostriatal Dopaminergic System |
| Chang-Ho Yun | Cardiopulmonary Coupling as a Sleep Quality Metrics |
| Singapore | Michael Chee | 1. Alterations in Decision Making Induced by Sleep Deprivation  
2. Neural Correlates of Reduced Processing Capacity in Sleep-Deprived Persons |
| Goh Yau Hong | Debate: Conservative Treatment is Superior to Surgery in the Management of OSA |
| June Chi-Yan Lo | Partial and Total Sleep Deprivation: Effects on Performance across Cognitive Domains, Individuals, and Circadian Phase |
| Pon Poh Hsu | Computer-Assisted Quantitative Upper Airway Analysis Following Modified Upppp for OSA |
| Taiwan | Fang-Chia Chang | Neuroendocrines on Epilepsy-Induced Sleep Disruptions |
| Ya-Wei Cheng | Emotion Processing in Sleeping Brain |
| Ching-Sui Hung | Local, Experience-Dependent Sleep/Wake Regulation and Brain Plasticity |
| Terry Bo-Jau Kuo | Performance of the Frequency Domain Indices with Respect to Sleep Staging |
| Li-Ang Lee | Changes of Snore Sound after Sleep Surgery |
| Hsueh-Yu Li | 1. Multilevel Surgery for OSA  
2. The Role of Nose in Sleep – Disorder Breathing Patients |
<p>| Cheng-Hui Lin | Anatomic Predictors of Incomplete Remission in Pediatric Sleep Apneics after Tonsillectomy and Adenoidectomy: A 3D-CT Analysis |
| Chia-Mo Lin | Taiwan Status of Sleep Education |
| Hsin-Ching Lin | Minimally Invasive OSA Surgery: Rationale, Pitfalls and Outcomes |
| I-Mei Lin | The Relationship between Depressive Symptoms, Sleep Arousal, and Autonomic Activities |
| Sheng-Yi Liu | Undetermined |
| Yu-Che Tsai | The Relationship of Sleep and Autonomic Activities in Hypertension |
| Pa-Chun Wang | Undetermined |
| Chang-Wei Wu | What Happens to Our Brain upon Awakening? |
| Chien-Ming Yang | Association between Treatment Outcome and Changes in Cognitive and Behavioral Variables Following CBT for Insomnia |</p>
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<tr>
<td>Thailand</td>
<td>Prasit Mahakit</td>
<td>1. Tongue Base Surgery for Moderate to Severe OSA</td>
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<td>2. Multilevel Surgery for OSA</td>
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<td>Chairat Neruntarat</td>
<td>Palatal Surgery for Obstructive Sleep Apnea</td>
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<td>Aroonwan Preuthipan</td>
<td>Congenital Central Hypoventilation Syndrome: Thailand Experience</td>
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<td>Turkey</td>
<td>Murat Aksu</td>
<td>Impact of Sleep Disorders on Occupational Health</td>
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<td>Sadik Ardiç</td>
<td>An Overview of Epidemiology of Sleep Among the Working Population</td>
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<td>Adile Oniz</td>
<td>Implicit Memory and Tactile Stimulations in Sleep</td>
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<td>Brain Responsiveness in Search of Active Brain</td>
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<td>Hikmet Yılmaz</td>
<td>Impact of Work Related Conditions on Sleep</td>
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<td>U.S.A.</td>
<td>Jiang-Fan Chen</td>
<td>Adenosine A&lt;sub&gt;2A&lt;/sub&gt; Receptors in Parkinson’s Disease and Neuroinflammation</td>
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<td>Resulting in Dysfunction of the Motor System and Poor Sleep</td>
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<td>Ying-Hui Fu</td>
<td>Clock Genes and Human Sleep Behavior Phenotypes</td>
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<td>Jun Lu</td>
<td>Dissecting the Basal Ganglia Control of Sleep-Wake Behavior</td>
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<td>Seiji Nishino</td>
<td>Etiological/Pathophysiological Mechanisms of Hypersomnia: from Anima Studies to Bedside</td>
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<td>Dinesh Pal</td>
<td>Changes in Anterior-Posterior Feedback Connectivity during Anesthesia and Sleep in Rat</td>
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<td>Denise Sharon</td>
<td>From Restless Legs Syndrome to Willis-Ekbom Disease: A Diagnostic and Treatment Conundrum</td>
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<td>Mark Opp</td>
<td>Cytokines, Neuroinflammation, and Sleep</td>
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## CURRENT EMPLOYMENT

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<tr>
<th>Time</th>
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<tr>
<td>APR 2006 – PRESENT</td>
<td>Sleep Disorders Centre</td>
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<tr>
<td></td>
<td>Princess Alexandra Hospital</td>
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<td>Scientific Director</td>
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## CURRENT TEACHING

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<tr>
<td>2006-CURRENT</td>
<td>Lecturer, Masters of Clinical Physiology, Griffith University. (2 Lectures per year).</td>
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## POSITIONS/OFFICE

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<tr>
<td>2008-CURRENT</td>
<td>President, Australasian Sleep Technologists Association</td>
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<tr>
<td>2009-CURRENT</td>
<td>Member, ASA Laboratory Accreditation Subcommittee</td>
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<tr>
<td>2006-CURRENT</td>
<td>Member, Queensland Health Sleep Disorders Program Executive Committee</td>
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<tr>
<td>2008-CURRENT</td>
<td>Member, Statewide Clinical Measurements Network</td>
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<tr>
<td>2010-CURRENT</td>
<td>Chair, Clinical Measurements Advisory Group</td>
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</table>

## MEMBERSHIPS

- Australasian Sleep Technologists Association
- Australasian Sleep Association
- American Academy of Sleep Medicine

## GUIDELINE PUBLICATIONS


## RECENT PUBLICATIONS


Curriculum Vitae

Name: Sheng-Yi Liu

Sex: Male

Citizenship: Taiwan

Position:
  Director, Department of Sleep Technology, Taiwan Society of Sleep Medicine
  Consultant, Sleep Laboratory, Taipei Veterans General Hospital
  Consultant, Brain Research Center, National Chiao Tung University

Education:
  Department of Health Education, National Taiwan Normal University (1967~1972)

Training:
  Stanford University Sleep Disorders Center, USA (1989)
  Wisconsin University Sleep Disorders Center, USA (1992)
  American Association of Sleep Technologists Annual Meeting (2005~2010)

Employments:
  Chief Technologist, Chest Department, Taipei Veterans General Hospital (1997~2003)
  Clinical Instructor, Department of Biomedical Engineering, National Yang Ming University (1994~2003)

Textbooks:
  Sheng-Yi Liu, Practice of Sleep Medicine, Ho-Chi Book Publishing CO., Taipei, Taiwan (2011)

E-mail: syliu@ms94.url.com.tw
CURRICULUM VITAE

Shiroh Isono, M.D., Ph.D.

Dr. Isono's areas of expertise and interest include respiratory physiology and sleep medicine, particularly the pathophysiology of upper airway maintenance in unconscious subjects. His clinical interests include perioperative airway management, and diagnosis and treatment of obstructive sleep apnea.

Degrees and other medical licenses
1984   M.D., National Medical Board (License to practice medicine in Japan)
1991   Instructor of Anesthesiology (Japanese Society of Anesthesiologists)
1995   Ph.D., Chiba University School of Medicine, Japan
2005   Authorized Physician of Japan Society of Sleep Medicine

Current Professional Position
June 2012-present Professor of Department of Anesthesiology,
Graduate School of Medicine, Chiba University

Research Experiences outside JAPAN
Aug 1990-Mar 1993 Research Fellow at Department of Medicine, University of Calgary
Mentor: John E Remmers, M.D., Ph.D.

Academic Activities
2007-present Board member of Japan Society of Sleep Medicine
2008-present Board member of Japanese Society of Anesthesiologists
2005-present Editorial board member, Journal of Applied Physiology
2009 January Editor, Anesthesiology

Awards
2009 Sixth Honorary Member Award (The American Academy of Dental Sleep Medicine)

Representative Original Article

Publications (English): 77 peer-reviewed original articles
Tomonori Iwasaki

Curriculum vitae
- May 1990 Graduated from School of Dentistry, Faculty of Dentistry, Kagoshima University
- April 1990 Entered Graduate School of Dentistry, Kagoshima University (Pediatric Dentistry)
- April 1994 Joined Kagoshima University of Dentistry as physician assistant
- August 2010 Lecturer of Kagoshima University Medical and Dental Hospital

Award
- May 2008 Award of the Excellent presentation, The 46th Conference of the Japanese Society of Pediatric Dentistry (Oomiya)
- September 2008 Award of the Excellent presentation, The 67th Annual Meeting of Japan Orthodontic Society (Chiba)
- September 2010 Award of the Excellent presentation, The 67th Annual Meeting of Japan Orthodontic Society (Yokohama)
- June 2012 Award of the Best presentation, The 37th Annual Meeting of Japanese Society of Sleep Research (Yokohama)
CURRICULUM VITAE

Name SHINTARO CHIBA
Date of Birth July 22 1961
Place of Birth Iwate, Japan

PRESENT POSITION

Associate Professor
Department of Otorhinolaryngology
Jikei University School of Medicine
3-25-8 Nishishinbashi, Minato-ku, Tokyo
105-8471 Japan

EDUCATION

2007 Ph.D., Otorhinolaryngology, Jikei University school of Medicine
2002 Sleep Physician's License in Japan (No. 140)
1994 Certificated Otorhinolaryngologist in Japan (No. 9382)
1989 Physician's License in Japan (No. 365573)
1989 M.D., Jikei University school of Medicine

PROFESSIONAL EXPERIENCE

2010-2012 Visiting Associate Professor
Sleep & Circadian Neurobiology Laboratory
Center for Narcolepsy
Stanford Sleep Research Center
Stanford University School of Medicine

Japan:
2007-present Associate Professor
Department of Otorhinolaryngology,
Jikei University School of Medicine, Tokyo, Japan
2001-2004 Director
Sleep Disorder Center,
Ota General Hospital, Kanagawa, Japan
1999-Present Director
Department of Otorhinolaryngology,
Ota General Hospital, Kanagawa, Japan
1991-1998 Clinical Fellow
Department of Otorhinolaryngology,
Jikei University School of Medicine, Tokyo, Japan
1989-1990 Resident
Department of Otorhinolaryngology,
Jikei University School of Medicine, Tokyo, Japan
PROFESSIONAL SOCIETIES

The Oto-Rhino-Laryngological Society of Japan
Japan Rhinologic Society
Japan Society of Stomato-Pharyngolory
Japanese Telemedicine and Telecare Association
The Japanese Society of Sleep Research
American Academy of Sleep Medicine
Makoto Kikuchi

personal curriculum vitae

1973 Graduate from Tokyo Dental college
1977 PhD course at Tokyo Dental College, Department of Orthodontics
1977 Private practice at Narita, Chiba
1977-1982 Visiting lecturer at Tokyo Wemen’s Medical University, Department of Dentistry
1977-1993 Visiting lecturer at Tokyo dental College, Department of Orthodontics
1991 Authorized dentist in Orthodontics and Authorized Orthodontic Instructor
1998 Cosmos center for sleep respiratory disordered at Narita, chiba
2000 American Board of Dental Sleep Medicine
2002 Japanese Board of Dental Sleep Medicine
2008 Japanese Board of Orthodontics & Dentofacial Orthopedics
Robert Stickgold is an associate professor of psychiatry at Beth Israel Deaconess Medical Center and Harvard Medical School. He received his B.A from Harvard University and his Ph.D. from the University of Wisconsin, Madison, both in biochemistry. His early research was on bacterial cell wall synthesis and bacterial DNA replication. He had post-doctoral fellowships at Stanford Medical School in neurochemistry (with Eric Shooter) and at Harvard Medical School in neurophysiology (with Stephen Kuffler) before becoming an assistant professor of physiology at the University of Massachusetts Medical School. He subsequently left this position to work in the private sector for several years, before taking his current position at Harvard, where he has been since 1990. He has published two science fiction novels, and over 100 scientific publications, including papers in *Science*, *Nature*, and *Nature Neuroscience*. His work has been written up in *Time*, *Newsweek*, *The New York Times*, *The Boston Globe Magazine*, and *Seed Magazine*, and he has given invited talks around the world, including Brazil, Germany, Portugal, Sweden, Switzerland, Japan, and The Netherlands. He has been a guest on *The Newshour with Jim Leher* and on NRP’s *Science Friday* with Ira Flato several times.

Research from his laboratory has led to a recognition of the many forms of sleep-dependent memory processing, including (i) the consolidation, cortical reorganization, and enhancement of both perceptual and motor procedural learning, (ii) the consolidation and stabilization of verbal declarative memory, (iii) the selective consolidation of emotional elements from complex scenes, (iv) the extraction and selective consolidation of the gist representation from sets of semantically related verbal memories, and (v) the discovery of complex rules within large data sets. It has also shown that these differing memory processes are dependent on specific sleep stages and patterns of brain activity, described in both EEG and fMRI studies.

His research has shown that one form of sleep-dependent memory processing is absent in patients with schizophrenia, in parallel with a disease-related decrease in the frequency of sleep spindles in their EEGs. Preliminary results suggest that normal sleep spindle activity and sleep-dependent memory processing can be restored pharmacologically. In parallel studies, he has shown the same deficit in sleep-dependent memory processing is patients with mild sleep apnea, correlated with the number of arousals occurring across the night.

Dream studies in his laboratory have demonstrated that objective (“manifest”) dream content can be manipulated experimentally, and, using this paradigm, he has shown that patients with dense amnesia, caused by bilateral medial temporal lobe damage, can be induced to dream about experiences for which they have no conscious (declarative) memory. He has further shown that sleep-dependent improvement on a spatial navigation task is limited to those individuals who report, on experimental probes, to have dreamed about the task, providing arguably the strongest evidence to date of a functional role of dreaming, specifically in sleep-dependent memory processing.

His current work looks at the nature and function of sleep and dreams from a cognitive neuroscience perspective, with an emphasis on the role of sleep and dreams in memory consolidation and integration. In addition to studying the normal functioning of sleep, he is currently investigating alterations in sleep-dependent memory consolidation in patients with schizophrenia, autism spectrum disorder, and PTSD. His work is funded by NIMH and Autism Speaks. He has recently consulted to and received research funding from Merck & Co., Sepracor Pharmaceuticals, and Actelion Pharmaceuticals, and has consulted to Epix Pharmaceutical and Eli Lilly.
David P. White, MD  
Short Bio

Dr. White graduated from Emory University Medical School and completed training in Internal Medicine and Pulmonary Disease at the University of Colorado Health Sciences Center. He has held a number of positions at various universities over his career and is currently a Clinical Professor of Medicine at the Harvard Medical School and the Chief Medical Officer for Philips Respironics. Some of his major accomplishments include being President of the American Academy of Sleep Medicine, Chairing the Task Force to write the research plan addressing sleep and its disorders for the National Institutes of Health, and being the Editor-in-Chief of the Journal SLEEP. He was also on the test writing committees for the American Board of Internal Medicine for both Pulmonary Disease and Sleep Medicine. Finally, his principle research interest has been the pathophysiology of disorders of breathing during sleep about which he has published over 150 original papers.
Dr. Michael Wei-Liang Chee

I. Professional Qualifications:

1993  Fellow, Academy of Medicine Singapore (Neurology)
1992  Diplomate of the American Board of Clinical Neurophysiology
1987  Membership of The Royal College of Physicians United Kingdom (MRCP (UK))
1983  Bachelors in Medicine, Bachelors in Surgery; MBBS (Singapore)

II. Awards and Fellowships:

2009  National Outstanding Clinician Scientist Award
2009  Fellow Of The Royal College Of Physicians Edinburgh FRCP(Edin)
2008  Singapore Translational Research Investigator Award
2005  BMRC-NMRC Clinician Scientist Investigator Award
2004  SingHealth Investigator Excellence Award
2003  Singapore General Hospital, Excellent Publication Award
2000  Fellow, Summer Institute in Cognitive Neuroscience; Dartmouth College, USA
1999  Singapore General Hospital Commendable Researcher Award
1996  National Medical Research Council, Senior Medical Research Fellowship concurrent with Academy of Medicine, Singapore, Traveling Fellowship Award (Special Fellow, Massachusetts General Hospital NMR Centre, Boston MA, USA)
1993  Parke Davis International Epilepsy Service Award
1992  First Prize, Cleveland Clinic Neuroscience Residents Day, HMDP scholarship in EEG and Clinical Neurophysiology, The Cleveland Clinic Foundation
1981  General Practitioners Student Essay Book Prize. First Place
1978  Public Service Commission Scholarship (Medicine)

III. Present status and professional experience:

  Professor, Cognitive Neuroscience Laboratory, Duke NUS Graduate Medical School
**Professional Experience:**

<table>
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<tr>
<th>Year</th>
<th>Position</th>
<th>Institution</th>
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<tr>
<td>2006</td>
<td>Professor</td>
<td>Duke-NUS Graduate Medical School</td>
</tr>
<tr>
<td></td>
<td>Consultant</td>
<td>Sleep Disorder Unit, SGH</td>
</tr>
<tr>
<td>2002</td>
<td>Clinician-Scientist</td>
<td>Cognitive Neuroscience laboratory, Singapore Health Services</td>
</tr>
<tr>
<td>1997</td>
<td>Principal Investigator</td>
<td>Cognitive Neuroscience laboratory, Singapore General Hospital (Senior Consultant from 2001)</td>
</tr>
<tr>
<td>1996</td>
<td>Special Fellow</td>
<td>Massachusetts General Hospital NMR Centre August 1996 to February 1997</td>
</tr>
<tr>
<td>1994</td>
<td>Consultant</td>
<td>Department of Neurology, Singapore General Hospital</td>
</tr>
<tr>
<td>1992</td>
<td>Senior Registrar</td>
<td>Department of Neurology, Tan Tock Seng Hospital</td>
</tr>
<tr>
<td>1990</td>
<td>Fellow</td>
<td>Section of Epilepsy and Sleep Disorders, The Cleveland Clinic Foundation, Ohio, USA</td>
</tr>
<tr>
<td>1988</td>
<td>Registrar</td>
<td>Department of Neurology, Tan Tock Seng Hospital</td>
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<tr>
<td>1985</td>
<td>Medical Officer</td>
<td>Department of Medicine III, Singapore General Hospital</td>
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<td>Department of Respiratory and general Medicine, Tan Tock Seng Hospital</td>
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<td>Department of Medicine I, Singapore General Hospital</td>
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<tr>
<td>1983</td>
<td>House Officer</td>
<td>Department of Medicine III, Singapore General Hospital</td>
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<tr>
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<td>Department of Surgery, Alexandra Hospital</td>
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<td>Department of Pediatrics, Alexandra Hospital</td>
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</tbody>
</table>
1. Name: In-Young Yoon

2. Date of Birth: September 10, 1963

3. Address:
   
   Department of Neuropsychiatry, 
   
   Seoul National University Bundang Hospital 
   
   166 Gumi-ro, Bundang-gu, Seongnam-si, Gyeonggi-do, 463-707

4. E-mail Address: iyoon@snu.ac.kr

5. Academic Background:
   
   1982 - 1990  M.D. degree from Seoul National University, College of Medicine, Seoul, Korea
   
   1994 - 1996  Master degree from Seoul National University, Graduate School, Seoul, Korea
   
   1996 - 1999  Ph.D. degree from Seoul National University, Graduate School, Seoul, Korea

6. Career
   
   1990 - 1991 Internship in Seoul National University Hospital
   
   1991 - 1995 Residency in Psychiatry, Seoul National University Hospital
   
   1995. 3.  Korean Board of Psychiatry
   
   1995 - 1996 Fellowship in Sleep Medicine, Seoul National University Hospital
   
   1996. 3, - 2001.7 Staff Psychiatrist in Yong-In Mental Hospital
   
   2001.7. - 2003, 2 Visiting Professor, University of California, San Diego
   
   2003. 3 - 2008.9. Assistant Professor, Department of Neuropsychiatry, Seoul National University 
   
   Bundang Hospital
2008. 10 - Present  Associate Professor, Department of Neuropsychiatry, Seoul National University
Bundang Hospital

2010. 6. 14  Completion of GCP training at Seoul National University Bundang Hospital

7. Membership:
Korean Medical Association
Korean Neuropsychiatric Association
Korean Society of Sleep Medicine
American Academy of Sleep Medicine

8. Recent Publications:
1) Increased striatal dopamine transporter density in moderately severe old restless legs syndrome patients.

2) Effects of Sleep Apnea Syndrome on Delayed Memory and Executive Function in the Elderly.


June Chi Yan LO

Contact Information

Address:  Cognitive Neuroscience Laboratory, Duke-NUS Graduate Medical School, 8 College Road, Singapore 169857

Email:    june.lo@duke-nus.edu.sg

Education

2011   Ph.D. (Psychology)  
       University of Surrey, UK

2005   M.Phil. (Psychology)  
       The Chinese University of Hong Kong

2003   B.S.Sc. (Psychology)  
       The Chinese University of Hong Kong

Current Employment

From 2012  
Position:    Research Fellow  
Institution:   Cognitive Neuroscience Laboratory, Duke-NUS Graduate Medical School, Singapore

Past Employment

2011  
Position:   Part-time Lecturer  
Institution:  Community College of City University, Hong Kong

2011  
Position:   Part-time Lecturer  
Institution:  HKU SPACE Po Leung Kuk Community College, Hong Kong

2008-2011  
Position:   Post-doctoral Research Fellow in Sleep, Circadian Rhythm, and Cognition  
Institution:  Surrey Sleep Research Centre, University of Surrey, UK

Title of project:  Cognitive Vulnerability Following Extended Wakefulness in Defined Genotypes: Effects of Sleep Duration on Sustained Attention, Executive Function and Novel Biomarkers

Research Interests

• The psychological and behavioural impact of sleep deprivation
• Sleep, cognitive performance, and health in elderly
• Sleep and memory consolidation
• Diurnal preference in Asian populations
CURRICULUM VITAE

Name: Takuro Kitamura, MD
Birth Date: March 26, 1970
Office Address:
Department of Sleep Medicine, Shiga University of Medical Science
Seta Tsukinowa-cho, Otsu, Shiga, JAPAN 520-2192
Nationality: Japanese
Specialty: SDB, Nasal Surgery, Sleep Surgery

Education:
1988-1994 University of Occupational and Environmental Health, School of Medicine, awarded MD

Medical Training:
1994-2000 Resident of Otolaryngology at University of Occupational and Environmental Health, School of Medicine
2000-2001 Kumamoto Rosai Hospital

Research and Academic Positions:
2001-2011 Assistant Professor, Department of Otolaryngology, University of Occupational and Environmental Health, School of Medicine
2011- Assistant Professor, Department of Sleep Medicine, Shiga University of Medical Science

Medical Certification
Otorhinolaryngology Diplomate, The Oto-Rhino-Laryngological Society of Japan
CURRICULUM VITAE

Name: LI Albert Martin

Present Post: Professor and Honorary Consultant, Department of Paediatrics, Prince of Wales Hospital, The Chinese University of Hong Kong, Shatin, Hong Kong

Dr. Li graduated from the University of Wales College of Medicine, Cardiff, United Kingdom and received his paediatric training at King’s College Hospital and Great Ormond Street Hospital, United Kingdom. He joined the Chinese University of Hong Kong as a lecturer in August 2001. His training took him back to the UK where he worked as a clinical research fellow at the Royal Brompton Hospital, London in 2002, under the guidance of Prof. Andy Bush.

His clinical and research interest is in respiratory and sleep medicine. He is currently the vice-president of the Asia Pacific Paediatric Sleep Alliance and vice-president of the Hong Kong Society of Sleep Medicine.

Academically, he recently completed his 5-year term as editorial board member of the Journal of Pediatrics. He is currently associate editor of the Hong Kong Journal of Paediatrics. He has published over 150 peer-reviewed articles. For 5 consecutive years, he was awarded “Best Clinical Teacher” by the medical faculty of CUHK.
Curriculum Vitae

Name: Lin, Cheng-Hui  
Sex: Male  
Citizenship: Taiwan  
Education: July 1995, MD, Taipei Medical College, Taipei  
July 2007, Master, Graduate Institute of Dental and Craniofacial Science, Chang Gung University (碩字 0544)  
Professional Appointment :  
June, 2012 : Assistant Professor, Plastic Surgery, CGMH.Taoyuan  
Board Certification :  
Dec. 2001, Surgeon Board of Taiwan, No. 004607  
Oct. 2003, Plastic Surgery Board of Taiwan, No. 366  
March 2004, Hand Surgery Board of Taiwan.  
March 2012, Sleep Medicine Board of Taiwan, No. 105.  
Licensers: Taiwanese License NO: 5933  
Lecturer, Department of Education, No. 088287 (2006 Aug)  
Professional Affiliations: Membership:  
Surgical Association of Taiwan, No. 2-4060  
Plastic Surgical Association, Taiwan, No. 366  
Hand Surgery Association  
Cleft Lip Palate Craniofacial Association, Taiwan (Secretary General)  
Taiwan Society of Sleep Medicine (Secretary General)  
American Cleft Palate Association  
Asian-Pacific Cleft Association  
World Association of Sleep Medicine  
American Association of Sleep Medicine  
Research Interest:  
1. Craniofacial Development, Growth and Deformation, 3D Image Analysis  
2. Craniofacial Surgery in Obstructive Sleep Apnea  
3. Computer Aided Surgical Simulation  
Research visits:  
1. Euclidean Distance Matrix Analysis (EDMA), Dr. Richtsmeiers’ Lab, Pensylvania State University, USA, Oct 2003  
2. Sleep Surgery, Sleep Center, Stanford University, Palo Alto, Claifornia, USA, October 2006; Sep 2007;  
3. Computer Assisted Surgical Simulation, Department of Maxillofacial Surgery, Methodist Hospital, Houston, Texs, USA, March 2011.  
Research Fellowship:  
1. Sleep Center, Stanford University Medical Center, September 2009– August 2010.
BRIEF CURRICULUM VITAE
AROONWAN PREUTTHIPAN, MD., FCCP.

EDUCATION:
1984 M.D., Faculty of Medicine, Ramathibodi Hospital,
Mahidol University, Bangkok, Thailand
1990 Diplomate Thai Board of Pediatrics
1992 Diplomate Thai Sub-Board of Pediatric Pulmonology

POST-GRADUATE TRAINING:
1992-1994 Visiting Faculty, Divisions of Pediatric Respiratory Sciences, Pediatric
Sleep and Breathing Disorders Center and Pediatric Intensive Care, the
Johns Hopkins Children’s Center, Baltimore, Maryland, USA.

PROFESSIONAL EXPERIENCE:
2011 - Present Director, Ramathibodi Hospital Sleep Disorder Center
2005-Present Professor and Director, Division of Pediatric Pulmonology, Ramathibodi
Hospital, Faculty of Medicine, Mahidol University

COMMITTEES:
• International Organizing Committee, the International Congress on Pediatric Pulmonology, 2003-present
• Executive Committee, Sleep Society of Thailand, 2010-present
• International Scientific Committee. The 7th Sleep Respiration Forum in Kyoto in conjunction
  with World Sleep 2011, October 13-14, 2011.
• Scientific Chair, Thai Association of Pediatric Pulmonology and Critical Care Medicine
  (TPRC) 2011-present

HONORS AND AWARDS:
2003 2003 Governors Community Service Award for the project, “Home Mechanical
Ventilation for Children in Thailand”, the Chest Foundation, the American
College of Chest Physicians
2012 President, Local Organizing Committee, the 11th International Congress on
Pediatric Pulmonology, Bangkok, Thailand

PUBLICATIONS:
33 articles and one chapter in English. 86 chapters in Thai. Editor of 12 Thai books.

INVITED SEMINAR:
14 lectures or symposiums in International Meetings in English
150 lectures or symposiums or workshops in Thai
BIOGRAPHICAL SKETCH

NAME
Takashi Kanbayashi

POSITION TITLE
Associate Professor

EDUCATION/TRAINING (Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Akita University School of Medicine, Akita, Japan</td>
<td>Ph.D.</td>
<td>1998</td>
<td>Neuroscience, Sleep, Pharmacology, Psychiatry</td>
</tr>
<tr>
<td>Akita University School of Medicine, Akita, Japan</td>
<td>M.D.</td>
<td>1990</td>
<td>Medicine</td>
</tr>
</tbody>
</table>

A. Positions and Honors

Positions and Employment

1990-1992  Resident in Neuropsychiatry, Akita University School of Medicine
1992-1998  Ph.D program, Akita University School of Medicine
1994-1996  Post-doctoral Research Fellow, Stanford University School of Medicine.
1998-2006  Assistant Professor, Akita University School of Medicine
2006-Present  Associate Professor, Akita University School of Medicine

Other Experience and Professional Memberships:

Japanese Society of Sleep Research
Sleep Research Society (USA)
Society for Neuroscience (USA)
CURRICULUM VITAE

Name  SEIJI NISHINO
Date of Birth  September 1, 1955
Place of Birth  Osaka, Japan
Visa Type  Permanent Resident of USA

PRESENT POSITION

Professor
Department of Psychiatry and Behavioral Sciences
Stanford University School of Medicine
1201 Welch Road, MSLS, P213
Palo Alto, CA  94305-5485    (650) 723-3724
E-mail: nishino@stanford.edu

EDUCATION

2004  Sleep Physician's License in Japan (No. 228)
1984-1990 Ph.D., Neuropsychiatry, Osaka Medical College
1988  Designated physician of Mental Health in Japan (No. 7859)
1982  Physician's License in Japan (No. 269441)
1982  M.D., Osaka Medical College

RESEARCH AND PROFESSIONAL EXPERIENCE

USA:
2007-present  Professor
Department of Psychiatry and Behavioral Sciences
Stanford University School of Medicine, Palo Alto, California
2001-2007  Associate Professor
Department of Psychiatry and Behavioral Sciences
Stanford University School of Medicine, Palo Alto, California
1996-2001  Senior Research Scientist
Department of Psychiatry and Behavioral Sciences
Stanford University School of Medicine, Palo Alto, California
1991-1996  Visiting Assistant Professor
Department of Psychiatry and Behavioral Sciences,
Stanford University School of Medicine, Palo Alto, California
1989-1991  Postdoctoral Research Fellow
Department of Psychiatry and Behavioral Sciences,
Stanford University School of Medicine, Palo Alto, California
1987-1989  Visiting Scholar
Department of Psychiatry and Behavioral Sciences,
Stanford University School of Medicine, Palo Alto, California

JAPAN:
2010- present Visiting Researcher
The Tokyo Metropolitan Institute for Neuroscience, Tokyo, Japan.
2001- present Visiting Professor
Anatomy, Showa University, School of Medicine, Japan
1991- present Visiting Lecturer
Neuropsychiatry, Osaka Medical College, Takatsuki, Japan
1985-1988 Research Fellow
Hayaishi Bioinformation Transfer Project, Research Development Corporation of Japan, Exploratory Research for Advanced Technology, Takatsuki, Japan
1982-1984 Resident
Neuropsychiatry, Osaka Medical College, Takatsuki, Japan

AWARDS
2003 Narcolepsy Network Scientist Award
1998-03 NIMH Mentored Research Scientist Developmental Award (K01)
1997 David Amar Award- Israel Sleep Research Society
1987 Award of Sankyou - Sankyou Seimei Science Foundation

PROFESSIONAL SOCIETIES

Neuroscience Society
Sleep Research Society
European Sleep Research Society
Japanese Sleep Research Society
New York Academy of Science

REVIEWER

2008-2010 NIH study BRS (biological Rhythm and Sleep) study section
02/2011 Temporary member for NIH, NPAS (Neural Basis Of Psychopathology, Addictions And Sleep Disorders Study Section)
2010-2011 Ad hoc member for NIH RFA-HL and Lasker Clinical Research Scholars Program study section

Lancet; Neurology; Annals of Neurology; Lancet Neurology; Sleep; Journal of Clinical Investigation; European Journal of Neurology; Clinical Neurophysiology; J. Neuroscience; Journal of Neurology, Neurosurgery & Psychiatry; European Journal of Neuroscience. J. Neurophysiology; Neuroscience; Clinical Pharmacology; Biological Psychiatry; Sleep Medicine, Journal of Clinical Sleep Medicine; Journal of Clinical Psychopharmacology; Neuropsychopharmacology; PNAS; FESEB Journal; Physiology and Behavior; PloSONE; Pharmacology, Biochemistry and Behavior; Journal of Psychiatric Research; Neuropharmacology; Brain Research; Sleep Research Online; Sleep Medicine Review; Life Science; Neuroscience letters; Prostaglandins, Trend in Pharmacological Sciences;
Trend in Biochemical Sciences; Israel Medical Association Journal; Expert Opinion on Investigational Drugs; Expert Opinion on Pharmacotherapy; American Journal of Physiology; Academic Psychiatry; Journal of Clinical Endocrinology and Metabolism; The Journal of Physiology; Journal of Neurophysiology; Human Genetics: Sleep and Biological Rhythms; Clinica Chimica Acta; European Journal of Neurology; Diabetes; Behavioral Brain Research; The Cochrane Library; Journal of Small Animal Practice; the Journal of Human Genetics; Acta Physiologica, Psychoneuroendocrinology; Journal of Psychiatric Research
Jean Jacques ASKENASY , M.D., Ph.D.

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Curriculum Vitae

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- Awards

Jean Jacques ASKENASY , M.D., Ph.D.

Department: Department of Neurology
Institution: The Assuta Hospital
Address: 20 Habarzel
City, State, ZIP: Tel-Aviv 69710
Country: Israel
Telephone: 972-9-9543354 / 972-9-9543242
FAX: 972-9-9572767
E-mail address: ajean@post.tau.ac.il jeani@bezeqint.net

Curriculum Vitae

BORN : Nov. 13, 1929

TRAINING

School of Medicine – University of Cluj –Roumania – MD 1954
Residence of Neurology – Neurological Institute of the Academy – 1956
University Parhon Bucharest – D. Sc. – 1969
Sackler School of Medicine TAU. Lecturer and Senior Lecturer 1976-79
Mount-Sinai School of Medicine NY University- Associated Prof. 1981
Pierre et Marie Curie University Professeur Agrege – Charcot Cl. 1986-
Sackler School of Medicine TAU. Professor of Neurology 1995

NOMINATIONS

Chairman of the Neurological Department at CFR2 Hospital Bucharest
Chairman of the Sleep Medicine Institute at Sheba Medical Center
President of the Israeli Sleep Medicine Association
Vice President of the Asian Sleep Research Society
Editor in Chief of the ASRS Newsletter
Member of the Appeal Commission - Court of Law - Rishon Lezion
Member of the Tel-Aviv Journalists Association
Member of the Superior Appeal Commission for Disabled IDF Staff
Chairman of the Superior Appeal Commission - Ministry of Health
Chairman of the Superior Appeal Commission of the Social Security
Membre d'honneur de la Societe Francaise de Neurologie
Associated Member of the American Academy of Neurology
Honorary Member of the Academy of Men of Science of Romania
Honorary Member of the Roumanian Society for Cell Biology
Member of the Editorial Boards of:
Int.J.of Sleep Medicine
Int. J. of Neurobiology
Int. J. Sleep Research On Line
Constitutive Member of the Israeli Chapter of Forensic Sciences

AWARDS

1964- La Medaille des Journées Internationales de Grenoble - France-
1981- Honor Citizen of the City of Yeruham – Israel-
1995- Medaille Pierre Castaigne – France-
1997- Merit Certificate of the Ministry of Education of Israel
1999- Officier de l'Ordre de Mono – Togo-
2001- Diplome d'honneur de l'AMIF – France
2003- Meritul Academiei Romane – Romania
2012- Crystal Trophy & Diploma of Excellence of Medical Life Gala 2012

Received his doctorate in medicine degree cum laude from the University of Cluj in Romania
1954. Appointed instructor of neurology in his 5 ler School of Medicine. Residence of
Neurology- Neurological Institute of Academy 1956. Doctor in Science at the University
C.I.Parhon Bucharest, 1969. Chairman of the dpt. of Neurology at the Univeristary Hospital CFR
II Bucharest. In 1973 neurologist at Ichilov Hospital-Tel-Aviv University. Researcher in
Neuroscience at Weitzman Institute 1974-1975.In 1981 was appointed Associated Prof. of
Neurology at Mount-Sinai School of Medicine NY University, where he worked together with
Prof. Melvin Yahr and Prof. Eliot Weitzman on the project Sleep in Parkinsonian patients. He subsequently was nominated Director of the Sleep Medicine Center at the Sheba Medical Center. In this quality he organized the courses in sleep medicine at the Tel-Aviv University. In 1986 was nominated Professeur Agrege at Pierre et Marie Curie University, Clinique Charcot. In 1995 was nominated Clinical Professor of Neurology at Sackler School of Medicine TAU. Member of the International Scientific Program Committee of the World Sleep Congress 2007 in Australia. Author of 9 books, 13 chapters in books, 91 original scientific articles & cas reports in the field of Neurology and Sleep Medicine, 29 invited lectures and 175 presentations at national and international congresses.

Publications

Books

1) VIOLENCE

AN INTEGRATED MULTIUVARIATEW STUDY OF HUMAN AGGRESSION
S.G.SHOHAM, JJM.. ASKENASY , G. RAHAV, F.CHARD, A.ADDI, M.ADDAD
CHANG-HO YUN, M.D., Ph.D.

Associate Professor
Department of Neurology and Clinical Neuroscience Center,
Seoul National University Bundang Hospital (SNUBH)

Dr. Chang-Ho Yun graduated Seoul National University College of Medicine in 1994, and finished his resident training and fellowship at the Department of Neurology, Seoul National University Hospital, Seoul, Korea. His area of expertise has been evolved from epileptology to sleep medicine. He is a Certified International Sleep Specialist by American Board of Sleep Medicine (2005). Now he is working as a sleep specialist and clinical researcher at SNUBH. Clinical research interests are to phenotype and quantitate individual trait-like sleep property, to define the genetic and epigenetic background, and to document the effects of sleep quality and quantity on the cognitive function and cardiometabolic health, participating as a co-investigator of the Korean Genome and Epidemiology Study at Ansan, South Korea.

