ALZHEIMER'S DISEASE (AD) AND DEMENTIA

Friday, March 27, 2020	
07:30-08:30	**E-Poster Presentations (Exhibition Area)**
08:30-10:10	**PRECLINICAL AND EARLY ALZHEIMER'S DISEASE (AD)**
Chairs:	**Martin Rossor**, UK | **Shira Knafo**, Spain
08:30-09:20	**Is subjective cognitive impairment itself a prelude to dementia?** *Capsule: In a chronic medical condition, early diagnosis becomes an issue when treatment is available that can alter its course. Regarding AD, there is hope that novel prevention strategies will have the capacity of slowing down the neurodegeneration. Such treatments may provide greatest benefit to patients at the stage of absent or minor cognitive impairment. This debate will focus on the central question, can (and should) AD be diagnosed in the stage of subjective cognitive deficits, although disease-modifying interventions are still unproven?*
08:30-08:40	Host:
08:40-08:55	Yes: **Babak Tousi**, USA
08:55-09:10	No: **Panteleimon Giannakopoulos**, Switzerland
09:10-09:20	Discussion and rebuttals
09:20-10:10	**Is cognitive reserve just a buzzword lacking scientific value?** *Capsule: The concept of reserve was established to account for the observation that a given degree of neurodegenerative pathology may result in varying degrees of symptoms in different individuals. There is a large amount of evidence on 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 strategies. This debate will focus on the important question, is reserve just another buzzword or is the phenomenon supported by convincing scientific evidence.*
09:20-09:30	Host: **Robert Perneczky**, Germany
09:30-09:45	Yes: **Panteleimon Giannakopoulos**, Switzerland
09:45-10:00	No: **Irena Rektorova**, Czech Republic
10:00-10:10	Discussion and rebuttals
10:10-10:25	***Coffee Break***
10:25-12:05	**PATHOPHYSIOLOGY OF AD**
Chairs:	**Nataliya Pryankova**, Ukraine **|** **Gabriel Vainstein**, Israel
10:25-11:15	**The development in understanding AD has not made an impact on patient care.** *Capsule: AD is characterized by cognitive deterioration, but non-cognitive behavioral symptoms are also frequent and often associated with more suffering than cognitive decline. Treatment of those symptoms may be difficult and challenging, and there is insufficient evidence to support treatment decisions. This debate will focus on the question whether the improved understanding of AD pathology has improved patient care and treatment, including non-cognitive symptoms.*
10:25-10:35	Host: **Ruth Itzhaki**, UK
10:35-10:50	Yes: **Judith Aharon**, Israel
10:50-11:05	No: **Sagrario Manzano**, Spain
11:05-11:15	Discussion and rebuttals
11:15-12:05	**Vascular lesions contribute to AD pathology.** *Capsule: Over several years, evidence has been accumulating that suggests vascular disease impacts on common causes of dementia, in particular AD. Modifiable vascular risk factors such as hypertension, diabetes, dyslipidemia and adiposity are all linked to AD. However, it is unclear whether these factors contribute to or promote AD pathology itself or just add vascular damage.*
11:15-11:25	Host: **Nick Fox**, UK
11:25-11:40	Yes: **Raj Kalaria**, UK
11:40-11:55	No: **David Knopman**, USA
11:55-12:05	Discussion and rebuttals

