CONy 2021 Virtual Congress Scientific Program

Program times refer to Central European Time (CET)

(as of September 24, 2021)

THURSDAY, SEPTEMBER 23, 2021		
14:00-15:20	**SESSION 1**	**HALL A**
14:00-14:10	Opening remarks by CONy President **Amos Korczyn**, Israel	
14:10-14:20	Welcome remarks by CONy Co-Chairs **Derk W. Krieger**, UAE, **Natan Bornstein**, Israel	
14:20-14:40	Frontotemporal dementia (FTD) overview **Bruce Miller**, USA	
14:40-15:00	RNA therapies in neurology **Aida Abu-Baker**, Canada	
15:00-15:20	Treating neurological autoimmune disease with mRNA vaccines **Ari Waisman**, Germany	
15:20-16:45	**SESSION 2**	**HALL A**
15:20-15:25	Introduction **Jera Kruja**, Albania	
15:25-15:45	Breaking the boundaries for stroke management **Derk W. Krieger**, UAE	
15:45-16:05	Small vessel disease of the brain **M. Edip Gurol**, USA	
16:05-16:25	The war of the worlds: Neurodegeneration and the adaptive immune system **Jesse Cedarbaum**, USA	
16:25-16:45	Dopamine and reward **Abdalla Bowirrat**, Israel	

THURSDAY, SEPTEMBER 23, 2021		
16:45-17:45	**SESSION 3**	**HALL A**
	Is alpha-synuclein (α-syn) a useful target for Parkinson's disease (PD) treatment?	
	Capsule: α-syn accumulation in neurons is a hallmark of PD and DLB, and has been suggested to be related to the pathogenesis of these diseases. It has also been claimed that if α-syn can be eliminated, the disease can be prevented or its progression slowed. Is this assumption correct?	
16:45-16:50	Introduction and Pre-Debate Voting: **Natan Bornstein**, Israel	
16:50-17:10	Yes: **Gennaro Pagano**, Switzerland	
17:10-17:30	No: **Amos Korczyn**, Israel	
17:30-17:45	Rebuttals, Discussion and Post-Debate Voting: Led by **Raul Arizaga**, Argentina; **Natan Bornstein,** Israel	

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THURSDAY, SEPTEMBER 23, 2021		
14:40-16:25	**ALZHEIMER'S ASSOCIATION AND CONy JOINT MEETING ON FTD - SESSION 1**	**HALL B**
Chairs:	**Amos Korczyn**, Israel; **Bruce Miller**, USA	
14:40-14:55	Alzheimer Association Presentation **Heather Snyder**, USA; **Claire Sexton**, USA	
14:55-15:15	Neuropsychological testing in FTD. **Adam Staffaroni**, USA	
15:15-15:25	Discussion	
15:25-15:45	Is FTD one entity or more? **Jonathan Rohrer**, UK	
15:45-15:55	Discussion	
15:55-16:15	The study of the genetics of FTD is over. Or is it just beginning? **Jennifer Yokoyama**, USA	
16:15-16:25	Discussion	
16:25-18:00	**ALZHEIMER'S ASSOCIATION AND CONy JOINT MEETING ON FTD – SESSION 2**	**HALL B**
Chair:	**Judith Aharon**, Israel	
16:25-16:45	Neuropathology in FTD: Overview **Lea Grinberg**, Brazil/USA	
16:45-16:55	Discussion	
16:55-17:15	Prodromal FTD **Eino Solje**, Finland	
17:15-17:25	Discussion	
17:25-17:45	FTD and ALS **Rebekah Ahmed**, Australia	
17:45-18:00	Discussion	

18:00-19:30	**ALZHEIMER'S ASSOCIATION AND CONy JOINT MEETING ON FTD – SESSION 3**	**HALL B**
Chair:	**Morris Freedman**, Canada	
18:00-18:20	Imaging biomarkers in FTD **Martina Bocchetta**, UK	
18:20-18:30	Discussion	
18:30-18:50	Emerging biomarkers in FTD **Adolfo Garcia**, USA	
18:50-19:00	Discussion	
19:00-19:30	Highlights of the Day **Amos Korczyn,** Israel	

