CONy 2020 Virtual Congress Scientific Program (Subject to changes)

THURSDAY, OCTOBER 29, 2020		
15:00-15:40	**PLENARY SESSION 1: OPENING**	**HALL A**
Chair:	**Natan Bornstein**, Israel	
15:00-15:10	Welcome remarks	
15:10-15:40	Forming beliefs and the human brain: Obstacles to truth **Tali Sharot**, UK	
15:40-16:10	**PLENARY SESSION 2**	**HALL A**
Chair:	**Martin Rossor**, UK	
	Emerging neuropathological comorbidities in aging - LATE, ARTAG and CTE **Lea Grinberg**, Brazil/USA	
16:10-17:00	**ALZHEIMER'S DISEASE AND DEMENTIA I**	**HALL A**
Chair:	**Martin Rossor**, UK	
	GWAS is unlikely to contribute significantly to Alzheimer's disease (AD) patients care.	
	Capsule: Genome-wide association studies (GWAS) looking at genetic variants in different individuals may identify variants associated with disease. These cross-national investigations have been applied widely to AD, identifying dozens of "disease-specific" alterations. Is the huge and expensive investment likely to help in patient care?	
16:10-16:30	Yes: **John Hardy**, UK	
16:30-16:50	No: **Amos Korczyn**, Israel	
16:50-17:00	Discussions and rebuttals	

17:00-20:30	**ALZHEIMER'S ASSOCIATION SATELLITE I: OVERCOMING THE IMPASSE IN DEMENTIA PREVENTION AND TREATMENT**	**HALL A**
Chair:	**Zaven Khachaturian**, USA	
17:00-17:15	Introduction from the Alzheimer's Association (AA) **Heather Snyder**, USA *Capsule: The AA leads the way to end AD and all other dementia — by accelerating global research, driving risk reduction and early detection, and maximizing quality care and support. The AA fosters and facilitates advances in scientific discussion through publications, conferences and collaborations, including our partnership with CONy to support the CONy Dementia Satellite.*	
17:15-17:45	The Complex Reality **Amos Korczyn**, Israel *Capsule: The overwhelming failure of the attempts to prevent or cure AD should lead us to rethink the problem. Is AD a real disease or is it a syndrome? Can we use genetic early onset AD as a template for the sporadic disease? What is the role of comorbidities in older people with dementia? Since AD is a multifactorial disorder can we expect to find a "silver bullet" that will cure AD? Is the idea of "the sooner the better" really valid?*	
17:45-18:00	Discussion	
18:00-18:30	Why the pathological phenotype is sufficient? **Bart De Strooper**, **UK** *Capsule: Traditional definition of AD consisted clinical features and pathological hallmarks of both amyloid-β and T. This is because the clinical manifestations are not very specific. The development of biomarkers, particularly PET-scans, showed that many individuals carry amyloid deposits yet without cognitive symptoms. Do we have enough data to be certain that they will definitely develop dementia?*	
18:30-18:50	Discussion	
18:50-19:20	Non Pharmacological Interventions **Miia Kivipelto**, Sweden *Capsule: Are environmental interventions (optimal CV control, diet, physical activity, etc.) likely to change the frequency of dementia and by how much? Have any of these factors been shown to prevent the occurrence of dementia or do they merely delay the onset, and if so, by how much? What is the number needed to treat (NNT in order to prevent one case? These are standard questions for drug treatment and should be also relevant for non-pharmacological interventions?*	
19:20-19:35	Discussion	
19:35-20:00	Animal Models of AD **Andrea Tenner**, USA *Capsule: Is the employment of young, genetically modified rodents without comorbidities, likely to lead us to find a cure for sporadic AD? Several treatments were beneficial in those animal models yet failed in humans. Are the models misleading us? How can we improve?*	
20:00-20:30	Discussion	

20:30-21:00	PLENARY SESSION 3	HALL A
Chair:	**Lea Grinberg**, Brazil/USA	
	Multimorbidity of the ageing brain **Johannes Attems**, UK	