He has also been contributing to the development of sleep medicine in Korea, working as a member of board of councils of the Korean Society of Sleep Medicine (KSSM) and Korean Sleep Research Society, and an executive editor of journal Sleep Medicine Research (www.sleepmedres.org), the official journal of KSSM published in English. He was a member of organizing committee for “9th World Congress on Sleep Apnea Seoul 2009.”
Dr. Bindu M. Kutty, Ph.D
Professor & Head
Department of NEUROPHYSIOLOGY
National institute of Mental Health and Neurosciences
(NIMHANS Deemed University),
Hosur Road, Bangalore 560029

General Secretary, Indian Society for Sleep Research (ISSR)
Teaching faculty, National Sleep Medicine Course, An Advanced Course by Indo-US Foundation for Sleep Education since 2006.
Member, Indian Sleep Medicine Board

Research areas of interest: Role of Subiculum in spatial learning and memory, Sleep and memory, Neuronal plasticity, Human sleep physiology and consciousness, Meditation and sleep

Publication (2005-2012)
6. Dayalan Sampath, Vaishnavi S., Dugalakshmi R., Bindu M. Kutty and
Name: Sunao Uchida
Date of Birth: December 4th, 1956
Gender: Male
Present Position: Professor, Faculty of Sport Sciences Waseda University

Education and Professional Experience:
- March, 1983: Graduated from Shiga University of Medical Science
- May, 1983: Licensee of Medical Doctor in Japan, #274527
- June, 1983 - September, 1984: Resident, Tokyo Medical and Dental University Hospital
- October, 1984 - June, 1986: Psychiatry Medical Staff, Asai Hospital
- July, 1986: Senior Resident, Research fellow, Tokyo Medical and Dental University
- July, 1990: Visiting Researcher, Department of Psychiatry, University of California at Davis, Supervisor: Prof. Irwin Feinberg
- December, 1992 - March 2003: Vice councillor of research, Division of Sleep Disorders Research, Tokyo Institute of Psychiatry
- April 2003 - Present: Professor, Faculty of Sport Sciences, Waseda University
- April 2007 - Present: Clinical Professor, Tokyo Medical and Dental University

Academic Title
- January, 1993: PhD (Medical Science), from Tokyo Medical and Dental University

Academic societies:
- Japanese Society of Psychiatry and Neurology
- Japanese Society for Sleep Research (Board member)
- Japanese Association of Sports Psychiatry (Chairperson)
- Japanese Society of Sport Medicine (Board member)
- Japanese Society for Clinical Neurophysiology (Board member)
- Japanese Society for Neuroscience
- Sleep Research Society (USA)
- Asian Sleep Research Society
- European Sleep Research Society
- American College of Sport Medicine

Licenses and others
- Medical Doctor (Japanese Government)
- Certified Physician of Sleep Medicine (Japanese Society of Sleep Research)
- Sports Doctor (Japan Sports Association)

Language:
- Fluent in Japanese and English
- Can read German

Research Interests:
- Sleep and Biological Rhythm,
- Sport Neuroscience,
- Sport Psychiatry
KAMALESH K. GULIA, Ph.D.
Female, Citizen of India, Married
Date of Birth: August 5th, 1964

ADDRESS (Office)
Scientist-D/ Neurophysiologist
Comprehensive Centre for Sleep Disorders
Biomedical Technology Wing
Sree Chitra Tirunal Institute for Medical Sciences & Technology
Thiruvananthapuram 695012, Kerala
India

EDUCATION


M.Sc. : 1986-1988, Zoology (Reproductive Physiology), Department of Zoology, University of Delhi, Delhi 110007.


PROFESSIONAL EXPERIENCE

JRF, SRF (CSIR) 1992-96 Neuroendocrinology Dept. of Zoology, Delhi Univ.
Research Associate 1998-04 Neurophysiology of sleep and sex behavior Dept. of Physiology, AIIMS, Delhi
(ICMR, CSIR)

Pool Officer (CSIR) 2004-06 Neurophysiology of sleep and sex behavior Dept. of Physiology, AIIMS, Delhi
(Scientist’s Pool)

International Res. Fellow (IBRO) 2006-07 Modern techniques in Neurophysiology Fukushima Medical School, Dept. Neurophysiology, Japan
Scientist

Visiting Scientist 2008 To demonstrate Telemetric technique Dept. Psychobiology, Univ Fed
(Brazil) (IBRO) Sao Paulo, Brazil

Scientist (WOS-A) 2009 Neurophysiology of Arousal system National Brain Research Centre
(DST) Manesar

Scientist-D / 2009- Neurophysiology of Sleep & Arousal system Sree Chitra Tirunal Inst for Medical
Neurophysiologist Sciences & Technology, Kerala
NAME
Jun Lu, MD, PhD.

POSITION TITLE
Associate Professor

eRA COMMONS USER NAME
junlubidmc

EDUCATION/TRAINING

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
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<tbody>
<tr>
<td>Forth Military Medical University</td>
<td>M.D</td>
<td>1979-1984</td>
<td>Medicine</td>
</tr>
<tr>
<td>Institute of Space Medico-Engineering</td>
<td>M.S</td>
<td>1985-1988</td>
<td>Endocrinology</td>
</tr>
<tr>
<td>Texas A &amp; M University</td>
<td>Ph.D</td>
<td>1990-1994</td>
<td>Zoology</td>
</tr>
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</table>

A. Personal Statement

My research has been focused on delineating neural circuitry regulating sleep-wake and motor behavior for 15 years. One of the main research interests is the neural circuitry regulating REM sleep and atonia. Loss of atonia is the hallmark of REM sleep behavior disorder (RBD) that precedes most common neural degenerative diseases such as Parkinson’s disease, Alzheimer’s disease, dementia with Lewy bodies and multiple system atrophy, by a decade. So understanding the neural control of atonia will likely help to understand not only neural mechanism of REM sleep but also neuropathology and causes and prevention of neural degenerative diseases. We have proposed that the REM-active sublaterodorsal tegmental nucleus (SLD) projects to and activates the inhibitory interneurons in the ventral horn, resulting in atonia, which is in sharp difference with the view is that the ventromedial medullar is the site of inhibitory premotor neurons. In the current proposal, to determine which premotor groups in the spinal cord and VMM are responsible for atonia, we will plan to use a novel technique DREADD (the designer receptors exclusively activated by designer drugs, for detail, see main texts of grant proposal). This technique allows us to activate and inhibit the selective neurons simultaneously in freely moving animals. By doing so, we will be able to determine the neural pathways, neuronal groups and neurotransmitters that are involved in regulation of atonia.

Our data support that the SLD glutamatergic neurons activate spinal cord inhibitory interneurons that then suppress the phasic activity of motor neurons.

B. Positions and Honors

Positions and Employment
1999-2003 Instructor, Dept. of Neurology, Beth Israel Deaconess Medical Center and Harvard Medical School.
2003-2010 Assistant professor, Department of Neurology, Beth Israel Deaconess Medical Center and Harvard Medical School.
2010- Associate professor, Department of Neurology, Beth Israel Deaconess Medical Center and Harvard Medical School.

C. Selected Peer-Reviewed Publications (15 max)

D. Research Support

Ongoing Research Support

1R01 NS062727 (Jun Lu, PI) 2/01/2009-1/31/2014
NINDS

Pontine control of motor behavior

The goal of this project is to delineate the pontine neural circuits regulating motor behaviors and their links to the basal ganglia and motor disorders such as cataplexy.
Completed Research Support

R01 NS051609 (Jun Lu, PI) 4/01/2005-3/31/2011

NINDS
Dopamine control of arousal,
   The goal of this project is to understand how dopamine regulates wakefulness. We will identify the wake-active dopamine cells in the ventral PAG and identify the afferents and efferents of these dopamine cells and examine the role of these wake-active dopamine cells in the regulation of wakefulness.

1R01 NS061841-01A2 (Jun Lu, PI) 9/15/2009-9/14/2010
NINDS
Pontine control of REM sleep and atonia
   The goal is to delineate the neural circuitry underlying REM sleep and muscle atonia.

Dr. Jun Lu is currently an associate professor of Neurology of Beth Israel Deaconess Medical Center and Harvard Medical School. He received his medical degree from Fourth Military Medical University, Xian, China and PhD from Texas A & M University, USA. His research of past 20 years has been on neural circuitry of circadian, sleep-wake and motor regulation. Recently, he and his colleagues have been working on neural control of basal ganglia control of sleep-wake and motor behavior. In this talk, he will outline and discuss their findings from knockout, lesion, tracing experiments in combination of sleep-wake and motor behavioral analyses, and underlying neural circuitry of the basal ganglia and dopamine control of sleep and wakefulness.
Michael Lazarus received his doctoral degree (Dr. rer. nat.) at the University of Würzburg in Germany. In 1999, he joined Prof. Osamu Hayaishi’s group at the Osaka Bioscience Institute with prestigious fellowships of the Takeda Science Foundation of Japan and the Alexander-von-Humboldt Foundation of Germany. He then moved to Harvard Medical School where, from 2002-2007, he was an Instructor in Prof. Clifford Saper’s Systems Neurobiology group to examine the differential role of prostaglandin E2 receptors in regulation of fever by establishing transgenic mouse models with conditional expression of each receptor.

In 2007, he returned to the Osaka Bioscience Institute, where he is now a Senior Scientist in the Department of Molecular Behavioral Biology. Dr. Lazarus is interested how prostaglandin and adenosine receptors in the brain regulate responses such as sleep and thermoregulation to circadian, homeostatic, and allostatic inputs. He uses molecular approaches such as conditional knockout or knockin mice and engineering adeno-associated viral vectors for focal deletion experiments and neuronal tract tracing of central pathways.

Selected publications:
Dr. Zhi-Li Huang graduated from Wannan Medical College in 1985, and was awarded a Sasakawa Fellowship to receive his PhD from Ehime University of Japan in 1999. Thereafter, he worked as a postdoctoral fellow for Japan Society for Promotion of Science, a senior research scientist and vice-head of Dept. of Molecular Behavioral Biology in the Osaka Bioscience Institute, Japan. In 2006, he became a professor, Department of Pharmacology and State Key Laboratory of Medical Neurobiology, Shanghai Medical College of Fudan University, China.

His research interest is the hypothalamic regulation of sleep-wake cycle with focus on the histaminergic system. He published more than 50 original and review papers in *Nature Neuroscience, PNAS, J Neuroscience, Progress in Neurobiology*, and so on. He serves as a reviewer for more than 10 international journals such as *PNAS, J Neuroscience, J Neurochemistry* and *Neuropharmacology*. 
Jiang-Fan Chen

Professor, Department of Neurology & Pharmacology, Boston University School of Medicine

My lab, the Molecular Neuropharmacology Lab at Department of Neurology, Boston University School of Medicine, has been studied the role of adenosine receptors in the development and treatment of neuropsychological disorders such as Parkinson’s disease, stroke and Huntington’s disease for the last 15 years and published >110 papers on adenosine receptor neurobiology. By coupling genetic knockout models with pharmacological approaches, my work aimed to elucidate the action and mechanisms of the adenosine $A_{2A}$ receptor under physiological and pathological conditions. The work from my lab advances the prospective of $A_{2A}$ antagonists as a novel treatment strategy for Parkinson’s disease, and contributes to the understanding of adenosine neurobiology in basal ganglia and neuropsychiatric disorders. Recently, in collaboration with Drs. Urade and Huang at Osaka Bioscience Institute, my lab has made an effort to generate brain region- and cell-type specific adenosine $A_{2A}$ receptor knockout models and uncovered important functions of the striatal $A_{2A}$ receptor in control of arousal and its intrinsic relationship with its control of motor and cognitive function.
WING Yun Kwok

Professor (Clinical), Department of psychiatry, The Chinese University of Hong Kong

OTHER POSITIONS:
- Assistant Dean (Student affairs) Faculty of Medicine
- Honorary Chief of Service of Department of Psychiatry (Shatin Hospital & Prince of Wales Hospital)
- Director of Sleep Assessment Unit
- President of the Hong Kong Society of Sleep Medicine
- Deputy Director of Centre for Clinical Trials on Chinese Medicine

RESEARCH INTERESTS:
- Sleep medicine
- Biological and cultural psychiatry
- Neuropsychiatry
- Transcultural psychopharmacology

SELECTED PUBLICATIONS:
CURRICULUM VITAE

Keisuke Suzuki, M.D., Ph.D.

OFFICE
Department of Neurology,
Dokkyo Medical University,
880 Kitakobayashi, Mibu,
Tochigi 321-0293, Japan

EDUCATION

1995-2001  Dokkyo University School of Medicine
Received Master in Medical Sciences degree in May, 2001.
2003-2007  Postgraduate Medical Course (Neurology) in Dokkyo University
School of Medicine
Received Doctor of Philosophy in March, 2007.

PROFESSIONAL AFFILIATIONS

2001-2003  Resident, Dokkyo Medical University Hospital
2007-2008  Assistant, Department of Internal Medicine, Dokkyo Medical
University Koshigaya Hospital
2008-2010  Research fellow, Section of Investigative Medicine, Imperial College
London, London, UK
2010-2012  Assistant, Department of Neurology, Dokkyo Medical University
2012-      Assistant Professor, Department of Neurology, Dokkyo Medical University

BOARD CERTIFICATION

2005  Board certified member of the Japanese Society of Internal Medicine
2007  Board certified neurologist of the Japanese Society of Neurology
2011  Fellow of the Japanese Society of Internal Medicine (FJSIM)
BOARD CERTIFICATION

2005  Board certified member of the Japanese Society of Internal Medicine
2007  Board certified neurologist of the Japanese Society of Neurology
2011  Fellow of the Japanese Society of Internal Medicine (FJSIM)

PUBLICATION

CURRICULUM VITAE
Yuichi Inoue, M.D., Ph.D.
Director of Japan Somnology Center, Japan Somnology Center
Neuropsychiatric Research Institute
Professor, Department of Somnology
Tokyo Medical University

DATE PREPARED: 2/3/2010

NAME: Yuichi Inoue

OFFICE ADDRESS: Japan Somnology Center
Neuropsychiatric Research Institute
1F TS Bldg., 1-24-10 Yoyogi, Shibuya-ku, Tokyo 151-0053
TEL: +81 (0)3 3374 9112
FAX: +81 (0)3 3374 9125

Department of Somnology
Tokyo Medical University
6-7-1 Nishi-Shinjuku, Shinjuku-ku, Tokyo 160-0023

HOME ADDRESS: 1-4-1-102 Minami-Otsuka, Toshima-ku, Tokyo 171-0005
TEL&FAX: +81 (0)3 3945 0959

EMAIL: inoue@somnology.com

PLACE OF BIRTH: Tottori Pref., Japan

DATE OF BIRTH: August 13, 1956

EDUCATION:
1982 BS Tokyo Medical University, Tokyo
1987 MD Tottori University Faculty of Medicine, Tottori Pref.

POSTDOCTORAL TRAINING:
1986 - 1988 Clinical Training National Tottori Hospital
1988 - 1990 Fellowship Tottori University Hospital

SPECIALTY: Somnology, neurovegetative research, Neuropsychopharmacology

CERTIFICATIONS:
Board certified Psychiatrist The Japanese Society of Psychiatry and Neurology
Board certified Sleep Disorder Physician The Japanese Society of Sleep Research

ACADEMIC APPOINTMENTS:
1990 - 1992 Research Associate of Neuropsychiatry Tottori University Faculty of Medicine
1992 - 1999  Assistant Professor of Neuropsychiatry  Tottori University Faculty of Medicine
1999 - 2003  Assistant Professor of Psychiatry  Juntendo University School of Medicine
2003 - 2008  Research Director  Neuropsychiatric Research Institute
2007 -  Professor of Mental Health  Tokyo Medical University
2008 -  Director of Japan Somnology Center  Neuropsychiatric Research Institute
2008 -  Professor of Somnology  Tokyo Medical University
2011 -  President  Yoyogi Sleep Disorder Center

HOSPITAL APPOINTMENTS:
1986 - 1988  Physician  National Tottori Hospital
1988 - 1999  Physician  Tottori University Hospital
1999 - 2003  Physician  Juntendo University Hospital
2003 - 2011  Physician  Yoyogi Sleep Clinic, Neuropsychiatric Research Institute
2007 - 2011  Physician  Tokyo Medical University Hospital
2011 - 2011  Physician  Yoyogi Sleep Disorder Center

OTHER PROFESSIONAL POSITIONS & MAJOR VISITING APPOINTMENTS:
Part-time Lecturer, Department of Psychiatry, Nara Medical University
Clinical Professor, Tokyo Medical & Dental University
Visiting Professor, Aichi Medical University
Visiting Professor, Kitasato University School of Medicine

PROFESSIONAL SOCIETIES INVOLVEMENTS:
The Japanese Society of Sleep Research  Board of Directors
The Japanese Society of Sleep Research  Board of Councilors
The Japanese Society of Sleep Research  Chairman of Educational Committee
The Japanese Society of Sleep Research  Chairman of Dentists Certification Committee
The Japanese Society of Sleep Research  Chairman of Medical Care Expenditure Committee
Japanese Society of Biological Psychiatry  Board of Councilors
Japan Society of Neurovegetative Research  Board of Councilors
Japanese Society for Chronobiology  Board of Councilors
Japan Society of Anxiety Disorder  Board of Councilors
World Association of Sleep Medicine  Assistant Secretary (2009-2011)
World Federation of Sleep Research Society  Programme Committee Co-chair
Japanese Journal of Sleep Medicine  Chief Editor
Sleep and Biological Rhythm  Associate editor (2012-)

AWARDS & HONORS:
1993  Tottori University Neuropsychiatry Award
1996  Tottori University Shimoda Award
2009  WASM Christian Guilleminault Award

PUBLICATIONS:

Chapters:


2. Inoue Y, Kishimoto A. “Research about mood stabilizers in Japan.” In: Recent advances in the research
Name Col Prasit Mahakit MD.

Date of birth 20 March 1960

Place of birth Trang

Marital Status Marriage Number of children 1

Nationality Thai

Home Address 321/9 Soi Amorn Nanglinchee road Yannawa Bangkok

Present Professional Position Deputy Director of department of Otolaryngology Phramongkutklao Hospital

Undergraduate Education
Name of Medical School Prince of Songkhla University
Degree M.D. Date of Obtained 1983

Post graduate Education
Institution Phramongkutklao Hospital Duration 3 years

Present and Past Professional Appointment
Degree and Certification BSc. M.D. Board of Otolaryngology
MSc. (Epidemiology) THAICERT, Chulalongkorn University
Board of Family Medicine Research fellow in Otolaryngology (Vanderbilt U., U.S.A.)
Visiting Doctor Kagoshima University, Kagoshima Japan
Visiting Doctor Kurume University, Kurume Japan
Visiting Doctor Johns Hopkins University MD. USA
Visiting Doctor Kurume University, Kurume
Japan
Visiting Doctor Johns Hopkins University MD.
USA
Member of Professional Association
Fellow of Royal College of Otolaryngology
Thailand
Fellow of Thai Facial Plastic Society
Fellow of Family Medicine Society

International Scientific Presentation
1993 The preliminary study of mucociliary Transport in sinusitis, allergic rhinitis and smoker
The Twelfth international symposium on infectious and allergy of the nose
November 1993, Seoul, KOREA
1995 The study of indoor inhalant allergens by prick test
The second Asian Pacific Congress of Allergy and Clinical Immunology
October 1995, Taipei, TAIWAN

Bibliography
Text Book
1997 Chapter 5 Immunotherapy
Pumhirun, P., Mahakit, P.
Rhinology (Asian Ed.) Bunnag, C. Editor
2004 Editor of textbook of Otolaryngology Head and Neck Surgery (Thai version)

International Publication
1994 The preliminary study of nasal mucociliary clearance in smoker, sinusitis and rhinitis Asian Pacific Journal of Allergy and Immunology
13; 119-121, 1995
2006 Oral Clindamycin 300 mg BID Compared with Oral Amoxicillin/Clavulanic Acid
Jingying Ye, female, professor of otolaryngology, director of sleep medicine center of Affiliated Beijing Tongren Hospital, Capital University of Medical Science, vice-president of Sleep Professional Association of the Chinese Medical Doctor Association, vice-president of Chinese Sleep Research Association and consultant specialist of the Central Health Care Committee. Professor Jingying Ye specializes in pharynx and larynx disease, and has some outstanding achievements in the research of obstructive sleep apnea hypopnea syndrome, especially in the sleep surgery. She has been in charge of several National Nature Science Foundation Project, and has ever won the second class of National Science & Technology Progress Award and the first class of National Education Ministry Award for Science & Technology Progress. Up to now, she has published over 100 articles and taken part in the writing of “Surgery for Sleep Breathing Disorder” and other 4 books as the assistant chief editor.
Curriculum Vitae

Sung Wan Kim

Work

Professor and Chairman, Head Professor
Department of ORL–HNS, School of Medicine, Kyung Hee University, Seoul, Korea
Director of Sleep Breathing Disorder Center, Kyung Hee University Hospital

Current Status

2009– International Surgical Sleep Society board member
2009– Sleep Technology Special Interest Group, Advisory board member
2008– Sleep Respiration Forum, Board Member
2007– Director of Planning committee, Korean Society of Sleep Medicine
2010– 20th IFOS World Congress 2013, Scientific Committee Member
2011– Director of Legislation Committee, Korean Academy of Asthma, Allergy
and Clinical Immunology

Past Status

2001–2011 Director of Various Committee, Korean Rhinologic Society
2009–2011 General Secretary, Korean Rhinologic Society
2006 General Secretary, Asian Research Symposium in Rhinology
2007–2009 Treasurer, 9th World Congress on Sleep Apnea,
2011 Vice–president, International Sleep Surgical Society Congress, Taiwan,
CURRICULUM VITAE

Name  SHINTARO CHIBA
Date of Birth  July 22 1961
Place of Birth  Iwate, Japan

PRESENT POSITION

Associate Professor
Department of Otorhinolaryngology
Jikei University School of Medicine
3-25-8 Nishishinbashi, Minato-ku, Tokyo
105-8471 Japan

EDUCATION

2007  Ph.D., Otorhinolaryngology, Jikei University school of Medicine
2002  Sleep Physician's License in Japan (No. 140)
1994  Certificated Otorhinolaryngologist in Japan (No. 9382)
1989  Physician's License in Japan (No. 365573)
1989  M.D., Jikei University school of Medicine

PROFESSIONAL EXPERIENCE

2010-2012  Visiting Associate Professor
Sleep & Circadian Neurobiology Laboratory
Center for Narcolepsy
Stanford Sleep Research Center
Stanford University School of Medicine

Japan:
2007-present  Associate Professor
Department of Otorhinolaryngology,
Jikei University School of Medicine, Tokyo, Japan
2001-2004  Director
Sleep Disorder Center,
Ota General Hospital, Kanagawa, Japan
1999-Present  Director
Department of Otorhinolaryngology,
Ota General Hospital, Kanagawa, Japan
1991-1998  Clinical Fellow
Department of Otorhinolaryngology,
Jikei University School of Medicine, Tokyo, Japan
1989-1990  Resident
Department of Otorhinolaryngology,
Jikei University School of Medicine, Tokyo, Japan
Jikei University School of Medicine, Tokyo, Japan

PROFESSIONAL SOCIETIES

The Oto-Rhino-Laryngological Society of Japan
Japan Rhinologic Society
Japan Society of Stomato-Pharyngology
Japanese Telemedicine and Telecare Association
The Japanese Society of Sleep Research
American Academy of Sleep Medicine
PD Dr. Ursula Voss

Vita

MA in psychology from J.W. Goethe-University Frankfurt, Germany, Ph.D. in experimental psychology from the University of Southern Mississippi, second dissertation (Habilitation) from the J.W. Goethe-University Frankfurt, Germany.

Lecturer and professor at J.W. Goethe-University Frankfurt, Northern Institute of Technology/Technical University Hamburg Harburg, Erfurt University, and Friedrich-Wilhelms-University Bonn (current position).

Research interests

- attention
- states of consciousness
- endocrine changes in sleep
- information processing and vigilance
- psychological sleep research
- stress and coping
- sleep in neurodegenerative disease

Publications


Curriculum Vitae

Dinesh Pal, Ph.D.
University of Michigan, Department of Anesthesiology, 1150 W Medical Center Drive, 7433 Med Sci Bldg 1, Ann Arbor, MI 48109, USA. Ph: 734-764-3326 FAX: 734-764-9332 Email: dineshp@umich.edu

Education and Training
1997 B.Sc. (Hons) Applied Zoology, Dyal Singh College, University of Delhi, Delhi, India.
1999 M.Sc. Zoology, Hansraj College, University of Delhi, Delhi, India.
2006 Ph.D. (Neurobiology), Jawaharlal Nehru University, Delhi, India.
2006-07 Postdoctoral scholar, School of Optometry, University of California, Berkeley, CA, USA.
2007-12 Research fellow, Department of Anesthesiology, University of Michigan, Ann Arbor, MI, USA.

Academic Appointments
2012 Research Investigator, Department of Anesthesiology, University of Michigan, Ann Arbor, MI, USA

Honors and Awards
2000 Junior research fellowship, Council of Scientific and Industrial Research, Delhi, India.
2003 Senior research fellowship, Council of Scientific and Industrial Research, Delhi, India.
2004 Training and travel award to attend 5th International Brain Research Organization (Asia-Pacific) Neuroscience School, Bangkok, Thailand.
2007 B. K. Anand research prize for the best paper in physiology published in a peer-reviewed journal, Association of Physiologists and Pharmacologists of India.
2008 Young Scientist medal, Indian National Science Academy, Delhi, India.
2008 Professor L.S.S. Kumar memorial award in Animal Sciences, Indian National Science Academy, Delhi, India.
2010 Travel award for highest-scoring scientific paper, Society for Neuroscience in Anesthesiology and Critical Care, San Diego, CA, USA.
2011 Travel award for highest-scoring scientific paper, Society for Neuroscience in Anesthesiology and Critical Care, Chicago, IL, USA.

Memberships in Professional Societies: 1) Sleep Research Society, 2) Indian Society for Sleep Research, 3) Society for Neuroscience, 4) Society for Neuroscience in Anesthesiology and Critical Care, 5) Society of Anesthesia and Sleep Medicine, 6) Association of Physiologists and Pharmacologists of India

Peer-Review Service: 1) Brain Research, 2) SLEEP

Recent Publications:
BIOGRAPHICAL SKETCH

NAME

Fang Han

POSITION TITLE

Professor of Medicine

EDUCATION/TRAINING

<table>
<thead>
<tr>
<th>INSTITUTION AND LOCATION</th>
<th>DEGREE (if applicable)</th>
<th>YEAR(s)</th>
<th>FIELD OF STUDY</th>
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<tr>
<td>Binzhou Medical University, Shan Dong Province</td>
<td>M.B.</td>
<td>1992</td>
<td>Medicine</td>
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<tr>
<td>Beijing University People’s Hospital</td>
<td>M.D.</td>
<td>1997</td>
<td>Pulmonary Medicine, Sleep Medicine</td>
</tr>
</tbody>
</table>

A. Positions and Honors.

Positions and Employment

2009-Professor, Department of Pulmonary Medicine, Beijing University People’s Hospital, Beijing, China
2001-2009 Associate Professor, Department of Pulmonary Medicine, Beijing University People’s Hospital, Beijing, China 1999-2001
1999-2001-Assistant Professor of Medicine, Case Western Reserve University, Cleveland, Ohio
1997-1999 Physician in charge, Department of Pulmonary Medicine, Beijing University People’s Hospital, Beijing, China

Editorial Board and Professional Memberships

Editorial Board

2001- Sleep and Breathing
2004- J of Foreign Medical Science (Section of Respiratory Diseases)
2008- Chinese Journal of Tuberculosis and Respiratory Medicine

Professional Positions and Memberships

2011- President, Chinese Sleep Research Society
2005 Member, American Academy of Sleep Medicine.
2005 Member, World Association of Sleep Medicine.
1998- Member, Chinese Medical Association
1996- Member, Chinese Sleep Research Society
2007, Member, International Academic Committee, World Association of Sleep Medicine Meeting (WASM, Thailand)
2007, Member, International Academic Committee, World Federation of Sleep Research and Sleep Medicine Society Conference (Australia)

PHS 398/2590 (Rev. 09/04) Page 2 Biographical Sketch Format Page
2009, Member, International Scientific Committee, 9th World Congress on Sleep Apnea (Korea)
2009, Member, International Scientific Committee, 6th Congress of Asian Sleep Research Society (Japan)
2013, Member, International Scientific Committee, the 5th World Congress on Sleep Medicine, World Association of Sleep Medicine (WASM) (Spain)

**Honors**

1998 Chinese Thoracic Society-Respironics Scholarship
2004 Young Investigator Award (First Prize), the Fourth Congress of Asian Sleep Research Society
2004 The best sleep research paper by Chinese Sleep Research Society
2005 Mini-fellowship for International Scholars (2005) by American Academy of Sleep Medicine
2005 Young Investigator Award in Clinical Research (First Prize), the first Congress of World Association of Sleep Medicine
2005 Best Paper Award, the Tenth Congress, Asian Pacific Society of Respirology
2006 Young Investigator Award, the Fifth Congress of Asian Sleep Research Society

**B. Selected peer-reviewed publications (in chronological order).**

(Publications selected from 100 peer-reviewed publications)

7. Han F. Narcolepsy, orexins and respiratory regulation. Sleep and Biological Rhythms. 2011; 9:44-51
Name:  Zhang Xilong  
Institute: The First Affiliated Hospital of Nanjing Medical University  

CURRICULUM VITAE  

Name:  Zhang Xilong       Sex: Male  
Date of Birth:  December 21, 1952  
Institute:  Department of Respiratory Medicine, The First Affiliated Hospital of Nanjing Medical University, Guangzhou Road 300, Nanjing 210029, China  

Education:  
1978-1982   Nanjing Medical College, Nanjing, China                   Student  
1983-1986   Xinqiao Hospital, The Third Medical College. Chongqing, China Postgraduate for MD  
1996-1998   Heart Institute of Japan, Tokyo Women’s Medical University, Tokyo, Japan Postgraduate for PhD  
2004-2004  Sleep Disorder Center, Division of Pulmonary/Critical Care & Sleep Medicine, Harper University Hospital, Wayne State University School of Medicine, Detroit, USA AASM mini-fellowship owner  

Major Research Experience:  
1998.10-2000.9  Dealing with research on “pathogenesis and treatment of obstructive sleep apnea syndrome” in Jinling Hospital, Nanjing, China  
2000.10--present  Dealing with research on pathogenesis of sleep apnea syndrome and its interventional treatment” in Department of Respiratory Medicine, The First Affiliated Hospital of Nanjing Medical University, China  

Research Award  
2003.9       Silver sponsorship of European Respiratory Society on research paper “Effect and significance of continuous positive airway pressure treatment on vascular endothelial function change in patients with obstructive sleep apnea syndrome.”  
2004.5       Mini-fellowship of American Association of Sleep Medicine  
2005.9       Silver sponsorship of European Respiratory Society on research paper “Effect of continuous positive airway pressure treatment on hypoadiponectinemia in patients with obstructive sleep apnea syndrome.”  
2005.10      The Third Class Award of Science and Technological Progress of Jiangsu Province about “Pathogenesis, diagnosisi and treatment of obstructive sleep apnea syndrome”  
2006.9       The Best Poster Award of The 8th World Congress of Sleep Apnea
continuous positive airway pressure treatment on hypo adiponectinemia in patients with obstructive sleep apnea syndrome.”

2005.10 The Third Class Award of Science and Technological Progress of Jiangsu Province about “Pathogenesis, diagnosisi and treatment of obstructive sleep apnea syndrome”

2006.9 The Best Poster Award of The 8th World Congress of Sleep Apnea

2012.2 The Third Class Award of Huaxia Medicine

Main Recent Publications

Books (Editor-in-chief)

《Sleep and sleep disorders》, 1st ed., Publication Society of Beijing Military Medicine, Beijing, 2005


Major Original Articles (1st & corresponding author)


Yuanming Luo is a professor of State Key Laboratory of Respiratory Disease of China. He is also vice director of sleep research group of Chinese Respiratory Society and is in charge of sleep disordered breathing center of Guangzhou Institute of Respiratory Disease. He studied at King’s College/University College of London between 1996 and 2001 under the guidance of Professor John Moxham and gained his PhD degree in 2001. His major research interests are investigating neural respiratory drive in both healthy subjects and patients, particularly patients with sleep disordered breathing and COPD.

Publication:
CURRICULUM VITAE

NAME: Motoo Yamauchi

PERSONAL DETAILS
Date and place of Birth: 18 October 1969, Nara, Japan
Nationality: Japan

WORK EXPERIENCE
July 2008 – present:
Assistant professor
Department of Respiratory Medicine, Nara Medical University, Japan

January 2008 – June 2008:
Medical staff
Department of Respiratory Medicine, Nara Medical University, Japan

October 2005 - December 2007:
Research fellow;
Division of Pulmonary, Critical Care and Sleep Medicine, Department of Medicine, Case Western Reserve University, Louis Stokes VA Medical Center, 10701 East Boulevard, K215, Cleveland, OH, 44106, USA

EDUCATION
2010 Ph.D., Nara Medical University, Nara, Japan
1995 M.D., Nara Medical University, School of Medicine, Nara, Japan
1988 Graduated from Unebi High School, Nara, Japan
Prof. Mary Ip is Chair Professor and Mok Hing-Yiu Endowed Professor of Respiratory Medicine at The University of Hong Kong, and Chief of Division of Respiratory Medicine in the Department of Medicine, at Queen Mary Hospital. She is also the Associate Dean (Education) at the Li Ka Shing Faculty of Medicine, University of Hong Kong.

In the past fifteen years, she has developed a strong clinical and research base in sleep related breathing disorders. Her initial population-based study on sleep disordered breathing in Hong Kong has paved the way for the awareness of this common but then unrecognized condition among the local population, and the subsequent flourishing of clinical service for sleep apnea. Her work now spans clinical, translational and basic research with focus on cardiovascular and metabolic aspects of obstructive sleep apnea.

Professor Ip has published extensively in international peer-reviewed journals on sleep related breathing disorders and other respiratory diseases. She is a regular expert reviewer for papers of international journals in respiratory/sleep medicine, and invited panel reviewer for local or overseas research grants and awards in the specialty. Currently, she is an Associate Editor of Chest and an Editorial Board member of Sleep Medicine.

Prof. Ip is an active member of the local, regional and international respiratory community. She is the current President of APSR, the Asia Pacific Society of Respirology. Previously, she has served as President to the Hong Kong Thoracic Society, Hong Kong Society of Sleep Medicine, and as International regent of the American College of Chest Physicians.
CURRICULUM VITAE
Denise Sharon, MD, PhD

Current Position:  Director, Comprehensive Sleep Medicine and Research Center of the Gulf Coast
Clinical Director, Advanced Sleep Center

Academic Affiliation:  Associate Professor of Medicine, Tulane University Medical School
1999 – to date  Clinical Assistant Professor of Pediatrics, Sleep Medicine Fellowship Faculty
Tulane School of Medicine, New Orleans, LA

Education:
1970-1973  Faculty of Medicine and Surgery, University of Siena, Italy. Preclinical studies.
1974-1979  Sackler School of Medicine, Tel-Aviv University, Israel. Thesis presentation and graduation 1981, M.D.

Professional Training:
1993 - 1995  Sleep Disorders Training, ASDA approved
Sleep Laboratory, Department of Psychiatry and Health Behavior MCG/VAMC, Augusta, GA
1989 - 1990  Internship in Psychology (APA approved) ICTR, The Devereux Foundation - Devon, PA
1980 - 1985  Residency in Psychiatry (PGY II-VI), Pardesia Psych. Hospital, Pardesia, Israel.
1979 - 1980  Residency in Psychiatry (PGY-I), Geha Psych. Hospital, Petah-Tikva, Israel.
1978 - 1979  Rotating Internship, Sheba Medical Center, Tel-Hashomer, Israel.

The last three hospitals are affiliated with the Sackler School of Medicine, Tel-Aviv Univ., Israel.

Medical License:  Israel, January 1981 (R/I/#014923)
Psychological License:  Texas License No. 25449, Alabama License No. 810 Georgia License No. 1695, Louisiana License No. 813

Credentials:
1992  American Assoc. of State and Provincial Psychology Boards Certification - Psychology
1997  American Board of Sleep Medicine Board Certified - Sleep Disorders
2007  Fellow of the American Academy of Sleep Medicine

Prior Sleep Medicine Related Experience:
2003-2006  Sleep Specialist, Premier Sleep Medicine Center, Baton Rouge, LA
2002-2005  Sleep Medicine Fellowship Program Co-Director Tulane School of Medicine, New Orleans, LA
1999-2005  Sleep Specialist, Tulane Hospital and Clinics, New Orleans, LA
1997-1998  Sleep Specialist, Sleep Medicine Associates of Texas, Dallas TX
1996  Independent Contractor, Sleep Consultants, Inc., Fort Worth, TX
1995-1996  Sleep MedicineTrainee, Sleep Medicine Associates of Texas, Dallas TX

Academic Appointments:
1994 - 1995  Clinical Instructor, Department of Psychiatry and Health Behavior, MCG Augusta, GA
1983 - 1985  Teaching Assistant in Psychiatry, Pardesia Psychiatric Hospital
Sackler School of Medicine - Pardesia, Israel

Research Appointments:
1996 - 1997  Research Work, Sleep Medicine Associates of Texas - Dallas, Texas
1991 - 1995  Research Associate/Quantitative Electroencephalographer, Laboratory of Psychoelectrophysiology,
Depart. of Psychiatry and Health Behavior, MCG
1987 - 1990  Research Associate, Laboratory of Psychiatric Electrophysiology
Department of Psychiatry - Temple Univ. Hospital, Philadelphia, Pennsylvania.
1986 - 1987  Research Team Dept. of Clin. Psychology Northwestern Univ. - Evanston, IL
1985 - 1985  Research Fellow at the Psychobiology Institute, Tel-Aviv University, Israel.

