



**PRELIMINARY SCIENTIFIC PROGRAM** (Subject to changes – as of February 11, 2024)

| THURSDAY, MARCH 21, 2024 |   |               |
|--------------------------|---|---------------|
| 08:10 – 10:50            | <b>Alzheimer's disease (AD): Biomarkers</b>   | <b>HALL B</b> |
| Chair:                   | <b><u>Marina Janelidze</u></b> , Georgia  |               |
| 08:10-09:00              | Biomarkers are useful in subjective cognitive complaints and should be tested in each patient   |               |
|                          | <i>Capsule:</i> Patients with SCC are at increased risk to develop dementia . it is important to identify who is at risk. Are there any biomarkers which van help?  |               |
| 08:10-08:20              | Moderator: <b><u>Tom Neylan</u></b> , USA<br>Introduction and Pre-Debate Voting   |               |
| 08:20-08:35              | Yes: <b><u>Paul Edison</u></b> , UK   |               |
| 08:35-08:50              | No: <b><u>Zvezdan Pirtosek</u></b> , Slovenia   |               |
| 08:50-09:00              | Discussion, Rebuttals and Post-Debate Voting  |               |
| 09:00-09:50              | Are serum markers such as phospho-tau useful in diagnosing AD ?   |               |
|                          | <i>Capsule:</i> <i>In a chronic medical condition, early diagnosis becomes important when treatment is available that can alter its course. Regarding AD , there is hope that drugs or prevention strategies will have the capacity of slowing down the neurodegeneration. Such treatments may provide greatest benefit to early stage since higher levels of functioning, independence, and quality of life will be maintained. Blood-based biomarkers would be critical in making early diagnosis accessible in routine clinical care. This debate will focus on the central question whether AD can (and should) be diagnosed early based on biomarkers measured in blood.</i> |               |
| 09:00-09:10              | Moderator: <b><u>Xiaoping Wang</u></b> , People's Republic of China<br>Introduction and Pre-Debate Voting   |               |
| 09:10-09:25              | Yes: <b><u>Robert Perneczky</u></b> , Germany   |               |
| 09:25-09:40              | No: <b><u>Arfan Ikram</u></b> , The Netherlands   |               |
| 09:40-09:50              | Discussion, Rebuttals and Post-Debate Voting  |               |
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| 09:50-10:50        | <b>Sleep, Alzheimer's and Dementia – session in cooperation with Alzheimer's Association</b>  |               |
| 09:50-09:55        | Moderators: <b>Claire Sexton</b> , USA; <b>Lea Grinberg</b> , USA   |               |
| 09:55-10:10        | Sleep as risk factor - evidence and interventions: <b>Sharon Naismith</b> , Australia   |               |
| 10:10-10:25        | Neuropathology and neuroimaging of sleep: <b>Neus Falgas</b> , Spain  |               |
| 10:25-10:40        | Sleep in clinical populations: <b>Tom Neylan</b> , USA  |               |
| 10:40-10:50        | Panel Discussion  |               |
|                    |   |               |
| <b>15:00-16:40</b> | <b>AD: Therapy</b>  | <b>HALL B</b> |
| Chairs:            | <b>Panteleimon Giannakopoulos</b> , Switzerland ; <b>Yvonne Freund-Levi</b> , Sweden  |               |
| <b>15:00-15:50</b> | Obstructive sleep apnea is detrimental in patients with dementia and should always be treated   |               |
|                    | <i>Capsule: An overwhelming body of work suggests that obstructive sleep apnea is more prevalent in patients with dementia and may be one of the risks for development of dementia. Whilst the exact mechanics of this bidirectional relationship are not fully understood, several studies advocate that early diagnosis, and early treatment of sleep apnea in patients with dementia may improve their quality of life, and possibly also decelerate the neurodegenerative process. In this debate the major limitations and/or potential contraindications, as well as the most promising aspects of OSA-treatment approach will be discussed.</i>  |               |
| 15:00-15:10        | Moderator: <b>Michael D. Geschwind</b> , USA<br>Introduction and Pre-Debate Voting  |               |