FRIDAY, SEPTEMBER 24, 2021		
14:00-16:30	**HEADACHE**	**HALL A**
Chair:	**Christopher Gottschalk**, USA	
14:00-14:50	**Migraine without aura originates outside of the brain**	
	Capsule: Migraine attacks have a peripheral and then a central phase; while cortical spreading depression (CSD) probably underlies the experience of aura and can trigger migraine pain, attacks of migraine without aura were suggested to start in the peripheral nervous system.	
14:00-14:05	Introduction and Pre-Debate Voting	
14:05-14:20	Yes: **Patricia Pozo-Rosich**, Spain	
14:20-14:35	No: **Hayrunnisa Bolay**, Turkey	
14:35-14:50	Rebuttals, Discussion and Post-Debate Voting	
14:50-15:40	**Post traumatic migraine is just migraine uncovered by trauma**	
	Capsule: Headache is the most common disorder reported after head trauma in both military and civilian populations. In both cases, a prior migraine history increases the likelihood of persistent post-traumatic migraine. Is traumatic brain injury (TBI) simply a trigger for the unmasking of an underlying disorder?	
14:50-14:55	Introduction and Pre-Debate Voting	
14:55-15:10	Yes: **Oved Daniel**, Israel	
15:10-15:25	No: **Hakan Ashina**, Denmark	
15:25-15:40	Rebuttals, Discussion and Post-Debate Voting	
Chair:	**Messoud Ashina**, Denmark	
15:40-16:30	**Low CSF pressure headache syndromes are frequent and often missed**	
	Capsule: Spontaneous or secondary intracranial hypotension (SIH) was considered rare when limited to patients with clearly orthostatic headache who have diffuse meningeal enhancement on MRI. Are we missing cases with less common presentations?	
15:40-15:45	Introduction and Pre-Debate Voting	
15:45-16:00	Yes: **Jose Miguel Lainez**, Spain	
16:00-16:15	No: **Dimos Mitsikostas**, Greece	
16:15-16:30	Rebuttals, Discussion and Post-Debate Voting	

16:30-20:20	**HEADACHE**	**HALL A**
Chair:	**Messoud Ashina**, Denmark	
16:30-17:20	**Estrogen is a safe and effective therapy for menstrually related migraine**	
	Capsule: Migraine activity frequently relates to hormone status. The incidence in women skyrockets at menarche and menstruation and drops off after menopause. For women with menstrually related migraine, should we be using estrogen preparations to mitigate the effects of endogenous hormone fluctuation – or is it dangerous?	
16:30-16:35	Introduction and Pre-Debate Voting	
16:35-16:50	Yes: **Simona Sacco**, Italy	
16:50-17:05	No: **Cristina Tassorelli**, Italy	
17:05-17:20	Rebuttals, Discussion and Post-Debate Voting	
17:20-18:10	**Monoclonal antibodies to CGRP or its receptor are effective and safe in treating migraine patients**	
	Capsule: Monoclonal antibodies against CGRP are effective in reducing migraine attacks. Should they be used for patients with heart disease, hypertension & hypercholesterolemia?	
17:20-17:25	Introduction and Pre-Debate Voting	
17:25-17:40	Yes: **Laine Green**, USA	
17:40-17:55	No: **Larry Robbins**, USA	
17:55-18:10	Rebuttals, Discussion and Post-Debate Voting	
Chair:	**Alan Rapoport**, USA	
18:10-19:00	**Monoclonal antibodies to CGRP can improve comorbid depression and anxiety in migraine**	
	Capsule: It is common to diagnose anxiety and/or depression in migraine patients, especially those with chronic headache. Do the monoclonal antibodies against CGRP work well to also reduce levels of depression and anxiety in these patients?	
18:10-18:15	Introduction and Pre-Debate Voting	
18:15-18:30	Yes: **Jack Schim**, USA	
18:30-18:45	No: **Andrea Harriott**, USA	
18:45-19:00	Rebuttals, Discussion and Post-Debate Voting	

19:00-19:50	**Targeting the CGRP ligand itself may be a more efficacious and/or safer option for migraine prevention than targeting the CGRP receptor** *Supported by an unrestricted educational grant from Teva Pharmaceuticals*
	Capsule: In recent years, CGRP has been shown to have a central role in migraine, and clinical improvement resulted from blocking it, either by eliminating it altogether or by blocking its receptors. Which method is advantageous?
19:00-19:05	Introduction and Pre-Debate Voting
19:05-19:20	Yes: **Piero Barbanti**, Italy
19:20-19:35	No: **Uwe Reuter**, Germany
19:35-19:50	Rebuttals, Discussion and Post-Debate Voting
19:50-20:20	Highlights of the day: **Oved Daniel**, Israel

FRIDAY, SEPTEMBER 24, 2021		
14:15-18:00	**ALZHEIMER'S ASSOCIATION AND CONy JOINT MEETING ON FTD – SESSION 4**	**HALL B**
Chair:	**Noa Bregman**, Israel	
14:15-14:35	Cultural issues in diagnosis FTD in low and middle income countries **Lina Zapata**, Colombia	
14:35-14:45	Discussion	
14:45-15:05	Psychiatric perspectives in FTD **Yolande Pijnenburg**, The Netherlands	
15:05-15:15	Discussion	
15:15-15:35	Why do people with FTD become artistic? **Adit Friedberg**, USA	
15:35-15:45	Discussion	
15:45-16:05	FTD and the law **Chiadi U. Onyike**, USA	
16:05-16:15	Discussion	
16:15-16:35	Advances towards disease-modifying MAPT-targeted therapeutics – obstacles and prospects **Leticia Toledo-Sherman**, USA	
16:35-16:55	Advances in the development of therapeutics for progranulin-deficient FTD **Laura Mitic**, USA	
16:55-17:15	Treatment for FTD: When will it become available? **Adam Boxer**, USA	
17:15-17:25	Discussion	
17:25-18:00	Highlights of the day **Heather Snyder**, USA; **Claire Sexton,** USA	