17:00-19:30	NEUROIMMUNOLOGY	HALL B
Chair:	**Hans Peter Hartung**, Germany	
17:00-17:50	**Is narcolepsy an autoimmune disorder which should be treated as such?**	
	Capsule: Narcolepsy is a relatively rare disorder and its cause is unknown. There is a genetic component, particularly the HLA system, which is important in immune regulation. This leads to the hypothesis that interference with the immune system may be helpful in patents.	
17:00-17:20	Yes: **Jacek Losy**, Poland	
17:20-17:40	No: **Angela Vincent**, UK	
17:40-17:50	Discussion and rebuttals	
17:50-18:40	**Treatment of CIDP - immunoglobulins or steroids first?**	
	Capsule: Many treatments have been advocated for CIDP, but the best accepted options are intravenous immunoglobulins and corticosteroids. Which treatment is best and should be applied first?	
17:50-18:10	Immunoglobulins: **Helmar Lehmann**, Germany	
18:10-18:30	Steroids: **Michael Lunn**, UK (Invited)	
18:30-18:40	Discussion and rebuttals	
18:40-19:30	**PANDAS (PANS/CANS) is a clinically distinct entity and needs early and prompt immunotherapy.**	
	Capsule: The nosology of pediatric acute neuropsychiatric syndromes like PANDAS remains controversial, and the evidence for immunopathology and in particular autoimmunity driven by streptococcal infection is still being questioned. Is PANDAS a distinct disorder and is prompt immunotherapy indicated for treatment?	
18:40-19:00	Yes:	
19:00-19:20	No: **Ming Lim**, UK	
19:20-19:30	Discussion and rebuttals	

17:00-19:30	**PARKINSON'S DISEASE I**	**HALL C**
17:00-20:20	**ALS**	**HALL D**
Chair:	**Ettore Beghi**, Italy	
17:00-17:50	**ALS should be considered a neurodegenerative disease rather than a neuromuscular disorder**	
	Capsule: ALS is now recognized to have clinical, histopathological and genetic overlap with fronto-temporal dementia. Brain-based pathology is consistently identifiable, yet ALS frequently continues to be classified alongside neuromuscular disorders of peripheral nerves, rather than among neurodegenerative disorders. This may have a detrimental impact on research funding and restrict optimal collaboration.	
17:00-17:20	Yes:	
17:20-17:40	No: **Monica Povedano Panades**, Spain	
17:40-17:50	Discussion and rebuttals	
17:50-18:40	**The study of mice has been detrimental to developing therapy for ALS.**	
	Capsule: ALS is a highly-selective neurodegeneration involving primarily motor neurons, possibly unique to humans. Twenty-five years since the development of the SOD1 mouse model of ALS, there are currently only two modestly disease-modifying therapies for the human disorder. Have these models helped or slowed the development of therapies?	
17:50-18:10	Yes:	
18:10-18:30	No: **Pamela Shaw**, UK	
18:30-18:40	Discussion and rebuttals	
18:40-19:30	**Patients should set the agenda for therapeutic trials in ALS.**	
	Capsule: There is an increasing drive of many grant-awarding bodies for applicants to demonstrate public and patient involvement. ALS patients are understandably desperate for effective therapy and frequently want to "try anything". Placebo-controlled trials may be problematic in rapidly-progressive diseases. 'Right-to-try' legislation challenges the traditional model of physician-as-expert, while unfiltered information disseminated through social media by 'expert patients' and self-appointed advocacy groups may adversely distort the research agenda.	
18:40-19:00	Yes: **Albert Ludolph**, Germany	
19:00-19:20	No:	
19:20-19:30	Discussion and rebuttals	

19:30-20:20	**ALS-Parkinsonism-dementia complex is due to toxins**
	Capsule: Pacific ALS-PDC may illuminate the causes of ALS, atypical parkinsonism and related disorders. ALS-PDC is a familial and sporadic neurodegenerative disease featured neuropathologically by a tau-dominated proteinopathy. Is ALS-PDC primarily a genetic disease? Others propose that environmental factors dominate the etiology of ALS-PDC.
19:30-19:50	Yes: **Peter Spencer**, USA
19:50-20:10	No: **Helmar Lehmann**, Germany
20:10-20:20	Discussion and rebuttals