| 15:10-15:25        | Yes: <b>Ivana Rosenzweig</b> , UK   |               |
| 15:25-15:40        | No: <b>Sharon Naismith</b> , Australia  |               |
| 15:40-15:50        | Discussion, Rebuttals and Post-Debate Voting  |               |
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| <b>15:50-16:40</b> | Is cognitive reserve a useful term?   |               |
|                    | <i>Capsule: The concept of reserve was established to account for the observation that a given degree of neurodegenerative pathology may result in varying severities in different individuals. There is a large amount of evidence on epidemiological risk and protective factors for neurodegenerative diseases and dementia, yet the biological mechanisms that underpin the protective effects of certain lifestyle and physiological variables remain poorly understood, limiting the development of more effective preventive and treatment strategies. Additionally, different definitions and concepts of reserve exist, which hampers the coordination of research and comparison of results across studies. Is cognitive reserve just another buzz word or is the phenomenon supported by enough scientific evidence?</i> |               |
| 15:50-16:00        | Moderator: <b>Robert Perneczky</b> , Germany<br>Introduction and Pre-Debate Voting  |               |
| 16:00-16:15        | Yes: <b>Yaakov Stern</b> , USA  |               |
| 16:15-16:30        | No: <b>Amos Korczyn</b> , Israel  |               |
| 16:30-16:40        | Discussion, Rebuttals and Post-Debate Voting  |               |
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| 17:00-18:40 | AD 3  | HALL B |
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| Chairs:     | <b>Judith Aharon</b> , Israel; <b>Milica G. Kramberger</b> , Slovenia   |        |
| 17:00-17:50 | Antiamyloid drugs have only a very limited effect and will not be clinically useful for most patients   |        |
|             | <i>Capsule: Several large clinical trials have demonstrated potential utility of amyloid-targeting approaches in slowing the progression of AD. These treatments may change the course of the disease in some people in the early stages, giving them more time to participate in daily life. However, while promising, these treatments have also been shown to have significant side effects and high cost. In this debate the major limitations as well as the most promising aspects of amyloid-targeting approach will be discussed.</i>   |        |
| 17:00-17:10 | Moderator: <b>John Hardy</b> , UK<br>Introduction and Pre-Debate Voting   |        |
| 17:10-17:25 | Yes: <b>Dorota Religa</b> , Sweden  |        |
| 17:25-17:40 | No: <b>Paul Edison</b> , UK   |        |
| 17:40-17:50 | Discussion, Rebuttals and Post-Debate Voting  |        |
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| 17:50-18:40 | Should lecanemab use be extended beyond 18 months?  |        |
|             | <i>Capsule: There is only one phase 3 randomized trial of lecanemab and one of donanemab. Both were undertaken to support FDA marketing approval; and both have uncontrolled, long-term extended treatment options provided for participants who completed the 18 month trials and wished to continue treatment. As the clinical effects of these antibodies are small and dropouts and adverse events fairly common a question arises about whether treatment should be continued beyond the length of the trials and whether any clinical benefit might become apparent over the long-term. Only a few hundred clinical trials patients have been exposed to these antibodies beyond 18 months; and no regular clinic patient in the USA or Japan could have been exposed to lecanemab for more than 8 months. Thus this issue is ripe for debate as evidence is sparse or absent. This debate might highlight what needs to be considered for better understanding of treatment.</i> |        |
| 17:50-18:00 | Moderator: <b>Zvezdan Pirtosek</b> , Slovenia   |        |
| 18:00-18:15 | Yes: <b>Dorota Religa</b> , Sweden  |        |
| 18:15-18:30 | No: <b>Lon Schneider</b> , USA  |        |
| 18:30-18:40 | Discussion, Rebuttals and Post-Debate Voting  |        |