FRIDAY, SEPTEMBER 24, 2021		
14:00-15:30	**PARKINSON'S DISEASE (PD) I**	**HALL C**
Chair:	**Angelo Antonini**, Italy	
14:00-14:45	**Clinical assessment in PD: Motor assessment is the key and nonmotor is marginal**	
	Capsule: PD is primarily a motor disorder, yet non-motor symptoms (NMS) become more widely recognized. How important are these NMS in the clinical assessment of the patient?	
14:00-14:05	Introduction and Pre-Debate Voting	
14:05-14:20	Yes: **Tanya Gurevich**, Israel	
14:20-14:35	No: **Ray Chaudhuri**, UK	
14:35-14:45	Rebuttals, Discussion and Post-Debate Voting	
14:45-15:30	**Is there a role for stereotactic ablation in movement disorders in the deep brain stimulation (DBS) era?**	
	Supported by an unrestricted educational grant from Insightec	
	Capsule: DBS was introduced as an alternative to ablative therapy for tremor. The reversibility of DBS and the ability to adjust the implanted stimulator was appealing. But over time the invasiveness, adverse events profile and the high cost of the procedure became apparent. The introduction of minimally invasive ablative treatments such as MRI guided focused ultrasound has raised the question of whether it is time to reintroduce ablative procedures as an alternative to DBS.	
14:45-14:50	Introduction and Pre-Debate Voting	
14:50-15:05	Yes: **Amos Korczyn**, Israel	
15:05-15:20	No: **Fiona Gupta**, USA	
15:20-15:30	Rebuttals, Discussion and Post-Debate Voting	

15:30-18:00	**PARKINSON'S DISEASE (PD) I**	**HALL C**
Chair:	**Stuart Isaacson**, USA	
15:30-16:30	**Device aided therapies in APD, a no brainer for earlier use?** *The session is sponsored by Britannia*	
	Capsule: Device-aided therapies (intraduodenal or transdermal levodopa, subcutaneous apomorphine or intracranial stimulation) are traditionally used for treatment of PD patients in the advanced stages. Should they be used earlier?	
15:30-15:35	Introduction and Pre-Debate Voting	
15:35-15:55	Yes: **Tobias Warnecke**, UK	
15:55-16:15	No: **Zvezdan Pirtosek**, Slovenia	
16:15-16:30	Rebuttals, Discussion and Post-Debate Voting	
16:30-17:15	**Molecular imaging is an expensive tool with poor external validity and not relevant for clinical use**	
	Capsule: The main manifestations of PD are the result of a dopamine deficiency, which can be demonstrated by molecular imaging, and then treated successfully by levodopa. But the therapeutic effect can be shown just as well without imaging.	
16:30-16:35	Introduction and Pre-Debate Voting	
16:35-16:50	Yes: **Pramod Pal**, India	
16:50-17:05	No: **Nicola Pavese**, UK	
17:05-17:15	Rebuttals, Discussion and Post-Debate Voting	
17:15-18:00	**Should PD patients carrying GBA mutations be treated differently from gene mutation negatives?**	
	Capsule: GBA mutations acting through lysosomal pathways are known to contribute to a minority of PD cases. Should there be an attempt to activate lysosomes in order to protect against PD?	
17:15-17:20	Introduction and Pre-Debate Voting	
17:20-17:35	Yes: **Leonidas Stefanis**, Greece	
17:35-17:50	No: **Jaime Kulisevsky**, Spain	
17:50-18:00	Rebuttals, Discussion and Post-Debate Voting	

18:00-18:45	**PARKINSON'S DISEASE (PD) I**	**HALL C**
Chair:	**Angelo Antonini**, Italy	
18:00-18:45	**Should continuous dopaminergic stimulation replace pulsatile once motor fluctuations develop?**	
	Capsule: Dopaminergic stimulation is the key treatment for PD. However, it is still debatable whether pulsatile stimulation contributes to motor complications. Should patients be treated with continuous therapies as soon as OFF episodes and dyskinesia emerge?	
18:00-18:05	Introduction and Pre-Debate Voting	
18:05-18:20	Yes: **Jaime Kulisevsky**, Spain	
18:20-18:35	No: **Nestor Galvez-Jimenez**, USA	
18:35-18:45	Rebuttals, Discussion and Post-Debate Voting	
18:45-19:45	**PARKINSON'S DISEASE (PD) I**	**HALL C**
	Deep brain stimulation and telemedicine *Supported by an unrestricted educational grant from Abbott*	
	Patients centered care and technology: How to combine **Rajesh Pahwa**, USA	
	Beyond traditional clinical measures for patients selection **Angelo Antonini**, Italy	
	Remote DBS programming is it feasible? **Fiona Gupta**, USA	
	Discussion	