Organizations:
American Academy of Sleep Medicine  Sleep Research Society
Organizations:
American Academy of Sleep Medicine  Sleep Research Society  
World Association of Sleep Medicine  National Sleep Foundation  
American Insomnia Association  Movement Disorder Society  
International RLS Study Group  RLS Foundation  
Southern Sleep Society  Louisiana Academy of Sleep Medicine  
American Psychological Association  LA Psychological Association - Baton Rouge Chapter

Other Activities - Sleep Medicine Related:
Reviewer for Sleep, Sleep Medicine, J of Clinical Sleep Medicine, Journal of Psychosomatic Research, American Journal of Medicine, Journal of Women’s Health
2010- 2012 Southern Sleep Society - President
2011- to date AASM ICSD task force
2012- to date AASM Coding and Compliance Committee
2008- 2010 AASM Nosology Committee
2007- 2010 SRS Membership Committee; vice-chair  2008-2010
2000 - 2010 AASM Fellowship Accreditation Committee
1999 - 2005 AASM Center Accreditation - Site Visitor
1998- to date International Restless Legs Syndrome Study Group – Executive Committee, 1998-2010 Treasurer

Abstracts and Publications – Sleep Medicine related:


Dahi H, Crowder C, Sharon D, Simakajornboon N. The Correlation between Subjective Sleep Perceptions from Post-Test Questionnaire and Objective Sleep Parameters in Patients with OSA. Sleep 28 (Suppl):A176, 2005.


Ronit Gilad, M.D.

A. Education

<table>
<thead>
<tr>
<th>Period of Study</th>
<th>Name of University</th>
<th>Subject</th>
<th>Degree or Professional License</th>
<th>Awarded</th>
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<tbody>
<tr>
<td>1978 - 1981</td>
<td>University of Rome, Rome Italy</td>
<td>Medicine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1981 - 1984</td>
<td>Sackler Faculty of Medicine, Tel Aviv University</td>
<td>Medicine</td>
<td>M.D.</td>
<td>1984</td>
</tr>
</tbody>
</table>

Title of Doctoral Dissertation: Low Dose Treatment with Amiodarone: Antiarrhythmic Efficacy and Adverse Side Effects

Name of Supervisors: Prof. Shlesinger and Dr. Feigl

Date of receiving Specialization Certificate: 1991 Neurology (No. 19213)

B. Further Studies

<table>
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<tr>
<th>Period of Study</th>
<th>Name of University</th>
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## B. Further Studies

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<th>Period of Study</th>
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<th>Subject</th>
<th>Degree or Professional License</th>
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<tbody>
<tr>
<td>1987 - 1991</td>
<td>E. Wolfson Medical Center School of Nursing</td>
<td>Clinical Neurology</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1989</td>
<td>Allyn Hospital, Jerusalem</td>
<td>Clinical Neurophysiology</td>
<td></td>
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</tr>
<tr>
<td>1992</td>
<td>Department of Physical Therapy, Sackler Faculty of Medicine, Tel Aviv University</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>1992</td>
<td>Sackler Faculty of Medicine, Tel Aviv University</td>
<td>Post Graduate Studies in Neurology</td>
<td></td>
<td></td>
</tr>
<tr>
<td>July 1995</td>
<td>Neurologische Klinik und Poliklinik der Technischem, Universitat Muchen, Germany</td>
<td></td>
<td>E.M.G.</td>
<td></td>
</tr>
<tr>
<td>August 1998</td>
<td>Columbia Presbyterian Medical Center New York, N.Y., U.S.A.</td>
<td></td>
<td>E.E.G. Electrophysiology</td>
<td></td>
</tr>
<tr>
<td>June 2003</td>
<td>University of Liverpool Liverpool, United Kingdom</td>
<td></td>
<td>E.E.G. Electrophysiology</td>
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## C. Academic and Clinical Experience

<table>
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<tr>
<th>Period</th>
<th>Name of Institution</th>
<th>Department</th>
<th>Rank/Function</th>
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<tbody>
<tr>
<td>1984 - 1985</td>
<td>E. Wolfson Hospital, Holon</td>
<td>Rotating Internship</td>
<td></td>
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<tr>
<td>1987</td>
<td>E. Wolfson Hospital, Holon</td>
<td>Neurology</td>
<td>Residency</td>
</tr>
<tr>
<td>1990</td>
<td>Arbarbanel Hospital, Bat Yam</td>
<td>Psychiatry</td>
<td>Residency</td>
</tr>
<tr>
<td>1989</td>
<td>E. Wolfson Hospital, Holon</td>
<td>Internal Medicine</td>
<td>Residency</td>
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<tr>
<td>Year</td>
<td>Position</td>
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</tr>
<tr>
<td>1990-1992</td>
<td>Neurology Instructor</td>
<td>E. Wolfson Hospital, Holon, Sackler Faculty of</td>
<td>Neurology Residency Program</td>
</tr>
<tr>
<td></td>
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<td>Medicine, Tel Aviv University</td>
<td></td>
</tr>
<tr>
<td>1991-2007</td>
<td>Neurology Senior Residency</td>
<td>E. Wolfson Hospital, Holon (Electrodiagnosis and</td>
<td>Neurology Senior Residency</td>
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<td>Electrophysiology)</td>
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<tr>
<td>1992-present</td>
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<td>E. Wolfson Hospital, Holon</td>
<td>Neurology</td>
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<td>(Electrodiagnosis and Electrophysiology)</td>
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<tr>
<td>1996-present</td>
<td></td>
<td>Board examiner for psychiatry and neurology specialization certification</td>
<td></td>
</tr>
<tr>
<td>1999-present</td>
<td></td>
<td>Member of the medical examination development committee</td>
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<tr>
<td>Sept 2002-present</td>
<td></td>
<td>Sackler Faculty of Medicine, Tel Aviv University</td>
<td>Neurology</td>
</tr>
<tr>
<td>2002-present</td>
<td></td>
<td>Sackler Faculty of Medicine, Tel Aviv University</td>
<td>Post graduate courses continuing education for neurology</td>
</tr>
<tr>
<td>2005-2006</td>
<td></td>
<td>Sackler Faculty of Medicine, Tel Aviv University</td>
<td>Head, Committee for post graduate teaching</td>
</tr>
</tbody>
</table>
2007-present E. Wolfson Hospital, Holon Neurology Deputy Head
2007-present Sackler Faculty of Medicine, Tel Aviv University Member, Teaching Committee for Medical Students
2007-present Sackler Faculty of Medicine, Tel Aviv University Member, Examination Committee for Medical Students

D. Active Participation in Scientific Meetings

<table>
<thead>
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<th>Year</th>
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<tr>
<td>1990</td>
<td>2nd International Meeting of the European Neurological Society,</td>
</tr>
<tr>
<td>1991</td>
<td>International Conference on Stroke, Geneva, Switzerland</td>
</tr>
<tr>
<td>1993</td>
<td>XIIIth International Congress of Electroencephalography and Clinical Neurophysiology, Vancouver, Canada</td>
</tr>
<tr>
<td>1994</td>
<td>4th International Meeting of the European Neurological Society, Barcelona, Spain</td>
</tr>
<tr>
<td>1995</td>
<td>4th International Conference on SLE, Jerusalem, Israel</td>
</tr>
<tr>
<td>1996</td>
<td>8th European Congress of Clinical Neurophysiology, Munich, Germany</td>
</tr>
<tr>
<td>1999</td>
<td>XIth International Electromyography and Clinical Neurophysiology, Prague, Czech Republic</td>
</tr>
<tr>
<td>2000</td>
<td>ECTRIM 2000 – 16th Congress of the European Committee for the Treatment and Research in Multiple Sclerosis, Toulouse, France</td>
</tr>
<tr>
<td>2006</td>
<td>American Academy of Neurology (AAN), 58th Annual Meeting, San Diego, CA, USA</td>
</tr>
<tr>
<td>2007</td>
<td>ECTRIM 2007 – 23rd Congress of the European Committee for the Treatment and Research in Multiple Sclerosis, Prague, Czech Republic</td>
</tr>
</tbody>
</table>
E. Academic and Professional Awards

<table>
<thead>
<tr>
<th>Year</th>
<th>Name of Institution</th>
<th>Award</th>
</tr>
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<tbody>
<tr>
<td>1995</td>
<td>Department of Neurology and Clinical Neurophysiology, Technische Universitat Muchen (Under: Dr. Bishof)</td>
<td>Fellowship</td>
</tr>
<tr>
<td>1995</td>
<td>Department of Neurology and Clinical Neurophysiology,</td>
<td>Fellowship</td>
</tr>
<tr>
<td>June 2003</td>
<td>E.E.G. Electrophysiology, University of Liverpool, Liverpool, United Kingdom</td>
<td>Fellowship</td>
</tr>
</tbody>
</table>

F. Membership in Professional Societies

<table>
<thead>
<tr>
<th>Year</th>
<th>Society</th>
<th>Country</th>
</tr>
</thead>
<tbody>
<tr>
<td>1987</td>
<td>Israel Medical Association</td>
<td></td>
</tr>
<tr>
<td>1987</td>
<td>Israel Neurological Society</td>
<td></td>
</tr>
<tr>
<td>1988</td>
<td>Israel Association of EEG and Clinical Neurophysiology</td>
<td></td>
</tr>
<tr>
<td>1988</td>
<td>Israel League Against Epilepsy</td>
<td></td>
</tr>
<tr>
<td>2007</td>
<td>Scientific Committee, Neurological Association of Israel</td>
<td></td>
</tr>
</tbody>
</table>

G. Students Supervised by Candidate

Specialization Certificate Candidate

<table>
<thead>
<tr>
<th>Year</th>
<th>Name of Student</th>
<th>Title of Project</th>
<th>Name of Hospital</th>
</tr>
</thead>
<tbody>
<tr>
<td>2008</td>
<td>Natalya Izikovitz, M.D.</td>
<td>Basic Science Study in Neurology</td>
<td>E. Wolfson Medical Center</td>
</tr>
</tbody>
</table>
CURRICULUM VITAE

PERSONAL DATA:
Anca- Herschkovitsch Marieta, MD, license nr.20169
Born: 1959.06.09-, Romania
Address: Israel, Shoham, and POB 2415
Citizenship: Romania, Israel

EDUCATION:
1978-1984 Faculty of General Medicine- University of Cluj, Romania.

ACTIVITY AND EXPERIENCE:
1984-1985 Internal ship, Jewish Hospital, Cluj, Romania.
1986 -1987 internal ship, Kaplan Hospital and Haemek Hospital, Israel
1987-1988 General Medicine Practitioner in Ambulatory and Emergency Service
1988-1990 General Medicine Practitioner in Geriatric Hospital, Harzfeld
1990- 1997 Residency, Department of Neurology, Sheba Medical Center
1993-1995 –Basic Science in Sleep Medicine, Sleep Laboratory, Sheba
1997-1998 Neurological Department, Wolfson Medical Center and Consultant Neurologist in Psychiatric Hospital Abarbanel –Bat Yam
1998 -2000 Fellowship in Movement Disorders, Sourasky Medical Center, Tel Aviv,
1999-Award of General Manager of Ministry of Health
2000-2002-Neurologist specialized in Movement Disorders, Movement Disorders Unit, Tel Aviv Medical Center:
1997-2012 Leumit & Maccabi Health Medical Organization
1999-2012 Medical consultant in Maarag laboratory for sleep and ADHD screening
2002-2004 Kupat Holim Clalit Health Medical Organization
2002- ongoing to present Head of Movement Disorders Clinic, Departement of Neurology, Edith Wolfson Medical Center, Holon 59100-Israel
1996-2012 Coordinator/Instructor for Israeli and American students in General Medicine and Nursing, Tel Aviv University
2002-2012-Membership in Movement Disorders Society, American Academy of Sleep Medicine, ASRA (Asian Sleep Research Society), ISMA (Israeli Sleep Medicine Association), 1998-2012 Membership in Israeli Neurological Association and EFNS 2000-2012- Medical Staff of Israeli Parkinson’s Disease Association, Huntington disease Israeli Association, Tourette syndrome Israel Association 2012- Ongoing medical consultant in Bituah Leumi Staff, Ramat Gan

**Published Articles:**

1-Tetrabenazine in Neuroacantocythosis –case studies –Cambridge Laboratory Limited – 2001  
2-Cross-sectional study of the prevalence of Parkinson’s disease in the Kibbutz movement in Israel - Neuroepidemiology 2002  
3-Olanzapine in Huntington’s disease -Acta Neurologica Scandinavica 2002  
4-Quality of sexual life in Parkinson’s disease –Parkinsonism and related disorders, 2002  
5-Natural history of Oppenheim’s Dystonia in Israel- Journal of Child Neurology 2003  
6-Rivastigimine for dementia in patients with Parkinson’s disease –Acta Neurologica Scandinavica 2003  
7-Globus pallidus internus deep brain stimulation for dystonic conditions- Movement Disorders, 2003  
9-Ropinirole in Gilles de la Tourette syndrome –Neurology, 2004  
11-Familial leucoencephalopathy, European Child Neurology 2009  
12-Botulinum toxin use in neurology - medicine Neurology( Israel) 2008

**Presented in Congresses:**

1-The protective effect of Periodic Leg Movement in Sleep on patients with Sleep Apnea Syndrome(Poster) –Asian Sleep Society, Tokyo, 1994
2- Cross-sectional study of the prevalence of Parkinson’s disease in the Kibbutz movement in Israel (Poster)–MDS Vancouver 1999
3- Trauma and tics (Poster)–International Congress on Parkinson Disease and Movement Disorders – Barcelona 2001
5- Effective Treatment Formula for Tourette Syndrome and different types of Tremor- Janssen, Cilag Congress Hamburg, 2002
6- Gyrokinetics: A preventive rehabilitation program in Parkinson disease, International Congress on Parkinson and Movement Disorders, Rome 2004
7- Atypical PSP, International Congress on Parkinson and Movement Disorders, New Orleans, 2005
8- Posttraumatic ADHD, Danube Symposia and World ADHD Congress, Wurzburg , 2007
9- Home Sleep Study-10 years of experience, WSC, Cairns, Australia, 2007
10- Botulinum toxin in tardive dystonia, MDS, Paris, 2009
11- Home polysomnography in sleep related movement disorders, MDS, Buenos Aires, 2010
12- Posttraumatic peroneal spasm – case report MDS Toronto 2011

Clinical trials:
-Rasagilin in Parkinson’s disease Phase1 and 2 clinical trial 1995-996
-Rasagilin in RLS, open trial, 2007
-Botulinum toxin type A in blepharospasm and torticollis – double blind clinical trial in 23 patients– Omnicare Israel /Merz company Germany 2001-2002.
Curriculum Vitae
Hirotaka Hara

Personal

Birthdate:  18.12.1964
Birthplace: Fukuoka, Japan
Marital Status: Married ; two children

Education and Degrees

1996-1999: Visiting research fellow, Department of Pathology, Tulane University Medical School, Louisiana.

1995: Ph.D. , Department of Otolaryngology, Yamaguchi University Graduate School of Medicine, Japan
1989: M.D. , Graduate Yamaguchi University School of Medicine, Japan.

Academic Positions

2004-present: Associate Professor, Department of Otolaryngology, Yamaguchi University Graduate School of Medicine.
1996-2004: Instructor, Department of Otolaryngology, Yamaguchi University School of Medicine.

Membership

The Oto-Rhino-Laryngological Society of Japan
The Japanese Society of Sleep Research
American Academy of Sleep Medicine
Asian sleep surgical society
Japanese Broncho-Esophagological Society
Japanese Laryngological Association
Japan Society of Stomato-pharyngology
The Japan Society for Head and Neck Surgery
Japan Society for Head and Neck Cancer
The Society of Practical Otolaryngology
The Society of Swallowing and Dysphagia of Japan

Prize

2004: Watanabe Memorial Award from the Ube Foundation
Curriculum Vitae

Name: Li-Ang Lee, 李立昂

Sex: Male

Language: Mandarin, Taiwanese and English

Education:
1994 Jul- 1999 Jun, School of Postbachelor Medicine, Kaohsiung medical college, Kaohsiung, Taiwan
1989 Jul-1993 Jun, School of Medical Technology, Chang Gung University, Taoyuan, Taiwan

Employment record:
(1) 2011 Jul – Present, Director, Division of Pediatric Otolaryngology, Department of Otolaryngology, Chang Gung Memorial Hospital
(2) 2009 Nov – Present, Assistant Professor, School of Medicine, Chang Gung University
(3) 2007 Jun – Present, Assistant Professor , Department of Otolaryngology, Chang Gung Memorial Hospital
(4) 2003 Jul – Present, Attending physician, Department of Otolaryngology, Chang Gung Memorial Hospital
(5) 1999 Jul – 2003 Jun, Resident, Department of Otolaryngology, Chang Gung Memorial Hospital
(6) 1998 Jul – 1999 Jun, Internship, Chang Gung Memorial Hospital

Professional Affiliation:
1. Taiwan Otolaryngological Society
2. Taiwan Society of Sleep Medicine
3. Taiwan Voice Society
4. American Academy of Otolaryngology-Head and Neck Surgery

Research Interest:
1. Sleep medicine, 2. Pediatric otolaryngology, 3. HPV, 4. Laryngology, & 5. Head and neck oncology
Curriculum Vitae

VICTOR ABDULLAH

July 2012
Place of Birth: Hong Kong
Nationality: British
Sex: Male
Religion: Roman Catholic
Language: Fluent command of written and spoken English and Cantonese

Mail Address: Department of ENT
G1C, United Christian Hospital
130 Hip Wo Street, Kwun Tong
Kowloon, Hong Kong SAR
Telephone: (852) 3513 5195
Fax: (852) 3513 5506
Email: abdlv@ha.org.hk

Qualifications: BSc (Hons), MBBS (London), FRCS (England), FRCS (Edinburgh), FHKAM (Otorhinolaryngology), FCSHK, FHKCORL
EDUCATION

Medical School
Charing Cross & Westminster Medical School
London, UK

1979 - 1985

Intercalated BSc(Hons)
Biochemistry & Pharmacology

Unit Courses in:
“Biochemical Bases of Human Diseases”
“Anaphylaxis and Allergy”

1981 - 1982

Full GMC Registration
August 1986

QUALIFICATIONS

BSc (Hons)       June 1982 (Second Class Upper)
MBBS (London)    June 1985
FRCS (England) in Otolaryngology May 1991
FCSHK            March 1993
FHKAM (Otorhinolaryngology) June 1994
FHKCORL          November 1996
FRCS (Edinburgh) March 1998
Awarded without examination

POST-REGISTRATION EXPERIENCE

Senior House Surgeon in Otolaryngology 30th September 1986 - 31st August 1987
to Mr SM Mady, Mr JR Knight & Mr ARJ Al-Sheikhli
Mayday University Hospital
Croydon, UK

Senior House Surgeon in General Surgery 1st September 1987 - 31st August 1988
to Mr JEH Pendower
Mayday Hospital
Croydon, UK

Otolaryngological experience at October 1988 – July 1992
The Royal National Throat, Nose & Ear Hospital
London, UK

Senior House Surgeon to 1st October 1988 - 30th April 1989
Mr CB Croft

Senior House Surgeon to 1st May 1989 - 30th June 1989
Mr CM Bailey & Mr NS Shah

Registrar in Otology and 1st July 1989 - 31st January 1990
General Otolaryngology to Mr CM Bailey & Mr NS Shah

Registrar in Head & Neck Surgery and General Otolaryngology to Mr. CB Croft
1st February 1990 - 31st January 1991

Registrar in General Otolaryngology to Mr DI Choa
Whittington Hospital
London, UK
(part of registrar rotation)

Acting Senior Registrar to Mr DI Choa
Whittington Hospital
London, UK

Registrar in Otology and General Otolaryngology to Mr CM Bailey, Mr NS Shah & Mr DI Choa
1st February 1992 - 30th June 1992

Medical and Health Officer Honorary Clinical Tutor
Co-Ordinator of:
Laryngology
Head & Neck (ENT) Surgery
Paediatric Otolaryngology
to Professor CA van Hasselt
Department of Surgery
The Chinese University of Hong Kong
Prince of Wales Hospital
Hong Kong
September 1992 - April 1995

Senior Medical and Health Officer Honorary Clinical Associate Professor
Co-ordinator of:
Laryngology
Head & Neck (ENT) Surgery
Paediatric Otolaryngology
Sleep Medicine
Voice Clinic
Dysphagia Clinic
to Professor CA van Hasselt
Department of Surgery
The Chinese University of Hong Kong
Prince of Wales Hospital
Hong Kong
May 1995 - December 1996

Unit Head SMO i/c Department of ENT
Alice Ho Miu Ling Nethersole Hospital
The Hospital Authority
Hong Kong
January 1997 – October 2000
Honorary Clinical Associate Professor
Department of Surgery
The Chinese University of Hong Kong
Prince of Wales Hospital
Hong Kong

Visiting Fellow in Head & Neck Reconstructive Surgery
February 1998 - May 1998
February 1998 - May 1998
Professor Sean Sellars
Groote Schur Hospital
Cape Town, South Africa

Visiting Fellow in Paediatric Otolaryngology
Professor Christopher Prescott
Red Cross Memorial Children’s Hospital
Cape Town, South Africa

Chief of Service in ENT
November 2000 – November 2004
Department of ENT
Alice Ho Miu Ling Nethersole Hospital
The Hospital Authority
Hong Kong

Honorary Clinical Associate Professor
Department of Surgery
The Chinese University of Hong Kong
Prince of Wales Hospital
Hong Kong

Cluster Co-ordinator, Clinical Services for ENT
October 2001 - November 2004
New Territories East Cluster
(Prince of Wales Hospital, Alice Ho Miu Ling Nethersole Hospital and North District Hospital)
The Hospital Authority
Hong Kong

Visiting Fellow (Observer)
March 2003
In Paediatric Otolaryngology
Great Ormond Street Hospital for Children
London, UK

PRESENT APPOINTMENT

Chief of Service in ENT
Since December 2004
Department of Otorhinolaryngology, Head and Neck Surgery
Kowloon East Cluster (United Christian Hospital and Tseung Kwan O Hospital)
The Hospital Authority
Hong Kong
(I was appointed Cluster Chief of Service in ENT of Kowloon East Cluster in January 2008.)
<table>
<thead>
<tr>
<th>Role</th>
<th>Institution/Committee</th>
</tr>
</thead>
<tbody>
<tr>
<td>Honorary Clinical Associate Professor</td>
<td>Department of Otorhinolaryngology, Head and Neck Surgery</td>
</tr>
<tr>
<td></td>
<td>The Chinese University of Hong Kong</td>
</tr>
<tr>
<td>Chief of Division of Paediatric Otorhinolaryngology</td>
<td>Department of Otorhinolaryngology, Head and Neck Surgery</td>
</tr>
<tr>
<td></td>
<td>The Chinese University of Hong Kong</td>
</tr>
<tr>
<td>Former Vice President</td>
<td>The Hong Kong College of Otorhinolaryngologists</td>
</tr>
<tr>
<td>Immediately Past President</td>
<td>The Hong Kong Society of Otorhinolaryngology, Head and Neck Surgery</td>
</tr>
<tr>
<td>Member</td>
<td>Court of Examiners</td>
</tr>
<tr>
<td></td>
<td>The Royal College of Surgeons of Edinburgh/The Hong Kong College of Otorhinolaryngologists Joint Intercollegiate Higher Specialist Examination</td>
</tr>
<tr>
<td>Member</td>
<td>Joint Chinese University of Hong Kong/New Territories East Cluster Clinical Research Ethics Committee</td>
</tr>
<tr>
<td>Editor</td>
<td>SENSES</td>
</tr>
<tr>
<td></td>
<td>News Magazine of The Hong Kong College of Otorhinolaryngologists (2006 – 2011)</td>
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<tr>
<td>Honorary Consultant in Otorhinolaryngology</td>
<td>New Territories East Cluster</td>
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<td></td>
<td>The Hospital Authority</td>
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<td>Council Member</td>
<td>Local Service Sub-Committee</td>
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<td>China Service Sub-Committee</td>
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<td>The Hear Talk Foundation</td>
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<tr>
<td>Council Representative</td>
<td>The Hong Kong Federation of Medical Societies (1995 - 2003)</td>
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<tr>
<td>Honorary Secretary</td>
<td>The Hong Kong Society of Otorhinolaryngology, Head and Neck Surgery</td>
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<tr>
<td>Member</td>
<td>Education Committee</td>
</tr>
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<td>The Hong Kong College of Otorhinolaryngologists (2001 - 2005)</td>
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<tr>
<td>Council Member</td>
<td>The Hong Kong College of Otorhinolaryngologists (2001 - 2005)</td>
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<tr>
<td>Honorary Clinical Advisor</td>
<td>ENT Commissioning Team</td>
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<td>Alice Ho Miu Ling Nethersole Hospital</td>
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<td>(1995 - 1997)</td>
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</table>
SPECIAL INTERESTS

Otology, Paediatric Otolaryngology, Surgery for Obstructive Sleep Apnoea and Interventional Bronchoscopy.

RECENT PUBLICATIONS

Leukotriene receptor antagonist in the treatment of childhood allergic rhinitis – a randomised placebo-controlled study
Albert M Li, V Abdullah, Tsen CS, Au CT, Lam HS, So HK, Chan MH, Leung AW, Chan IF, Lam CW, Ng PC
Pediatric Paumonol. 2009 Nov;44(11);1085-92

High-level expression of early growth response-1 and association of polymorphism with total IgE and atopy in allergic rhinitis adults
Iris HS Chan, Dennis LY Lee, Osn YM Ho, Eddy WY Wong, Yvonne TO Lam, Nelson LS Tant, Michael HM Chan, V Abdullah, Chun K Wong and Christopher WK Lam

Natural history and predictors for progression of mild childhood obstructive Sleep apnoea
Li AM, Au CT, Ng SK, V Abdullah, Ho C, Fok TF, Ng PC, Wing YK
Thorax 2009 Sep 23

Parapharyngeal space neuroglial heterotopia with tumoral differentiation
Joseph Y Chan, V Abdullah, CA van Hasselt, Tong MC
International Journal of Paediatric Otorhinolaryngology
Extra 2010 (155-8)

Facial Plastic Surgery in Otorhinolaryngology
V Abdullah
The Hong Kong Medical Diary, Vol 15, No. 4, April 2010, P.4-6

Epidemiology of obstructive sleep apnoea syndrome in Chinese children:
A two-phase community study
Li AM, So HK, Au CT, Ho C, Lau J, Ng SK, V Abdullah, Fok TF, Wing YK
Thorax. 2010 Nov;65(11):991-7

Otitis Media with Effusion and Hearing Loss in Cleft Lip and Palate Children in Chinese
Kwan WM, V Abdullah, Liu K, van Hasselt CA, Tong MC
Dr Hsu Pon Poh is the Chief & Senior Consultant, ENT department, Changi General Hospital in Singapore.

He has special interest on sleep disordered breathing. He has pioneered the technique of Computer-assisted Video-endoscopic Quantitative Upper Airway Analysis for SDB. He has lectured and contributed to peer-reviewed journals on this subject.

He heads the CGH Integrated Sleep Services, which is the first of its kind in Singapore and comprises of multiple surgical, medical, allied health specialties with 23 members. CGH Accredited Sleep Lab is one of the busiest lab in Singapore.

Dr Hsu has won 16 awards locally and internationally. Dr Hsu headed CGH Research & Clinical trials unit from 2004-2009, CTRU has conducted 160 Pharmaceutical trials with secured contracts of more than 20 million dollars, 158 Hospital Principal Investigator’s trials till date.

In 2009, Dr Hsu spearheads the new International Liaison unit which is formed to promote and enhance the International collaborations between CGH and International reputable Academic Medical Centers in the areas of service development, research & education collaborations.

A strong supporter of patient-centric approach to healthcare, Dr Hsu places much emphasis on patient’s education and ensures that his patients receive appropriate treatment.
Curriculum Vitae

NAME          Sato Honma
NATIONALITY  Japanese

EDUCATIONAL QUALIFICATION
Doctor of Philosophy; March 1976 (Physiology)
Doctor of Medicine; June, 1972
Resident; Pediatrics (at Hokkaido University Hospital, Sapporo, Japan)

EDUCATION
1966-1972   Hokkaido University School of Medicine
1972-1976   Hokkaido University Graduate School of Medicine  (Physiology)

FELLOWSHIPS AND POSITIONS
1976-1977   Resident, Department of Pediatrics, Hokkaido University Hospital
1977-1978   Postdoctoral Fellow, Dept. of Anatomy, Hokkaido University School of Medicine
1978-1979   Postdoctoral Fellow, Dept. of Neurochemistry, Max-Planc-Institute for Biophysicalchemistry, (Goettingen, Germany)
1979-1981   Postdoctoral Fellow, Dept. of Anatomy, Hokkaido University School of Medicine
1981-1989   Instructor, Dept. of Physiology, Hokkaido University School of Medicine
1989-1992   Lecturer, Dept. of Physiology, Hokkaido University School of Medicine
1992-2007   Associate Professor, Dept. of Physiology, Hokkaido University School of Medicine
2007-2012   Professor, Dept. of Physiology, Hokkaido University Graduate School of Medicine
2007-       Dept. of Chronomedicine, Hokkaido University Graduate School of Medicine

MEMBERSHIP OF SOCIETIES
Member of Science Council of Japan
Science Council of Japan, Basic Medicine Committee member (vice chair)
Science Council of Japan, Section of Functional Medical Sciences (chair)
Society for Research on Biological Rhythm (Secretary 1998-2000)
Society for Neuroscience
Japanese Society of Physiology (Counselor)
Japanese Society of Chronobiology (Board of Directors)
The Japan Neuroscience Society (Board of Directors)
Japanese Society of Sleep Research (Counselor)

EDITORIAL WORK
2004-2010   Associate Editor of Journal of Biological Rhythms

AWARDS
2000. May 13  Aschoff’s Rule Award  (SRBR meeting at Amelia Island  FL)
2010. March 29  Prominent Scientific Achievement Award (Hokkaido University Graduate School of Medicine)
Michihiro Mieda, PhD
Associate Professor
Department of Molecular Neuroscience and Integrative Physiology
Graduate School of Medical Science
Kanazawa University

Education
1988-1992 Faculty of Science, University of Tokyo, BSc
1992-1994 Department of Biochemistry and Biophysics, Graduate School of Science, University of Tokyo, MSc
1994-1997 Department of Biochemistry and Biophysics, Graduate School of Science, University of Tokyo, PhD

Research and Professional Experience
2008-present Associate professor, Department of Molecular Neuroscience and Integrative Physiology, Graduate School of Medical Science, Kanazawa University
2004-2007 Assistant Professor, Department of Molecular Neuroscience, Medical Research Institute, Tokyo Medical and Dental University
2001-2004 Postdoctoral Fellow, Department of Genetics, University of Texas Southwestern Medical Center
1998-2001 Research Scientist, Laboratory for Developmental Gene Regulation, RIKEN Brain Science Institute
1997 Research Associate, Department of Physiology, Medical School, Keio University

Awards
2001-2004 Human Frontier Science Program Long-Term Fellowship
2006-2009 Human Frontier Science Program Career Development Award

Membership of Academic Society
1999-present The Japan Neuroscience Society
2006-present Japanese Society For Chronobiology
2009-present Physiological Society of Japan
2011-present Society for Neuroscience, USA
2009-present Editorial board member for “CNS & Neurological Disorders-Drug Targets”
Kyoung Jin Lee

Director, Center for Cell Dynamics and Dept. of Physics at Korea University,
Sungbuk-Ku, Anam-Dong 5-1, Seoul, Korea 136-713

- **PhD in Physics**, 1994, Dept. of Physics, The University of Texas at Austin, TX, USA
- **Research Associate**, 1994-95, Dept. of Physics, Princeton University, Princeton, NJ, USA
- **Professor**, 1996-current, Dept. of Physics, Korea University, Seoul, Korea

**Research Interest: biophysics of neural systems and cardiac wave dynamics**

Broadly speaking, I am interested in biophysical problems in which nonlinear dynamics plays an essential role. These are ubiquitous in nature: by all means, "living" creatures are better be dynamical!

The most significant problem in this regard is perhaps biological neural networks (or brains!). They have remarkable computational capabilities and "minds" that are proven very difficult to emulate in conventional digital computers. Over many years, researchers have learned that understanding distributed nonlinear dynamical activity of neuronal networks is essential in deciphering how they perform cognitive tasks and represent, store, and process information. Currently, we have a limited understanding along this line of investigation.

Another important example is cardiac fibrillation. Scientists and cardiologists all agree that spontaneous or induced spatiotemporal nonlinear wave activity of cardiac cell populations (or cardiac waves) are responsible for various type of cardiac arrhythmia including fibrillations that cause a sudden cardiac death. Subsequently, some utmost concerns are to understand why these collective waves emerge suddenly and to characterize their biophysical properties for the use in preventing or removing them.

Concerning the above two challenging problems, my laboratory is exploring several different biophysical issues based on in vitro experiments with up-to-date imaging techniques and electronics, and with computer simulations. Some key issues are:

- Recurrent neural (electrical and calcium) activity in cultures of cortical cells:
imaging techniques and electronics, and with computer simulations. Some key issues are:

- **Recurrent neural (electrical and calcium) activity in cultures of cortical cells:**

  Spatially distributed neuronal networks covering a large domain exhibit synchronized activity of 'bursts' of spikes. They seem to be a generic property of any growing network of neurons in development. Recurrent synchronized bursting activity are often hypothesized as a basis for neural computation and memory. On the other hand, the neural bursting activity is also believed responsible for the abnormal brain activity such as epileptic seizure. Among others, our research will focus on the dynamic re-organization of in vitro neural circuits for various patterned stimulations.

- **Calcium waves in network of astroglial cells and their interactions with neurons:**

  In mature neuron-glia co-culture systems, populations of neurons often exhibit globally synchronized fast 'calcium spikes' which match with the neural bursts almost in one-to-one fashion. Interestingly, however, the glia in the same co-culture support much slower calcium wave activities. In fact, a growing set of evidences augments the role of glia as regulatory agents of neuronal activity, undertaking active roles in brain neural computation. We are interested in characterizing and modeling spontaneous and induced (electrical and calcium) spatiotemporal activities that arise in co-cultures of neurons and glia, and associate them with the learning ability of the neural circuits.