FRIDAY, OCTOBER 30, 2020		
15:00-16:45	**ALZHEIMER'S ASSOCIATION SATELLITE II**	**HALL A**
Chair:	**Robert Perneczky**, Germany	
15:00-15:25	Amyloid in AD **David Knopman**, USA *Capsule: Are βA and tau wrong treatment targets in sporadic, late onset AD, given the disappointments with human anti-amyloid studies, although they were successful in eliminating amyloid significant clinical benefit were not seen? Since βA deposition is very prevalent in aging and is not necessarily associated with dementia, how do we know that it is a worthwhile therapeutic target? Could other biomarkers, such as synaptic loss or neurodegeneration add specificity?*	
15:25-15:35	Discussion	
15:35-16:00	To futility or not – when and how should futility analysis be applied? **Rema Raman**, USA *Capsule: AD treatment exploratory studies are excessively costly and long. Should the studies always be continued till the planned end? Is discontinuation always justified and ethical when treatment seems non-effective during the study?*	
16:00-16:10	Discussion	
16:10-16:35	Neuropathology of dementia **Lea Grinberg**, Brazil/USA *Capsule: What can pathology contribute to our understanding, given that autopsies come very late in the disease course and show mixed pathology in most cases?*	
16:35-16:45	Discussion	
16:45-19:25	**ALZHEIMER'S ASSOCIATION SATELLITE III**	**HALL A**
Chair:	**Peter Whitehouse**, USA	
16:45-17:10	Is APOE4 a potential treatment target, given that we do not understand its mechanism? **Daniel M. Michaelson**, Israel *Capsule: It is now almost 30 years since the identification of APOE polymorphism as important genetic determinant of AD, yet the underlying mechanism is still unknown and it is not even clear whether APOE4 is toxic or just less protective than APOE3. And, should APOE4 related dementia be designated as a separate disease?*	
17:10-17:25	Discussion	
17:25-17:50	Is neuroinflammation a useful potential therapeutic target? **Michael Heneka**, Germany *Capsule: Examination by pathologists demonstrate the existence of inflammation in the brain of patients with dementia and this is*	

	supported by imaging, genetic, and neurochemical studies. However, attempts to ameliorate the condition of patients have largely failed. Does that mean that the inflammatory processes are just epiphenomena, or perhaps have different roles in early and late stages of the disease? Could it be that inflammation has both beneficial and toxic effects?
17:50-18:05	Discussion
18:05-18:30	Fear and loathing in AD trials **Lon Schneider**, USA *Capsule: The cases of Aducanumab, albumin/IVIG exchange, and oligomannurarate show the difficulties in preforming and interpreting data. What is the way forward? Are the targets wrong or are the other methods used mistaken? Failure of design, methods, execution or analysis?*
18:30-18:45	Discussion
18:45-19:10	The need for multiple targets, outcomes, and approaches. **Vladimir Hachinski**, Canada *Capsule: The typical patient with Alzheimer's Disease harbor 8 pathologies, hence we need to target more than one mechanism. Cognition, motion and emotion are closely inter-related and should be considered for a composite outcome measure that would increase the likelihood of a positive result. The high risk dementia prevention strategy that prevails, needs to be complemented by population level strategies, where most of the gains can be made in the near future.*
19:10-19:25	Discussion

20:30-21:00	**PLENARY SESSION 4**	**HALL A**
Chair:	**Amos Korczyn**, Israel	
20:30-21:00	Functional neurological disorders **Stoyan Popkirov**, Germany	

15:00-17:30	HEADACHE I	HALL B
Chair:	**Christopher Gottschalk**, USA	
15:00-15:40	**Headache devices will replace medications for the acute and preventive treatment of migraine and cluster headache.**	
	Capsule: Headache devices are proliferating rapidly in the headache medicine field; there is hope that they will provide an alternative therapeutic option for patients with migraine and cluster headache. How strong is the evidence?	
15:00-15:15	Yes: **Jose Miguel Lainez**, Spain	
15:15-15:30	No: **Giorgio Lambru**, UK	
15:30-15:40	Discussion and rebuttals	
15:40-16:20	**Correcting the derangement in sleep architecture is sufficient to treat cluster and migraine headache without medication.**	
	Capsule: Migraine and cluster headache patients who do not sleep well develop more frequent and severe headaches. Would optimal sleep therapies ever be good enough to take the place of medication for the treatment of these headaches, or is sleep impairment just an epiphenomenon?	
15:40-15:55	Yes: **Bojana Zvan**, Slovenia	
15:55-16:10	No: **Brian E. McGeeney**, USA	
16:10-16:20	Discussion and rebuttals	
16:20-17:00	**The safety and efficacy of CGRP mAbs are known well enough for physicians to recommend them for long-term use** This session is supported by an unrestricted educational grant by Teva Pharmaceuticals Teva are not responsible or involved in the content of the presentation	
	Capsule: CGRP is a potent vasodilator and there was early concern about blocking it in patients that may have an impending stroke or myocardial infarction. CGRP is also involved in many other processes such as bone and wound healing as well as cardiovascular homeostasis and gastrointestinal function. Are these drugs safe enough?	
16:20-16:35	Yes: **Lars Edvinsson**, Sweden	
16:35-16:50	No: **Rob Cowan**, USA	
16:50-17:00	Discussion and rebuttals	