FRIDAY, SEPTEMBER 24, 2021		
14:00-16:15	**MULTIPLE SCLEROSIS (MS)**	**HALL D**
Chair:	**Olaf Stüve**, USA	
14:00-14:45	**Serum NfL should replace brain MRI in monitoring MS disease activity**	
	Capsule: NFL is a marker of neurodegeneration, which has been shown to be elevated in MS. Can it reflect disease activity in the same way that is done by repeated MRI scans?	
14:00-14:05	Introduction and Pre-Debate Voting	
14:05-14:20	Yes: **Ide Smets**, The Netherlands	
14:20-14:35	No: **Jacek Losy**, Poland	
14:35-14:45	Rebuttals, Discussion and Post-Debate Voting	
14:45-15:30	**Optical coherence tomography (OCT) adds to diagnostic certainty in MS**	
	Capsule: OCT is frequently impaired after optic nerve lesions, including clinical or subclinical neuritis. It has been shown to be impaired in MS. Can it add to the certainty of the diagnosis in patients who fulfill the McDonald criteria?	
14:45-14:50	Introduction and Pre-Debate Voting	
14:50-15:05	Yes: **Frederike Oertel**, Germany	
15:05-15:20	No: **Alexander U. Brandt**, USA	
15:20-15:30	Rebuttals, Discussion and Post-Debate Voting	
15:30-16:15	**Is it possible to balance beneficial effects of disease modifying therapies (DMT) on the course of MS with fears concerning potential risks to the fetus or child?**	
	Capsule: The modern drugs against MS have presumed disease modifying effects, yet their safety to the fetus is still unknown. Should patients planning pregnancy be encouraged to use them?	
15:30-15:35	Introduction and Pre-Debate Voting	
15:35-15:50	Yes: **Kerstin Hellwig**, Germany	
15:50-16:05	No: **Celia Oreja-Guevara**, Spain	
16:05-16:15	Rebuttals, Discussion and Post-Debate Voting	

16:15-18:15	**MULTIPLE SCLEROSIS (MS)**	**HALL D**
Chair:	**Klaus Schmierer**, UK	
16:15-17:00	**Remyelination/neuroprotection are realistic prospects in people with MS** *Supported by an unrestricted educational grant from Sanofi*	
	Capsule: While the existing drugs are very efficacious against MS relapses, disability results from the demyelination and neurodegeneration. Can these be expected to respond to therapy?	
16:15-16:20	Introduction and Pre-Debate Voting	
16:20-16:35	Yes: **Konrad Rejdak**, Poland	
16:35-16:50	No: **Alicja Kalinowska**, Poland	
16:50-17:00	Rebuttals, Discussion and Post-Debate Voting	
17:00-17:45	**We should test for myelin oligodendrocyte glycoprotein (MOG) and aquaporin-4 AQ4 antibodies in all patients with inflammatory spinal cord and/or optic nerve lesions**	
	Capsule: Myelin oligodendrocyte glycoprotein (MOG) and aquaporin-4 (AQ4) antibodies are seen in patients suffering from demyelinating diseases, sometimes mistaken for MS. Can we diagnose these diseases in confidence or should all patients suspected of suffering from MS be tested for the occurrence of these antibodies?	
17:00-17:05	Introduction and Pre-Debate Voting	
17:05-17:20	Yes: **Ruth Dobson**, UK	
17:20-17:35	No: **Dimitrios Karussis**, Israel	
17:35-17:45	Rebuttals, Discussion and Post-Debate Voting and rebuttals	
17:45-18:15	Highlights of the day: **Klaus Schmierer**, UK	

FRIDAY, SEPTEMBER 24, 2021		
14:00-17:00	**STROKE**	**HALL E**
Chair:	**Laszlo Csiba**, Hungary	
14:00-14:45	**The risk of rethrombosis in COVID positive stroke patients is very high. Should reperfusion therapy be followed by immediate anticoagulation?**	
	Capsule: Some patients with stroke related to acute COVID-19 infection show thrombi in multiple arterial territories, including the intracranial vessels. There is some suggestion that the early use of anticoagulation following an acute stroke may reduce the risk of recurrence. Early anticoagulation in a large stroke may also increase the risk of hemorrhagic conversion. This debate will focus on the advantages and disadvantages of very early anticoagulation in acute stroke in patients with active COVID-19 infections.	
14:00-14:05	Introduction and Pre-Debate Voting	
14:05-14:20	Yes: **Seby John**, UAE	
14:20-14:35	No: **Derk W. Krieger**, UAE	
14:35-14:45	Rebuttals, Discussion and Post-Debate Voting	
14:45-15:30	**Can we mitigate the risk of stroke due to tobacco consumption by other methods of tobacco use without combustion?**	
	Capsule: Smoking tobacco is an established risk factor for stroke as well as for other cardiovascular and non-cardiovascular diseases. The best way to prevent smoking related disease (and stroke) is to stop smoking and even better not to start smoking at all. However it is very difficult for actual smokers to kick the habit. This panel is intended to review and debate whether other methods of tobacco use (without combustion) could affect smoking related disease and whether those methods should be accepted by regulatory authorities.	
14:45-14:55	Introduction and Pre-Debate Voting: **Dov Gavish**, Israel	
14:55-15:07	Yes: **Nebojsa Tasic**, Serbia	
15:07-15:19	No: **Dov Gavish**, Israel	
15:19-15:30	Rebuttals, Discussion and Post-Debate Voting	