- **Complex oscillatory cardiac wave reentries (spiral waves) and their instabilities:**

  Ventricular fibrillation (VF) is one of the most deadly cardiac arrhythmia during which different parts of the heart beat asynchronously, thus the heart cannot pump blood properly. It remains the leading cause of death among industrialized nations, and in the United States alone, for example, there are about 300,000 sudden cardiac deaths associated with VF each year. A growing body of evidence suggests that VF is a 'dynamics disease' that is caused by cardiac spiral wave instabilities. Self-rotating spiral waves in heart tissue are life-threatening because their fast rotation frequency (5-10 Hz) drives an abnormally rapid contraction known as ventricular tachycardia (VT), which subsequently becomes unstable and decays to VF after a few rotations even in healthy tissue. During the last several years, my laboratory has pioneered in studying cardiac spiral waves and their transition to 'alternans,' a harbinger of VF, by developing an in vitro cardiac system and bringing in a new phase contrast imaging technique that non-invasively visualizes mechanical waves of contractile motion. Continuing on the previous effort, currently we are exploring various cardiac wave instabilities leading to VF, and characterize important dynamic properties such as 'restitution curve' and 'dispersion relation' under various pharmacological conditions.
Synchronization and phase resetting of biological master clock, SCN:

The biological master clock, suprachiasmatic nucleus (SCN) functions as the master circadian clock, an essential component of all mammals. The key building blocks responsible for the rhythm generation are the individual SCN neurons (clock cells). The core elements of the individual clock is now well known to be the transcription and translation feedback loops of 'clock

Nonequilibrium pattern formation in general
Building simple biological neural networks on multi-electrode array

Awards and Honors

- Presidential Young Investigator (2000)
- Grant, Nat'l Creative Research Initiatives (1998~current)

Three Major Publications (as of 2012.05.07)

<table>
<thead>
<tr>
<th></th>
<th>Paper title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Competing patterns of signaling activity in dictyostelium discoideum</td>
<td>Lee, KJ, Cox, EC</td>
</tr>
<tr>
<td></td>
<td>PHYSICAL REVIEW LETTERS (76,7), 1174-1177 (1996)</td>
<td>Goldstein, RE</td>
</tr>
<tr>
<td>2</td>
<td>Wave pattern selection in an excitable system</td>
<td>Lee, KJ</td>
</tr>
<tr>
<td></td>
<td>PHYSICAL REVIEW LETTERS (79,15), 2907-2910 (1997)</td>
<td></td>
</tr>
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<td>3</td>
<td>Complex-periodic spiral waves in confluent cardiac cell cultures induced by localized inhomogeneities</td>
<td>Hwang, SM Kim, TY Lee, KJ</td>
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<td></td>
<td>PNAS, USA (102,29), 10363-10368 (2005)</td>
<td></td>
</tr>
</tbody>
</table>
Ying-Hui Fu
Professor, Department of Neurology
School of Medicine
University of California San Francisco
Fu & Ptáček Laboratories, MC 2922
1550 Fourth St., Room 548B
San Francisco, CA 94158-2324
USA

EDUCATION
1976-1980 - National Chung-Hsing University, Taichung, Taiwan, BS, Food Science
1981-1986 - Ohio State University, PhD, Biochemistry & Molecular Biology
1987-1989 - Ohio State University, Postdoc, Molecular Biology
1990-1993 - Baylor College of Medicine, Postdoc, Human Genetics

EMPLOYMENT
PRINCIPAL POSITIONS HELD
1980-1981 - Research Assistant, Food Industry Research Institute, Taiwan
1993-1995 - Scientist, Millennium Pharmaceutical Inc., Boston, MA
1995-1997 - Senior Scientist, Darwin Molecular Corp., Seattle, WA
1997-2002 - Research Associate Professor, University of Utah, Salt Lake City, UT
2002-2006 - Associate Professor, University of California, San Francisco
2006-pres - Professor, University of California, San Francisco

ANCILLARY POSITIONS HELD
2004-pres - University of California, San Francisco, Member, Tetrad Program
2004-pres - University of California, San Francisco, Member, Pharmaceutical Sciences & Pharmacogenomics Program
2002-pres - University of California, San Francisco, Member, Program in Biological Sciences
2002-pres - University of California, San Francisco, Member, Neuroscience Program
2002-pres - University of California, San Francisco, Member, Biomedical Sciences Program

1997-2003 - University of Utah, Member, Human Molecular Biology Program

1997-2003 - University of Utah, Member, Interdepartmental Program in Neuroscience

PROFESSIONAL ACTIVITIES

MEMBERSHIP IN PROFESSIONAL ORGANIZATIONS

American Society of Human Genetics

Society for Neuroscience

Society for Research on Biological Rhythms

SERVICE TO PROFESSIONAL PUBLICATIONS

2001 Ad hoc reviewer - Science

2002 Ad hoc reviewer - Wellcome Trust Grants

2003 Ad hoc reviewer - Wellcome Trust Grants

6/2003 Ad hoc reviewer - Human Molecular Genetics

9/2003 Ad hoc reviewer - Human Molecular Genetics

2/2005 Ad hoc reviewer - Sleep

3/2005 Ad hoc reviewer - Journal of Biological Rhythm

7/2005 Ad hoc reviewer - Journal of Biological Rhythm
CURRICULUM VITAE

Ji Ho Choi, M.D., Ph.D.,
Certified International Sleep Specialist, RPSGT

Work
Department of Otorhinolaryngology Head and Neck Surgery, College of Medicine, Korea University, Ansan hospital
516 Gojan-Dong, Danwon-Gu, Ansan-City, Gyeonggi-Do, 425-707, Republic of Korea

Faculty
- 2009.3 ~ present ; Clinical Assistant Professor, Department of Otorhinolaryngology Head and Neck Surgery, Ansan Hospital, Korea University Medical Center
- 2007.3 ~ 2009.2 ; Clinical Instructor, Department of Otorhinolaryngology–Head and Neck Surgery, Ansan Hospital, Korea University Medical Center
- 2005.3 ~ 2007.2 ; Clinical Fellow, Department of Otorhinolaryngology–Head and Neck Surgery, Ansan Hospital, Korea University Medical Center

Certification and License
- Korea Medical License : 2000
- Korean board of Otorhinolaryngology–Head and Neck Surgery : 2005
- Certified International Sleep Specialist (ABSM, USA) : 2006
- Registered Polysomnographic Technologist (USA) : 2007

Publication [SCI(E)]

1) Choi JH, Kim SN, Cho, JH.
Prof. Soichiro Miyazaki, MD, PhD

Prof. Miyazaki was born in Ehime, Japan 1954. He graduated from Akita University School of Medicine 1979. Prof. Miyazaki has been a chairman of Department of Sleep Medicine, Shiga University of Medical Science, since 2004. He is an executive committee member of Japan Society of Sleep Research. He is a member of Oto-Rhino-Laryngological Society of Japan. Prof. Miyazaki’s major interest is in Sleep Medicine and Sleep Apnea Syndrome. He also spends much effort for public education of sleep medicine.
Satoru Miyauchi

Birth Date: 1954/09/24 (Tokyo, Japan)

Research fields: Psychophysiology, Non-invasive measurements of human brain function

Current Major Researches:
Simultaneous EEG recording with fMRI
Psychophysiological study on dreaming
Visual imagery, Visual attention, Crossmodal Attention, Body image

Professional background:
2006- : Research Manager, Kobe Advanced ICT Research Center, National Institute of Information and Communications Technology, Kobe, Japan
1998 - 2005: Senior Researcher, Kansai Advanced Research Center, National Institute of Information and Communications Technology, Kobe, Japan
1993 - 1997: Senior Researcher, Communications Research Laboratory, Tokyo, Japan
1990 - 1993: Assistant Professor, National Institute for Physiological Sciences, Okazaki, Japan
1988 - 1990: Visiting Scholar, Department of Psychology, Brown University, Providence RI, USA
1986 - 1988: Assistant Professor, Department of Psychology, Waseda University, Tokyo, Japan

Teaching experience:
1998- Visiting professor, Department of Brain Science and Systems Engineering, Kyushu Institute of Technology
2000- Part-time lecturer, Graduate School of Human and Environmental Studies, Kyoto University
1992-2008 Part-time lecturer, Graduate School of Information Science and Electrical Engineering, Kyushu University

Academic background:
1990: Ph. D., Toho University (Japan), Medical Sciences
1981: Master Degree. Waseda University (Japan), Psychology
1977: BA Waseda University (Japan), Psychology
CURRICULUM VITAE

Yawei Cheng
Social Neuroscience Laboratory
Institute of Neuroscience
National Yang-Ming University
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Taiwan, R.O.C.

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Fax: +886-2-2826-4903
Email: ywcheng2@ym.edu.tw

EDUCATIONS:
06/2007 – Ph.D. Institute of Neuroscience, School of Live Science, National Yang-Ming University
07/1996 – M.D. School of Medicine, Chang-Gung University

POSITION HELD
02/2011–present  Associate Professor, Social Neuroscience Laboratory, Institute of Neuroscience, National Yang-Ming University
02/2008–01/2011 Assistant Professor, Social Neuroscience Laboratory, Institute of Neuroscience, National Yang-Ming University
04/2008–present Attending Physician, Department of Physical Medicine & Rehabilitation, National Yang-Ming University Hospital
02/2005–01/2006 Postdoctoral Fellow, Social Cognitive Neuroscience Laboratory, Institute for Learning and Brain Sciences, University of Washington, Seattle (Advisor: Jean Decety)
09/2000–05/2006 Doctoral Research, Laboratory for Cognitive Neuroscience, Institute of Neuroscience, National Yang-Ming University (Advisor: Ovid Tzeng)
07/1997–06/2001 Residency, Department of Physical Medicine & Rehabilitation, National Taiwan University Hospital
07/1996–06/1997 Residency, Department of Neurology, National Taiwan University Hospital
10/1989–06/1996 Medical College of Chang Gung University

AWARDS AND HONORS:
2011 Wu Da-You Memorial Award and Young Investigator Grant from National Science Council, Taiwan
2009 The 20th Ten Outstanding Young Women, Taiwan
2006 Award of Junior Physicians by Rehabilitation Medicine Funds of Prof. I-Nan Lien
2006 Award of Taipei Medical Wang-Fang Neuroscience, Neuroscience Society of Taiwan
Ching-Sui (Bessy) Hung 洪菁穗

Brain Research Center
National Yang-Ming University, Taiwan

Integrated Brain Research Lab,
Taipei Veterans General Hospital, Taiwan

Tel: 886-920166571
Email: bessyhung@gmail.com

Education

Sleep Research Centre (SRC), Dept. of Human Sciences, Loughborough University, Loughborough, Leicestershire, U. K.
Supervisors: Jim Horne, DSc, PhD & Clare Anderson, PhD
Director of Research: Kevin Morgan, PhD

09/2000-02/2003  MSc. in Neuroimaging
1 Dept. of Psychology, National Chung-Cheng University (CCU), Chiayi, Taiwan.
2 Integrated Brain Research Unit (IBRU), Dept. of Medical Education and Research, Taipei Veterans General Hospital (Taipei-VGH), Taipei, Taiwan
Supervisors: 蔡玲玲 Ling-Ling Tsai, PhD & 謝仁俊 Jen-Chuen Hsieh, MD, PhD

09/1995-06/1999  B.A. in Journalism
Dept. of Journalism, National Cheng-chi University (NCCU), Taipei, Taiwan

Research Experiences

Functional Brain Imaging (MEG, EEG, fMRI)
04/2012-present  MEG/EEG/fMRI study in Schizophrenia / Sleep
Post-doctoral fellow in the Brain Research Centre, National Yang-Ming University, Taipei, Taiwan
Supervisor: Jen-Chuen Hsieh, MD, PhD.

Sleep Research (human/animal)
11/2008-02/2012  High-Density (hd) EEG Study in Human:
Post-doctoral research associate at the Center for Sleep and Consciousness, University of Wisconsin-Madison, United States of America
Supervisor: Giulio Tononi, MD, PhD
Collaborators: Simone Sarasso, PhD; Fabio Ferrarelli, MD, PhD; Brady Riedner, PhD; Giulio Bernardi, MD (project 1) & Jan Grosch, MD (project 2)

Full-time research assistant at the Sleep Laboratory, CCU, Chiayi, Taiwan
Supervisor: Ling-Ling Tsai, PhD

Professional Affiliations

• Sleep Research Society (2007-present)
• Taiwan Sleep Medicine Society (2002-present)
Seiichi Nakata, MD, PhD  Associate Professor

Office: Department of Otorhinolaryngology
Second Hospital, Fujita Health University School of Medicine
3-6-10 Otobashi, Nakagawa-Ku, Nagoya, 454-8509 Japan

Education:

College, Medical Degree

Kochi University School of Medicine
Nankouku, Kochi Prefecture, 1983-March 1989

Internship
Okazaki Municipal Hospital
Okazaki, Aichi Prefecture, 1989-1990

Residency
Nagoya University Graduate School of Medicine
Nagoya, Aichi Prefecture, 1990-1992
Kariya Toyota General Hospital
Kariya, Aichi Prefecture, 1992-1993

Research History:

Washington University
St. Louis, Mo, 1994-1996
(Research fellow, Research in central auditory system)

Job History:
Nagoya University Graduate School of Medicine
Nagoya, Aichi Prefecture, 1996
Nagoya First Red Cross Hospital
Nagoya, Aichi Prefecture, 1996-1999
Nagoya University Graduate School of Medicine
Nagoya, Aichi Prefecture, 1999-2010
Second Hospital, Fujita Health University School of Medicine
Nagoya, Aichi Prefecture, 2010-present
Research:
(1) Research grant from Grants-in-Aid for Scientific Research to study the physiology of nasal function under a sleep. Research conducted with Seiichi Nakata MD, PhD, at Nagoya University, 2006-2008
(2) Research grant from Suzuken Memorial Foundation to study the effectiveness of Pillar® Palatal Implant on mild OSAS patients Research conducted with Seiichi Nakata MD, PhD, at Nagoya University, 2006-2007

Professional Organization:
American Academy of Sleep Medicine
The Japanese Society of Sleep Research
The Oto-Rhino-Laryngological Society of Japan
Japan Society of Somato-pharyngology
Japan Rhinologic Society

Licence/Certifications:
Medical Doctor license number: 327153 1989~present
A doctorial degree: 2550 (Nagoya University) 1997~present
Chairat Neruntarat, MD
Professor, Chair
Department of Otolaryngology
Faculty of Medicine
SrinaKharinwirot University
President, Sleep Apnea Association, Thailand
President, Snoring & Sleep Disorders Foundation, Thailand
Sadık Ardıç M.D.

- Medical Doctor (Hacettepe University, Medical Faculty) 1-July-1980
- Pulmonary Specialist (Hacettepe University, Medical Faculty) 10-April-1986
- Assistant Prof. (Anadolu University, Medical Faculty) 11-November-1986
- Associate Prof. (Anadolu University, Medical Faculty) 9-October-1990
- Associate Prof. (İnönü University, Medical Faculty) 11-August-1991
- Sleep Medicine Fellow (Baylor College Of Medicine, Sleep Disorders Center, Houston, Texas, USA) September 1991-1992
- Associate Prof. (İnönü University, Medical Faculty) 1992-April 1993
- Head Of Pulmonary Medicine (SSK Ankara Research and Training Hospital) April 1993-2000
- Cheif Of Pulmonary Medicine (SSK A SSK Ankara Research and Training Hospital) 7. September 2000
- Cheif Of Pulmonary Medicine (SB.Disclaimer YB. Research and Training Hospital) November. 2005-
- President of Turkish Sleep Medicine Society(2007-2011)
- Executive Member of Turkish Sleep Medicine Society(2011-)

Publications


CURRICULUM VITAE

Prof. Murat AKSU, M.D.

He is the professor of Neurology in Erciyes University Medical Faculty. He completed his medical education in Hacettepe University in Ankara and residency in Erciyes University, Kayseri. He earned his associate professor degree in 2000 and professor degree in 2006. He studied as fellow in Neurology in 1998-1999 in National Institutes of Neurology, Bethesda-Maryland, USA and worked as investigator in same Institute in 2003-2005. He is the founder and the chair of Sleep Disorders and Research Unit in Erciyes University. He is the professor of Neurology in Erciyes University and also the head of Neurology and Sleep Disorders units in Acibadem Kayseri Hospital. He has more than 60 publications and 90 oral and poster presentations. He is the elected president of Turkish Sleep Medicine Society and president of Sleep Disorders Study Group of Turkish Neurology Society.
CIRRICULUM VITAE
AND LIST OF PUBLICATIONS RELATED WITH SLEEP DISORDERS

CIRRICULUM VITAE
Name, surname: HİKMET YILMAZ
Date and Place of Birth: April 14, 1964 Arapgir-Malatya, Turkey
Nationality: Turkish
Degree: Professor
Affiliation and Official Address: Celal Bayar University, School of Medicine, Department of Neurology, Section of Epilepsy and Sleep Disorders, 45010, Manisa, Turkey

Education (Degrees, dates, universities):

<table>
<thead>
<tr>
<th>Degree</th>
<th>Field</th>
<th>University</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>High licence</td>
<td>Medical Faculty</td>
<td>İSTANBUL UNIVERSITY MEDICAL CERRAHPAŞA FACULTY</td>
<td>1988</td>
</tr>
<tr>
<td>Doctorate</td>
<td>Neurology</td>
<td>İNÖNÜ UNIVERSITY MEDICAL FACULTY</td>
<td>1996</td>
</tr>
</tbody>
</table>

Career/Employments (Employers, positions and dates):

<table>
<thead>
<tr>
<th>Degree of employment</th>
<th>Place of employment</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>General practitioner Dr.</td>
<td>Karakoçan Merkez Sağlık Ocağı, Karakoçan, Elazığ</td>
<td>1988-1990</td>
</tr>
<tr>
<td>General practitioner Dr.</td>
<td>Vehbi Bolak Kan Merkezi, Balıkesir</td>
<td>1990-1991</td>
</tr>
<tr>
<td>General practitioner Dr.</td>
<td>Balıkesir Devlet Hastanesi, Acil Birimi, Balıkesir</td>
<td>1991-1992</td>
</tr>
<tr>
<td>Researcher Dr.</td>
<td>Medical Faculty, İnönü University</td>
<td>1992-1996</td>
</tr>
<tr>
<td>Assistant Professor Dr.</td>
<td>Medical Faculty, Celal Bayar University</td>
<td>1996-2002</td>
</tr>
<tr>
<td>Associate Professor Dr.</td>
<td>Medical, Celal Bayar University</td>
<td>2002-2009</td>
</tr>
<tr>
<td>Professor Dr.</td>
<td>Medical, Celal Bayar University</td>
<td>2009-.......</td>
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</table>

Membership of Scientific Organizations:
The European Neurological Society
International League Against Epilepsy Society
**Membership of Scientific Organizations:**
The European Neurological Society
International League Against Epilepsy Society
Turkish Neurological Society
Turkish Sleep Medicine Society
Movement Disorders Society

**Specialization (specify)**
Sleep disorders, Epilepsy, Neuro-otology, Neuro-ophthalmology and Clinical Neurophysiology

**List of publications related with sleep disorders**

1. **Yılmaz, H.**, İşkesen, İ. “Late daytime naps may cause drowsiness after coronary bypass graft operation in postoperative first week”. *Neurosciences, Vol. 12 (4); 348-349 (2007).*


PA-CHUN WANG M.D., MSc (王拔群教授)
CURRICULUM VITAE

Education and Training

Doctor of Medicine, China Medical College, Taichung, Taiwan
Master of Science in Health Policy and Management, Harvard School of Public Health, Boston, MA, USA
Research Fellowship in Otolaryngology, Massachusetts Eye and Ear Infirmary, Harvard Medical School, Boston, MA, USA
Executive Master Program in Business Management, College of Management, National Taiwan University, Taipei, Taiwan

Professional Assignment

Chief, Quality Management Center, Cathay General Hospital, Taipei, Taiwan
Chief, Department of Otolaryngology Head Neck Surgery, Cathay General Hospital, Taipei, Taiwan
Fellow, Taiwan Joint Commission of Hospital Accreditation and Healthcare Quality
Professor, Fu Jen Catholic University School of Medicine
Adjunct Professor: School of Public Health, China Medical University, Taipei
Medical University School of Medicine; and National Central University
Clinical Professor, School of Medicine, National Yang-Ming University, Taipei, Taiwan

Professional Qualification

Member, Collegium Oto–Rhino–Laryngologicum Amicitiae Sacrum
Member, The International Otopathology Society a.k.a Schuknacht Society
International Member, The American Academy of Otolaryngology- Head and Neck Surgery
Member and Board Certified Specialist in Otolaryngology, The Otolaryngological Society of Taiwan
Certified Professional in Healthcare Quality (CPHQ), Healthcare Quality Certification Board (HQCB) of the National Association for Health Quality (NAHQ)
Certified Advanced Quality Manager, The Association for Healthcare Quality, R.O.C.

Professional and Social Services

Board Director, Taipei Sinfonieta and Philharmonic Orchestra Fundation
Board Director, Taiwan Otolaryngological Society
Board Director, The Association for Healthcare Quality, R.O.C.
Board Director, Taiwan College of Healthcare Executive

Research Interest

Otology and neurotology
Outcomes in otolaryngology diseases
Quality of care
Patient safety
Health policy and management
Curriculum vitae

Personal Data
Name: Tomomi Tsunematsu Ph.D.
Sex: female
Date of Birth: 15. Dec. 1983
Nationality: Japanese

Address Correspondence: Division of Cell Signaling, Okazaki Institute for Integrative Bioscience, National Institutes of Natural Sciences, Okazaki, Aichi 444-8787, Japan

Education
2002-2006 College of Biological Science, Second Cluster of Colleges, University of Tsukuba
2006-2008 Graduate School of Comprehensive Human Sciences, University of Tsukuba
2008-2011 Department of Physiological Sciences, School of Life Science, The Graduate University for Advanced Studies

Career
2011- JSPS Postdoctoral Fellow, National Institute for Physiological Sciences
Mark R. Opp, Ph.D.

Dr. Opp obtained his Ph.D. in Zoology from Washington State University, Pullman WA, in 1987. After postdoctoral training in the laboratory of Jim Krueger, Dr. Opp was recruited to the Department of Psychiatry at the University of Texas Medical Branch, Galveston, TX. In 2001, Dr. Opp moved to the University of Michigan, where he had appointments as Professor in the Departments of Anesthesiology, and Molecular & Integrative Physiology, as well as in the Neuroscience Graduate Program. Dr. Opp became the Vice Chair for Basic Research and holder of the UW Medicine Research & Education Endowed Chair in Anesthesiology in the Department of Anesthesiology & Pain Medicine at the University of Washington in 2010. Dr. Opp has served as Secretary Treasurer, President-elect, President and past President of the Sleep Research Society. He serves as associate editor for SLEEP and is on the editorial board for Sleep Medicine, Reviews and Brain, Behavior and Immunity. In addition to providing peer-review for many journals, Dr. Opp is a member of the Neuroendocrinology, Neuroimmunology, Rhythms and Sleep Initial Review Group of NIH.

Dr. Opp directs a research program funded by the National Institutes of Health to answer questions related to sleep-immune interactions. One unanswered fundamental question is whether the changes in sleep during infection aid in the recovery process. Conversely, chronic insufficient sleep impairs immune function. Our research has demonstrated that interactions among cytokines (interleukin-1) and neurotransmitters (serotonergic, GABAergic, cholinergic, hypocretinergic systems), play a role in the regulation of natural sleep and in altered sleep during infection. Current projects focus on: 1) prior sleep history as a determinant of clinical outcome in response to systemic infections such as sepsis, and 2) the mechanisms by which insufficient sleep produces CNS inflammation. These studies aim to answer mechanistic questions of how it is that infection alters CNS processes and why chronic insufficient sleep is associated with cardiovascular disease, insulin resistance, diabetes, and obesity.
CURRICULUM VITAE

PERSONAL DATA

Name: Fang-Chia Chang, Ph.D.
Citizenship: Taiwan, Republic of China

EDUCATION

1989  B.S.  (Pharmacy) Kaoshiung Medical College, Kaoshiung, Taiwan ROC
1991  M.S.  (Pharmacology) National Cheng-Kung University, Tainan, Taiwan ROC
1999  Ph.D.  (Neuroscience) University of Texas Medical Branch at Galveston, Galveston, TX

POSTDOCTORAL TRAINING

1999 – 2000  Postdoctoral Research Associate, Department of Pharmacology, Southern Illinois University School of Medicine, Springfield, IL

ACADEMIC APPOINTMENTS

2000 – 2004  Assistant Professor, Department of Chinese Medicine, China Medical University, Taichung, Taiwan ROC
2004 – 2005  Associate Professor, Department of Chinese Medicine, China Medical University, Taichung, Taiwan ROC
2005 – 2008  Assistant Professor, Department of Veterinary Medicine, National Taiwan University, Taipei, Taiwan ROC
2008 – 2012  Associate Professor, Department of Veterinary Medicine, National Taiwan University, Taipei, Taiwan ROC
2012 –  Professor, Department of Veterinary Medicine, National Taiwan University, Taipei, Taiwan ROC

Publications:

1 Pei-Lu Yi, Ying-Ju Chen, Chung-Tien Lin, Fang-Chia Chang*. Occurrence of Epilepsy at Different Zeitgeber Times Alters Sleep Homeostasis Differently in Rats. *Sleep* 2012 (in press).
5 Yi-Tse Hsiao, Pei-Lu Yi, Chia-Ling Li, Fang-Chia Chang*. Effect of Cannabidiol on Sleep Disruption Induced by the Repeated Combination Tests consisting of Open Field and Elevated Plus-maze in Rats. *Neuropharmacology* 2012; 62: 373-384.
7 Fang-Chia Chang*. Effects of traditional Chinese medicine in insomnia treatment: the possible
CURRICULUM VITAE

His research interests include applied brain biophysics, brain responsiveness and sleep, conscious states (i.e. sleep and anesthesia), information processing in the brain, multidisciplinary and translational studies in the cognitive science incorporating memory, sleep and pathological processes. He is the chair of Department of Biophysics, Faculty of Medicine, Dokuz Eylul University. He is a member of the Brain Dynamics Research Center. He is the associate editor in the journal of “Sleep and Biological Rhythms”. He has organized a number of congress and workshops on Brain biophysics and Neuroscience areas, and supported 13th World Congress of Psychophysiology- The Olympics of the Brain in Istanbul. He has participated in the initiative of The US Turkey Advanced Study Institute on Global Healthcare Grand Challenges indicating the future prospects of brain research as well as neural engineering. He has organized the Turkish Japanese Sleep Forum highlighting research capacity in sleep and biorhythms. He is a MC member of NEUROMATH (European COST action on Neural Signal Processing).

He is a national infrastructures expert of Turkish Research Council, TUBITAK for EC. Additionally he is a delegate of European Strategic Forum on Research Infrastructures (ESFRI), as well as being chair of Health and Food Strategy Working Group. He has represented Turkish Sleep Society in The 6th Congress of Asian Sleep Research, the 34th Annual Meeting of Japanese Society of Sleep Research, and the 16th Annual Meeting of Japanese Society for Chronobiology. Furthermore he has participated to ASRS Summit and Symposium in Okinawa, 2009. He is the associate-editor of Sleep and Biological Rhythms Journal and is the member of the editorial Editorial Board of World Journal of Anesthesiology. He reviews manuscripts for a number of journals ranging from Brain Research to Neuropsychologia. He is the founding member of the proposed initiative of “International Sleep Science and Technology Association (ISSTA” which is being launched in 2012).

1. Name: Murat Özgören
2. Birthday: 20/07/1966
3. Qualification: Prof. Dr.
4. Education

<table>
<thead>
<tr>
<th>Degree</th>
<th>Dept./Programme</th>
<th>University</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Graduate</td>
<td>Medical Faculty</td>
<td>Ege University</td>
<td>1992</td>
</tr>
<tr>
<td>Doctorate</td>
<td>Biophysics</td>
<td>Ege University</td>
<td>1999</td>
</tr>
</tbody>
</table>

5. Academic Qualifications and Works:

<table>
<thead>
<tr>
<th>Rank</th>
<th>Institution</th>
<th>Department</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assist. Prof.</td>
<td>Dokuz Eylul University, Medical Faculty Biophysics Dept.</td>
<td>2004</td>
<td></td>
</tr>
<tr>
<td>Assoc. Prof.</td>
<td>Dokuz Eylul University, Medical Faculty Biophysics Dept.</td>
<td>2005</td>
<td></td>
</tr>
<tr>
<td>Prof.</td>
<td>Dokuz Eylul University, Medical Faculty Biophysics Dept.</td>
<td>2010</td>
<td></td>
</tr>
</tbody>
</table>
CURRICULUM VITAE

Yawei Cheng
Social Neuroscience Laboratory                          Telephone: +886-2-2826-7912
Institute of Neuroscience                               Fax: +886-2-2826-4903
National Yang-Ming University                           Email: ywcheng2@ym.edu.tw
155, Sec. 2, St. Linong, Dist. Beitou
Taipei 112, Taiwan, R.O.C.

EDUCATIONS:
06/2007 – Ph.D. Institute of Neuroscience, School of Life Science, National Yang-Ming University
07/1996 – M.D. School of Medicine, Chang-Gung University

POSITION HELD
02/2011 – present  Associate Professor, Social Neuroscience Laboratory, Institute of Neuroscience, National Yang-Ming University
02/2008 – 01/2011 Assistant Professor, Social Neuroscience Laboratory, Institute of Neuroscience, National Yang-Ming University
04/2008 – present Attending Physician, Department of Physical Medicine & Rehabilitation, National Yang-Ming University Hospital
02/2005 – 01/2006 Postdoctoral Fellow, Social Cognitive Neuroscience Laboratory, Institute for Learning and Brain Sciences, University of Washington, Seattle (Advisor: Jean Decety)
09/2000 – 05/2006 Doctoral Research, Laboratory for Cognitive Neuroscience, Institute of Neuroscience, National Yang-Ming University (Advisor: Ovid Tzeng)
07/1997 – 06/2001 Residency, Department of Physical Medicine & Rehabilitation, National Taiwan University Hospital
07/1996 – 06/1997 Residency, Department of Neurology, National Taiwan University Hospital
10/1989 – 06/1996 Medical College of Chang Gung University

AWARDS AND HONORS:
2011 Wu Da-You Memorial Award and Young Investigator Grant from National Science Council, Taiwan
2009 The 20th Ten Outstanding Young Women, Taiwan
2006 Award of Junior Physicians by Rehabilitation Medicine Funds of Prof. I-Nan Lien
2006 Award of Taipei Medical Wang-Fang Neuroscience, Neuroscience Society of Taiwan
PROFESSIONAL QUATIFICATION

• Physiatrist

PROFESSIONAL MEMBERSHIPS

• Taiwan Academy of Physical Medicine and Rehabilitation
• Organization of Human Brain Mapping
• Society for Neuroscience (SfN)
• Society for Social Neuroscience (S4SN)
• Cognitive Neuroscience Society

RESEARCH INTEREST AND SKILLS:

• Social neuroscience
• Human mirror system
• Emotion regulation
• Empathy and sympathy
• Moral reasoning
• Autism spectrum disorders
• Psychopathy
• Developmental affective neuroscience
• Intersubjectivity
• fMRI, MEG, EEG/ERP, NCS/EMG, eye-tracking

INVITED TALKS

• 2011 Taiwan Society of Biological Psychiatry: Empathy in Autism: Reduced pain threshold mediates somatosensory response to the pain of others.
• 2011 Institute of Occupational Therapy, National Cheng-Kung University: Broken mirror theory of autism: from mirror neurons to empathy.
• 2011 Department of Psychiatry, National Taiwan University Hospital: The neural correlates of empathy imbalance in autism spectrum disorders.
• 2010 Institute of Cognitive Sciences, National Cheng-Kung University: Social neuroscience of empathy.
• 2010 Social Neuroscience and Its Benefits to Mental Health, Schizophrenia Translational Research Center (STRC), Seoul National University, Korea: Expertise modulates the perception of pain in others.

PUBLICATIONS:

Journal Papers
5. Chen CY, Yang CY, Cheng Y* (2012) Sensorimotor resonance is an outcome but not a platform to anticipating harm to others. Social
Curriculum Vitae (2012.6.10)

Name
Given name  Ken-ichi  Family name  Honma

Date of Birth
November 10, 1946

Nationality
Japanese

Educational Qualification
Doctor of Medicine :  June, 1971
Ph. Degree of Medicine :  March 1977

Education
1965.4-1971.3  Hokkaido University School of Medicine (Medical Doctor)
1973.4-1977.3  Postgraduate Course of Physiological Sciences, Hokkaido University Graduate School of Medicine (Ph.D. for Medicine)

Profession
1971.4-1973.3  Clinical Fellow at Hokkaido University Hospital (Psychiatry)
1977.4-1980.1  Assistant Professor, Hokkaido University School of Medicine (Physiology)
1980.2-1982.4  Lecture, Hokkaido University School of Medicine (Physiology)
1982.5-1991.5  Associate Professor, Hokkaido University School of Medicine (Physiology)
1992.1-2000.3  Professor, Hokkaido University School of Medicine (Physiology)
2000.4-2010.3  Professor, Hokkaido University Graduate School of Medicine (Physiology)
2005.4-2009.3  Dean, Hokkaido University Graduate School of Medicine and School of Medicine, Education and Research Council member of Hokkaido University
2009.4-2012.3  Director, Research and Education Center for Brain Science of Hokkaido University
2010.4-2012.3  Professor emeritus and Specially Appointed Professor, Hokkaido University
2012.4-  President of the Kei-Ai Cooperation Aggregate, Sapporo Hanazono Hospital

Membership of Academic Society (major)
International Union of Physiological Societies (2000-2006  Section Leader for Chronophysiology)
World Federation of Societies for Chronobiology (2002-2011 President)
Asian Society for Sleep Research (Secretary in General)
Science Council of Japan (Associate Member)
Japanese Society for Chronobiology (Council Member; 2005-2010 President)
Japanese Society for Sleep Research (Vice President, Editor-in-Chief of SBR)
Japanese Society of Physiology (Former Council Member)
CURRICULUM VITAE

“Her research interests are application of cognitive processes, learning, brain dynamics, electrophysiology and oscillations, brain pathologies. She is a group psychotherapist and psychodramatist. She was the secretary of National Biomedical Engineering Congress-2009, which was bridging Engineering and Medical Faculties. She was in organizing committee of the two Brain Biophysics Workshops in Izmir and local organizing committee of 13th World Congress of Psychophysiology- The Olympics of the Brain in Istanbul (2006). She is a member of Brain Dynamics Research Center of Dokuz Eylul University. She is in DEU-BAP, TUBITAK, and COST Projects as a coordinator, advisor, and researcher.”

1. **Name:** Adile Öniz (Tınç)
2. **Birthday:** 17 Nisan 1967
3. **Qualification:** Assoc. Prof. Dr. (MD, PhD)
4. **Contact Information**
   +90 232 4124498 (Office)
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   e-mail: adile.oniz@deu.edu.tr
5. **Education:**

<table>
<thead>
<tr>
<th>Degree</th>
<th>Dept./Programme</th>
<th>University</th>
<th>Year</th>
</tr>
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<tbody>
<tr>
<td>Graduate</td>
<td>Medical Faculty</td>
<td>Ege University</td>
<td>1990</td>
</tr>
<tr>
<td>Doctorate</td>
<td>Biophysics</td>
<td>Dokuz Eylul University</td>
<td>2006</td>
</tr>
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6. **Academic Qualifications and Works:**

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<tr>
<th>Position</th>
<th>Institution and Department</th>
<th>Years</th>
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<tbody>
<tr>
<td>Assist. Prof.</td>
<td>Dokuz Eylul University, Medical Faculty Biophysics Dept.</td>
<td>2008 - 2010</td>
</tr>
<tr>
<td>Assoc. Prof.</td>
<td>Dokuz Eylul University, Medical Faculty Biophysics Dept.</td>
<td>2010-</td>
</tr>
</tbody>
</table>
CURRICULUM VITAE

Akira Tamura, MD

PERSONAL INFORMATION
DATE OF BIRTH: 9 May 1959
PLACE OF BIRTH: Nobeoka
NATIONALITY: Japanese
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PROFESSIONAL ADDRESS: Internal Medicine 2, Faculty of Medicine, Oita University, Idaigaoka 2-3-1, Hasama-machi, Yufu 879-5593, Japan, E-mail: akira@oita-u.ac.jp

EDUCATION
COLLEGE/UNIVERSITY:
1978-84 MD, Medical College of Oita (currently, Faculty of Medicine, Oita University)

BOARD CERTIFICATION
1999 Board Certified Member of the Japanese Society of Internal Medicine
2001 Board Certified Member of the Japanese Circulation Society
2001 Senior Fellow of Japanese Society of Interventional Cardiology

PRESENT POSITION
Associate Professor, Internal Medicine 2, Faculty of Medicine, Oita University

PREVIOUS PROFESSIONAL POSITIONS
1996-2002 Assistant Professor of Internal Medicine 2, Faculty of Medicine, Oita University
2002-2005 Lecturer of Internal Medicine 2, Faculty of Medicine, Oita University

BIBLIOGRAPHY
Publication – Journal (SAS-related papers)
Baoyuan Chen

Chen Baoyuan, MD. FCCP. Director of the sleep centre, Department of pulmonary Medicine, Tianjin General Hospital, Tianjin Medical University. Member of Standing Committee of Society of Respiratory Medicine, Chinese Medical Association, in charge the Sleep Breath Disorder Group. Member of Standing Committee of Society of Respiratory Medicine, Chinese Chest Physician Association. Chairman of Tianjin Respiratory Association, Tianjin Medical Association. Member of Standing Committee or board for 10 medical journals. He is a national senior researcher and Doctor in the field of sleep apnea, intermittent hypoxia, asthma with gastroesophageal reflux, and pulmonary infection disease in China. The titles of his publications was more than 100 in Chinese and English with more than 600 citations to his work. As a chief editor, five medical books were published in the last 5 years.
Curriculum Vitae

Name: Dai Yumino

Institution & Location:
Department of Cardiology, Tokyo Women’s Medical University
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Education:
1998 M.D.       Juntendo University, Tokyo, Japan
2008 Ph.D.    Tokyo Women’s Medical University, Tokyo, Japan

Post-doctoral Training:
1998-2000    Internal Medicine, Tokyo Women’s Medical University.
2000-2002    Clinical fellow in Cardiology, Tokyo Women’s Medical University.
2002-2003    Department of Cardiology, Kosei Hospital
2003-2004    Department of Cardiology, Toranomon Hospital
2004-2006    Assistant Professor, Department of Cardiology, Tokyo Women’s Medical University.
2006-2009    Research fellow in Centre for Sleep Medicine and Circadian Biology, University of Toronto, Canada
2009-present    Assistant Professor, Department of Cardiology, Tokyo Women’s Medical University.
4. Clinical Experience

Clinical Cardiology

2000-2006年 Heart Failure Unit, Interventional Cardiology, Coronary Care Unit, Electrophysiology/Cardiac Pacing Laboratory, Echocardiogram Laboratory

Main Clinical Interest

2009年 Heart Failure Unit

5. Teaching Experience

2009年 厚生労働省認可 臨床研修医指導医
2010年 Tutor Education

6. Memberships

日本内科学会（認定医）
日本循環器学会（専門医）
日本心臓病学会
日本心不全学会
日本睡眠学会
日本老年医学会
American Heart Association
American Thoracic Society

7. Main Research Themes

Advanced Heart Failure
Right-sided Heart Failure
Sleep Apnea
Chronic Obstructive Pulmonary Disease
Pulmonary Hypertension
Non-invasive Positive Pressure Ventilation (CPAP/BiPAP/ASV)

8. Investigators of Multicenter study

1. Acute Decompensated Heart Failure Syndromes Registry (ATTEND)
2. Sleep and Cardiovascular Disease National Prospective Cohort Study (SCCS)
3. Multicenter Automatic Defibrillator Implantation Trial: Reduce Inappropriate Therapy (MADIT-RIT, as a research coordinator)
9. **External Reviewer for the following International Journals**
   1. American Journal of Respiratory and Critical Care Medicine
   2. Chest
   3. Circulation
   4. Journal of the American College of Cardiology
   5. American Journal of Cardiology
   6. Nature Clinical Practice Cardiovascular Medicine
   7. Journal of Computational Neuroscience
   8. American Journal of Hypertension
   9. Sleep and Biology Rhythm
   10. Journal of Cardiology

10. **List of Publications**

**Peer Reviewed Papers (published or in press)**


Curriculum Vitae

Jee Hyun Kim, MD, PhD

Personal Data:
Born: January 26, 1974, Seoul, Korea

Education:
1992 – 1998 Ewha Womans University School of Medicine
2000 – 2003 Ewha Womans University Graduate School, B.S
2003 – 2006 Ewha Womans University Graduate School, Ph.D.