12:05-12:15	***Short Break***
12:15-13:15	**Industry Supported Symposium**
13:15-14:15	***Lunch Break***
13:15-14:15	**Meet the Expert |**
14:15-15:45	**RISK AND PROTECTIVE FACTORS**
Chairs:	**Yvonne Freund-Levi**, Sweden |
14:15-15:00	**Does aerobic exercise protect cognition?** *Capsule: Lifestyle changes have been suggested for dementia prevention. Physical activity engagement has repeatedly been associated with preserved cognition and lower risk for cognitive decline and dementia among older adults. Whether physical activity is neuroprotective or if it mitigates enhanced risk for dementia via other factors is less well understood. This debate will discuss whether physical activity protects cognitive function and whether we know enough about the phenomenon to design effective interventions.*
14:15-14:25	Host: **Miia Kivipelto**, Sweden/UK
14:25-14:40	Yes: **Catherine Robb**, UK
14:40-14:55	No: **Naji Tabet**, UK
14:55-15:00	Discussion and rebuttals
15:00-15:45	**Is deafness a causative risk factor to dementia?** *Capsule: Hearing loss is associated with increased risk for dementia. Other data suggest that there may be a causal link between deafness and cognitive decline but hearing loss may merely be an early symptom of neurodegenerative changes.*
15:00-15:10	Host: **Gill Livingston**, UK
15:10-15:25	Yes: **Sergi Costafreda**, UK
15:25-15:40	No: **Lev Kruglov**, Russia
15:40-15:45	Discussion and rebuttals
15:45-16:00	***Coffee Break***
16:00-17:30	**NEW DEFINITIONS AND APPROACHES**
Chairs:	**Homa Ebrahimi**, Iran |
16:00-16:45	**Is the new NIA-AA research definition of AD helpful?** *Capsule: The 2011 NIA-AA definition of AD was based on clinical symptoms. However, the new 2018 definition eliminates the use of clinical phenotypes and rather depends on biological manifestations like whether amyloid-β (Aβ) and tau pathology or neuroimaging evidence of neurodegeneration exist, regardless of clinical manifestations. Is this change useful?*
16:00-16:10	Host: **Edson Amaro**, Brazil
16:10-16:25	Yes:
16:25-16:40	No: **Mee Park**, South Korea
16:40-16:45	Discussion and rebuttals
16:45-17:30	**Will big data help us to find cure for dementia?** *Capsule: In recent years there has been a surge towards big data approaches in AD, following a general trend in other disciplines. However, not everyone is convinced and the debate on the merits of big data for identifying effective treatments for AD is in full swing. That big collections might generally be useful is not the issue. It was suggested that we can let the data speak for itself. But what does the amassed data actually explain about the underlying pathophysiology and how can it help us identify new drug targets? This debate will focus on the promise of big data initiatives for finding a cure for AD.*
16:45-16:55	Host: **Stefano Sensi**, Italy
16:55-17:10	Yes: **John Gallacher**, UK
17:10-17:25	No:
17:25-17:30	Discussion and rebuttals
17:30-19:00	**FRONTOTEMPORAL DEMENTIA (FTD)**
Chairs:	**Mina Ryten**, UK | **Raul Arizaga**, Argentina

17:30-18:15	**The term Frontotemporal Dementia (FTD) is no longer of value.** *Capsule: FTD is the second most common cause of young-onset dementia, which includes several distinct, heterogeneous sub-syndromes. FTD is characterized by progressive deficits in behavior, language, and executive function, but individual symptoms vary considerably and the underlying pathology is also heterogeneous. Over the last decades, the nomenclature of this group of disorders has undergone several iterations. This debate focusses on the question whether the term FTD is still relevant or if it should be replaced.*
17:30-17:40	Host: **Janine Diehl-Schmid**, Germany
17:40-17:55	Yes:
17:55-18:10	No: **James Rowe**, UK
18:10-18:15	Discussion and rebuttals
18:15-19:00	**All patients with a diagnosis of FTD should have genetic testing.** *Capsule: Approximately 40 percent of affected individuals with FTD have a family history that includes at least one other relative diagnosed with a neurodegenerative disorder. Three genes account for the majority of mutation-associated hereditary FTD cases, including C9orf72, GRN and MAPT and others. Furthermore, a very small percentage of people with sporadic FTD have a mutation in a known FTD gene. This debate focuses on the question if this knowledge about the genetics of FTD is reason enough to perform genetic testing (and counselling) in all patients diagnosed with the disease.*
18:15-18:25	Host: **Julie Williams**, UK
18:25-18:40	Yes: **Lea Grinberg**, Brazil/USA
18:40-18:55	No:
18:55-19:00	Discussion and rebuttals