17:00-17:30	How are the CGRP monoclonal antibodies being used today? This session is supported by an unrestricted educational grant by Teva Pharmaceuticals Teva are not responsible or involved in the content of the presentations *Supported by an unrestricted educational grant from Teva* **Christopher Gottschalk**, USA

17:30-20:10	**HEADACHE II**	**HALL B**
Chair:	**Alan Rapoport**, USA	
17:30-18:10	**Vestibular migraine – does it exist?**	
	Capsule: Vestibular migraine is a term used to describe episodic vertigo occurring in migraine patients; but should it be a distinct diagnosis, or simply a sensory manifestation, or even an aura, of migraine?	
17:30-17:45	Yes: **Teena Shetty**, USA	
17:45-18:00	No: **Morris Levin**, USA (Invited)	
18:00-18:10	Discussion and rebuttals	
18:10-18:50	**Estrogen containing contraceptives are safe in women with migraine with aura.**	
	Capsule: Migraine with aura has been associated with increased risk of ischemic stroke in women. Prior studies have shown a further increase in risk in women using combined hormonal contraceptives (CHCs). This has led to guidelines recommending against use of CHCs in this population. Should these recommendations be changed?	
18:10-18:25	Yes: **Pooja Dassan**, UK	
18:25-18:40	No: **Christopher Gottschalk**, USA	
18:40-18:50	Discussion and rebuttals	
18:50-19:30	**Will telemedicine make headache office visits redundant?**	
	Capsule: Telemedicine turned out to be very useful during the corona virus epidemic. Will it make in-person consultation redundant?	
18:50-19:05	Yes: **Messoud Ashina**, Denmark (Invited)	
19:05-19:20	No: **Jess Ailani**, USA	
19:20-19:30	Discussion and rebuttals	

19:30-20:10	**Is CGRP just a grand placebo?**
19:30-19:45	Yes: **Pravin Thomas**, UK
19:45-20:00	No: **Patricia Pozo-Rosich**, Spain
20:00-20:10	Discussion and Rebuttals

15:00-17:00	**EPILEPSY I**	**HALL C**
Chair:	**Martin Brodie**, UK	
15:00-15:40	**Ambulatory video-EEG monitoring can replace in-hospital video-EEG.**	
	Capsule: Outpatient ambulatory video-EEG devices are now widely available. Are they a reasonable substitute for inpatient monitoring? Can they provide the same information? Should this be done first before considering in hospital assessment? What are the risks involved?	
15:00-15:15	Yes: **Antonio Gil-Nagel**, Spain	
15:15-15:30	No: **Ilan Blatt**, Israel	
15:30-15:40	Discussion and rebuttals	
15:40-16:20	**Combination antiepileptic drug (AED) therapy should be offered immediately after failure of a single antiepileptic drug.**	
	Capsule: Though monotherapy has advantages, there is clear evidence from randomized trials that polytherapy affords improvement to many patients, and reduces mortality as well. If combination therapy is used, when should it be implemented?	
15:40-15:55	Yes: **Martin Brodie**, UK	
15:55-16:10	No: **Manjari Tripathi**, India	
16:10-16:20	Discussion and rebuttals	
16:20-17:00	**Antidepressant drugs should be avoided if possible in epilepsy.**	
	Capsule: Many antidepressant medications can provoke seizures in animals, and concerns have been raised that these drugs may trigger seizures in some patients. Is the efficacy of these agents sufficient to warrant their use, given potential risks?	
16:20-16:35	Yes: **John Duncan**, UK	
16:35-16:50	No: **William Theodore**, USA	
16:50-17:00	Discussion and rebuttals	

17:00-20:20	**EPILEPSY II**	**HALL C**
Chair:	**Michael Sperling**, USA	
17:00-17:40	**Psychotherapy improves outcome in "psychogenic" seizures.**	
	Capsule: In patients with "psychogenic" seizures, spontaneous remission rates are quite high and patient adherence to therapy quite low. Is there evidence that psychotherapy provides long-term benefit?	
17:00-17:15	Yes: **William Curt LaFrance**, USA	
17:15-17:30	No: **Daniel Goldenholz**, USA	
17:30-17:40	Discussion and rebuttals	
17:40-18:20	**Combination therapy should 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?	
17:40-17:55	Yes: **Matthew Walker**, UK	
17:55-18:10	No: **Alla Guekht**, Russia	
18:10-18:20	Discussion and rebuttals	
18:20-19:00	**Cryptogenic SE should be treated with immunomodulation as soon as it is diagnosed.**	
	Capsule: NORSE and FIRES are epilepsy syndromes resistant to treatment with conventional AED and may require immune modulation for cessation of seizures. Should patients be presumptively treated with immunosuppressive agents early in the course of illness when status epilepticus has no known cause?	
18:20-18:35	Yes: **Larry Hirsch**, USA	
18:35-18:50	No: **Matthias Koepp**, UK (Invited)	
18:50-19:00	Discussion and rebuttals	
19:00-19:40	**The newer AED are more effective than the established ones.**	
	Capsule: Over the past 20 years a number of new antiseizure drugs have been introduced around the world as adjunctive treatment and subsequently as monotherapy for pharmacoresistant and newly diagnosed epilepsy. Have they improved overall outcomes in terms of seizure freedom and so proved value for money?	
19:00-19:15	Yes: **Andreas Schulz-Bonhage**, Germany	
19:15-19:30	No: **Martin Brodie**, UK	