Postgraduate Training:
1998 – 1999 Ewha Womans University Hospital Internship
1999 – 2003 Department of Neurology, Residency
Ewha Womans University Hospital, Seoul, Korea
2003 – 2005 Fellowship in Neurology, (Epilepsy and sleep disorder section)
Samsung Medical Center, Seoul, Korea
2006-2007 Postdoctoral fellowship in Stanford sleep clinic, Stanford University, CA, USA
2009.3 Cognitive behavioral therapy minifellowship in UPENN, PA, USA

Professional experiences:
2005 – 2006 Clinical instructor, Department of Neurology,
Ewha Womans University Hospital, Seoul, Korea
2007-2008 Clinical instructor, Department of Neurology,
Ewha Womans University Hospital, Seoul, Korea

Current Position: Assistant professor, Dept. of Neurology
Dankook University Hospital
Sungkyunkwan University College of Medicine
Cheonan, Korea
**Board Certification:**
1998       Korean Medical Board
2003       Korean Board of Neurology
2006       Certified International Sleep specialist Board (AASM)

**Professional Organization:**
Korean Neurological Association, member
American Association of Sleep Medicine, member
Secretary General, Korean Sleep research Society
Scientific committee, Korean Neurological Association
International Scientific Committee, Organization for 2012 Asian sleep research Society
Terry Bo-Jau Kuo, M.D., Ph.D. is Professor and Director of Institute of Brain Science, National Yang-Ming University, Taipei, Taiwan. His research interest ranges from medical engineering, cardiovascular physiology, neurophysiology, to sleep medicine. Prof. Kuo has also developed original techniques related brain science research including both hardware and software. Prof. Kuo has published more than 100 research articles, and patented more than 50 of his inventions. Prof. Kuo was the recipient of numerous awards in academic and research, including the 2004 Ten Outstanding Young Individuals in Taiwan. Prof. Kuo is also a key medical educator promoting clinician scientist training in Taiwan.
Hiroshi Hagiwara

1) a brief description of my study(ies)
   We are researching lifestyle design and successful aging focusing on biological signals such as human physiological function, physiological factor, and behavioral activity. In lifestyle design research, our aim is to propose new lifestyle habits by developing technologies in relation to sleep and alertness that are affected by physical stimulus from environment. In successful aging research, our aim is to improve and maintain flexible physical control capabilities by developing technologies in relation to training and relaxation of mind and body.

2) my brief CV.
   Hiroshi Hagiwara received the B.S. degree from Osaka University, Japan, in 1977 and the Ph.D. degree from Kansai Medical University, Japan, in 1997. He worked at Matsushita Electric Works Co.Ltd., and moved to Ritsumeikan University, Japan, where he is a professor in the Department of Human and Computer Intelligence in the College of Information Science and Engineering. His research interests include physiological engineering and ergonomics.
Curriculum Vitae

I-Mei Lin Ph.D.

• **Education**
  Ph.D. National Chung Cheng University, Department of Psychology

• **Academic position**
  Assistant Professor, Department of Psychology, Kaohsiung Medical University, Taiwan

• **Experience**
  Research Scholar  San Francisco State University (2006.08 - 2007.09)
  Biofeedback certified for Biofeedback Certification International Alliance (BCIA, BCB, 2010–present)
  Taiwan Clinical Psychologist

• **Research interest**
  1. Cardiac autonomic and emotion regulation
  2. Heart rate variability biofeedback in anxiety, depressive disorder, insomnia, and cardiovascular disease
  3. Neurofeedback in major depressive disorder
CURRICULUM VITAE

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EDUCATION

2008               Certified Doctor of the Japanese Society of Psychiatry and Neurology
                   (No. 06623)
2004               Certified Doctor of the Japanese Society for Morita therapy (No. 89)
2003               Certified Doctor of the Japanese Society for General Hospital Psychiatry
                   (No. 327)
2002               Certified Doctor of the Japanese Society of Sleep Research (No. 39)
1999               Certified Psychiatrist of Ministry of Health, Labor and Welfare (No. 11576)
1995               Ph.D., Psychiatry, Jikei University school of Medicine
1991-1995          The Jikei University School of Medicine, Post-graduate (Ph.D.) course
1988               Physician's License in Japan (No. 317647)
1988               M.D., Jikei University school of Medicine
1982-1988          The Jikei University School of Medicine, M.D course

PROFESSIONAL EXPERIENCE

2012-Present   Assistant Professor
               Department of Psychiatry, the Jikei University
               Katsushika Medical Center, Japan
2011-2012      Visiting Assistant Professor
               Stanford Sleep and Circadian Neurobiology Laboratory
               Department of Psychiatry and Behavioral Sciences
               Stanford University School of Medicine
2001-2011      Assistant Professor
               Department of Psychiatry,
               Jikei University School of Medicine, Tokyo, Japan
1990-2001  Clinical Associate  
Department of Psychiatry, 
Jikei University School of Medicine, Tokyo, Japan

1988-1990  Resident  
Department of Psychiatry, 
Jikei University School of Medicine, Tokyo, Japan

PROFESSIONAL SOCIETIES

The Japanese Society of Psychiatry and Neurology  
The Japanese Society of Psychosomatic Medicine  
The Japanese Society of Sleep Research  
The Japanese Society for General Hospital Psychiatry  
The Japanese Society for Morita therapy  
The Japan Association of Ambulatory Psychiatric Service
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ISA OKAJIMA, Ph.D.

PERSONAL INFORMATION
Name          Isa OKAJIMA
Date of Birth  April 28th, 1979
Place of Birth Tokyo, Japan
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Address (Institute)  Department of Somnology
                     Tokyo Medical University,
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EDUCATION
Mar. 2008  Doctor of Philosophy, Clinical Psychology
           Department of Psychological Science
           Health Sciences University of Hokkaido, Hokkaido, JAPAN
Mar. 2005  Master of Arts, Clinical Psychology,
           Health Sciences University of Hokkaido, Hokkaido, JAPAN
Mar. 2003  Bachelor of Arts, Psychology,
           Nihon University, Tokyo, JAPAN

CAREER HISTORY
2009-present  Assistant Professor
              Department of Somnology, Tokyo Medical University
2008-present  Research Worker
              Japan Somnology Center, Neuropsychiatric Research Institute
              Full-time Clinical Psychologist
              Medical Corporation for Yoyogi Sleep Disorder Center
2008-2011    Full-time Clinical Psychologist
              Yoyogi Sleep Clinic, Neuropsychiatric Research Institute
2005-2008    Research Assistant
              Department of Psychological Science, Health Sciences University of Hokkaido,
              Hokkaido, Japan
              Assistant for Experiment or Research, and Analysis of Experimental and Research
              Data
2003-2005    Teaching Assistant
              Department of Nursing and Social Services, Health Sciences University of
              Hokkaido, Hokkaido, Japan
Department of Nursing and Social Services, Health Sciences University of Hokkaido, Hokkaido, Japan

CERTIFICATION

Clinical Psychologist, No.21252
Certificate of the Japan Society of Certified Clinical Psychologist

Expert Behavior Therapist, No. 45
Certificate of the Japanese Association of Behavior Therapy

Industrial Counselor, No. 07010682
Certificate of the Japan Industrial Counselors Association

AWARDS

Sleep Research Award

Poster Prize

Poster Award

Stress Science Award

Uchiyama Memorial Award

GRANTS

2010-present Grant-in-Aid for Young scientists (B)
Japan Society for the Promotion of Science (JSPS), Tokyo, JAPAN.

2008-2009 Grant-in-Aid for Young scientists (Start-up)
Japan Society for the Promotion of Science (JSPS), Tokyo, JAPAN.

2007 Grant-in-Aid for JSPS Fellows
Japan Society for the Promotion of Science (JSPS), Tokyo, JAPAN.

PROFESSIONAL MEMBERSHIPS/AFFILIATIONS

Association for Behavioral and Cognitive Therapies
Japanese Association of Behavior Therapy
Japanese Association for Cognitive Therapy
Japanese Psychological Association
Japanese Society of Psychosomatic Medicine
Japanese Society of Sleep Research
The Japanese Society for Child and Adolescent Psychiatry
Curriculum Vitae

Chien-Ming Yang

National Chengchi University
Department of Psychology
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Wunshan District, Taipei City 116
Taiwan

Current Affiliation
Professor
National Chengchi University, Department of Psychology/The Research Center for Mind, Brain, and Learning

Visiting Scholar
University of California, Berkeley, Institute of East Asian Studies/Department of Psychology

Education
1999/02: Ph.D., Psychology
The City University of New York, Graduate School and University Center
Joint track of Clinical Psychology of Neurobehavioral Disorders

1998/02: Master of Philosophy, Psychology
The City University of New York, Graduate School and University Center

1989/06: B.S., Psychology
National Chengchi University, Taipei, Taiwan

Membership
American Psychological Association (1992-present)
American Academy of Sleep Medicine (1993-present)
Society for Psychophysiological Research (1996-2003, 2008-present)
International Organization of Psychophysiology (2007-present)
Taiwan Psychological Association (1989-present)
Taiwan Society of Sleep Medicine (2002-present)
Taiwan Association of Clinical Psychology (2002-present)
Taiwan Society of Sleep Medicine (2002-present)
Taiwan Association of Clinical Psychology (2002-present)

Professional Service
- Executive Board, Taiwan Association of Clinical Psychology (2005-2007, 2009/08-present)
- Executive Board, Taiwan Society of Sleep Medicine (2002/04-present)

Editorial Board
- Review of Psychology Frontier (2012.7--)
- Formosa Journal of Mental Health (2010.12--)
- The Journal of Kaohsiung Behavioral Sciences (2010--)

Honor/Award
- 2010/8-2013/7: Distinguished Professor, National Chengchi University
- 2010: National Chengchi University Awards for Outstanding Research
- 2007: National Chengchi University Awards for Research Excellence
- 2006: National Chengchi University Distinguished Teaching Award
- 2006: Ta-You Wu Memorial Award, National Science Council
- 1999: American Sleep Disorders Association Young Investigator Award: Honorable Mention
CURRICULUM VITAE

NAME
Yuichi Kamei (August 29, 1962.)

ADDRESS
Laboratory Medicine, National Center of Neurology and Psychiatry, National Center Hospital
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EDUCATION
1988 MD Yamanashi Medical University
1996 PhD Yamanashi Medical University

EMPLOYMENT
1988-1993 Yamanashi Medical University
1993-2008 National Center of Neurology and Psychiatry, Kohnodai Hospital
2008-2010 National Center for Global Health and Medicine, Kohnodai Hospital
2010- National Center of Neurology and Psychiatry, National Center Hospital
Quality is difficult to define. Yet, patients, healthcare professionals and administrative bodies have a reasonable expectation that quality is ensured. More often than not, the term “quality control” is used to describe the laboratories actions to ensure quality. Unfortunately, quality control is but one aspect of a structure to ensure quality in the laboratory. Under a Total Quality Management framework, a number of systems are essential to ensuring quality within the sleep laboratory. These systems are planning, processes, control, assessment and improvement. All of these systems are closely interlinked and require each to be functioning well in order to achieve quality. This session will examine the Total Quality Management framework in relation to a sleep laboratory. Particular attention will be paid to quality control and various actions that can be used in the pre-analytic, analytic and post-analytic areas in the laboratory. Recently, a standard specific for sleep disorders services has been developed from the ISO15189:2007 Medical laboratories - particular requirements for quality and competence document. This new interpretation of ISO15189 for sleep disorders services will be presented as a laboratory model for ensuring quality in this discipline.
Obstructive sleep apnea syndrome (OSAS) in children is not rare and is recognized as a major health problem. Currently, the primary treatment advocated for OSAS in children is surgical treatment of the airway. Unfortunately, surgery's success rate is relatively low because the obstruction site of the upper airway varies considerably.

Therefore, an examination extending from the nose to the hypopharynx is crucial to precisely locate the obstruction. This examination can be carried out using a variety of techniques, including nasopharyngoscopy, fluoroscopy, pressure measurements, CT and MRI.

But, these method are not always well correlated with either the respiratory disturbance or the location of the obstruction. For the aforementioned reasons, establishing a specific method to simultaneously evaluate the morphology and function of the whole upper airway is indispensable to successful treatment of OSAS in children.

To better evaluate the relationship between respiratory function and airway morphology, a 3-dimensional (3D) model of each subject's airway was constructed from computed tomography data and used to create computational fluid dynamics (CFD) models of respiratory status during quiet respiration. The results of CFD may be more reliable and can be evaluated more precisely than a morphological evaluation alone.

On the other hand, efficacy of some orthodontic treatment for OSAS of children is suggested. However, enough consensus for these treatment is not obtained because an evaluation of the airway ventilation condition was difficult. Therefore I evaluated efficacy of these treatment using CFD.

I describe clinical application to a diagnosis and treatment of OSAS of children using CFD.
NASAL BREATHING AND FACIAL GROWTH IN PEDIATRIC OSA IN JAPAN

Shintaro Chiba

Associate Professor, Department of Otorhinolaryngology, The Jikei University School of Medicine

Background: OSA is recognized as multifactorial disease. Especially, Adult OSA patients in Japan, small jaw is very important factor as same as obesity. But in children, it is recognized that adeno-tonsillar hypertrophy and obesity are the major risk factors, and an association between facial structure and OSA in children is unclear.

It is thought that Maxillo-facial growth depends on not only genetic factors but also environmental factors, for examples, eating habits, nasal obstruction and so on.

And we think one of the treatments for the retrognathia(maxillofacial growth abnormalities) would be adeno-tonsillectomy for nasal breathing disorder.

Objective: To evaluate maxilla-facial growth in children after adeno-tonsillectomy retrospectively.

Methods: 58 pediatics, 2002 to March, 2007, diagnosed as OSA by nPSG or Serous Otitis Media. Retrospectively we compare the facial growth between 1. OSA with Adeno-tonsillectomy group (N=21). 2. No OSA with Adeno-tonsillectomy group: habitual tonsillitis (N=29) and 3. OSA with no Adeno-tonsillectomy group (N=8 as controls), using cephalogram at 1st visit and 2 years later.

Results: 1. In OSA surgery group, SNA angle (from 80.8 to 84.0 p=0.008, Japanese Norm is 81.4 plus minus 3.3) increased after adeno-tonsillectomy. However in OSA non-surgery group and otitis media surgery group, they didn’t change significantly. 2. SNB angle (from 75.6 to 77.7 p=0.003, Japanese Norm is 76.2 plus minus 2.9) increased after adeno-tonsillectomy. However in OSA non-surgery group and otitis media surgery group, they didn’t change significantly. 3. There are similar results in Fx, Just in OSA with surgery group, There is significant increase after surgery.

Conclusion: Adeno-tonsillectomy was supposed to normalize maxillofacial growth in children with OSA. There is a possibility that the early intervention for the children with OSA can improve their maxillofacial growth and prevent to transmigrate to adult OSA. Now, We have to re-make the indication of Adeno-tonsillectomy from viewpoint of prevention of OSA.
CAN WE PREVENT OSA?

Makoto Kikuchi

Director, Orthodontics, Cosmos Center for Sleep Breathing Disorders

One of the causes of sleep apnea syndrome is known as the abnormal of craniofacial morphology. However how the abnormality of craniofacial morphology affects on the sleep apnea syndrome is unclear. Some cases i.e. Crouzon disease has been reported as sleep apnea caused by genetic abnormality, however which is resulted by premature fusion of coronal suture.

Patients we are treating is almost genetically normal, who are obese and/or craniofacial abnormal in general. I would like to discuss why these normal people become sleep apnea syndrome from the stand point of "Functional Matrix Hypothesis", which was originated by Prof. Melvin Moss back in 1950’s.

“Functional Matrix Hypothesis” could be explained as follows.
There are many relatively independent functions at craniofacial skeleton. Functions like visions, smell, listening, breath, conversation, mastication, deglutition, digestion need the proper soft tissues to accomplish the function, which need the proper skeletal elements to support and protect the soft tissues. Therefore soft tissue means not only muscles and tendons but also glands, nerves, vessels, teeth, and sinuses. One set of soft tissue and skeletal element relating one function is called functional cranial component. Total skeletal element is called skeletal unit and total soft tissue is called functional matrix. Each skeletal unit exists relatively independent, because the functions of the soft tissue are different. The generation, growth and maintenance of one skeletal unit depend upon the amount and quality of the functional matrix related to the skeletal unit. For instance, the primary growth-promoting potential to the skeletal unit is the functional matrix and the bone grows as a secondary reaction.

From the stand point of “Functional Matrix Hypothesis”, I would like to propose a hypothesis that sleep apnea during childhood makes the mandible small and positioned backward resulted in worsening sleep apnea and thoracic negative pressure affects the shape and form of the craniofacial skeleton. Finally we would like to discuss the possibility of how we could prevent the obstructive sleep apnea.
Most of the major psychiatric disorders, including all of the DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, 4th Edition) axis one disorders list dysregulations of sleep as one of their defining characteristics. Invariably, the assumption has been that the sleep disorder is a consequence of the psychiatric disorder. But a range of studies now raises the question of the extent to which independent sleep disorders can contribute to, or even cause, psychiatric illness.

Studies of children comorbid for sleep apnea and attention deficit-hyperactivity disorder (ADHD) indicate that correcting the apnea with adenotonsillectomy can reduce or eliminate the ADHD. Similar studies in adults with sleep apnea and major depression report that treatment of the apnea with CPAP (continuous positive airway pressure) can reduce or eliminate the depression.

Anecdotal reports provide striking evidence of sleep’s role in bipolar disorder as well. In these instances, sleep’s influence is not on the development or resolution of the disorder, but on the switching between states, with sleep deprivation triggering the onset of episodes of mania.

Most provocative is the hypothesis that post-traumatic stress disorder (PTSD) is a memory disorder, and, more specifically, a consequence of defective sleep-dependent memory processing. The hypothesis makes two major claims. First, it argues that PTSD develops when normal mechanisms of post-encoding memory processing fail to convert an isolated, emotionally charged, episodic memory of a traumatic event into an integrated, semanticized, and emotionally modulated narrative story of the trauma. Second, it proposes that the mechanisms that normally perform this processing of traumatic memories are largely sleep dependent, and that it is the failure of these sleep-dependent memory processes that finally leads to PTSD.

Recent studies demonstrating sleep’s role in the normal processing of emotional memories, as well as in the extraction of gist representations and “rules” from larger sets of memories, provide evidence for the proposed role of sleep in normal trauma processing, and comparisons of dream studies in normal subjects and patients with PTSD support the argument that the classic repetitive dreams of PTSD, which replay traumatic events in near-veridical form, are a reflection of this failed sleep-dependent memory processing.

Together, these wide ranging investigations suggest that much more attention should be paid to the roles of sleep in the etiology and treatment of psychiatric disorders.
PHENOTYPING IN THE PATHOGENESIS OF OBSTRUCTIVE SLEEP APNEA: IMPLICATIONS FOR FUTURE THERAPIES

David P. White, MD

Clinical Professor of Medicine, Harvard Medical University

Obstructive sleep apnea is a common disorder with important adverse consequences for afflicted individuals. A better understanding of apnea pathophysiology could lead to improved therapies with the potential to individualize treatment. Deficient pharyngeal anatomy is a common characteristic in patients with obstructive sleep apnea. However, measures of anatomy, generally Pcrit, predict little of the variability in apnea presence and severity. Thus other traits are important as well and likely include: 1. Upper airway muscle responsiveness and effectiveness during sleep: the upper airway response. 2. Arousal threshold to a respiratory stimulus. 3. Loop Gain, a measure of ventilator control stability or instability. We have developed a graphical, mathematical model that incorporates these four traits which can predict who does and does not have obstructive sleep apnea based on these physiologic characteristics. Manipulation of individual traits can affect apnea severity based on predictions from the model. Examples include decreasing loop gain with oxygen or acetazolamide, increasing arousal threshold with a hypnotic or changes in upper airway anatomy i.e. surgery or mandibular advancement. Although not fully tested, several other observations have resulted from the model: 1. REM predominant apnea occurs in individuals with reasonably poor anatomy, but a substantial upper airway response during NREM sleep. Loss or reduction in the upper airway response during REM sleep likely leads to airway collapse. 2. Some patients have a negative upper airway response i.e. increasing respiratory drives leads to decreased airway patency. Unless this trait can be altered, such patients will likely be difficult to treat with approaches other than CPAP or major manipulation of anatomy. By measuring these traits and utilizing the graphical model, apnea pathophysiology can be better understood and new therapeutic strategies designed.
NEURAL CORRELATES OF REDUCED PROCESSING CAPACITY IN SLEEP-DEPRIVED PERSONS

Wei Liang Michael Chee

Principal Investigator, Neuroscience & Behavioral Disorders Program, Duke-NUS Graduate Medical School Singapore

Being alert and quickly responsive to salient stimuli is something we take for granted when we are healthy and sleeping well. A sleep-deprived person is slower to respond, less capable of directing attention to a selected target and finds it more difficult to avoid being distracted. In addition, despite our best efforts to stay awake, we experience more frequent lapses of attention / microsleeps.

When sleep-deprived the rate at which we perceive visual stimuli is also affected. All these phenomena have neural correlates which we can demonstrate using fMRI.

Finally, persons vary how sleep deprivation affects them. The extent to which one can maintain activation of fronto-parietal brain regions during the sleep-deprived state may predict resistance to processing capacity declines.
DYSFUNCTION OF DOPAMINE AND SEROTONIN SYSTEM IN RLS

In-Young Yoon

Associate Professor, Department of Neuropsychiatry, Seoul National University Bundang Hospital

Background
Dopamine and serotonin dysregulation is suspected in restless legs syndrome (RLS), considering clinical experiences that dopaminergic drugs are effective in controlling RLS symptoms and selective serotonin reuptake inhibitors have been associated with the risk of RLS.

Methods
Drug-naïve elderly patients with RLS and normal controls were enrolled in these studies. RLS was diagnosed according to the criteria of the National Institute of Health, and its severity was evaluated using the International RLS Study Group (IRLSSG) Severity Scale. Dopamine transporter density (DAT) availability and D2 receptor density in the striatum were measured using \([^{123}\text{I}]2\beta\)-carbomethoxy-3\(\beta\)-(4-iodophenyl) tropane single-photon emission computed tomography (SPECT) and \([^{123}\text{I}]\)iodobenzamide SPECT. The availability of serotonin transporter (SERT) was assessed in the pons and medulla via \([^{123}\text{I}]\)-2\(\beta\)-carbomethoxy-3\(\beta\)-(4-iodophenyl) tropane (\(\beta\)-CIT) SPECT.

Results
Dopamine transporter density of patients with RLS was increased in the caudate (\(P = 0.037\)), posterior putamen (\(P = 0.041\)), and entire striatum (\(P = 0.046\)) compared with that of normal controls. The availability of SERT was similar in the RLS group and the control group, but IRLSSG Severity Scale scores increased with decrease of SERT availability in both the pons (\(\beta = -0.50, P = 0.009\)) and the medulla (\(\beta = -0.42, P = 0.03\)).

Conclusions
Dysregulation rather than simple upregulation or downregulation of central dopaminergic neurotransmission may underlie the pathogenesis of RLS, and decreased dopaminergic neurotransmission may cause moderate to moderately severe RLS in the elderly. An increase of serotonergic neurotransmission in the brainstem may exacerbate RLS, possibly via dual modulations on striatal dopaminergic neurotransmission and on the activities of spinal motorand sensory neurons.
Brain functions and performance vary across the day and deteriorate during sleep deprivation, but the nature of the contribution of circadian rhythmicity and sleep to different cognitive functions is unclear. To assess the effects of repeated partial sleep deprivation (PSD), acute total sleep deprivation (TSD), and circadian phase on performance across multiple cognitive domains, we conducted a 2 × 12-day cross-over study on 36 healthy individuals (18 males; mean ± SD of age = 27.6 ± 4.0 years). Repeated PSD was induced by restricting sleep opportunity to six hours per 24 hours for seven days, and this was followed by an acute TSD period in which participants stayed awake for approximately 40 hours. In the Control condition, participants were given a 10-h sleep opportunity for seven days before TSD. To assess genetically determined differences in response to sleep loss across cognitive domains, the sample was stratified for the rs57875989 polymorphism in the coding-region of PER3, a member of the PERIOD gene family, which plays a role in circadian rhythmicity and confers cognitive susceptibility to TSD.

Throughout the protocol, performance in seven cognitive domains was assessed by a 40-min test battery. Here, we focus on three domains: (1) Self-reported Alertness as indicated by the reverse of the Karolinska Sleepiness Scale score, (2) Sustained Attention as indicated by the performance in the Psychomotor Vigilance Task and Sustained Attention to Response Task, and (3) Working Memory and Executive Functions as measured by the Verbal 1-, 2-, and 3-Back Tasks.

Repeated PSD resulted in significant deterioration in performance in all cognitive domains, but surprisingly, the effect size (Cohen’s $f^2$) was large for Alertness and medium for Sustained Attention, but was only small for Working Memory and Executive Functions. The effects of acute TSD were large on all domains, but interestingly, in the same order as the effects of PSD.
Importantly, the effects of PSD carried over to the subsequent acute TSD period and were modulated by circadian phase such that the largest effects were observed in the morning hours during the initial part of the TSD period, then became progressively smaller to nearly absent in the wake maintenance zone just prior to the onset of melatonin secretion, and then increased again during the circadian night. Finally, we observed that genotype-dependent differences were not largest for those domains most affected by sleep deprivation, i.e. Alertness and Sustained Attention, but instead, were most pronounced for the Working Memory task with the highest load on Executive Functions, i.e. Verbal 3-Back, and particularly so in the circadian morning after a night of TSD.

These findings challenge the notion that cognitive functions supported by the prefrontal cortex, such as Executive Functions, are most vulnerable to sleep deprivation, but support models of cognitive control in which Sustained Attention and Executive Functions can deteriorate independently of each other. These findings have implications for understanding how sleep debt and circadian rhythmicity interact to determine waking performance across cognitive domains and individuals.
THE INCIDENCE AND RESPIRATORY PATTERN IN PEDIATRIC SLEEP APNEA

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Introduction:
Sleep disordered breathing (SDB) occurs in children of all ages, from neonates to adolescents. It is thought to be most common in children around school period when the tonsils and adenoids are physiologically enlarged in relation to the growing airway size. Based on some recent reports about epidemiology of pediatric SDB, the International Classification of Sleep Disorders, Second Edition (ICSD-2) chose that the prevalence of obstructive sleep apnea (OSA) as approximately 2 percent in otherwise-normal young children. However, each previous reports adopted different diagnostic criteria, age of the subjects and testing approaches, and there are marked variability in prevalence of pediatric OSA. When sleep test were applied to all the participants with respiratory events and were scored according to the American Academy of Sleep Medicine (AASM) scoring rule (2007) and the ICSD-2 diagnostic criteria of pediatric OSA (obstructive apnea-hypopnea index: OAHI>=1), it is anticipated that the prevalence of children around school period might be well above 2%. Regarding Asian countries, epidemiological studies on pediatric SDB have been performed in some countries, but not in Japan.

Methods:
We investigated the prevalence of SDB in Japanese kindergartners (3-6 years) and children in the lower grades of elementary school (6-8 years). All 261 participants’ caretakers completed questionnaires (Child Sleep Checklist and OSA-18). An otolaryngologist examined children with regard to the presence or absence of nasal diseases and tonsillar hypertrophy. Nasal resistance was measured by rhinomanometry employing the active anterior method. All children underwent overnight in-home respiratory recordings of airflow, respiratory effort, and oximetry. Respiratory events were scored according to the AASM scoring rules (2007). In addition kindergartners had an X-ray to evaluate adenoid hypertrophy.

Results:
The mean of total AHI and OAHI were 1.9±2.7/1.4±1.3 and 0.8±2.2/0.4±0.6 in 3-6 years/6-8 years children. And the estimated prevalence of SDB (OAHI>=1) were 14.6% and 9.4%. Central apnea (CA) accounted for 67.7% among all respiratory events. Between the SDB group and normal group, significant differences were found in frequency of habitual snoring and degree of adenoid and tonsillar hypertrophy.

Discussion:
The prevalence of SDB in Japanese kindergartners and lower graders of elementary school were considerably high by according to the ICSD diagnostic criteria and the AASM scoring rules. Central apneas are frequently found even in healthy children in this study. It may be necessary to make a clear diagnosis between obstructive and central apnea for pediatric SDB screening.
CARDIOVASCULAR COMPLICATIONS IN CHILDREN WITH SLEEP-DISORDERED BREATHING DISORDER

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Sleep-disordered breathing disorder is a spectrum of conditions ranging from simple primary snoring, upper airway resistance syndrome to the more severe end of obstructive sleep apnoea (OSA). Childhood OSA is a common condition, and along with the global obesity epidemic its prevalence rate will likely inflate further. It is well established that if OSA remains untreated a variety of metabolic, cardiovascular and neurocognitive deficits will result. In this talk, the presenter will concentrate on the cardiovascular aspects of OSA, and share with you research work carried out examining blood pressure, ventricular function and endothelial function in children with OSA. There is also emerging evidence to suggest baseline OSA severity correlates with blood pressure levels at 4-year follow-up, reinforcing the importance to diagnose the condition promptly. Early and effective treatment is able to reverse a great proportion of these potential complications.

As much attention is paid to OSA, further studies are also necessary to delineate the clinical importance of primary snoring, which has been placed in the milder end of the sleep-disordered breathing spectrum. In our current clinical practice, in the absence of any obvious anatomical abnormalities in the upper airways, snoring in children is not an indication for treatment in majority of the cases. However, there is literature support that snoring on its own is not as benign as once thought. Primary snoring has been found to lead to reduced intelligence quotient, poor attention, memory, language skills and emotional liability. The presenter’s research group has also documented elevated nighttime blood pressure in children with primary snoring compared to normal controls. In part of this talk, further evidence of cardiovascular complication associated with primary snoring will be presented. If further evidence confirms the occurrence of significant morbidity in children with primary snoring, then finding appropriate therapeutic regimens will be our next research goal.
CURRENT HYPOPNEA SCORING CRITERIA UNDERSCORE PEDIATRIC SLEEP DISORDERED BREATHING

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Objectives To compare the results of scoring polysomnograms using AASM criteria versus using the Stanford scoring criteria in pediatric patients. Methods Polysomnograms (PSGs) were performed on children who were referred for evaluation of suspected sleep disordered breathing (SDB) over a one year period. The polysomnograms were blindly scored using both AASM and Stanford scoring criteria. The Stanford scoring criteria was previously developed using esophageal manometry readings. The diagnosis of SDB was based on the Stanford criteria. Ninety-nine patients underwent SDB post treatment follow-up evaluations during the study period and had repeat polysomnograms which were also scored in a blinded fashion. Results During the time allocated, 209 (83 girls) subjects with suspected SDB underwent clinical evaluation and polysomnograms which were blindly scored. Two-hundred-seven of the studies were considered positive for SDB based on Stanford criteria versus 39 studies based on AASM criteria. Scatterplot analyses showed that the AASM apnea hypopnea index (AHI) was skewed and significantly lower compared to the Stanford AHI. Ninety-nine children (9 with SDB diagnosed by AASM scoring criteria) had follow up clinical evaluation and PSGs after SDB treatment. All of these children showed improvement in their clinical presentation, the Stanford AHI and oxygen saturation percentage during sleep. Of note, 91% of these appropriately treated children would not have been identified as having SDB if AASM criteria had been used. When comparing the clinical information to PSG scoring, there was a positive correlation between SDB related clinical symptoms and anatomic risk factors for SDB independently of the scoring rules. Conclusion 207/209 subjects in this study presented with clinical symptoms and anatomic risk factors that are suggestive of abnormal breathing during sleep. The AASM scoring criteria recognized 12.7% of these subjects, while the Stanford criteria recognized the presence of abnormal breathing in all of the 207 children. Referring physicians treated the 207 children for SBD. Available follow-up clinical evaluation and PSG data (n=99) showed that all subjects had polysomnographic and clinical improvement post treatment. Ninety-one percent of children would not have been diagnosed nor treated for SDB if AASM scoring criteria had been used. When comparing the AASM criteria to the Stanford criteria, differences between hypopnea scoring is the main factor. The AASM hypopnea definition requiring a 30% nasal cannula amplitude drop plus a 4% oxygen desaturation, or 50% amplitude drop and 3% oxygen desaturation or 3 second EEG arousal, may be detrimental to the recognition of SDB in children.
CONGENITAL CENTRAL HYPOVENTILATION SYNDROME: THAILAND EXPERIENCE

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Congenital central hypoventilation syndrome (CCHS) is a rare sleep disordered breathing characterized by alveolar hypoventilation and autonomic dysregulation. A PHOX2B mutation is required to confirm the diagnosis. Four cases of CCHS with PHOX2B mutations have been diagnosed and received treatment in Thailand. Male:female was 2:2. Three patients were noticed to develop cyanosis in the newborn period. Associated findings included pulmonary hypertension (3 patients), seizure (2 patients), dysphagia (2 patients). None had Hirschsprung nor neural crest tumor.

Polysomnography performed in all patients showed significant oxygen and carbon dioxide abnormalities markedly in nonREM sleep. The age at diagnosis was ranged 3 to 17 months. Three patients received invasive nighttime ventilations via tracheostomies. One patient whose parents refused tracheostomy received noninvasive nighttime ventilation via a full face mask. Three patients survive with a good quality of life. The oldest patient who participates in normal school and family activities is currently 7 years old.
SYMPTOMATIC NARCOLEPSY AND HYPERSOMNIA, HYPOCRETIN/OREXIN INVOLVEMENTS


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Narcolepsy is a chronic sleep disorder characterized by excessive daytime sleepiness (EDS), cataplexy, hypnagogic hallucinations and sleep paralysis (i.e. narcolepsy tetrad). A major breakthrough in narcolepsy research was recently made through the identification of orexin (hypocretin) deficiency in narcolepsy-cataplexy. Orexins are hypothalamic neuropeptides involved in various fundamental hypothalamic functions including, sleep-wake control, energy homeostasis, autonomic and neuroendocrine functions. Orexin containing neurons are located exclusively in the lateral hypothalamic area (LHA). Since orexin deficiency in narcolepsy is also tightly associated with human leukocyte antigen (HLA)DR2/DQ6(DQB1*0602) positivity, an acquired cell loss of orexin containing neurons with autoimmune process is suggested in “so-called” idiopathic cases of narcolepsy. “Idiopathic narcolepsy” has been used for the cases with narcolepsy unassociated with apparent radiographical or clinical evidence of brain pathology apart from sleep-related abnormalities. The symptoms of narcolepsy can occur during the course of other neurological conditions (i.e. symptomatic narcolepsy). Inherited disorders, tumors and head trauma were the three most frequent causes for symptomatic narcolepsy. Other causes include multiple sclerosis (MS), vascular disorders and encephalitis. Cerebrospinal fluid (CSF) Orexin-A (hypocretin-1) measures were carried out in some recent cases with symptomatic narcolepsy and moderate decreases in orexin levels were seen in a large majority of these cases. Excessive daytime sleepiness (EDS) in these symptomatic cases was sometimes reversible with an improvement of the causative neurological disorder and with an improvement of the orexin status. Recently, we found that several symptomatic narcoleptic cases with MS show unique bilateral symmetric hypothalamic lesions associated with significant orexin ligand deficiency. In addition, these patients often share the clinical characteristics of neuromyelitis optica (NMO) and the detection of NMO-IgG (or anti aquaporin-4 (AQP4) antibodies), suggesting a new clinical entity. Further studies of the involvement of the orexin system in symptomatic narcolepsy and EDS are helpful to understand the pathophysiological mechanisms for occurrence of EDS and cataplexy.
QUESTIONS POST HYPOCRETIN / OREXIN DISCOVERY

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The discovery of hypocretin/orexin as an arousal peptide imply a number of questions in the pathophysiology of hypersomnia occurring in various diseases:

1. Is hypocretin neurotransmission system impaired in the excessive sleepiness encountered in: Brain damage, epilepsy, Alzheimer's disease, Parkinson's disease, depression, neurodegenerative conditions, Celiac Disease, Uremia, Mononucleosis, Influenza, peripheral neuropathy, neuromuscular disorders, chronic pain syndromes, RLS and PLM, Kleine Levin Syndrome, sleep-related breathing disorders (SRBD), gastro-esophageal reflux, Fibromyalgia, Hypothyroidism and Overweight.

2. Strokes, tumors, cysts, abscesses, hematomas, vascular malformations and multiple sclerosis plaques of the following brain structures: hypothalamus. brainstem reticular activating system, locus ceruleus, dorsal raphe, basal forebrain, thalamus and cortex produce excessive daytime sleepiness. Is hypocretn system involved in all these areas?

3. Many biochemical agents have an interactive awakening effect, such as: catecholamines, acetylcholine, histamine metabotropic glutamate receptor agonists, adenosine receptor antagonists, CRH, TRH, neuropeptide Y and ghrelin, leptin, and glucose. How dor these systems work with the hypocretin system and cordinate wakefulness?
CARDIOPULMONARY COUPLING AS A SLEEP QUALITY METRICS

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Numerous exogenous and endogenous factors influence whether sleep or wake is achieved, for how long it is maintained, and for what reason it is interrupted or terminated [1-3]. Improved clinical phenotyping of sleep or sleep disorders is a key to improve diagnosis, prognosis, and treatment approaches. Although the visual scoring of EEG from polysomnography is a standard method for evaluating sleep quality, it is not readily available, resource-dependent, and inadequate for long term monitoring. Furthermore, clinical correlations between daytime complaints and polysomnographic metrics of disease severity are not always straightforward, due in part to trait-like inter-subject variability, largely subjective complaints, and variable intrinsic tolerance to sleep disruption. Complementing standard metrics with novel sleep architecture models may greatly improve diagnosis (disease sub-typing and individualized severity metrics) and treatment (objective outcome measures and treatment monitoring techniques) of sleep disorders. Given the patient-by-patient variability and complexity of sleep, and its bi-directional interactions with virtually every organ system, there is an urging need for developing novel, quantitative, and personalized metrics of sleep architecture, with extension to long-term ambulatory monitoring. One key example of such a computational advancement is the electrocardiogram (ECG)-based cardiopulmonary coupling (CPC) analysis [4,5].