15:30-16:15	**Should GLP1 agonists be part of the treatment in stroke prevention in patients with diabetes?** *Sponsored by Novo Nordisk*
	Capsule: Stroke prevention strategies have focused on management of traditional risk factors and the use of antithrombotic medications. Glucagon-like peptide 1(GLP-1) agonists are a new class of medications in the treatment of diabetes. Early studies suggest that they may have potent effects on slowing atherosclerosis. Is there sufficient evidence for the use of GLP-1 agonists in the prevention of stroke or TIA in patients with diabetes?
15:30-15:35	Introduction and Pre-Debate Voting
15:35-15:50	Yes: **Natan Bornstein**, Israel
15:50-16:05	No: **Daniel Bereczki**, Hungary
16:05-16:15	Rebuttals, Discussion and Post-Debate Voting
16:15-17:00	**Patients requiring acute stroke care should be transferred to the closest hospitals rather than to an accredited stroke center**
	Capsule: Time from symptoms-onset is an important factor for best outcome in reperfusion strategies. It is also known that the risk of complications is lower when treatment is initiated in stroke centers with experience and qualified personnel. Bypassing nearby hospitals may however result in delays that may affect good outcome.
16:15-16:20	Introduction and Pre-Debate Voting
16:20-16:35	Yes: **Amal Al Hashmi**, Oman
16:35-16:50	No**: Derk Krieger**, UAE
16:50-17:00	Rebuttals, Discussion and Post-Debate Voting

17:00-20:00	**STROKE**	**HALL E**
Chair:	**Ashfaq Shuaib**, Canada	
17:00-17:45	**Lifestyle changes including exercise and diet are essential components of secondary stroke prevention program**	
	Capsule: It is widely accepted that obesity, poor diet and lack of exercise are risk factors for stroke, but are rarely enforced to prevent stroke recurrence. Should we be more proactive or is it useless?	
17:00-17:05	Introduction and Pre-Debate Voting	
17:05-17:20	Yes: **Vida Demarin**, Croatia	
17:20-17:35	No: **Bartlomiej Piechowski-Jozwiak**, UAE	
17:35-17:45	Rebuttals, Discussion and Post-Debate Voting	
17:45-18:35	**In wake-up strokes, CT perfusion is sufficient for decision making for thrombolysis**	
	Capsule: Recent reports suggest that carefully selected patients presenting with acute stroke symptoms upon awakening ("wake-up stroke") show benefit with thrombolysis. Selection requires advanced imaging including MRI or CT-Perfusion (CTP). Patients in the original studies required only MRI for selection. There are emerging data that CTP may be sufficient for selection of wake-up stroke patients for successful thrombolysis. Does the convenience and better availability of CT allow for recommending CTP instead of MRI?	
17:45-17:50	Introduction and Pre-Debate Voting	
17:50-18:05	Yes: **Ashfaq Shuaib**, Canada	
18:05-18:20	No: **Patrik Michel**, Switzerland	
18:20-18:35	Rebuttals, Discussion and Post-Debate Voting	

18:35-19:20	**Blood pressure control in acute ischemic stroke patients receiving reperfusion therapy is critically important**
	Capsule: Blood pressure (BP) is almost always elevated following acute stroke and its autoregulation is impaired but spontaneous decrease is expected. Pharmacotherapy is suggested only by extreme values, but the optimal BP values, the length and intensity of blood pressure control after reperfusion therapy, are not well established.
18:35-18:40	Introduction and Pre-Debate Voting
18:40-18:55	Yes: **Georgios Tsivgoulis** Greece
18:55-19:10	No: **Csaba Farsang**, Hungary
19:10-19:20	Rebuttals, Discussion and Post-Debate Voting
19:20-20:00	Highlights of the day: **Ashfaq Shuaib**, Canada

SATURDAY, SEPTEMBER 25, 2021		
14:00-15:30	**ALZHEIMER'S DISEASE AND DEMENTIA**	**HALL A**
Chair:	**Robert Perneczky**, Germany	
14:00-14:45	**Primary age-related tauopathy (PART): Is it part of the AD spectrum?**	
	Capsule: PART is a relatively recent concept that was introduced as a pathologic diagnosis to describe brains with mild to moderate neurofibrillary tangle pathology (Braak stage ≤ 4) without significant β-amyloid burden. However, focal tau accumulation is a frequent finding in older individuals, and most people with PART do not develop dementia. Higher Braak stage is associated with older age and more severe cognitive impairment and the rate of cognitive deterioration seems to be slower compared to patients with AD. Therefore, it is still unclear whether PART is a distinct entity or merely early-stage AD. This debate will cover this important question.	
14:00-14:05	Introduction and Pre-Debate Voting	
14:05-14:20	Yes: **Lea Grinberg**, USA/Brazil	
14:20-14:38	No: **Magda Tsolaki**, Greece	
14:38-14:50	Rebuttals, Discussion and Post-Debate Voting	
14:50-15:05	Do sleep disturbances contribute to neurodegeneration in AD? **Lea Grinberg**, USA / Brazil	
15:05-15:30	Break	

