CONy 2022 Virtual Congress Scientific Program

Program times refer to Central European Time (CET)

FRIDAY, MARCH 25, 2022		
14:00-17:20	**ALZHEIMER'S DISEASE (AD) AND DEMENTIA**	**HALL E**
Chair:	**Robert Perneczky**, Germany	
14:00-14:50	**Is intracellular tau a better treatment target than extracellular tau**	
	Capsule: The microtubule-associated, axonal tau protein accumulates both in the intra- and the extracellular space in AD and other tauopathies. Current disease-modifying immunotherapies mostly target the extracellular protein to inhibit the spreading of tau pathology in the brain. So far, all clinical studies focusing on extracellular tau have failed. The present debate will focus on the pros and cons of extra- vs intracellular tau as a promising treatment target.	
14:00-14:10	Introduction and Pre-Debate Voting	
14:10-14:25	YES: **Einar Sigurdsson**, USA	
14:25-14:40	NO: **Ravi Jagasia**, Switzerland	
14:40-14:50	Discussion, Rebuttals and Post-Debate Voting	
14:50-15:40	**Viruses are a major cause of amyloid deposition in the brain**	
	Capsule: Strong evidence exists for an association between herpes simplex virus type 1 (HSV1) and AD. Very recent epidemiological studies indicate that antiviral treatment for serious HSV1 infections confers protection against AD/dementia, supporting a causal role for the virus.	
14:50-15:00	Introduction and Pre-Debate Voting	
15:00-15:15	YES: **Ruth Itzhaki**, UK	
15:15-15:30	NO: **Magda Tsolaki**, Greece	
15:30-15:40	Discussion, Rebuttals and Post-Debate Voting	
15:40-16:30	**Should findings from monogenic dementias inform sporadic disease research?**	
	Capsule: After age, genes are the second-greatest factors associated with risk of AD. Children of parents with dementia have an increased lifetime risk of symptomatic AD, with greatest risk in those with a maternal history or with two affected parents, in particular if they inherited copies of the APOE ε4 allele. However, in contrast to familial AD, the sporadic form is not associated with mutations in the PSEN1, PSEN2 or APP genes and there is a debate on the usefulness of evidence from monogenic disease to inform sporadic AD research, to be discussed here.	
15:40-15:50	Introduction and Pre-Debate Voting	
15:50-16:05	YES**: Johannes Levin**, Germany	

16:05-16:20	NO: **Amos D. Korczyn**, Israel	
16:20-16:30	Discussion, Rebuttals and Post-Debate Voting	
16:30-17:20	**Is it too early to praise the benefits of gene editing for treatment of sporadic AD?**	
	Capsule: Familial AD is linked with mutations on the PSEN1, PSEN2 and APP genes, resulting in overexpression of pathological amyloid protein. The CRISPR/Cas9 genome editing tool is a powerful technology for correcting inconsistent genetic signatures, offering promising new treatment options for mutation carriers. However, sporadic AD is caused by impaired amyloid clearance from the brain without relevant overproduction. Therefore, it is questionable if gene editing is also promising in sporadic cases, which will be discussed in the present debate.	
16:30-16:40	Introduction	
16:40-17:10	**George Perry**, USA	
17:10-17:20	Discussion	
17:20-19:50	**ALZHEIMER'S DISEASE (AD) AND DEMENTIA**	**HALL E**
Chair	**Jonathan Kennedy**, UK	
17:20-18:10	**Should a person living with advanced stage dementia be allowed to request medical aid in dying while they are still competent, but have it triggered when cognitive function worsens?**	
	Capsule: A person develops advanced stage dementia. Years prior, before (s)he had dementia, she expresses in writing that (s)he does not want to live with this stage dementia. Is it ethical to allow advance care directives that instruct physicians to hasten own death when cognitive problems worsen?	
17:20-17:30	Introduction and Pre-Debate Voting	
17:30-17:45	YES: **Andrew Peterson**, USA	
17:45-18:00	NO: **Marie Nicolini**, USA	
18:00-18:10	Discussion, Rebuttals and Post-Debate Voting	

18:10-19:00	**Does the evidence support a role for the gut microbiome in dementia pathogenesis?**
	Capsule: The gut microbiota comprises a complex community of microorganism species residing in the human gastrointestinal system. Changes of the microbiome may not only be associated with various gut disorders but also with brain diseases such as Parkinson's disease and AD. The increased permeability of the gut and blood-brain barrier induced by microbiota dysbiosis may mediate or affect AD pathogenesis, and imbalances in the gut microbiota could induce neuroinflammation. The present debate will focus on the question if there is enough evidence available to suggest a significant role of the gut microbiome for AD development.
18:10-18:20	Introduction and Pre-Debate Voting
18:20-18:35	YES: **Nikhil Sharma**, UK
18:35-18:50	NO: **Julian Griffin,** UK
18:50-19:00	Discussion, Rebuttals and Post-Debate Voting
19:00-19:50	**Do vascular lesions contribute directly to AD pathology?**
	Capsule: Epidemiological, clinical, and pathological evidence suggest a considerable overlap between cerebrovascular lesions and AD. Both pathologies could have additive and/or synergistic effects on cognitive decline and dementia. Cerebral amyloid angiopathy and small vessel disease are the most frequent vascular pathologies in older age and AD. However, there is an ongoing debate if vascular lesions contribute directly to amyloid pathology, which will be discussed in the present debate.
19:00-19:10	Introduction and Pre-Debate Voting
19:10-19:25	YES: **Ophir Keret**, Israel
19:25-19:40	NO: **Raj Kalaria** UK
19:40-19:50	Discussion, Rebuttals and Post-Debate Voting