CPC analysis measures employs Fourier-based methods to analyze the degree of coupling between two physiologic streams, heart rate variability (HRV) and respiratory tidal volume modulation of R-wave amplitude (ECG-derived respiration, EDR). It is an EEG-independent, readily repeatable (ECG recording alone), automated and scorer-independent. Based on the physiology that the higher level of coupling between autonomic (HRV) and respiratory stream (EDR) is achieved during stable sleep, CPC analysis puts out the relative amount of stable and unstable non-rapid eye movement (NREM) sleep. High frequency coupling (HFC) is the marker of stable sleep, and low frequency coupling (LFC) that of unstable sleep. We have previously demonstrated that HFC and LFC more closely correlates with CAP rather than conventional EEG sleep stages [5]. A propensity of sleep instability measured by CPC has been demonstrated in fibromyalgia, depression and chronic nocturnal hypoxia [6-8]. Pediatric OSA is associated with reduced HFC that is negatively correlated with respiratory disturbance index [9,10].

In conclusions, current findings support a potential role of CPC analysis to determine sleep
quality. CPC analysis could be a convenient, cost-effective, non-invasive biomarker that can measure the sleep quality with a possible extension to long term monitoring.

References


An important development in recent years has been, understanding the importance of sleep in health and diseases. Adequate amount of sleep is key to our well being and for prevention of many lifestyle diseases. We bring forward a fine homeostatic regulation between sleep and temperature for better sleep.

Regulation of body temperature and sleep are two important physiological mechanisms. Interestingly both these functions are controlled by some common basal forebrain structures which include the preoptic area and the medial septum commonly referred as preoptic -basal forebrain areas. Lesion of medial, lateral and ventrolateral preoptic areas, median preoptic nucleus and medial septum decrease sleep. On the other hand body temperature increases by lesion of medial and lateral preoptic areas and reduces by medial septal lesions. Local warming of preoptic area increases sleep, whereas cooling increases wakefulness. Low and high ambient temperature disturb sleep.

Given a choice, rats prefer to stay at ambient temperature of 27 °C. Increasing the ambient temperature from cold to warm within a range of 18-34 °C, linearly increases sleep and body temperature in rats and sleep is maximum at 30 °C. This ambient temperature related increase in sleep particularly paradoxical sleep is not observed after specific destruction of warm sensitive neurons of the preoptic area. These lines of evidence bring into focus integration of sleep and thermoregulatory mechanisms by preoptic-basal forebrain areas. Thus the interplay between sleep and thermoregulatory processes within the preoptic-basal forebrain areas bring about a fine homeostatic control of two vital physiological mechanisms.
MEDITATION AND SLEEP: AN EVALUATION OF THE ROLE OF VIPASSANA MEDITATION IN PROMOTING SLEEP.

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Meditative practices have been shown to integrate brain functions and thereby help to attain harmony between body and mind. Vipassana meditation is one of India’s ancient meditative techniques rediscovered by Sri Buddha almost 2500 years ago. Vipassana meditation involves the strategy of mindfulness to achieve the absorption of the mind and the meditators learn to notice and witness the perceptions of the senses and the thoughts arising in the mind without reacting to them and can focus the attention on bodily activities in their true perspective, in their true nature. Mindfulness meditative techniques influence the brain functions in different ways and at different levels. We have observed enhanced alpha coherence and P300 amplitude in vipassana meditators. Similarly, the sleep architecture and EEG power of various frequency bands associated with different sleep states were also different in the practitioners of vipassana meditation when compared to non meditators. The senior practitioners of vipassana meditation (with more than five years of meditative practices) showed enhanced slow wave sleep, REM sleep states and more number of sleep cycles. Additionally we have observed a stable sleep organization, among elderly meditators (between 50-60 years of age) when compared to age matched non meditator subjects. We have also observed a sleep state dependent autonomic modulation (necessary to maintain sleep organization) among the senior practitioners of vipassana meditation. Similarly the vipassana meditators showed almost three fold increase in melatonin levels which is reported to enhance the sleep quality and structure.

A detailed study of REM sleep characteristics among senior practitioners of vipassana meditation have shown significant enhancement in total REM episodes, REM density, phasic and tonic REM events, number of REM bursts and burst duration, enhanced theta alpha power during REM sleep etc. These are suggestive of meditation influence on various neural plasticity events, associated with heightened cortical activity leading to heightened orientation and inner alertness, enhanced dream recall or dreaming, higher states of consciousness etc. The study highlights the importance of meditative techniques
such as vipassana meditation in the maintenance of sleep organization as a proper mean to attain enhanced physical health, mental performance & state of well being.
EXERCISE EFFECTS ON SLEEP PHYSIOLOGY

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In this talk, I will review past studies on “exercise effects on sleep”. I will also review exercise effect on somatic physiology, which potentially affect sleep quality. Then, we will discuss effects of chronic exercise on somatic physiology and how these changes affect sleep quality.

In its early days, sleep research largely focused on central nervous system (CNS) physiology using standardized tabulations of several sleep-specific landmark electroencephalogram (EEG) waveforms. This method has enabled the observation and inspection of numerous uninterrupted sleep phenomena. Research on the effects of exercise on sleep began, in the 1960’s, with a focus primarily on sleep related EEG changes (CNS sleep). Those early studies only found small effects of exercise on sleep. However, more recent sleep research has explored not only CNS functioning, but somatic physiology as well. Sleep should be affected by daytime exercise, as physical activity alters circadian pacemaker, endocrine, autonomic nervous system (ANS) and other somatic functions. Since endocrinological, metabolic and autonomic changes can be measured during sleep, it should be possible to assess exercise effects on somatic physiology in addition to CNS sleep quality, evaluated by standard polysomnographic (PSG) techniques. Additional measures of somatic physiology have provided enough evidence to conclude that the auto-regulatory, global regulation of sleep is not the exclusive domain of the CNS, but is heavily influenced by inputs from the rest of the body.
ROLE OF SLEEP IN ORGANIZING CENTRAL NETWORKS FOR SEXUAL FUNCTIONS

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Sleep is a complex phenomenon involving dynamic interaction of several neural networks in various areas of the brain. Acute sleep deprivations stimulate male sexual behaviour, and slow wave sleep is increased after the sexual activity in animal models. In healthy males, penile erections is an important component of rapid eye movement (REM) sleep during entire life both pre- and post-partum. A prominent expression of REM sleep during early development signifies its importance in networks formation involved in several physiological functions including sexual functions. In CNS, areas like the preoptic area, septum, amygdala, temporal lobe etc. contribute to information processing for sleep and sexual behaviour. Hypersexuality is also encountered in subjects with temporal lobe epilepsy, and in Kluver-bucy syndrome in monkeys. In various sleep related disorders, like Kleine-lewin syndrome hyper-sexuality is observed. Atypical sexual behaviour (sexsomnia) is noted during sleep in confusional arousal parasomnias. On the other hand, a decreased libido is common in narcoleptic patients. Local microinjection of orexin-A at the preoptic area activate sexual behaviours in animal model. In the current decade, wherein sleep disturbances are more frequently encountered, the function and regulation of sleep is portrayed in a novel prospective. In this scenario, it is challenging to understand the mechanism for functional integration in the in-vivo systems and to examine the neuronal networking involved in central processing and control of these functions.
DISSECTING THE BASAL GANGLIA CONTROL OF SLEEP-WAKE BEHAVIOR

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Researchers recently have made substantial progress toward understanding the roles of dopamine and the basal ganglia (BG) in the control of sleep-wake behavior. In this talk, I will outline recent advancements regarding BG and dopaminergic modulation of sleep through the BG and extra-BG sites. Our main hypothesis is that dopamine promotes sleep by its action on the D2 receptors in the striatum. This hypothesis implicates dopamine depletion in the BG (such as in Parkinson's disease) in causing frequent nighttime arousal and overall insomnia. Furthermore, the arousal effects of stimulants (methamphetamine and cocaine) may be linked to the ventral periaqueductal gray (vPAG) dopaminergic circuitry targeting the extra-BG sleep-wake network.
ROLE OF ADENOSINE A_{2A} RECEPTORS IN THE NUCLEUS ACCUMBENS FOR SLEEP-WAKE REGULATION

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Adenosine promotes sleep through the activation of A_{2A} receptors. A_{2A} receptors are densely expressed on striatopallidal neurons of the basal ganglia, where dopamine D_{2} receptors are co-expressed with A_{2A} receptors and involved in motor function, habit formation, and reward/addictive behaviors, all activities that require wakefulness. Abilities to maintain arousal are compromised under low dopamine conditions such as Parkinson’s disease, but the extent to which A_{2A} receptors in the basal ganglia contribute to the regulation of sleep and wakefulness is not known. We investigated the role of A_{2A} receptors in the basal ganglia for wakeful consciousness by using powerful tools for site-specific gene manipulations, including conditional A_{2A} receptor knockout mice based on the Cre/lox technology; focal A_{2A} receptor knockdown in rats through the local infection with adeno-associated virus carrying short-hairpin RNA of A_{2A} receptors; and modulation of neuronal activity through in-vivo stimulation and inhibition with genetically engineered receptor-channel systems, e.g., designer receptors exclusively activated by a designer drug (DREADD). Our studies have revealed that the arousal effect of caffeine critically depends on A_{2A} receptors on neurons in the shell of the nucleus accumbens and that transient activation of these neurons promotes sleep. These observations imply that A_{2A} receptors in the nucleus accumbens are key structural elements for the control of sleep and wakefulness. The ventral striatum has the unique capability to integrate behavioral functions and thus, it is an ideal site where sleep and wakefulness are regulated by processes that require consciousness. We believe that motivation may be considered as an important fundamental principle by which sleep and waking are regulated (Lazarus, M., et al., Trends Neurosci., doi: 10.1016/j.tins.2012.07.001)
KEY ROLES OF DOPAMINE D₂ RECEPTOR IN THE SLEEP-WAKE REGULATION

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Dopamine (DA) has been reported to regulate processes responsible for the generation of complex movements and emotions, cognition, reward processing, and drug addiction. However, the role assigned to DA in sleep–wake regulation has been relatively limited. The main DA receptors (R) in the brain are D₁R and D₂R. To clarify the roles of DA receptors in the sleep–wake regulation, D₂R knock-out (KO) mice and pharmacological manipulation were used. We found that the D₂R blockade could reduce the sleep latency and increase amounts of non-rapid eye movement (non-REM, NREM) sleep. Modafinil is the most potent wake-promoting medicine for enhancing the extracellular DA level in the nucleus accumbens and the prefrontal cortex, and for increasing wakefulness. By using D₂R KO mice and D₁R antagonist, we demonstrated that D₁R and D₂R are essential for the arousal effect of modafinil, with D₂R being the receptor of primary importance. Compared with wild-type (WT) mice, D₂R KO mice exhibited a significant decrease in wakefulness, with a concomitant increase in NREM and REM sleep, especially during the first 4 h after lights off. When the KO mice were subjected to a cage change, the latency to sleep in the KO mice decreased to half of the level for WT mice. The D₂R antagonist raclopride mimicked these effects in WT mice. When GBR12909, a DA transport inhibitor, was administered intraperitoneally, it induced wakefulness in WT mice, but its arousal effect was attenuated to one-third of the D₂R KO mice. These results indicate that D₂R plays an essential role in the maintenance of wakefulness. Recently, we are employing focal RNA interference in WT mice and rescue technology for D₂R in the KO mice to clarify the key regions expressing D₂R in the brain for arousal control.
THE IMPLICATION OF NIGROSTRIATAL DOPAMINERGIC SYSTEM IN RBD

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Background
REM sleep behavior disorder (RBD) is frequently associated with Parkinson’s disease (PD), but other findings, such as effectiveness of clonazepam without influence on the dopaminergic transmission and little treatment effect of dopaminergic agonists, suggest that dopaminergic system may not play a key role in the pathogenesis of RBD.

Methods
Patients with RBD, patients with PD and normal controls were enrolled in the study. RBD was diagnosed based on the minimal diagnostic criteria of the International Classification of Sleep Disorders. All the participants performed single-photon emission computed tomography imaging 3 h after injection of $[^{123}]$FP-CIT for dopamine transporter (DAT) analysis. During REM sleep of RBD patients, tonic and phasic EMG activities were measured on the polysomnography (PSG), and we evaluated correlations between tonic and phasic EMG activities and regional DAT densities in the caudate, putamen and striatum.

Results
Compared with the PD group, the RBD group had significantly higher DAT binding in the whole striatum and in each subregion. The RBD patients showed a trend of lower binding in the striatum than the normal controls ($P = 0.07$), and the significance was revealed in the putamen ($P = 0.02$). However, in 11 individual cases of the 14 RBD patients, DAT densities in the putamen still remained within the normal range. There was no correlation between EMG activities and DAT densities in the RBD patients.

Conclusions
Only a small proportion of RBD patients showed the reduction of dopaminergic projects in the striatum that has been a universal finding in PD and other synucleinopathies. Furthermore, there was no correlation between muscle activities during REM sleep and striatal DAT densities in RBD patients. This data may suggest that neuronal degeneration of the nigrostriatal dopaminergic pathway could be involved, but is not essential for the development of RBD.
DREAM-ENACTING BEHAVIOR IS ASSOCIATED WITH IMPAIRED SLEEP, SEVERE HEADACHE-RELATED DISABILITY AND DEPRESSIVE SYMPTOMS IN MIGRAINE PATIENTS

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Background: Sleep disturbances and daytime sleepiness are more common in migraine sufferers than in headache-free individuals. In addition, the frequent occurrence of migraine attacks during rapid eye movement (REM) sleep, nightmares and visual hallucinations have been reported in patients with migraine. Several characteristic recurrent dream patterns often occur in patients with migraine, and they may be used as a diagnostic aid for migraine. These findings suggest the involvement of REM sleep regulation in migraine. REM sleep behavior disorder (RBD) is a parasomnia characterized by loss of normal muscle atonia and dream-enacting behaviors (DEB) during REM sleep, often leading to injury to the patient or their bed partner. RBD predominantly affects males over 50 years of age in association with neurodegenerative diseases. In contrast, early onset cases (under 50 years of age) are associated with antidepressants, narcolepsy and brain lesions affect REM sleep regulation. However, the link between migraine and REM sleep abnormality or RBD has yet to be clarified.

Objective and Methods: To investigate the frequency and clinical correlates of DEB in migraine patients, we assessed patients with migraine (n = 212, mean age 33.8 years) and headache-free control subjects (n=140, mean age 33.1 years) who were under 50 years of age in a cross-sectional, case-control study between June and November 2010 at the Department of Neurology, Dokkyo Medical University Hospital. We only analysed migraine patients and controls under 50 years old in this study because the characteristics of early onset RBD are still unclear, and the enrolled patients with migraine were relatively young. The Japanese version of the RBD screening questionnaire was used, and subjects who scored 5 or greater were defined as having DEB. All patients completed the Migraine Disability Assessment (MIDAS) questionnaire, the Beck Depression Inventory (BDI)-II, the Pittsburgh Sleep Quality Index (PSQI) and the Epworth Sleepiness Scale (ESS).

Results: A significantly increased frequency of DEB was found in migraine patients compared with controls (25.0 % vs. 14.3 %). Migraine patients with DEB showed higher MIDAS, BDI-II, PSQI and ESS scores than those without DEB. Apart from
minor tranquilizers, there was no significant difference in the medication use rate between migraine patients with and without DEB. Caffeine intake was less frequent in migraine patients with DEB than those without, but the average daily caffeine intake (cup/day) was similar. Migraine patients with DEB had an increased rate of current and past smoking compared with those without. Dose-dependent effects of smoking calculated by the Brinkman index did not differ between the groups.

Conclusion: Our study demonstrates an increased frequency of DEB in migraine patients, which was the significant predictor of headache-related disability, depressive symptoms and poor sleep quality. DEB may reflect increased brain excitability in migraine due to brainstem involvement.
CLINICAL SIGNIFICANCE OF REM SLEEP BEHAVIOR DISORDER PARKINSON’S DISEASE

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REM sleep behavior disorder (RBD) has been widely known as a prodrome of alpha-synucleinopathy including Parkinson’s disease (PD) and dementia with Lewy bodies (DLB). Meanwhile, RBD is well known to appear after the onset of motor symptoms in PD. Undoubtedly, development of dementia is the most critical problem in the disease course of PD. One longitudinal study showed that the cumulative incidence of dementia steadily increased with advancing age and/or prolongation of PD morbidity. In that study, 27.0% of the PD patients were diagnosed as having dementia at baseline and the rate increased to 45.0%, 56.0%, and 60.0% after 4, 8, and 12 years, respectively. Of note, recent studies have suggested a possibility that RBD symptom in PD becomes a risk factor for the development of dementia in PD (PDD). With this regard, we previously reported that the rate of the number of the ones showing stage REM without atonia (RWA), a physiological basis of the appearance of RBD, is significantly higher in PD patients with nocturnal visual hallucination, which is one of important prodromes of PDD, than those without. As for autonomic function, cardiac meta-iodobenzylguanidine (MIBG) uptake manifesting cardio-myosympathetic function in PD patients with clinical RBD and PD patients with dementia (PDD) is comparably lower compared to PD patients without either RBD or dementia.

Inspired by these findings, we made a 21.4±10.8 months of longitudinal follow-up survey focusing on the development of dementia among patients with clinical RBD (n=27), with REM sleep without atonia (RWA) but without having RBD symptoms (subclinical RBD; n=23), and with normal REM sleep (n=27). Mean age, gender distribution, MMSE score at the baseline did not differ among the three groups.

Kaplan Myer curves indicated that the occurrence of dementia in the PD group with clinical RBD was significantly faster than that in the PD group with normal REM sleep (p = 0.013). A Cox hazard regression analysis revealed that development to PD with dementia was significantly associated with the presence of RBD (hazard ratio: 14.1, p = 0.017) but not with subclinical RBD, and none of the other PD related variables were not associated with the development of PDD.

These findings suggest that prodromal symptoms of PDD are present even in RBD affected PD patients without dementia. In addition, clinical RBD symptoms but not subclinical RBD is thought to be associated with the development of dementia in PD.
MULTILEVEL SURGERY FOR SDB

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SDB is one of a common health problem affecting many millions people. It seems like a disease of severity in continuum, simple snorer, UARS, and sleep apnea. Simple snorer usually lead to social embarrassment and sleep quality impairment of bed partner. For UARS, patient usually lack of energy in daytime. Regarding OSA, it depend on severity of disease. The moderate and severe OSA patient frequently complaint of excessive daytime sleepiness, impaired performance, disturbed cognition and emotion. In long-term consequence, it increase cardiovascular events and disturbed neurobehavioral activity. The most common site of obstruction are located in pharynx (nasopharynx, oro-pharynx and/or hypopharynx). The goal of surgery for simple snorer or mild OSA is to decrease snoring sound for bed partner, so the treatment modality should be less invasive. In contrary, the goal of surgery for moderate to severe OSA as alternative method for CPAP has to has highly effective enough to abate short-term consequence as neurobehavioral event and to prevent long-term consequence as cardiovascular events.

Multilevel surgery is the main idea to do surgery for SDB patient. Patient selection for each surgery is crucial. In simple snorer, UARS and mild OSA; palatal implant, radio-frequency or oral appliance are among treatment of choice, except patient who had indication to do other surgery as septoplasty/tonsillectomy or sinus surgery, UPPP might be the choice of combined surgery modality. Regarding to moderate and severe OSA including mild OSA with history of cardiovascular event, CPAP is a gold standard of treatment. In patient who denied CPAP, there many alternative modality such as medial glossectomy/tongue suspension/tonsillectomy or hyoid suspension is deserved for patient who had hypertrophic tongue base, tonsillar hypertrophy or narrowing of hypopharynx. Patient who have maxillofacial skeleton disproportion, especially maxilla/mandibular retrusion, MMA is the most effective method. For morbid obesity, Barisctic surgery is able to decrease AHI significantly. Nasal surgery is not only improve nasal patency but also able to reduce snoring improve quality of sleep and may be increase CPAP compliance in selected patient.
Surgery is an important treatment for obstructive sleep apnea, however, some patients do not respond very well to the surgery which is considered to be the main obstacle of this kind treatment. The critical factor to improve the surgical treatment outcome of OSA is accurate preoperative prediction resulting from sufficient evaluation. OSA patients usually have anatomy abnormalities and functional disorder of upper airway, accordingly, both anatomy and function of upper airway need to be evaluated before surgery. First, anatomy characteristics need to be evaluated by fibrolaryngoscope and three-dimensional CT etc. It needs to be analyzed if the structural abnormalities conform to the results of polysomnograph, and if the abnormalities can be corrected by surgery. On the other hand, the activity deficiency of upper airway dilators need to be evaluated so that to assess if the deficiency of upper airway dilators can be compensated by surgery. The common evaluating method of upper airway dilators is intramuscular EMG which is inconvenient to routine clinical use because it is invasive. However, some items of polysomnograph can be used to reflect the activities of upper airway dilators, which include lowest nocturnal oxygen saturation, AHI of REM and NREM period, etc. In all, the key point of successful prediction for surgery outcome is accurate evaluation of anatomy and function characteristics of upper airway.
ADVANCEMENT OF SLEEP SURGERY IN ASIA

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Sleep surgery is a very important option for treatment of patients with obstructive sleep apnea (OSA), particularly for those who have failed or cannot tolerate positive airway pressure (PAP) therapy. In considering cultural difference, economic burden, and insurance system, between western and eastern countries, PAP therapy may not be an attractive in Asian area.

Since OSA has been introduced in Asia, surgical treatment has been more frequently applied to the patients. In some country, OSA has been recognized by publics to belong to ENT field.

Uvulopalatopharyngoplasty (UPPP) was developed in 1970th from Japan. After then, most of sleep surgery were confined to UPPP and nasal surgery until the end of 1990. After then, LASER was introduced in sleep surgery which was laser assisted uvulopalatoplasty. And in the year of 2000, radiofrequency surgery and pillar implant has been introduced in sleep surgery. But those were still confined into palatal surgery alone. Until this time, most sleep surgeons didn’t have enough knowledge about the sleep apnea itself. Snoring was the most frequent symptoms which patients wanted to be treated and doctors wanted to treat.

Since the beginning 20th, new information for the sleep apnea has been widely recognized by sleep doctors and sleep surgeons. Education of OSA patients was changed and treatment modality of OSA has been widely changed. Surgery has moved from palatal surgery alone to multilevel surgery including skeletal surgery. PAP treatment was become widely used.

Various techniques to reveal the obstruction site has been developed to enhance the success rate of sleep surgery. Sleep videofluoroscopy, sleep multi-detect CT, and sleep endoscopy were introduced. These techniques gave sleep surgeon more dynamic and precise information.

Moreover, various techniques for surgery have been developed in Asian area to reduce the complications and enhance the efficacy. Modified or extended uvulopalatal flap, relocation pharyngoplasty, modified cautery-assisted palatal stiffening operation, uvular muscle resection, palatal muscle resection, anterior palatoplasty, have been reported in Asian country. According to the result of obstruction site evaluation,
multilevel surgery has become more prevalent and short-term and long-term results have been reported. Modified maxillomandibular advancement for the oriental patients was developed.

Even though the standard surgical methods has not been established yet because of short duration and difficulty of analysis of surgical results, many sleep surgeons are trying to find out the good indication of each surgery and multiple combined surgery.

Now, there are a lot of Asian sleep surgeons who are trying to find out the proper surgical treatment for the Asian sleep apnea patients in all Asian countries. Therefore I have no doubt they will find the best option of surgical treatment in the near future.
Objective: Indication of UPPP on Japanese OSA patients.

Design: Retrospective clinical study

Subjects and methods:
1: we enrolled 98 OSA patients who treated by UPPP and undergone PSG pre and postoperatively.
2: We compared the effect about following different methods of postoperative management. (using BiPAP with fullface mask, nasal airway, none)
   1. How many rate of success of UPPP?
   2. What are predictors of UPPP?
   3. Which method of postoperative management is effective?

Results:
1. AHI decreased from 48.1 to 16.9/hr. Surgical success rate (AHI<20 & 50% decrease or more) is just 40.0% in according to Chicago criteria 1999
2. Almost parameter showed significant decrease, especially Obstructive apnea, but in some case Hypopnea increased postoperatively.
3. The independent predictors of UPPP are: 1:Severity (30<AHI<75/hr), 2:Tonsil size (>2/3), 3:Small Jaw (Facial Axis>85), 4: Obesity (BMI<26).
4. BiPAP with fullface mask is most effective, however, some patients could not use BIPAP.

Conclusion:
1. We have to make accurate evaluation for OSA patients preoperatively. PSG(including esophageal pressure measurement) is needed because we have to select patients who showed pure Obstructive events. And analysis of facial structure using Cephalometry or 3DCT cepharo is needed too. Because many Japanese OSA patients have small Jaw. Upper airway structure is affected by both structure soft tissue and facial bone.
2. We have to pay attention safety methods of postoperative management.
CONSCIOUS AWARENESS IN DREAMS: PROOF-OF-PRINCIPLE

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We will present data from a novel approach allowing to induce a change from primary to secondary consciousness in dreams. The design of the experiment is based on our prior work on lucid dreaming which suggested that a particular frequency in the EEG, namely the 40 Hz band (36 – 45 Hz) plays a major role in gaining reflective insight into the surreal character of the ongoing dream.

This incorporates two important steps: a demonstration that an externally applied low voltage electrical current is effective in changing the ongoing EEG and evidence that the dream content is altered as a function of the applied current.

We report on 13 subjects who spent 3 consecutive nights at the Goettingen sleep laboratory of the Department of Neurophysiology. Transcranial alternating current stimulation (tACS) was performed only after 3 am and only during REM phases. In nights 1 and 3, subjects were stimulated with 40 Hz or 2 Hz currents, counterbalanced for time of night. In night 2, participants received sham stimulations.

Our analyses show that the external application of a weak electrical current changes the ongoing cortical activity as evidenced by EEG recordings. This effect was traceable for the 40 Hz but not for the 2 Hz condition which is most likely due to the high delta power that is naturally present during REM sleep. Regarding subjective correlates of induced EEG changes during REM sleep, we observed increased dissociative thought specific to 40 Hz stimulation. Dissociative thought has been shown to be typical for lucid dreaming, especially in young children.

We interpret this finding as strong support for the 40 Hz band hypothesis stating that frequencies around 40 Hz are somehow involved in higher order or secondary consciousness. Moreover, results suggest a causal relationship between lucid dreaming and 40 Hz activity in fronto-temporal areas of the brain.

Although the method itself has been shown to influence cognitive performance by other laboratories, this is the first demonstration of an effect on the ongoing EEG. We consider our results a proof-of-principle, and trust that it will encourage further research into the possibilities and boundaries of low current electrical stimulation of the brain during sleep.
CHANGES IN ANTERIOR-POSTERIOR FEEDBACK CONNECTIVITY DURING ANESTHESIA AND SLEEP IN RAT

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Anesthetic-induced unconsciousness is characterized by a preferential inhibition of anterior-posterior feedback connectivity while feedforward connectivity is less suppressed [1,2]. General anesthetics suppress cortical acetylcholine (ACh) release [3,4]. We hypothesized that the differential suppression of feedback connectivity and the decrease in cortical ACh are causally related. In order to test the hypothesis, ACh levels in prefrontal cortex (PFC) and monopolar EEG were measured before, during and after anesthesia (propofol and sevoflurane). Symbolic transfer entropy (STE) was used to characterize the changes in directional feedback-feedforward connectivity. The mean ACh levels during intravenous propofol infusion showed a statistically significant decrease as compared to the wake state. Compared to propofol infusion, there was a non-significant (38.3%) increase in the mean ACh levels during post-propofol period. The changes in feedback connectivity closely approximated changes in ACh levels. There was a sharp decline of approximately 35% in feedback connectivity at the onset of anesthesia. The feedback connectivity remained suppressed during the anesthetized state and started recovering to the basal levels at the return of consciousness. In another set of experiments, ACh agonist (nicotine) was microinjected into PFC during propofol anesthesia and changes in EEG and directional connectivity were measured. Nicotine caused a full reversal of feedback connectivity accompanied by EEG signs of cortical arousal.

The inhalational anesthetic, sevoflurane, produced the effects similar to propofol i.e., simultaneous decrease in PFC ACh levels and feedback connectivity with the onset of anesthesia, and recovery to basal levels after return of consciousness.

In order to support our hypothesis regarding cholinergic regulation of anterior-posterior connectivity, we recorded monopolar EEG across spontaneous sleep-wake cycle. STE was used to characterize the changes in directional feedback-feedforward connectivity. Connectivity in both directions was high in the wake state (high cholinergic tone), depressed during slow wave sleep (low cholinergic tone) and returned to baseline during rapid eye movement sleep (high cholinergic tone). The bi-directional suppression of connectivity is similar to previous reports of surgical anesthesia as opposed to anesthetic-induced unconsciousness alone [1]. These data
further suggest that directional brain connectivity is regulated by cortical ACh and highlight the phenotypic relationship of sleep and anesthesia.

These preliminary data suggest a correlative and possibly causal relationship between anesthesia-induced changes in brain connectivity and PFC ACh levels. In support of this interpretation, anterior-posterior connectivity is depressed during slow wave sleep which is characterized by low cholinergic tone. We therefore conclude that directional connectivity in the anterior-to-posterior direction is suppressed during both sleep and anesthesia as a result of depressed cortical ACh.

References:

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DREAMS VERSUS HALLUCINATIONS

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Visual imagery during sleep state is physiologic and was called dream (D). Visual imagery during wake state is pathologic and was called hallucination (H). Thirty percent of Parkinson’s disease (PD) patients display H.

The common features of visual imagery in PD-H patients and dream subjects are: a) prosopagnosic faces (cannot be identified); b) bizarre (illogical). These phenomena are explained by the findings in single positron emission computerized tomography (SPECT) in PD-H patients. It was shown the coexistence of hypoperfusion in the right fusiform gyrus with hyperperfusion in the right superior and middle temporal gyri-(high rBGF) (Oishi et al.). The hyperperfusion proves the energy consumed by the hallucinating areas. The hypoperfusion of right fusiform gyrus prove the disturbed “What” and “Where” identification and recognition of human faces and categories of objects, it means the bizarre prosopagnosic faces.

The major difference between the hallucinations of the PD patients versus dreamers is the consciousness of the visual imagery. The dreamers are conscient when awakened that the visual imagery was false. The PD-H are lacking a clear consciousness of the unreal visual imagery. Upon arousal from a dream, the switch to the new state of wakefulness, allows consciousness to interpret the imagery as false. Upon ending the hallucination the disruption in “What” and “When” areas of the brain, do not allow a clear consciousness of the surrounding reality. The structural changes of PD-H are justified by the advanced age, long term disease and dopaminergic therapy; cognitive degradation; daytime sleepiness; Lewy body in subcoeruleus nuclei and disturbed balance of the DA, 5HT, NE & Ch receptors.

Conclusions: The visual imagery of H and D are internally generated by the brain and projected externally. The biochemical constellation of the DA, 5HT, NE & Ch receptors when creating the dream phenomenon in the sleep state are physiologic. The presence of such a constellation during the wake state, proves the disruption of the what and where mechanism of vision by a diseased brain. The presence of REM in in sleep is associated with normal consciousness at arousal. The presence of “REM like state” in wakefulness is associated with confusion.
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Principle and Efficacy of Transvenous Phrenic Nerve Stimulation for Cheyne-Stocks Respiration in Patients with Chronic Heart Failure

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BACKGROUND

Cheyne-Stokes respiration (CSR) may accelerate progression of congestive heart failure (CHF) and is associated with poor survival. Phrenic nerve stimulation (PNS) may interrupt CSR and improve CHF outcomes. We report the pioneer clinical use of transvenous PNS in CHF patients with central sleep apnea and CSR.

METHODS

Twenty-three CHF patients with central sleep apnea and CSR were enrolled. A single stimulation lead was placed at the junction between the superior vena cava and brachiocephalic vein or in the left pericardiophrenic vein. PNS stimulation was performed using the Eupnea System software (Cardiac Concepts Inc., Minnetonka, MN). Respiratory properties were assessed prior to and post-PNS. PNS was assessed at a maximum of 10 mA.

RESULTS

No adverse events were seen under maximum normal stimulation parameters for a maximum single 12 hour sleep cycle. Phrenic nerve stimulation was able to reproducibly slow the rate of breathing in a predictable manner and raise end-tidal expiratory CO2. When PNS was applied following a series of central sleep apneic events, a trend towards stabilization of breathing and heart rate, as well as improvement in oxygen saturation, was seen. There was a significant improvement in indices of apnea/hypopnea, central apnea, oxygen saturation and sleep efficiency after PNS versus pre-PNS (all P <0.01).

CONCLUSION

Unilateral transvenous PNS should be a safe and feasible treatment and by effectively improving CSR.
Measurement of esophageal pressure could be affected by changes in lung volume and airflow; therefore, esophageal pressure may not be the best technique for assessment of neural drive in patients with OSA whose pathophysiological changes include variation of airflow and lung volume. The diaphragm EMG recorded from multipair esophageal electrodes is free from artifacts originated from changes of airflow and lung volume. We found that the pattern of neural drive assessed by esophageal pressure differed from neural drive measured by diaphragm EMG. Neural respiratory drive decreased rather than increased during apnea events in patients with OSA. The diaphragm EMG recorded from multipair esophageal electrode was able to accurately distinguish central from obstructive sleep apnea and hypopnea. Conventional polysomnography with recording of chest-abdominal movement overestimated the frequency of central apnoea events and that about 30% of central apnoea events determined by chest-abdominal movement could not be approved by diaphragm EMG.

The genioglossus is the most important upper airway dilator muscle whose function could be reflected by its electrical activity. Genioglossus EMG could be reliably recorded from fine wire electrodes during overnight polysomnography in both patients with OSA and normal subjects. We found that neural drive to the genioglossus decreased during NREM sleep compared to wakefulness and further decreased during REM sleep. Both genioglossus EMG and diaphragm EMG decreased at the beginning of an apnea event and then increased gradually until resumption of airflow. The gap between peak of genioglossus EMG and diaphragm EMG decreased during apnea events when compared with normal breathing. We conclude that the reduction of neural drive to both the diaphragm and the upper airway muscles may be responsible for pathophysiological changes of obstructive sleep apnea.

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In polysomnography, each single apnea is classified into three groups, obstructive apnea, central apnea, and mixed apnea according to the presence of respiratory effort. Sometimes we see these three types of apnea coexisting during a night; however we diagnose by which type of apnea is predominant. Thus, if most of the respiratory events are obstructive apnea and/or mixed apnea, it would be OSAS, and if central apnea can be seen frequently we diagnose it as CSAS.

Cherniack has suggested in 1979 that the interaction of respiratory output to the upper airway and diaphragm may determine the expression of apnea types, such as central and obstructive. Thus, individuals may manifest apneas with both obstructive and central components. The relative proportion of these components would depend on individual factors, which may be genetic or secondary to a medical condition.

With this concept, we have been focusing on resting breathing pattern variability during wakefulness since we consider resting breathing might be not only a window to explore the central respiratory control system, but also a new tool to distinguish clinically important OSAS phenotypes.

In this session, first, I will present the findings in a mouse model of irregular breathing including recurrent apneas which is C57BL/6J mouse, and propose that there exist genetic mechanisms that could determine loop gain in the respiratory control system, in addition, pharmacologic treatment can modify the ventilatory behavior as well as loop gain. Second, I will show that irregular breathing during wakefulness and poor adherence to CPAP were seen in mixed apnea dominant OSAS which is one of the particular phenotypes of OSAS. Lastly, I will introduce the possibility of breathing irregularity during wakefulness as a marker for CPAP acceptance in patients with obstructive apnea dominant OSAS (pure OSAS).
FROM RESTLESS LEGS SYNDROME (RLS) TO WILLIS-EKBOM DISEASE: A DIAGNOSTIC AND TREATMENT CONUNDRUM

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Objectives:
At the end of the talk, participants should:
   1. Understand the proposed changes in the diagnosis of RLS for adults and children
   2. Recognize the confounding effect of “mimics” and co-morbidities
   3. Learn about available tools in the diagnosis of RLS and their limitations
   4. Know the current guidelines for RLS treatment

Introduction:
Clinical diagnoses based on the patients’ reports are subject to bias. This bias becomes more evident in research where methodology requirements limit the role of clinical experience. A lifelong familial disease course with possible remissions and exacerbations, with symptoms that can range from minimal to severe and treatment that can relieve the symptoms or worsen them compound the difficulty. With a limited understanding of its genetics and pathophysiology, Willis – Ekblom Disease (RLS) is a condition that presents such diagnostic, epidemiological, management and treatment challenges.

Methods:
The talk will review the following: RLS diagnostic tools and criteria for adults and children with the resulting epidemiology data; changes in RLS criteria as proposed by IRLSSG; RLS “mimics” and co-morbidities and current methodology for diagnosing RLS. New insights into the pathogenesis of RLS will be considered. In addition, available data and guidelines on management and treatment will be reviewed.

Discussion:
The discussion will focus on the pathophysiology of RLS as it affects diagnosis, epidemiology and management and on the applicability of different questionnaires and tools in the clinical practice and in research.
RLS AND PLMS, TWO PHENOTYPES OF THE SLEEP LEG MOVEMENT DISORDER (SLMD)

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The unification of Restless leg syndrome (RLS) and Periodic leg movement during sleep (PLMS) under a common tent is necessary due to the common epidemiological, clinical, etiological, biochemical and genetic criteria.