16:30-19:15	**ALZHEIMER'S DISEASE AND DEMENTIA**	**HALL A**
Chair:	**David Knopman,** USA	
16:30-17:15	**Will upcoming anti-amyloid treatments also fail?**	
	Capsule: There is ample evidence that AD (co-)pathology is the most prevalent pathological change in older individuals with dementia, and there is a credible correlation between AD-type pathology and cognitive/clinical decline. However, studies also show that this relationship is weaker in the oldest old. The assumption of clear-cut dementia subtypes is put into question by biomarker and neuropathological research suggesting that a substantial proportion of clinically 'pure' AD cases have mixed pathology at autopsy and that β-amyloid is commonly found in cognitively normal older adults. This debate will focus on the key question whether β-amyloid is a central characteristic of AD and if future anti-amyloid treatments also doomed to fail.	
16:30-16:35	Introduction and Pre-Debate Voting	
16:35-16:50	Yes: **David Knopman**, USA	
16:50-17:05	No: **Jesse Cedarbaum**, USA	
17:05-17:15	Rebuttals, Discussion and Post-Debate Voting	
17:15-18:00	**Will genetics play a key role in the diagnosis and clinical management of AD and in the discovery of new drug targets?**	
	Capsule: Mutations in the PSEN1, PSEN2 and APP genes cause familial AD with an early onset following a Mendelian inheritance pattern. For sporadic late-onset AD, the APOE ε4 allele was identified three decades ago as the main susceptibility genetic factor. Large genome-wide association studies have more recently identified over 30 common genetic loci with smaller effects. Furthermore, rare variants are also known that are associated with the disease. These advances have led to an improved understanding of the biological pathways underpinning disease pathogenesis. This debate will focus on the relevance of genetics in developing more effective AD treatment options.	
17:15-17:20	Introduction and Pre-Debate Voting	
17:20-17:35	Yes: **Richard Pither**, UK	
17:35-17:50	No: **George Perry**, USA	
17:50-18:00	Rebuttals, Discussion and Post-Debate Voting	

18:00-18:45	**Do existing clinical trial data support the effectiveness of Aducanumab treatment in AD?**
	Capsule: Positive news is relatively rare in AD research. Twenty years have passed since the last drug approval in this area. Since then all clinical trials have failed, despite some compounds showing initial promising results. Aducanumab is the first new drug with a potential for disease-modification that has a real chance for approval. After the initial announcement of a failed trial, the study data were re-analysed and have shown a positive signal which has led to approval of the drug. This debate will discuss whether the available data support the clinical effectiveness of Aducanumab.
18:00-18:05	Introduction and Pre-Debate Voting
18:05-18:20	Yes: **Jeff Cummings**, USA
18:20-18:35	No: **Peter Whitehouse**, USA
18:35-18:45	Rebuttals, Discussion and Post-Debate Voting
18:45-19:15	Highlights of the day: **David Knopman**, USA

SATURDAY, SEPTEMBER 25, 2021		
14:00-15:30	**NEUROIMMUNOLOGY**	**HALL B**
Chair:	**Dimitrios Karussis**, Israel	
14:00-14:45	**All patients with neuromyelitis optica spectrum disorder (NMOSD) should receive regulatory-approved medications proved by clinical trials. Patients who are stable on not approved immunotherapies should be switched to regulatory approved medications**	
	Capsule: Since the identification of NMOSD as a disease separate from multiple sclerosis, patients have been treated by immune suppression by drugs such as azathioprine. Recently, new drugs have been developed and found to be effective in NMOSD by RCT's and these received approval. Should patients who have been stable on non-specific off-label drugs be switched to the new, more expensive medications?	
14:00-14:05	Introduction and Pre-Debate Voting	
14:05-14:20	Yes: **Uros Rot**, Slovenia	
14:20-14:35	No: **Dimitrios Karussis**, Israel	
14:35-14:45	Rebuttals, Discussion and Post-Debate Voting	
14:45-15:30	**AQP4-IgG NMOSD is an exclusively relapsing disease (as opposed to a progressive disease)**	
	Capsule: The natural history of NMOSD is still being explored. While most patients have a disease characterized by relapses, it is still unknown whether chronic progressive form of the disease also occurs.	
14:45-14:50	Introduction and Pre-Debate Voting	
14:50-15:05	Yes: **Nikolas Grigoriadis**, Greece	
15:05-15:20	No: **Friedemann Paul**, Germany	
15:20-15:30	Rebuttals, Discussion and Post-Debate Voting	