Epidemiological studies revealed 9-20% RLS and 4-11% PLMS prevalence in the corresponding age group (elderly).

Clinically the RLS is the sensory expression, while PLMS is the motor expression of a movement phenomenon related to sleep. RLS is characterized by arousals caused by the urge to move a limb. PLMS is characterized by movements that cause arousals or fragmentation of the sleep continuum.

Etiologically RLS and PLMS have common causes such as: Parkinson's disease, iron deficiency, peripheral neuropathy, sleep apnea; varicose vein or venous reflux; uremia, diabetes, thyroid disease; hypoglycemia, magnesium deficiency, folic acid deficiency; auto-immune disorders: Sjögren's syndrome, celiac disease, rheumatoid arthritis, fibromyalgia; drugs: antidopaminergic, antihistamines, antidepressants, antipsychotics, anticonvulsants, opiates withdrawal.

Biochemically SLMD are strongly influenced by the dopamine and iron systems (Allen R. et al), (Clemens S. et al). Positron emission tomography and functional magnetic resonance imaging in autopsy series and cerebrospinal fluid showed low iron levels in the substantia nigra and other areas of the brain (Godau J. et al). Iron is an essential cofactor for the formation of L-dopa, the precursor of dopamine. The sensory and motor expressions of SLMD can be improved by L-Dopa administration.

Genetically, a common gene is affected in RLS and PLM, BTBD9 on chromosome 6p21.2 (Stefansson et al.). Other locuses found to be involved in SLMD were on chromosome 12q;14q; 9p; 20p; 2p and 16p12.1. Genes which were not yet found in PLMS, but were found in RLS are MEIS1, BTBD9 and MAP2K5 (Winkelmann et al.) and PTPRD (Ding Li. et al)

CONCLUSION: The sleep leg movement disorder (SLMD) is a movement disorder with two phenotypic components: sensory defined as Restless Leg Syndrome (RLS) and motor defined as periodic limb movements (PLMS).
There is a complex relationship between epilepsy and sleep as seizures may be exacerbated by sleep deprivation and some seizures mainly occur during sleep, disrupting normal sleep architecture and causing excessive day-time sleepiness.

Primary sleep disorders such as obstructive sleep apnea may aggravate epilepsy and treatment of these sleep disorders can improve seizure control.

The occurrence of seizures during sleep have been noted since a long time. Epileptic seizures can occur at any stage of NREM sleep but more frequent during lighter stages (stage 1 and 2) than deep stages.

Sleep disturbances in patients with epilepsy may be secondary to AEDs, nocturnal seizures, or asleep disorder such as sleep apnea or restless leg syndrome.

Certain epilepsy syndromes characteristically occur in sleep, such as frontal lobe epilepsy, temporal lobe epilepsy, benign childhood epilepsy with centro-temporal spikes, and certain others following awaking, such as Juvenile myoclonic epilepsy (JME).

Differentiating between NREM parasomnias and nocturnal frontal lobe seizures remains a challenge but there are semi logical features that facilitate differentiation. There are also similarities between NREM parasomnias and nocturnal frontal lobe seizures which may also co-exist in the same individual, suggesting that there may be a common pathogenic background for the two disorders.

Accurate identification and diagnosis of sleep disorders as well as epilepsy is clinically important to ensure optimal treatment of both epilepsy and sleep disorders.

JME is a common type of idiopathic generalized epilepsy syndrome which can occur shortly after awaking and often precipitated by sleep deprivation. Studies concerning the association of JME with sleep are scarce but some recent studies stressed the alterations in sleep quality and prevalence of sleep disorders in JME.
SLEEP MOVEMENTS AND ADHD

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The relationships between sleep and attention-deficit/hyperactivity disorder (ADHD) are complex and are routinely overlooked by practitioners. Motricity and somnolence, the most consistent complaints and objectively measured sleep problems in children with ADHD, may develop as a consequence of multidirectional and multifactorial pathways.

ADHD is most common in childhood, with about 30 - 60 % of patients continuing to be affected into adulthood. About 80 % of children who need medication for ADHD still need it as teenagers, and about 50 % need it as adults.

An estimated 25-50% of children and adolescents with ADHD experience problems with sleep. The most common sleep problems reported in ADHD include delayed sleep onset, sleep or bedtime resistance, prolonged tiredness upon waking and daytime sleepiness. Higher incidences of sleep disorders such as restless legs syndrome, periodic limb movement disorder and sleep-disordered breathing have been reported in pediatric ADHD populations compared with control populations.

Sometimes, medications for ADHD and/or co-morbid disorders may also contribute to sleep disturbances.

Sleep problems may exacerbate ADHD symptoms (neurobehavioral performance is reduced, more errors are made with sleep loss) and interventions targeted at ensuring adequate sleep (including behavioural, dietary, specific pharmacological agents for treatment-induced insomnia, melatonin) could in turn attenuate symptoms associated with ADHD.

Daily cycles of wakefulness and sleep are regulated by coordinated interactions between regulating neural circuitry. Wakefulness is regulated by two processes: the homeostatic drive for sleep and the endogenous circadian pacemaker.

Normal cycling between discrete behavioral states is mediated by the combined influence of a sleep need that increases with continued wakefulness and an intrinsic circadian oscillation.
The basal ganglia (BG) are involved in numerous neurobiological processes that operate on the basis of wakefulness, including motor function, learning, emotion and addictive behaviors. It is considered that the BG might play an important role in the regulation of wakefulness.

Over the last decade substantial progress have made toward understanding the roles of dopamine and BG in the control of sleep–wake behavior. There are recent advancements regarding dopaminergic modulation of sleep through the BG and extra-BG sites. The main hypothesis is that dopamine promotes sleep by its action on the D2 receptors in the BG and promotes wakefulness by its action on D1 and D2 receptors in the extra-BG sites. This hypothesis implicates dopamine depletion in the BG (such as in Parkinson's disease) as causing frequent nighttime arousal and overall insomnia. Furthermore, the arousal effects of psychostimulants (methamphetamine, cocaine, and modafinil) may be linked to the ventral periaqueductal gray (vPAG) dopaminergic circuitry targeting the extra-BG sleep–wake network.

The lower BP values for DAT in the midbrain suggest that dopamine signaling in subjects with ADHD is altered. Altered dopamine signaling might have a causal relationship to motor hyperactivity and might be considered as a potential endophenotype of ADHD.

Whether metabolic or neurological pathways common to both sleep and ADHD may be disrupted, and whether targeting treatments to these pathways may simultaneously improve both ADHD and sleep symptoms, needs further elucidation.
APPLICATION OF ACOUSTIC ANALYSIS OF SNORING SOUNDS FOR DYNAMIC EVALUATION OF UPPER AIRWAY MORPHOLOGY OF OSAS PATIENTS

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Obstructive sleep apnea (OSA) is a common form of sleep-disordered breathing characterized by repetitive episodes of partial or complete upper airway obstruction. It usually causes sleep fragmentation, reduced blood oxygen levels, and excessive daytime somnolence. Cognitive deficits, impaired psychosocial wellbeing, reduced driving competence, cardiovascular morbidity, and mortality have been reported. Because the clinical significance of OSA is increasing, more exact diagnosis for successful treatment is required.

Multiple factors like enlarged tonsils, nasal disease, body weight, age and craniofacial morphology causes OSAS. But it is well known that the Key to the successful treatment of OSAS, especially for surgical treatment, is upper airway evaluation. The upper airway is from the nose down to the vocal folds, and the lower airway has a cartilage framework that makes it a semi-rigid tube, while the upper airway has no such framework. Each part of the upper airway tract can cause obstruction.

Upper airway collapse several areas in varying degrees, especially during sleep. It has been reported that the imaging results while awake do not necessarily reflect conditions during sleep, when tone of the upper airway dilating muscles is decreased. Then we should evaluate the upper airway morphology both static and dynamic manner.

There has been reported that dynamic MRI and sleep endoscopy are both useful for detecting the obstruction site of upper airway during sleep, but the problem is the difficulty for performing the examination. Snoring is the most frequent complaint of patients with OSAS; hence, application of acoustic analysis of snoring sounds for determining a method of differentiation between simple snoring and snoring accompanied with OSAS is important. Also, application of acoustic analysis of snoring sounds for OSAS treatment should be useful. Furthermore, the analysis of snoring sounds is a non-invasive method. We has been perform 3D-CT examination instead of examine the cephalometry as a static evaluation and sleep nasoendoscopy and snoring analysis for dynamic evaluation of upper airway morphology.

In this study, we will show that the usefulness of snoring analysis and 3D-CT examination for dynamic and static evaluation of upper airway morphology of OSAS patients.
CHANGES OF SNORE SOUND AFTER SLEEP SURGERY

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Background: Snoring is the most prevalent symptom in patients with sleep-disordered breathing (SDB) and can be a public noise problem that may disturb or disrupt snorers’ and their sleep partners’ sleep. Loudness of snoring is significantly associated with severity of obstructive sleep apnea (OSA) that may contribute to the development of cardiovascular disease and neurocognitive dysfunction. Accordingly, the monitoring of snoring is an important step for patients with SDB.

Objective: This study was aimed at describing an innovative technique in assessment of snoring sounds and investigating the energy types of the full-night snoring sounds in male patients with OSA.

Patient and Method: Twenty male OSA patients prospectively underwent snoring sounds recording during 6 hours of in-lab overnight polysomnography and their parallel digitally recorded snoring sounds were processed and analyzed by a new sound analytic program — Snore Map®. We transformed the 6-hour snoring sound power spectra into the energy spectrum and classified it as snore map type 1 (monosyllabic low-frequency snore), type 2 (duplex low- & mid-frequency snore), type 3 (duplex low- & high-frequency snore), and type 4 (triplex low-, mid-, & high-frequency snore). The interrater and test-retest reliabilities of snore map typing were assessed. The snore map types and their associations among demographic data, subjective snoring questionnaires, and polysomnographic parameters were explored.

Results: The mean age of OSA patients was 38 ± 9.1 years, the average body mass index (BMI) was 26.9 ± 2.8 kg/m², and the mean neck circumference was 39.5 ± 2.2 cm. The interrater reliability of snore map typing was nearly perfect (κ = 0.87) and the test-retest reliability was high (r = 0.71). The snore map type was proportional to the body mass index (r = 0.63, P = 0.003) and neck circumference (r = 0.52, P = 0.018). The BMI was significantly lowest in type 1 snorers (P = 0.026), whereas the other demographic characteristics, snoring questionnaire scores, polysomnographic
parameters, and severity of OSA were insignificantly different among all the groups (all $P > 0.05$). The AHI was fairly correlated with the total-maximal intensity (40-2,000 Hz, $r = 0.49$, $P = 0.038$) and high-frequency-maximal intensity (851-2,000 Hz, $r = 0.56$, $P = 0.016$) after adjustment for BMI and neck circumference. In addition, snore map type 3-4 was significantly associated with severity of OSA ($r = 0.52$, $P = 0.026$) after adjustment.

**Conclusions:** This is the first study investigating OSA patients’ snoring using the graphic analysis of full-night snoring sound energy. Objective evaluation of snoring sounds indicated that the highest intensity of the special-band snores (40 Hz-2,000 Hz or 851 Hz-2,000 Hz) are reliable parameters to predict the AHI among male OSA patients; however, these predictors could not be measured by current subjective questionnaires appropriately. Energy typing of full-night snores by the Snore Map® provides an easy and reliable method to classify the severity of SDB and its value to clinical applications such as differentiation of primary snoring and OSA, and changes after OSA treatment needs a further investigation.
MIDAZOLAM SEDATION SLEEP ENDOSCOPY: WHAT CAN WE SEE NOW AFTER 20 YEARS

Dr. Victor Abdullah

Department of Otorhinolaryngology, Head and Neck Surgery, United Christian Hospital

Objective: This study examines subjects’ level of consciousness with Bispectral Analysis in sedation endoscopy of the upper airway.

Study Design: A prospective study.

Setting: University Hospital.

Subjects and Methods: Bispectral Analysis levels recorded in natural sleep of 43 obstructive sleep apnoea patients during overnight polysomnographic sleep study were directly compared with the levels recorded during Midazolam Sedation Sleep Endoscopy in the same subjects. The possible muscle relaxation effect of midazolam was examined via surface chin electrodes. 50 obstructive sleep apnoea patients with supine Muller Maneuvre findings were also compared with soft tissue dynamics during Midazolam Sedation Sleep Endoscopy.

Results: In our study of the 43 patients with Bispectral Analysis during natural sleep and Midazolam Sedation Sleep Endoscopy, a predominance of Bispectral Analysis values indicating N1 and N2 sleep is observed during the sedation study. Midazolam fails to achieve deeper levels of sleep with minimal of N3 and no convincing Rapid Eye Movement. As N1 and N2 are the stages during which maximal dynamic activities occur, and they make up an average of 70.5% of total sleep time, from 210 sleep studies at our laboratory, the present technique would be ideal as a surgical assessment tool. No muscle relaxation effect could be detected at our protocol dose of midazolam. The supine Muller Maneuver findings were significantly different from those observed during sedation sleep.

Conclusion: These findings support the value of sleep endoscopy as an efficient and informative technique for the examination of upper airway dynamics relevant to focused surgical planning.
COMPUTER-ASSISTED QUANTITATIVE UPPER AIRWAY ANALYSIS FOLLOWING MODIFIED UVULOPALATAL FLAP AND LATERAL PHARYNGOPLASTY FOR OBSTRUCTIVE SLEEP APNOEA

Pon-Poh Hsu

Chief & Senior Consultant, ENT, Changi General Hospital, Singapore

Objectives: The study aims to perform static and dynamic quantitative assessment of the anatomical changes of the upper airway before and after modified uvulopalatal flap and lateral pharyngoplasty and comparison of the improvement in airway dimensions, collapsibility and extent of normalization to that of control patients.

Design: Prospective case-control study

Setting: Computer-assisted quantitative measurement is used to compare upper airway parameters before and after modified uvulopalatal flap and lateral pharyngoplasty in patients with obstructive sleep apnoea.

Participants: Patients with obstructive sleep apnoea diagnosed on sleep study and failed positive airway pressure therapy.

Main Outcome Measures: Sleep study results, upper airway parameters and symptom score following surgery and its comparison to normal patients to assess the degree and extent of normalization.

Results: 35 study and 32 control subjects were recruited and completed the study. All the retropalatal airway dimensions like area, transverse diameter, longitudinal diameter and collapsibility showed statistically significant improvement following surgery. The success rate of this surgery is 43% (15 out of 35) overall, 58% (14 out of 24) for patients with isolated palatal obstruction and only 9% (1 out of 11) for patients with multi-level obstruction. Comparing OSA to the control subjects, there are obvious and logical differences in their biostatistics, sleep study parameters and airway dimensions. The postoperative OSA retropalatal longitudinal diameter has a higher tendency of normalizing to be comparable to those of control patients.

Conclusions: Modified uvulopalatal flap and lateral pharyngoplasty is an effective surgical technique for the treatment of obstructive sleep apnoea. The surgery increases the resting retropalatal dimensions and reduces the retropalatal collapsibility.
MULTI-OSCILLATOR SYSTEM OF MAMMALIAN CIRCADIAN CLOCK

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The master circadian clock of mammals is localized in the hypothalamic suprachiasmatic nucleus (SCN) which is a hierarchical multioscillator clock. Molecularly, a transcriptional and translational feedback loop involving several clock genes generates circadian rhythms in each cell. At least three pacemakers have been identified in different subregions of the SCN, since clock gene expression rhythms in these areas are transiently uncoupled after an abrupt shift of light-dark cycles or exposing to different photoperiods. Luciferase reporter and highly sensitive bioluminescence monitoring systems, such as a cooled EM-CCD camera, enabled us to monitor circadian rhythms of clock gene expression for a long-term at cellular levels as well as at tissue levels. In cultured SCN slices, undamped circadian clock gene expression rhythms were detected, indicating coupling among regional pacemakers. On the other hand, several brain tissues exhibit circadian rhythms only for several cycles ex vivo, suggesting that cellular rhythms are either desynchronized or damping out without inputs from the SCN.

Under prolonged temporal isolation, humans often exhibit an internal desynchronization between rhythms in sleep-wakefulness and those in melatonin or deep body temperature, suggesting that sleep-wakefulness rhythm is regulated by another pacemaker than light-entrainable one in the SCN. However, neither the localization nor the molecular mechanism is clarified for this pacemaker. Chronic methamphetamine treatment induces SCN-independent rhythms in rats and mice, the behavioral pattern of which is similar to the internal desynchronization of humans. Using this animal model, we explored the brain areas that directly regulate behavioral rhythms. We monitored clock gene expression rhythms in cultured brain tissues of Per2-luciferase transgenic rats which were treated with methamphetamine chronically. The treatment shifted bioluminescence rhythms in several areas of central dopaminergic systems. By monitoring clock gene expression ex vivo, we could rule out masking effects of behavior on gene expression and monitor autonomic oscillation in specific brain area.
DISSECTION OF NEURAL MECHANISMS UNDERLYING CIRCADIAN PACEMAKERS USING BRAIN REGION/CELL-SPECIFIC BMAL1 DEFICIENT MICE

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The suprachiasmatic nucleus (SCN) is the primary circadian pacemaker in mammals and entrains to the environmental light. It is composed of multiple types of neurons, and neuronal network properties are integral to its function. As a first step to understand the principle of the SCN network, we generated mice in which Bmal1, an essential clock component, is deleted specifically in the Avp-producing neurons, one of the primary neuronal types in the SCN. These mice showed lengthening of circadian period (by approximately 1 hour) and the activity period (by approximately 5 hours) in constant darkness. Thus, circadian oscillators in Avp neurons may regulate coupling of thousands of neurons in the SCN to determine circadian period.

In addition to the light, temporal restriction of feeding can also entrain circadian behavioral and physiological rhythms in mammals. These changes in biological rhythms are postulated to be brought about by a food-entrainable oscillator (FEO) that is independent of the SCN. However, the molecular machineries of FEO have remained elusive. We report here that mice with a nervous system-specific deletion of Bmal1 had a marked deficit in entrainment of locomotor activity by periodic feeding, accompanied by reduced food intake and subsequent loss of body weight. These mice exhibited a nearly normal light-entrainable activity rhythm, because deletion of the Bmal1 gene in the SCN was only partial. These findings suggest that an SCN-independent FEO in the nervous system requires Bmal1 and plays a critical role in adaptation of circadian locomotor activity and food intake to periodic feeding.
CIRCADIAN PHASE WAVES AND LONG-RANGE NETWORKS

IN SUPRACHIASMATIC NUCLEUS

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The suprachiasmatic nucleus (SCN) is the master clock in mammals governing the daily rhythms. It is composed of thousands of clock cells that have their own intrinsic periods varying over a wide range (20~28 h). Despite this heterogeneity, the clock cells in an intact SCN nucleus maintain a coherent 24h periodic rhythm (i.e., frequency-locking). On the other hand, generically populations of coupled nonlinear oscillators can exhibit some form of phase synchronization when the coupling strength of cell-to-cell interaction exceeds a critical value. Therefore, some interesting spatiotemporal phase dynamics can exist within the SCN. This study examined how the clock cells are connected to each other and how their phases are organized in space by monitoring the cytosolic free calcium ion concentration of clock cells using the calcium binding fluorescent protein, cameleon. Extensive analysis of 18 different organotypic slice cultures of rat SCNs showed that the SCN calcium dynamics is coordinated by many phase-synchronizing sub-networks of long-range neurites as well as by diffusively propagating phase waves. Our analysis on the image data quantifying Per1-d2EGFP expression levels of individual SCN clock cells of mice, which was recently published by Enoki et al. (J. Neuroscience Methods, 2012), reveals a very similar property. Taken all together, the existence of long-range subnetworks and circadian phase waves seem to be a generic property of SCN. The networks appear quite extensive and far-reaching, and potentially have a role of expediting the conduction of circadian phase information throughout the SCN.
CLOCK GENES AND HUMAN SLEEP BEHAVIOR PHENOTYPES

Ying-Hui Fu
Professor, Department of Neurology, University of California

Behavioral studies using model organisms are stimulating and relatively approachable since the researchers can, for the most part, control both external and internal variables. These studies have provided us novel insights into the genetic nature of many interesting behaviors. Predictably, gaps remain when trying to translate what scientists learn in model organisms to something applicable or useful for human health conditions. In contrast to model organisms, studying behavior in humans such as sleep requirement and preferred sleep-wake times is a daunting task due to the complexity of confounding co-morbidities, environmental factors, and the polygenic nature of behavioral phenotypes.

Like most organisms, humans exhibit daily behaviors that are regulated in a circadian (24-hour) manner. Early pioneers of circadian biology research, such as Jurgen Aschoff, observed that human behaviors under constant conditions exhibited rhythms with an approximately 24-hour periodicity (Aschoff, et al., 1971). Aschoff’s studies in humans revealed the existence of an endogenous biological time-keeping mechanism. Decades later, studies from model organisms uncovered a molecular clock comprised of many genetic players that governs the circadian oscillation of physiology and behaviors through complex methods of regulation (Dunlap, 2004, Hastings, et al., 2008). Not surprisingly, there are shared molecular mechanisms for the molecular clock amongst diverse organisms, including Neurospora, Drosophila, rodents and humans. However, there are also salient differences. For instance, core clock mechanisms are presumably more widely integrated with other forms of physiological regulation in metazoans compared with unicellular organisms due to the inherent complexity of intercellular interactions. In addition, while molecular differences between vertebrates and invertebrates cannot be overlooked (Hardin, 2011, Lowrey and Takahashi, 2011), differences between closely related species, such as rodents and humans, are also expected. Notably, rodent circadian periods average approximately 23.5 hours (Lowrey and Takahashi, 2011), whereas the human average is slightly longer than 24-hours.

Identification of the underlying genetic basis of human circadian rhythm behaviors was not possible until the characterization of the first Mendelian circadian trait, Familial Advanced Sleep Phase (FASP), in 1999 (Jones, et al., 1999). This study began with meticulous phenotypic characterization of a 69 year old woman who had life-long early sleep-wake onset, which led to
the identification of a large family segregating this behavior. This story pioneered the field of human sleep genetics, including the search for rare Mendelian single gene/mutation forms and genome-wide association studies aimed at discovering novel variants in larger populations. Since the initial FASP findings, other human circadian/sleep phenotypes have been attributed to underlying genetic components, such as Familial Natural Short Sleep (FNSS) (He, et al., 2009, Zhang, et al., 2011). Therefore, studies of rare and extreme Mendelian behavioral traits have established a foundation for identifying human genetic components for circadian rhythms and sleep behaviors, which then provide further opportunities in understanding the molecular mechanisms of these behaviors.

Until a human genetics approach was applied to investigate human sleep behavior traits, the genetic components regulating these behaviors remained mysterious. Steady advances in the last decade have dramatically improved our understanding of the genes involved in circadian rhythmicity and sleep regulation. Finding these genes presents new opportunities to use a wide range of approaches, including in vitro molecular studies and in vivo animal modeling, to elevate our understanding of how sleep and circadian rhythms are regulated and maintained. Ultimately, this knowledge will reveal how circadian and sleep disruption contribute to various ailments and shed light on how best to maintain and recover good health.
According to the recent five years data (from 2006 to 2010) of the National Health Insurance Corporation (Korea), patients who receiving care for their sleep problem nearly doubled and the total hospital bills also increased by approximately 2.4-fold. Of various sleep problems, insufficient sleep, poor sleep hygiene, insomnia and obstructive sleep apnea syndrome (OSAS) were relatively common. Sleep education can be helpful in improving various symptoms in Korean patients with sleep disorder.

# Educational tips for better sleep hygiene

<table>
<thead>
<tr>
<th>Time</th>
<th>Tips</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morning</td>
<td>Get up from the bed at the same time every day.</td>
</tr>
<tr>
<td></td>
<td>See the sunlight after wake up (The light helps to regulate biological clock).</td>
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<tr>
<td>Day ~ Evening</td>
<td>If you need to take a nap, try to keep it less than 30 minutes before 3 p.m.</td>
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<td></td>
<td>Do not have any stimulant (e.g., caffeine, nicotine, etc.) or alcohol within six hours of bedtime.</td>
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<tr>
<td></td>
<td>Avoid any hard exercise within six hours of bedtime.</td>
</tr>
<tr>
<td>Night</td>
<td>Go to bed only when sleepy.</td>
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<tr>
<td></td>
<td>Get out of bed if you did not fall asleep after 20 minutes.</td>
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<tr>
<td></td>
<td>Avoid sedatives or sleeping pills, or use them carefully.</td>
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<td></td>
<td>Take a shower or bath in warm water before bedtime.</td>
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<td></td>
<td>Keep the bedroom environment dark, quiet, and comfortable.</td>
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<tr>
<td>Others</td>
<td>If you have any symptom related with sleep disorder, consult a sleep specialist.</td>
</tr>
<tr>
<td></td>
<td>Maintain a regular schedule (e.g., get up, go to bed, meals, exercise, etc).</td>
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SLEEP EDUCATION IN JAPAN

Soichiro Miyazaki

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Now the world is running for 24 hours by 7days. Taiwan, Korea and Japan are the shortest sleep countries in the world. Several epidemiological studies indicated that more than 20% of general adult are suffering from insomnia in Japan. However, only 5% of them visited hospitals. Due to the new social attention and documented high prevalence of sleep disorder, the need for sleep healthcare may increase. According to the Japanese national survey by NHK , more than half of subjects felt unsatisfied with their sleep. In the past 40 years, people turn to staying up later and later in night, and the sleep time has consequently decreased . Another questionnaire survey in 2002 was focused on what people will do with subjective sleep problems, among some countries in Europe and Asia. The result showed that around 53.4% people tried nothing, while only 30.8% people would consult doctors. Far fewer would seek formal medical attention or would go to see a physician when encountering sleep problems in Asia than in Europe.

These data emphasize the responsibility of academic organizations, medical doctors and other healthcare workers to sleep medicine.

For these seven years, we had developed sleep instructor courses for the people who are interested in sleep. The primary course offers 6 hours lectures which include mainly sleep mechanism and sleep hygiene explanation. The senior course offers 24 hours advanced sleep medicine lectures such as sleep science, sleep related diseases, sleep sociology and group discussion. Also we teach them how to make a presentation related to sleep for the general people. Course attendants are certified after examination.

Last three years, we have started sleep medicine lecture for medical and nursing students. The duration of each lecture is 90 minutes and the number of lectures are 14 times. The contents are history of sleep medicine, sleep mechanism, sleep related biorhythm, sleep and sport, aging and sleep, sex and sleep, shift work and sleep and group discussion on sleep related social problem.

In this panel discussion, I focus on the sleep education and promotion of sleep awareness in Japan.
SEEING A DREAMING BRAIN -BRAIN ACTIVATION ACCOMPANYING RAPID EYE MOVEMENTS DURING REM SLEEP-

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It is well known that our eyes move frequently and there is a high incidence of vivid dreams during REM sleep. Although there is a long-standing debate about whether REMs are functionally equivalent to waking saccades\(^1,2\) and about whether there is a relationship between the visual content of a dream and REMs in humans, the relationship between dreaming and REMs is still unclear.

Another important question concerning REM sleep is whether ponto-geniculo-occipital (PGO) waves occur in humans. PGO waves, named for the brain structures where these waves appear most prominently (the pons, lateral geniculate nucleus, and occipital cortex), are one of the phasic events of REM sleep that include REM bursts and have been observed in a variety of mammals. It is thought that PGO waves are related to several important brain functions such as sensorimotor integration, dreaming, learning, development of the visual system and so on. Although the existence of PGO waves in humans has been suggested by several lines of studies, there is a need for higher spatial and temporal resolution measurements to elucidate the cortical–subcortical functional neuroanatomy of PGO waves.

**Methods:**
To identify the neural substrate of REMs in humans, we conducted simultaneous functional magnetic resonance imaging (fMRI) and polysomnographic recording during REM sleep\(^3\). In addition, three control fMRI experiments were conducted during which subjects were required: (1) visually-guided saccade under the illuminated condition, (2) self-paced saccade under the illuminated condition, (3) self-paced saccade in total darkness.

**Results and Discussion:**
Event-related fMRI analysis time-locked to the occurrence of REMs revealed that the pontine tegmentum, ventroposterior thalamus, primary visual cortex, putamen and limbic areas (the anterior cingulate, parahippocampal gyrus and amygdala) were
activated in association with REMs. Whereas self-paced and visually-guided saccades under the illuminated conditions induced strong activation in the visual cortex, saccades in total darkness showed no activation in the visual cortex.

These results suggest that:

(1) The activation of the pontine tegmentum, ventroposterior thalamus, and primary visual cortex time-locked to REMs strongly indicates the existence of PGO-waves in humans.

(2) The activation of the primary visual cortex time-locked to REMs provides neural evidence for the link between REMs and dreaming.

(3) The activation of the parahippocampal gyrus and amygdala accompanying REMs suggests that REMs and/or their generating mechanism are not merely an epiphenomenon of PGO waves, but may be linked to the triggering activation of these areas.

(4) More importantly, these activities occur not tonically throughout REM sleep but phasically time-locked to REMs.

Furthermore, recent findings from EEG study⁴ and REM sleep behavior disorder (RBD) patients⁵ also suggest that REMs scan dream imagery. In conclusion, it seems reasonable to consider that there are at least two types of REMs. One is REMs to scan dream imagery, the other is REMs directly driven by PGO-waves.

References:
WHAT HAPPENS TO OUR BRAIN UPON AWAKENING?

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Sleep plays an important role in regulating our cognitive functions, which keep our mental and physical conditions refresh in each day, but until now we can only disclose part of mysteries inside sleep. How sleep modulates our brain functions can generally be revealed by observing the brain networks upon awakening. To this goal, we observed functional networks before and after a two-hour sleep. Our findings indicated that a general recovery of cortical connections and the reduced signal fluctuations support the hypothesis of rebooting cognitive functions after sleep.
ALTERATIONS IN DECISION MAKING INDUCED BY SLEEP DEPRIVATION

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Voluntary curtailment of sleep for social or economic reasons is pervasive. Behavioral studies indicate that while there is inter-individual variation, most individuals will behavioral changes after a single night of total sleep deprivation.

Loss of sleep can affect risky decision-making and alter our relative valuation of social and monetary rewards. Strategic changes in decision-making - a tendency to select less effortful choices can occur. Contrastingly, delay discounting, a measure of impulsiveness appears not to be affected.

Knowing what fails when we are sleep deprived can help us better prepare for the changes that we might encounter when having to stay up all night to work.
Plasticity is a fundamental feature of the nervous system that underlies neural development, adaptation, and learning. However, what is not often considered is that plasticity may come at a price. As studies recently showed with anatomical, molecular, and electrophysiological approaches, waking plasticity in cortical circuits results in a net increase in synaptic strength. And growing evidence indicates that synaptic strength needs to be renormalized by sleep. But what happens if plasticity is extended by increasing the duration of learning beyond the normal waking day? In this talk, we will discuss a study in humans which used high-density EEG to demonstrate EEG patterns of extending plasticity in specific brain circuits. 16 subjects recorded with high-density EEG completed two experiments during which they stayed awake for $\geq 24h$ practicing a language task (audio book, AB) or a visuo-motor task (driving simulation, DS). Both conditions resulted in global increases in resting wake EEG theta power at the end of 24h of wake, accompanied by increased sleepiness. Moreover, wake theta power and amplitude of large theta waves showed regional, task-dependent changes, increasing more over left frontal derivations in AB, and over posterior parietal regions in DS. These local changes in wake theta power correlated with similar local changes in sleep low frequencies including SWA. Thus, extended use/plasticity of specific circuits results in a local slowing of the wake EEG in those regions, followed by more intense sleep over the same areas.
Nasal obstruction as an independent risk factor for obstructive sleep apnea syndrome (OSAS) plays a critical role in the pathogenesis. It has been known that continuous positive airway pressure (CPAP) of the standard treatment for OSAS has a prognostic benefit of long-term use. However, CPAP is often not tolerated in patients of symptomatic nasal obstruction presumably with severely narrowed nasal airways and the CPAP intolerance adversely affects the prognosis in these patients. In general, when conservative treatment for nasal obstruction fails, the nasal surgery can be performed to correct the obstructive lesion. Friedman et al. demonstrated that nasal surgery improves the daytime sleepiness score in patients with OSAS and decreases the optimal CPAP level by substantially diminishing nasal resistance. According to our previous reports, the nasal surgery has a mainly positive effect on the severity of OSAS and its related symptoms. However, to our knowledge, the outcomes of surgery for these patients have not been investigated systematically. In this context, we have hypothesized that the nasal surgery can be recommended for the CPAP intolerance in OSAS patients with symptomatic nasal obstruction. Therefore, the aim of this study was to find not only the clinical indications of the nasal surgery for the CPAP intolerance in these OSAS patients, regarding the nasal resistance prior and posterior to the surgery, but also CPAP compliance after nasal surgery. In method, case-control study was performed between the male apnea patients undergoing nasal surgery: surgery group (n=40) and the pair-matched apnea patients for age, sex, body mass index, and race: control group (n=38). The surgery group suffering from nasal obstruction could not use continuous positive airway pressure, and the CPAP group free from nasal obstruction could use it successfully. AS for the results, in surgery group, surgery significantly decreased the nasal resistance and Epworth sleepiness scale scores without changing the apnea–hypopnea index. Surgery significantly increased the nadir of oxygen saturation and shortened the apnea–hypopnea duration. Although all of the surgery group failed to use positive airway pressure preoperatively, 29 of 40 patients (73%) were able to use it postoperatively. For both groups, the cutoff nasal resistance for differentiating the failure of positive airway pressure and its success was 0.33 Pa/cm³/s. In conclusion, isolated nasal surgery is effective for an intolerance of positive airway pressure in sleep apnea with nasal obstruction presumably by decreasing nasal resistance.
Obstructive sleep apnea (OSA) is a common condition characterized by recurrent episodes of upper airway obstruction during sleep. Patients may have detrimental consequences and the management of OSA is complex and challenging. Many patients respond to medical treatments with weight loss, body position training, oral appliances and continuous positive airway pressure (CPAP), when medical intervention fails, surgical option is the alternative to improve the upper airway patency. Since the introduction of uvulopalatopharyngoplasty (UPPP) many palatal surgical procedures have been introduced to alleviate upper airway obstruction in patients with OSA. Palatal surgery is now the largest, established surgical approach to OSA, however, most procedures are invasive, destructive, painful, and associated with a significant morbidity. Recently, minimally invasive surgeries also achieved the same results as the invasive procedures but with less postoperative complications. The response is 40% to 80%, defined as a > 50% reduction of the AHI or AHI < 20/hr postoperatively. Long-term follow-up is necessary because some initially successfully treated patients will relapse in the long term.
TONGUE BASE SURGERY FOR MODERATE TO SEVERE OSA

Prasit Mahakit

Consultant, Department of Otolaryngology, Phramongkutklao Army Hospital

In moderate and severe OSA patients, CPAP is the gold standard of treatment but it has poor compliance (60-85%). For patient who denied CPAP or non compliance, there are many modality as alternative method depending on site of obstruction. Patient who have redundant soft palate, enlargement of tongue base but have normal skeleton and hypopharynx. UPPP and tongue base surgery is appropriate for this group of patient. Tongue base surgery consist of medial glossectomy to reduced tongue volume and tongue traction suturing to tightening base of tongue to mandible.

This surgery would provide adequate retro lingual space preventing pharyngeal airway collapse during sleep. Patient who has velo–oro-hypopharyngeal narrowing which co-existent skeleton hypoplasia as retrognathia, maxilla-mandibular advancement MMA)is optimal modality of treatment with high success rate(85-90%). Regarding tongue base surgery, long term success rate is 70-80% depend on patient selection, identified various site of narrowing, multi level surgery with site specific, minimize morbidity and patient cooperation especially weight control and maintain a healthy life-style. Lifetime follow up is highly recommended.
LIMITATIONS AND COMPLICATIONS OF SKELETAL SURGERY FOR OSA

Sung Wan Kim

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Skeletal surgery for obstructive sleep apnea (OSA) aims to provide more space for the tongue in the oropharynx, to limit posterior collapse during sleep. These procedures include genioglossus advancement (GA), hyoid myotomy suspension (HMS), and maxillomandibular advancement (MMA). These skeletal surgery except MMA is usually combined with palatal soft tissue surgery (so called multilevel surgery), for example, palatal surgery plus GA and/or HMS. Multilevel surgery seems to have a higher success rate than unilevel surgery has. Maxillomandibular advancement (MMA) employs a different conceptual approach, whole upper airway reconstruction, because MMA can widen the entire upper airway with one procedure.

In spite of their higher success rate, there are a lot of limitations in the skeletal surgery.

The specific indications for the higher success rates of these surgical procedures have not been revealed according to various evaluation methods. Very few data has been existed for the indication of each procedure. No standard method in the evaluation of obstruction site in OSA patients. Every procedure for the evaluation of obstruction site has some limitation to be a standard method. The skeletal surgery can make a relapse as soft tissue surgery has. There is no study to decrease the relapse rate. And skeletal surgery needs longer learning curve than soft tissue surgery which result that very few surgeon use these techniques. The result of surgery is more affected by surgeon’s technique.

The complications of skeletal surgery are more aggressive than soft tissue surgery if occurred.

GA by various techniques can make the hematoma, tooth injury, anesthesia of tooth and lower lip, dental deformity and tooth color change. But most of them are temporary or correctable. Some procedures, such as modified mortise technique or sliding osteotomy, can make the weak mandible and susceptible to fracture. HMS is also able to make seroma, wound dehiscence, voice change, and swallowing difficulty. But most of them may be disappeared spontaneously within 6 months postoperatively. MMA is more aggressive procedure which can make an airway obstruction by hematoma and swelling just after the surgery. This procedure also can result in
malocclusion and inevitable facial shape change.

In spite of limitations in sleep surgery, surgery remains an important therapeutic consideration and inevitable treatment option in many patients. Therefore, to achieve increased success rates for surgical treatments, future studies must focus on evaluation methods for finding the obstruction site and on addressing the complex interactions of the numerous factors that cause airway obstruction. The search for more precise indication for each surgical method in each evaluation method should also result in better surgical success rates. In addition, experts in sleep surgery need to educate and encourage the beginners in sleep surgery for them to do right and effective surgery. These may be the only, or at least the best, ways to increase success rates and avoid unnecessary procedures in OSA patients.
AN OVERVIEW OF EPIDEMIOLOGY OF SLEEP AMONG THE WORKING POPULATION

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Epidemiology of sleep disorders has received great attention in the last 20-30 years. Recent investigations have shown the association between sleep duration, sleep disorders and mortality and morbidity related to accidents and cardiovascular and metabolic diseases. Due to the increased demands to work for 24 hours in various industries more on more people stay awake and work during sleep hours. Sleep deprivation has important health consequences. Four hours of sleep loss can produce comparable sedation and reduce performance to levels equal to legal intoxication (i.e., ≥ 0.1 breath ethanol concentration). A recent study among Japanese physicians found medical incident (19%) within the last month, which was associated with sleep deprivation and insomnia. Among nurses shift work disorder (32.4–37.6% with different assessment methods) was associated with intervals between shifts <11 h and number of nights worked in the last year. Trouble sleeping was associated with an increased risk of work injury, mostly among women and in processing and manufacturing and those who work rotating shifts. Excessive daytime sleepiness (20-25%) was much higher in occupations including medical residents, shift workers, and transportation workers. Prevalence of short sleep duration (≤6 h/day), among civilian employed workers increased from 24.2% to 29.9% from 1985-90 to 2004-07, with higher prevalence among transportation/warehousing, manufacturing, and public administration industries. Short sleep duration was associated with self-rated poor health and elevated body mass index (BMI). Insufficient sleep can affect job performance and workplace safety, which are important occupational health considerations and gained increased attention after the success in minimizing the traditional occupational hazards — toxic chemicals, temperature extremes, and noise in the developed countries. Shift work has been linked to the increased risk of cancer including breast and stomach, due to reduced melatonin production, which has antimitogenic and oncostatic activity, and type 2 Diabetes due to a mismatch of circadian rhythms triggering a cascade of biological changes that have potential diabetogenic effect. However a recent systematic review has questioned these findings and suggested further research with detailed information on shift work and confounders in large prospective cohort studies from a wider variety of occupations. Meta-analysis of currently available epidemiological studies found the pooled estimates of RR due to shift work for low birth weight and small for gestational age as
(1.27, 95%CI 0.93-1.74) and (1.12, 95%CI 1.03-1.22), respectively. While most of the studies focused on shift work, poor sleep has been associated with occupational and health-related problems, such as an increased risk of accidents, mortality, and illnesses, including, for example, coronary heart disease, diabetes, and mental disorders, workplace absence due to sickness and with reduced productivity. Measures of job stress, such as perceived stress, hectic work, high job demands, working under time pressure, low job control, high job strain, low social support at work, bad atmosphere at work, role conflicts, effort-reward imbalance, job dissatisfaction, low levels of interest in job, and job insecurity are associated with sleep disorders. Repeated measurements provide strong evidence for a substantial and sustained decrease in sleep disturbances following retirement.
IMPACT OF SLEEP DISORDERS ON OCCUPATIONAL HEALTH

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It is obvious that sleep affects all aspects of human life. Moreover, we have a quite large knowledge about sleep and circadian rhythm effects on energy metabolism. Therefore sleep is one of the major factors of occupational health.

Human kind has been programmed as hunting-feeding- working in daytime and sleeping during night time. However industrialized society can not tolerate to stop working and not producing during the night time. So some people have to work during the night time and sleep during the day time opposite to what genes tell us to do. Shift working is a major problem in aspects of sleep in occupational health. However some sleep disorders other than circadian rhythm also affect patients’ job performance. For example sleep related respiratory diseases and excessive daytime sleepiness are very common among truck and bus drivers and it is very important for their occupational health. In our study, it is found that 30 % of bus drivers in Turkey has excessive daytime sleepiness. Another study showed that excessive daytime sleepiness is very common in factory workers in Turkey.

There are several other studies which prove that sleep deprivation affects the job performance. Most of these studies were designed in medical workers like nurses, intern doctors or parameds.

In fact particular sleep disorders have been described in particular occupations. A good example for that issue is obstructive sleep apnea syndrome is a common disorder in bus drivers. Therefore sleep disorders should be considered as a main factor which affect the job performance. Moreover some of the occupations have a strong relationship with particular sleep disorders.
The total number of cases of occupational accidents related to sleep disorders has decreased since 1990 in Turkey. According to epidemiological studies, the number of workers who complain sleep disorders rather than other medical problems has been increasing and there are many more workers experiencing sleep problems related to working conditions. Overtime work, extra-working, shift working, irregular working, noisy environment, poor ventilation, bad ambient temperature, bad ambient light, bad working order, insufficient breaks and other factors related to work place affect the individual’s sleep. In addition, some studies have shown that measures of job stress, such as perceived stress, hectic work, high job demands, working under time pressure, low job control, high job strain, low social support at work, bad atmosphere at work, role conflicts, effort-reward imbalance, job dissatisfaction, low levels of interest in job, and job insecurity are associated with sleep disorders. Briefly, there are many studies showing the relationship between the quality of sleep and working conditions and our results are in agreement with the results of these studies. Also our epidemiological study (TAPES) showed that average sleep duration and efficiency of sleep were decreased at working population. However, studies on relatively small or selective samples, examined non-standard measures for the assessment of job-related factors, or did not take adequate account of potential confounding factors such as sociodemographic factors, physical and psychological health status, or important occupational risk factors such as shift work and working hours. Our findings highlight the strong association between workplace conditions and sleep disturbances and suggest that bad workplaces conditions that are prone to sleep disorders may have a detrimental effect on employees’ sleep, even if employees are not directly concerned by the phenomenon. Given the high prevalence of sleep disorders related to bad workplaces conditions observing in developed and developing countries. So, if we want to improve labor productivity; we have to pay more attention for workplaces conditions and we need more prospective studies to understand the relationship between working conditions and sleep characteristics.
EPIDEMIOLOGY OF OSA IN WORKING POPULATION – TAIWAN CHAPTER

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Sleep-disordered breathing (SDB) is a prevalent disorder among the middle-aged that can seriously compromise a patient’s quality-of-life. Patients of SDB may suffer from symptoms ranging from snoring to apnea (obstructive sleep apnea syndrome, OSAS). They have higher risks of developing cardiovascular complications and neurocognitive dysfunctions. The SDB can also raise the risk of accidents in traffic and working places.

Due to insufficient capacity and long waiting time for overnight polysomnography (PSG), we developed a two-tier screening model, using a sensitive questionnaire in the first-tier to exclude innocent subjects and the more specific oximeter in the second-tier to identify severely diseased subjects for early PSG. (Sleep Breath 2011;15:447–454). Other prediction algorithms such as MMTS (multiclass Mahalanobis-Taguchi system, Computational and Mathematical Methods in Medicine DOI:10.1155/2012/212498) and the integration of a rough set (RS) with the Mahalanobis distance (MD) (Expert System and Application 2011;38:7828-7836) were attempted to further simplify diagnostic procedures and reduce costs.

Our domestic prevalence study revealed that severe snoring was reported by 8.8% middle-aged employees, while severe sleepiness was reported by 8.5% of respondents. The body-mass index (BMI; kg/m2) (r = -0.1488, p = 0.0008) and age (r = -0.1468, p = 0.0010) were significantly associated with the severity of snoring (r = -0.1488, p = 0.0008). (FJJM 2007;5(2):67-80)

Shift work is frequent and necessary in the modern society. Shift workers suffer from worse sleep quality than regular day workers. We used the Pittsburg Sleep Quality Index (PSQI), Epworth Sleepiness Scale (ESS), and Snore Outcomes Survey (SOS) to survey 1719 randomly selected employees from a factory. Severe snoring occurred in 12.1% and severe daytime sleepiness in 11.9% of the employees. Caffeine-containing soft drinks were commonly used by shift-workers (p < 0.05). Shift-workers performed poorly in terms of general sleep quality and sleepiness, and had more snoring problems (p < 0.05). (FJJM 2007;5 (4):189-202) We found 2-5
days rotating shift is the worst schedule for the workers to tolerate, exposing employees to a higher possibility of SDB, poorer general health and sleep quality. A better shift schedule and coping strategy are very important to improve the general health of employees and the productivity of cooperate. (Indian J Sleep Medicine 2007;2:135-138)

General health status can also be seriously compromised by sleep disorder, Our quality-of-life, QoLF) study on Taiwanese adult patients with sleep-disordered breathing (SDB) shows that patients with SDB have worse performance in almost all dimensions of general health status. Daytime sleepiness, rather than apnea, is a major quality-of-life determinant. (Otolaryngology–Head and Neck Surgery 2006;135:421-426). Patients with obstructive sleep apnea (OSA) may experience unfavorable psychologic symptoms such as depression and anxiety. Our previous study confirmed that psychologic symptoms, such as depressed mood among OSA patients can be successfully relieved by surgical intervention. (Laryngoscope 2004;114:1098–1102)
OPTOGENETIC MANIPULATION OF OREXIN NEURONAL ACTIVITY AFFECTS SLEEP/WAKEFULNESS STATE IN MICE

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Edward S. Boyden and Akihiro Yamanaka

Orexin/hypocretin is a neuropeptide produced in the neurons sparsely distributed in the lateral hypothalamic area. The mice lack prepro-orexin or orexin-producing neurons (orexin neurons) show phenotypes, a fragmentation of sleep/wakefulness and sudden attack of muscle weakness. These symptoms are characteristic in human sleep disorder, narcolepsy. These suggest that orexin neurons have an important role in the maintenance of arousal.

To study physiological significance of orexin neuronal activity on maintenance of wakefulness, optogenetic manipulation of orexin neurons was incorporated. We previously generated orexin/halorhodopsin transgenic mice which enable inhibit orexin neuronal activity in vivo. However, halorhodopsin-induced inhibition did not last for more than one minute, probably due to desensitization.

In present study, to archive long lasting silencing of orexin neuronal activity in vivo, orexin/archaerhodopsin-3 transgenic mice were generated. In these mice brain, the orexin neurons specifically express green light-driven proton pump, archaerhodopsin-3. Slice patch clamp analyses of orexin neurons showed that green light illumination completely ceased firing of archaerhodopsin-3-expressing orexin neurons during illumination for up to one hour. In the early dark period (active period), one hour continuous silencing of orexin neuronal activity in vivo induced an increase in total time in slow wave sleep. Although inhibition of orexin neurons for one hour induced a fragmentation of sleep/wakefulness state, cataplexy was not observed. On the other hand, light illumination had no effect on sleep/wakefulness pattern in the light period (an inactive period).

These results suggest that orexin neuronal activity plays an important role in the maintenance of wakefulness especially in the active phase (in the early dark period).
The relationship between sleep and the immune systems has been recognized for millennia; Aristotle wrote in the 4th century BC that fever is commonly accompanied by feelings of tiredness and fatigue. In the late 19th and early 20th centuries, sleep alterations through the course of infection were recognized, and Toth and Krueger systematically explored infection-induced alterations in sleep during the 1980s. These, and many other observations demonstrate that sleep is altered when the immune system is activated. Only recently, however, has it been recognized that sleep loss has a negative impact on the immune system. For example, epidemiologic studies demonstrate that chronic insufficient sleep is associated with increased incidence of cardiovascular disease, hypertension, obesity, metabolic syndromes, insulin resistance, and type 2 diabetes mellitus. The common thread among all these pathologies is neuroinflammation.

The brain was long thought to be an immune-privileged site, but we now know that immunomodulators and their receptors are present in the central nervous system. Immunomodulators function in regulatory/modulatory roles for many behavioral and physiological processes. Important immunomodulators that have been extensively studied with respect to actions in the brain are the cytokines interleukin-1 (IL-1), IL-6, and tumor necrosis factor- (TNF). IL-1, IL-6, and TNF have been implicated in the regulation/modulation of sleep-wake behavior, feeding, sexual behavior, locomotor activity, and other complex neural processes. Gene and protein expression of these cytokines is by even brief periods of sleep disruption, providing the biologic basis for chronic neuroinflammation associated with chronic insufficient sleep.

New animal models and molecular-genetic approaches have made it possible to manipulate cytokine systems in brains of freely-behaving laboratory rodents. Use of these techniques promises to advance our understanding of relationships between sleep loss and inflammation, and provide answers to questions of how chronic insufficient sleep leads to pathologies that have such a negative impact on society.
Controversial sleep disruptions (e.g. poor nighttime sleep and daytime somnolence), which might result from the distinct types of epilepsy and/or the recurrent time of seizure, are common in epilepsy patients. Sleep is regulated by the homeostatic factors, which mediate sleep propensity, and the circadian oscillator, a clocklike mechanism. However, how epilepsy occurred at different zeitgeber time (ZT) points alters sleep regulation remains unknown. In this symposium, we will systematically explore the sleep disruptions and the underlying mechanisms of neuroendocrines by delivering amygdaloid kindling epilepsy, which resembles the temporal lobe epilepsy (TLE), at different ZT points—ZT0, ZT6 and ZT13. The full-blown epilepsy only occurred when kindling delivered. Our results demonstrated that kindled epilepsy occurring at ZT0 and ZT13 predominantly affected the homeostatic factors, whereas ZT6-kindling stimuli altered the circadian oscillator. ZT0-kindling decreased rapid eye movement (REM) and non-REM (NREM) sleep, which was mediated by corticotrophin-releasing hormone (CRH), but did not alter the rhythm of sleep fluctuation. On the other hand, ZT13-kindling enhanced interleukin-1 (IL-1) and consequently increased NREM sleep, without altering the sleep-wake fluctuation. Nevertheless, the expression of Per1 in the suprachiasmatic nucleus (SCN) of the hypothalamus and the rhythm of sleep fluctuation were respectively shifted 6 and 2 hours in advance when kindling stimulation was delivered at ZT6. The shift of sleep circadian rhythm induced by ZT6-kindling was blocked by administration of hypocretin receptor antagonist, SB334867, into the SCN, indicating the involvement of hypocretin. These observations suggest that the occurrence of epilepsy at different ZT points alters sleep processes differently and reveal the role of neuroendocrines.
RESPONSIVENESS OF THE BRAIN TO EXTERNAL STIMULATIONS VIA NEW METHODOLOGICAL APPLICATIONS

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The brain is neither idle nor unconscious during sleep. Indeed there is great deal of cognitive processing during this period. However there has been lack of available methods and cumulative experiments to display these properties of brain during sleep. Recently a number of research teams have started focusing on this task and some insight has been provided. In order to monitor continuous and ever changing conditions of the brain one candidate approach is “brain responsiveness”. The recent methodological applications may shed light onto the multidimensional dynamicity of brain responsiveness. These include new generation extended polysomnographies, Bispectral Index devices, Thermal monitoring and functional near-infrared spectroscopy. Furthermore continuous and adaptive stimulations of the brain enable a very dynamic and ever changing brain conditions during sleep from milliseconds to hours. The measurement of these dynamic responses may explain further the vigilance, the biological rhythms and brain’s cognitive functions. Furthermore these applications are not isolated for only sleep but can be potentially used to elucidate dynamic brain within various conscious states such has coma and anesthesia. Lastly there is a huge room to explore on the brain-body interactions and also metabolism, which in turn may explain some of the biological rhythmic changes. There is a more complex behavior and interaction between brain and the body for the metabolic and vigilant status than current state of knowledge.
EMOTION PROCESSING IN SLEEPING BRAIN

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Given that emotional stress can influence sleep physiology and dream contents and that affective memories are selectively facilitated by rapid eye movement (REM) sleep, the neural mechanism of emotion processing in sleep remains to be determined. Considering mismatch negativity (MMN) as the indicator of automatic sensory processing in sleeping brain, N1/P2 complex as the indicator of auditory information processing, and post MMN Positivity (PMP) as the indicator of involuntary attention switching, the present study was to investigate the emotion processing in sleeping brain.

Using the mismatch negativity (MMN) paradigm, this study enrolled twelve healthy adults while emotionally spoken syllables “dada” with happy prosodies as the standard and fearful prosodies as the deviant along with their corresponding nonvocal sounds were presented during sleeping. Results showed that no significant difference in sleep efficiency in both emotional and nonvocal sessions. N1/P2 complex, as an indicator of auditory information processing, could be detected in all sleep stages with stronger amplitude and shorter latency in emotional session. The sensitivity and specificity of emotional MMN had robust detection in sleep with larger amplitude and earlier latency in sleep. Post MMN positivity (PMP), as an indicator of involuntary attention switch, was robustly detected in all sleep stages with larger amplitude than in wakefulness. Emotional PMP latency in REM was the earliest among sleeping and wakefulness stages. In contrast, the corresponding nonvocal MMN was abolished in sleep except for stage 1. The findings demonstrate that emotional MMN could be preserved in sleeping brain. Emotional MMN could be a direct neurophysiological evidence to support emotion processing during sleep.

Key words: Emotion; voice; REM sleep; Mismatch Negativity (MMN)
HOW TO REALIZE A CAUSALITY OF SEQUENTIAL EVENTS OF BODILY FUNCTIONS WITH SLEEP

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It is well documented that certain brain functions are more active in sleep than in wakefulness. For example, pituitary hormones such as growth hormone and prolactin are released at night, which are under the control of hypothalamic hormones and dopamine. Melatonin, a pineal hormone, is also secreted at night. However, it is not clear whether these endocrine activities are directly related with sleep or independent of sleep but only temporally coincident with sleep under steady state conditions. To explore a causal relationship between nocturnal activities and sleep, we have introduced a specific experimental set-up.

Temporal order of physiology and behaviors in humans are regulated by the circadian pacemaker located in the hypothalamic suprachiasmatic nucleus (SCN). The circadian pacemaker generates an intrinsic oscillation which is entrained by a day-night alternation. The photic signals reach the SCN through the retino-hypothalamic tract and produce the phase-dependent phase shifts in the SCN circadian rhythm. Depending on the internal phase-angle difference between the circadian pacemaker and overt rhythms expressed in a variety of functions, some endocrine and autonomic activities are more pronounced at the time when sleep is most likely to occur than the time when wakefulness is most likely. As a result, these activities are apparently coincided with sleep in terms of time.

Forced desynchrony protocol under temporal isolation is invented to reveal a causal relationship between sleep or wakefulness and brain activity in question. By this protocol, sleep-wake cycles of a subject are artificially phase-shifted by several hours and fixed at a certain time of day. Immediately after the shift, the circadian rhythm of brain activity (e.g., hormone secretion) is measured. If the circadian rhythm of the brain activity is also shifted immediately, a causal relationship is highly plausible. If not, the two phenomena are independent.

For the last 20 years, we have examined the temporal relationship of several brain functions with sleep and found that the most, if not all, functions are more or less independent of sleep and mainly regulated by the circadian pacemaker. Furthermore, there are a number of the so-called peripheral circadian oscillators, each of which directly drives a particular function including behavior and phase-reset by the circadian pacemaker in the SCN. According to the current understanding, the temporal order of physiological functions is regulated by the circadian system in which the peripheral as well as central clocks are involved. This idea may provide a new approach to the so-called “sleep-related” phenomena.
IMPLICIT MEMORY AND TACTILE STIMULATIONS IN SLEEP

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The concept of brain dynamics/responsiveness and cognitive processes in different conscious states (i.e. awake, sleep, anesthesia, coma etc.) is currently a topic of debate. Accordingly, sleep is one of the complex and curious states of the brain and different types of measurement methods should be applied in order to investigate the sleep related dynamic brain activities. These methods can be classified as subjective (i.e psychometric tests) and objective (i.e electrophysiology, neuroimaging etc.).

In this session, as an example to both categories, results will be presented from different projects that are aimed at understanding cognitive processes (and brain responsiveness) during sleep. One such case is the explicit and implicit memory measurements via verbal batteries as a subjective method and the other one is the effect of tactile stimulations during sleep as an objective method. Both of these research initiatives, modification of memory tests and preparation of new designs, collecting data and analyses have been performed at Sleep Dynamics Research Laboratories of Dokuz Eylul University Department of Biophysics (SDRL-DEU).

For the memory measurements, a novel multi-blocked memory assessment design (named Modified Multi Block Design (MMBD)) was utilized by using meaningful and meaningless words in three conditions. Modified versions of Auditory Verbal Learning Test and Word Recognition Test were consecutively applied (1) before, (2) during and (3) after sleep sessions. The implicit memory was measured by means of a modified Word Stem Completion Test during sleep.

For the brain responsiveness to somatosensory stimuli, electrophysiological responses were recorded by using Evoked Potentials (EP) and Event Related Potentials (ERP) experimental designs. The aim of this study was to investigate the brain responsiveness via non-painful tactile stimulations during sleep.

In the light of these different experimental designs, it could be indicated that both subjective and objective approaches provide valuable information. Additionally, these findings help to paint the landscape that sleep is a dynamic process of the brain which does not imply that cognitive processing disappear.
ASSOCIATION BETWEEN OBSTRUCTIVE SLEEP APNEA AND CORONARY ARTERY DISEASE

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Much attention has recently been paid to links between sleep disordered breathing (SDB) and cardiovascular diseases, including hypertension, arrhythmias, coronary artery disease (CAD), heart failure, aortic dissection, pulmonary hypertension, and stroke. Obstructive sleep apnea (OSA), which is a chronic condition characterized by repetitive episodes of upper airway collapse, apnea, and arousal during sleep, is the most frequent SDB, with an approximate prevalence of 10 - 20% (defined as an apnea-hypopnea index ≥5) in the general adult population. The prevalence of OSA is up to 2-fold greater in subjects with CAD than in those without CAD. OSA is also frequently observed in subjects with coronary spastic angina. Physicians should therefore proactively screen for OSA in patients with CAD. OSA may be associated with nocturnal myocardial ischemia, nocturnal onset of acute coronary syndrome, an increased risk for restenosis after percutaneous coronary intervention, and increased mortality and morbidity in subjects with CAD. OSA-induced hypoxia (hypoxia-reoxygenation), hypercapnea, sympathetic activation, and hemodynamic stress (excessively negative intrathoracic pressure up to -80 cmH₂O and resultant increased cardiac transmural pressure) can cause inflammation, oxidative stress, and vascular endothelial injury, all of which could contribute to the development of atherosclerosis and an increased risk for cardiovascular mortality and morbidity. Nasal continuous positive airway pressure therapy, the first-line therapy for OSA, may reduce cardiovascular events in OSA subjects with CAD. However, there have so far been no prospective, randomized, controlled trials of the effects of treatment of OSA on the risks of developing CAD and the cardiovascular mortality and morbidity in subjects with CAD, and such trials are also ethically quite difficult to design and undertake. The precise effects of OSA on CAD and the merits of the treatment of OSA in CAD subjects remain to be further clarified.
OBSTRUCTIVE SLEEP APNEAN AND HYPERTENSION – A MULTICENTER STUDY IN MAINLAND OF CHINA

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To investigate the relationship between OSA and hypertension in Chinese population of OSA patients. A multicenter study was finished with 20 hospitals in different area of China.

The aim of the study: To investigate the prevalence of hypertension among sleep apnea patients and the associated factors. To define the association between daytime blood pressure and severity of OSA in Chinese population in mainland of China.

Methods: A total of 2297 patients(male 1310,female 211)from 20 teaching hospitals were enrolled in this study. Medical history interview, blood pressure measurement and an in-hospital polysomnogram were performed for all the patients. Questionnaires of sleep apnea and hypertension were finished by all the patients. SPSS 11.0 software package was used for data analyzing and prevalence rates of hypertension were compared by chi-square test.

Results:
Part ① The prevalence of hypertension in the apnea hypopnea index(AHI)<5/h group was 23.5%, but in AHI≥5/h group was 49.3%, the difference being significant. The prevalence of hypertension in the group with obstructive sleep apnea hypopnea syndrome(OSAHS) was 56.2%, but was 63.7% and 39.4% in the groups with and without hypertension family history, respectively. The odds ratio of hypertension in AHI≥5/h group was 3.167 times higher than in the AHI<5/h group(OR=3.167,95% CI 2.953-5.426,P<0.01). The prevalence of hypertension increased with AHI increases, and it was the highest in subjects with AHI of 16-20/h, but deceased slowly in subjects with AHI of 66-70/h.

Part ② All the average daytime, nighttime, evening and morning BPs are correlated with AHI positively and negatively with nadir nocturnal oxygen saturation. The ratios of nighttime/daytime and morning/evening average BP are correlated with AHI positively. The ratio of nighttime/daytime systolic BP becomes “reversed BP dipping” pattern until the classification of Severe while the ratio of nighttime/daytime diastolic BP becomes reversed at Moderate.
Conclusions:
① The prevalence of hypertension in subjects with sleep apnea was higher than those without sleep apnea in Chinese population of OSA patients. There was a close relationship between sleep apnea syndrome and the prevalence of hypertension. Sleep apnea was an independent factor for hypertension after control for the confounding factors.

② OSA can result higher BP levels at all time of a day. The ratios of nighttime/daytime and morning/evening BP increase with increased AHI. The increasing of diastolic BP, which inclines to rise more quickly, is not parallel with systolic BP increasing. OSA severity was associated with daytime blood pressure until AHI of 61–65, providing evidence for early OSA management, especially in OSA patients with concomitant hypertension.
Obstructive sleep apnea has been considered as one of the important risk factor for ischemic stroke as well as cardiovascular disorder. The prevalence of OSA in ischemic stroke patients is significantly higher than in the general population although it is variable due to the heterogeneity of the research method and the patients. Causal relationship between acute ischemic stroke and sleep apnea has not been clear yet. However, the in-vitro studies of thrombocyte or endothelial function under repetitive intermittent hypoxia as well as the well known epidemiologic studies showed sleep apnea is strongly associated with ischemic stroke and may influences vascular risk factors through direct or indirect mechanisms.

This lecture will review existing literature on the epidemiology and pathophysiology of sleep apnea in patients with ischemic stroke. In addition, treatment of sleep apnea and the potential improvements of functional outcome responding to the treatment will be discussed.
PERFORMANCE OF THE FREQUENCY DOMAIN INDICES WITH RESPECT TO SLEEP STAGING

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Objective: To compare computerized staging using spectral analyses of various electrophysiological signals with manual sleep staging.

Methods: Sleep recordings from 21 normal subjects were scored by an experienced rater and by a dichotomous algorithm. The performance of the spectral indices was assessed by the largest kappa value (LKV).

Results: Theta/beta power ratio of the electroencephalogram, high frequency power (8-58 Hz) of the electromyogram (PEMG), mean R-R interval, and total power (0-16 Hz) of the body acceleration (PACCE) had high (> 0.5) LKVs when differentiating between waking and sleep. To differentiate sleep with (stage 2 and slow wave sleep) and without (rapid eye movement and stage 1 sleep) spindles, sigma/beta power ratio had high LKVs. PEMG had a medium (> 0.25) LKV to separate rapid eye movement from stage 1 sleep whereas delta/beta power ratio had a high LKV to separate stage 2 and slow wave sleep.

Conclusion: The frequency components of electroencephalogram perform well in identifying sleep, sleep with spindles, and slow wave sleep. Electromyogram, heart rate, and body acceleration offer high agreement only when differentiating between wakefulness and sleep.

Significance: The human-machine agreement is acceptable with spectral parameters, but heart rate and body acceleration still cannot substitute for electroencephalogram.
We developed a technique for estimating sleep level without using electroencephalograms (EEGs). Using a Lorenz plot (LP) of the intervals between heartbeats, known as RR intervals (RRIs) on electrocardiograms (ECGs), during sleep, we confirmed that the distribution on the LP changes according to sleep level. To evaluate the changes in these distributions, RRIs are projected on an LP on both a $y = x$ axis and a $y = -x$ axis, and the shifting of the mean (center C) and standard deviation (area S) is analyzed for each sleep level. A time series value of sleep level is compared with center C and area S. When the sleep level shifts to deeper levels, the variations in center C and area S decrease. Conversely, variations increase when sleep level shifts to lighter levels. In the case of continuously deep levels of sleep, variations in center C remain negligible but variations in area S increase.

Results suggest that there are three characteristic relationships between evaluation indexes (center C, area S) from the LP and sleep level transitions. When sleep level deepens, both center C and area S stabilize. When sleep level lightens, evaluation values do not stabilize. In addition, when sleep levels are steady, center C stabilizes even though area S fluctuates. Therefore, the two evaluation indexes are linked to sleep level transitions, and based on these three patterns, the transition between sleep levels using LP values can be estimated. Standard deviation (SD) can be used as a measure of the stability of the evaluation indexes, and threshold indexes can be defined for quantitative evaluation. Threshold indexes were determined as stability. The threshold index for SD of center C was defined using discriminant analysis. Moreover, the stability of area S was defined by two threshold values because the variation of area S was larger than that for center C.

We also compared heart rate variability (HRV) and sleep level. Center C showed progress toward light sleep levels and area S showed the transition phases toward deep sleep. A concordance rate of 60.9% between the estimated values and actual transitional sleep levels was obtained for all-night sleep. Therefore, transitional sleep levels can be evaluated based on HRV using the LP.
THE RELATIONSHIPS BETWEEN DEPRESSIVE SYMPTOMS, SLEEP ARousAL, AND AUTONOMIC ACTIVITIES IN MAJOR DEPRESSIVE DISORDER

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Objective: Autonomic dysregulation is taken as one of the psychopathological mechanisms for major depressive disorder (MDD) with sleep disturbances. The purposes of this study were: (1) to explore the relationships between depressive mood, sleep quality, pre-sleep arousal, and autonomic activations in MDD patients with sleep disturbance; and (2) to examine the treatment effectiveness of heart rate variability (HRV) biofeedback in MDD patients with sleep disturbance.

Methods: Study 1: Thirty-three MDD patients (36.33 ± 13.75 years, 69.2% were female) completed the Beck Depression Inventory II, Pittsburgh Sleep Quality Index (PSQI), and Pre-Sleep Arousal Scale (PSAS); and then were measured the 5-min baseline of HRV. Study 2: Seven MDD patients (40.29 ± 14.13 years; four were female) attended the six weeks HRV-biofeedback program, and the changes of HRV were be examined.

Results: Poor sleep quality was positively related to total PSAS score, physical arousal and cognitive arousal scores; and negatively related to SDNN and HF. Total PSAS and physical arousal scores were positively related to LF/HF ratio. The MDD patients with high PSQI had lower SDNN than those with low PSQI. After treatment, there were significant decreases in PSQI and PSAS, as well increases in SDNN, LF and HF.

Conclusions: MDD patients with poor sleep quality had lower HRV and parasympathetic activations; higher pre-sleep physical arousal related higher sympathetic activations. HRV-biofeedback is a useful treatment strategy for improving sleep quality and the cardiac autonomic balance.
COMPARISONS OF SHORT TERM EFFICACY BETWEEN INDIVIDUAL AND GROUP COGNITIVE - BEHAVIORAL THERAPY FOR PRIMARY INSOMNIA

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The purpose of this study was to compare the efficacy of individual and group cognitive-behavioral therapy for insomnia (CBT-I) in outpatients with primary insomnia diagnosed by DSM-IV-TR. The participants were 20 individually treated (I-CBT-I) and 25 treated in a group therapy format (3-5 patients per group) (G-CBT-I), which showed no significant difference about demographic variables between groups. The same components of CBT-I, stimulus control therapy, sleep restriction therapy, cognitive therapy and sleep hygiene education, were applied on both groups. The short term outcome (4 weeks after the treatment) was measured by sleep logs, actigraphy, the Japanese version of Pittsburgh Sleep Quality Index (PSQI-J) and Dysfunctional Beliefs and Attitudes about Sleep Scale Japanese version (DBAS-J) and was compared between I-CBT-I and G-CBT-I. The results indicated that CBT-I was effective in improving subjective and objective sleep parameters and subjective sleep evaluations for both individual and group treatment. However, I-CBT-I resulted in significantly better improvements over G-CBT-I, in 1) objective and subjective sleep onset latency time, 2) objective sleep efficacy and moving time during sleeping, 3) overall sleep quality and duration of actual sleep time in PSQI-J, 4) consequences of insomnia, control and predictability of sleep, sleep requirement expectation and sleep-promoting practices in DBAS-J. The present study suggested a superiority of I-CBT-I over G-CBT-I in clinical settings.
THE EFFECT OF COGNITIVE BEHAVIORAL THERAPY FOR INSOMNIA ON VULNERABILITY TO DEVELOPMENT AND RELAPSE OF INSOMNIA

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It is suggested that the stress-induced sleep reactivity is vulnerability to development and relapse of insomnia (Drake et al., 2004). Furthermore, it is suggested that the dysfunctional beliefs about sleep is core beliefs on chronic insomnia (Morin et al., 2003). Nevertheless, the most effective techniques of CBT-I on insomnia is behavioral techniques. We examined the impact of CBT-I on stress-induced sleep reactivity and dysfunctional beliefs about sleep, and the relationship between those changes and sleep improvements.

Outpatients with chronic insomnia who were seen in our sleep clinic were received a biweekly six-session CBT-I. CBT-I included psycho-education, sleep hygiene, relaxation, stimulus control, sleep restriction, contingency management, and coping for worry. Subjective measures including the following variables were evaluated at the baseline and at the end of the treatment; the Pittsburgh Sleep Quality Index (PSQI), the Athens Insomnia Scale (AIS; a cut-off score is 6), the Ford Insomnia Response to Stress Test (FIRST), and the Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS). The FIRST is a self-administered questionnaire to assess stress-induced sleep reactivity and the DBAS assess sleep-related dysfunctional beliefs.

The scores of all the scale decreased significantly at the end of the treatment. 71% participants had the AIS score of 6 points or lower at the end of treatment. Although the score of the DBAS also decreased significantly, there were not the relationship between its change and sleep improvement.

CBT-I improves not only the insomnia symptoms of chronic insomnia, but also stress-induced sleep reactivity which predicts an onset and the relapse of insomnia. Furthermore, the change of dysfunctional beliefs might not relate to sleep improvements.
ASSOCIATION BETWEEN TREATMENT OUTCOME AND CHANGES IN COGNITIVE AND BEHAVIORAL VARIABLES FOLLOWING CBT FOR INSOMNIA

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Cognitive Behavioral Therapy for Insomnia (CBT-I) has been well documented to be an effective treatment for insomnia in different clinical populations. It consists of multiple components that are designed to change sleep-related psychological and behavioral factors associated with insomnia. However, the association between changes in these factors and treatment effect has not been well studied. The findings of two studies exploring the treatment effect of CBT-I conducted in Taiwan will be presented. The first study compared the treatment outcomes of CBT-I only (N = 32), CBT-I combined with medication (N = 71), and medication only (N = 23). The results showed that all three treatments are effective in improving total sleep time (TST) and wake after sleep onset (WASO). The two groups received CBT-I showed better outcomes in sleep efficiency (SE) and the overall rating of insomnia severity as measured by the insomnia severity index (ISI). The second study examined the association between treatment outcome and reduction in dysfunctional sleep beliefs as measured by the Dysfunctional Beliefs and Attitude about Sleep Scale (DBAS), maladaptive sleep-related practices measured by the Sleep Hygiene Practice Scale (SHPS), and pre-sleep arousal levels measured by the Pre-sleep Arousal Scale (PSAS) by CBT-I treatment in primary insomnia (PI; N = 103) and in insomnia comorbid with anxiety and/or depression (CI; N = 25). For PI group, improvement in ISI correlated with decrease of both somatic and cognitive pre-sleep arousal as well as reduction of arousal-related behavior and maladaptive sleep environment. For CI group, ISI improvement correlated with change of scores on the factor I (perceived consequences of insomnia) and factor II (control over sleep) subtests of the DBAS, both somatic and cognitive pre-sleep arousal, arousal-related behavior and poor sleep environment. The results suggest that CBT-I is an effective treatment for insomnia. The treatment effect is primarily associated decrease of arousal probably partially through the reduction of arousal related behaviors. Change in dysfunctional sleep beliefs however seems to be particularly important for insomnia with comorbid psychiatric conditions.
COGNITIVE BEHAVIOR THERAPY FOR SECONDARY INSOMNIA

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Insomnia has a high co-morbidity with psychiatric disorders and chronic medical disorders. Cognitive behavior therapy for insomnia (CBT-I) has proved effective not only in people with primary insomnia but also in people with insomnia co-morbid with psychiatric and medical disorders. Many people with Parkinson’s disease (PD) report difficulties falling asleep or staying asleep, insufficient sleep duration, or nonrestorative sleep. Previous studies reported a high incidence (42-98%) of sleep disturbances in patients with PD. Many PD patients show improvement of motor function after sleep (sleep benefit), which indicates that sleep affects motor control in PD. The co-morbid insomnia has significant negative impact on physical function and on quality of life. Treatment for this co-morbid insomnia is important, but the use of hypnotics should be done carefully, as they have undesirable side effect such as causing falls, sedation and cognitive deficits. CBT-I is a non-pharmacological therapy to avoid using hypnotics in PD patients. However, CBT-I has rarely has tested in those with PD. The present pilot trial was aimed at studying the efficacy of CBT-I in patients with PD. Using a single-case design, a 2-weekly session CBT-I intervention protocol was tested. All patients demonstrated positive treatment response on a measure of insomnia. CBT-I can be effective in treating insomnia associated with PD. I will discuss the utility and the limitation of CBT-I in patients with PD.