16:30-20:00	**NEUROIMMUNOLOGY**	**HALL B**
Chair:	**Brian Weinshenker**, USA	
16:30-17:15	**MOG-IgG is a highly specific and reliable indicator of a specific CNS inflammatory disorder**	
	Capsule: MOG-IgG is an autoantibody that has been associated with demyelination in animal models. Recently, it has been associated with a variety of syndromes, including NMOSD, recurrent optic neuritis, acute disseminated encephalomyelitis and certain forms of autoimmune encephalitis? However, the specificity, especially at low titers, is controversial. How specific is MOG-IgG and should this influence recommendations for testing?	
16:30-16:35	Introduction and Pre-Debate Voting	
16:35-16:50	Yes: **Yael Hacohen**, UK	
16:50-17:05	No: **Eoin Flanagan**, USA	
17:05-17:15	Rebuttals, Discussion and Post-Debate Voting	
17:15-18:00	**Targeted monoclonal antibodies are superior to conventional immunotherapies for myasthenia gravis (MG)**	
	Capsule: MG is an immune disorder caused by specific antibodies, which have conventionally been treated by non-specific immunosuppressants. Recently, specific monoclonal antibodies have been developed. Are they superior to the well-known drugs?	
17:15-17:20	Introduction and Pre-Debate Voting	
17:20-17:35	Yes: **Brian Weinshenker**, USA	
17:35-17:50	No: **Joab Chapman**, Israel	
17:50-18:00	Rebuttals, Discussion and Post-Debate Voting	
18:00-18:45	**All patients with chronic inflammatory demyelinating polyneuropathy (CIDP) should be tested for autoantibodies to nodal proteins because the results influence therapy**	
	Capsule: CIDP is caused by a variety of antibodies, in some cases targeting nodal or paranodal proteins. Is identification of these patients important?	
18:00-18:05	Introduction and Pre-Debate Voting	
18:05-18:20	Yes: **Brian Weinshenker**, USA	
18:20-18:35	No: **Joab Chapman**, Israel	
18:35-18:45	Rebuttals, Discussion and Post-Debate Voting	

18:45-19:30	**A therapeutic trial of corticosteroids is an effective strategy in individuals with suspected immune-mediated autoimmune encephalitis regardless of the identification of pathogenic autoantibodies**
	Capsule: Immune-mediated encephalitis is being recognized as a heterogeneous disorder, involving different autoantibodies. In many cases, identifying the specific antibody is delayed or unsuccessful. Should finding the antibody be attempted before therapy is initiated or should therapy not be delayed?
18:45-18:50	Introduction and Pre-Debate Voting
18:50-19:05	Yes: **Avi Gadoth**, Israel
19:05-19:20	No: **Anastasia Zekeridou**, USA
19:20-19:30	Rebuttals, Discussion and Post-Debate Voting
19:30-20:00	Highlights of the day: **Brian Weinshenker**, USA

SATURDAY, SEPTEMBER 25, 2021		
14:00-15:30	**EPILEPSY**	**HALL C**
	Supported by an unrestricted educational grants from Neurelis and SK Life Science	
Chair:	**Alla Guekht**, Russia	
14:00-14:45	**Home/ambulatory seizure detection devices should be regularly recommended for people with epilepsy**	
	Capsule: Several devices have been developed that detect seizures when worn in the home environment. Do they offer an advantage to patients? Should we regularly advise using them?	
14:00-14:05	Introduction and Pre-Debate Voting	
14:05-14:20	Yes: **Michael Sperling**, USA	
14:20-14:35	No: **Sandor Beniczky**, Denmark	
14:35-14:45	Rebuttals, Discussion and Post-Debate Voting	
14:45-15:30	**Are newer antiseizure medications preferable to older ones when they are based on the same molecules?**	
	Capsule: Several drugs were developed using older molecules as a starting point. For example, brivaracetam is based on levetiracetam which is based on piracetam; eslicarbazepine shares a similar structure as oxcarbazepine, which is similar to carbamazepine. Should these newer, generally more expensive molecules be used in preference to the older drugs?	
14:45-14:50	Introduction and Pre-Debate Voting	
14:50-15:05	Yes: **Jacqueline French**, USA	
15:05-15:20	No: **Martin Brodie**, UK	
15:20-15:30	Rebuttals, Discussion and Post-Debate Voting	

16:30-20:00	**EPILEPSY**	**HALL C**
	Supported by an unrestricted educational grants from Neurelis and SK Life Science	
Chair:	**Michael Sperling**, USA	
16:30-17:15	**Do antiseizure medications increase the risk of depression and suicide in people with epilepsy?**	
	Capsule: Most antiseizure medications have mandatory labeling by regulatory authorities indicating that they are associated with an increased risk of suicidality. This warning has impacted drug development and patient perception of these medications. Is this warning justified when treating people with epilepsy?	
16:30-16:35	Introduction and Pre-Debate Voting	
16:35-16:50	Yes: **Marco Mula**, UK	
16:50-17:05	No: **Martin Holtkamp**, Germany	
17:05-17:15	Rebuttals, Discussion and Post-Debate Voting	
17:15-18:00	**Should antiseizure medication be routinely prescribed for prophylaxis in people with brain tumors?**	
	Capsule: Many people with benign and malignant brain tumors develop seizures. Should we routinely prescribe prophylactic antiseizure medication to all or nearly all patients diagnosed with brain tumors, even though seizures have not occurred?	
17:15-17:20	Introduction and Pre-Debate Voting	
17:20-17:35	Yes: **Ilan Blatt**, Israel	
17:35-17:50	No: **Ivan Rektor**, Czech Republic	
17:50-18:00	Rebuttals, Discussion and Post-Debate Voting	
18:00-18:45	**Should combination therapy be used as first line treatment for status epilepticus (SE)?**	
	Capsule: Success rates diminish for treating SE with failure of each successive drug that is administered. Furthermore, the longer seizures last, the harder it is to control them. Can we improve outcome by aggressively using polypharmacy as initial therapy in SE?	
18:00-18:05	Introduction and Pre-Debate Voting	
18:05-18:20	Yes: **Martin Holtkamp**, Germany	
18:20-18:35	No: **Alla Guekht**, Russia	
18:35-18:45	Rebuttals, Discussion and Post-Debate Voting	

18:45-19:30	**Is it better to combine drugs with different mechanisms of action than combine drugs with similar mechanisms of action?**
	Capsule: Combination therapy is often used to treat resistant seizures. Do drugs with different mechanisms of action have synergistic effects and are therefore more effective?
18:45-18:50	Introduction and Pre-Debate Voting
18:50-19:05	Yes: **Firas Fahoum**, Israel
19:05-19:20	No: **William Theodore**, USA
19:20-19:30	Rebuttals, Discussion and Post-Debate Voting
19:30-20:00	Highlights of the day: **Alla Guekht**, Russia

SATURDAY, SEPTEMBER 25, 2021		
14:00-15:30	**PARKINSON'S DISEASE (PD) II**	**HALL D**
	Session supported by unrestricted grants from Abbott, Adamas, Amneal, Brittania, Kyowa Kirin, Merz, Sunovion, Supernus	
Chair:	**Stuart Isaacson**, USA	
14:00-14:45	**Anti-emetic pretreatment is not necessary for apomorphine initiation**	
	Capsule: When apomorphine is initiated, antiemetic pretreatment has frequently been used to reduce nausea and vomiting? But is it necessary?	
14:00-14:05	Introduction and Pre-Debate Voting	
14:05-14:15	Pro: **Stuart Isaacson**, USA	
14:15-14:25	Con: **Fernando Pagan**, USA	
14:25-14:45	Rebuttals, Discussion and Post-Debate Voting	
14:45-15:30	**An adenosine 2a receptor antagonist should be used before adjunctive dopaminergic therapies for OFF**	
	Capsule: Despite extended-release formulations and adjunctive dopaminergic polypharmacy, many patients continue to experience OFF time. Non dopaminergic therapies can modulate striatal outflow pathways. Striatal adenosine receptor are overactive in PD, increasing bradykinesia and OFF. Should a specific adenosine 2a antagonist, be used before adding dopaminergic adjunctive therapies?	
14:45-14:50	Introduction and Pre-Debate Voting	
14:50-15:00	Pro: **Laxman Bahroo**, USA	
15:00-15:10	Con: **Daniel Kremens**, USA	
15:10-15:30	Rebuttals, Discussion and Post-Debate Voting	

16:30-18:45	**PARKINSON'S DISEASE (PD) II**	**HALL D**
	Session supported by unrestricted grants from Abbott, Adamas, Amneal, Brittania, Kyowa Kirin, Merz, Sunovion, Supernus	
Chair:	**Stuart Isaacson**, USA	
16:30-17:15	**Is amantadine extended-release first line therapy for OFF?**	
	Capsule: Balancing dyskinesia while treating OFF episodes in PD often presents a clinical trade-off between the two. A familiar treatment for dyskinesia is amantadine, but a bedtime administered extended-release formulation has FDA approval for the treatment OFF episodes in addition to the treatment of dyskinesia. Does this formulation have a place as a first line treatment for OFF episodes in patients with current, prior, or at higher risk of dyskinesia as a way to address this trade off?	
16:30-16:35	Introduction and Pre-Debate Voting	
16:35-16:45	Yes: **Rajesh Pahwa**, USA	
16:45-16:55	No: **Yasar Torres-Yaghi,** USA	
16:55-17:15	Rebuttals, Discussion and Post-Debate Voting	
17:15-18:00	**Extended release levodopa formulations should be begun at first emergence of OFF**	
	Capsule: Dopaminergic stimulation is the key treatment for PD. However, it is still debatable whether pulsatile stimulation contributes to the development of motor complications. Should patients be treated with levodopa therapies that minimize fluctuation index as early as possible or only when motor fluctuations are troublesome?	
17:15-17:20	Introduction and Pre-Debate Voting	
17:20-17:30	Pro: **Danielle Larson**, USA	
17:30-17:40	Con: **Jill Farmer**, USA	
17:40-18:00	Rebuttals, Discussion and Post-Debate Voting	

18:00-18:45	**Botulinum toxin for sialorrhea in PD** *Supported by an unrestricted educational grant from Merz*
	Capsule: Sialorrhea is an undertreated nonmotor symptom that impacts patients and their caregivers. The physical, emotional, and social consequences of untreated sialorrhea should be routinely queried. When troublesome, botulinum toxin is first-line therapy for sialorrhea. Agree?
Moderator:	**Laxman Bahroo**, USA
18:00-18:05	Introduction: Sialorrhea in PD: **Laxman Bahroo, USA**
18:05-18:20	Anatomy, physiology, and clinic approach to injection: **Jill Farmer**, USA
18:20-18:35	Clinical trial evidence and application to patient selection: **Fernando Pagan**, USA
18:35-18:45	Discussion

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(as of September 24, 2021)

SUNDAY, SEPTEMBER 26, 2021		
14:00-17:00	**E-Posters Viewing and Visit the Exhibition**	