19:30-19:40	Discussion and rebuttals
19:40-20:20	**Should surgery be offered to patients after failure of two AED?**
	Capsule: Epidemiological studies suggest that drug failure is quite likely once two agents have failed to control seizures. On the other hand, the literature contains numerous reports of response to drug therapy in patients formerly considered drug resistant. Are the ILAE guidelines supported by the evidence?
19:40-19:55	Yes: **Zeljka Petelin Gadže**, Croatia
19:55-20:10	No: **Ettore Beghi**, Italy
20:10-20:20	Discussion and rebuttals

15:00-16:45	**SLEEP**	**HALL D**
Chair:	**Hugh Selsick**, UK (Invited)	
15:00-15:35	**Obstructive sleep apnea (OSA) diagnosis: A small home system is enough.**	
	Capsule: OSA is a common disorder, especially in the group of 35-65 years of age. In this group the frequency can be high: 10-45%. The diagnosis and treatment should not be delayed. What is the best method and which is the fastest type for the right diagnosis?	
15:00-15:15	Yes:	
15:15-15:30	No: **Arthur Kurvers**, The Netherlands	
15:30-15:35	Discussion and rebuttals	
15:35-16:10	**OSA should always be treated by CPAP.**	
	Capsule: There are several OSA types; should they all be treated the same way?	
15:35-15:50	Yes:	
15:50-16:05	No: **Monique Vlak**, The Netherlands	
16:05-16:10	Discussion and rebuttals	
16:10-16:45	**Restless leg syndrome (RLS) diagnosis can be made by history alone while polysomnography (PSG) is NOT mandatory.**	
	Capsule: RLS is a serious sleep disorder which can be diagnosed using the right questions. Others maintain that the correct diagnosis requires specific sleep measurements.	
16:10-16:25	Yes: **Guy Leschziner,** UK	

16:25-16:40	PSG is mandatory: **Panagis Drakatos,** UK
16:40-16:45	Discussion and rebuttals

16:45-19:40	**MULTIPLE SCLEROSIS I**	**HALL D**
Chair:	**Cris Constantinescu**, UK	
16:45-17:20	**Are MS patients at increased risk for developing cancer?**	
	Capsule: Whether people with MS are at higher risk of developing cancer has not been definitively established. The increased rate of general comorbidity would indicate a higher risk of cancer as well. However, some registers have not found an association. Could it be that there is higher risk of specific cancers, but not all cancers? And can newer highly potent immunosuppressive treatments modify the long term risk?	
16:45-17:00	Yes: **Ali Manouchehrinia**, Sweden	
17:00-17:15	No: **Melinda Magyari**, Denmark	
17:15-17:20	Discussion and rebuttals	
17:20-17:55	**MS is a primary progressive disease in all cases, but some patients have superimposed relapses.**	
	Capsule: Patients with clinically isolated syndrome have been shown to have significant cortical changes in their brains. Subcortical asymptomatic alterations have also been described. Does that mean that MS is basically a degenerative disease with superimposed clinical flare-ups ("relapses") as epiphenomena or is MS an inflammatory disease of the brain with only secondary degeneration?	
17:20-17:35	Yes: **Antonio Scalfari**, UK	
17:35-17:50	No: **Mark Freedman**, Canada	
17:50-17:55	Discussion and rebuttals	
17:55-18:30	**Cognitive decline is sufficient to define transition to secondary progressive multiple sclerosis (SPMS).**	
	Capsule: There is no biomarker that indicates when a patient has transitioned from relapsing-remitting MS (RRMS) to SPMS, and consequently SPMS is a retrospective diagnosis, based primarily on motor disability. The period of diagnostic uncertainty separating RRMS and SPMS diagnoses often lasts years. Is cognitive decline sufficient to define this change?	
17:55-18:10	Yes: **Klaus Schmierer**, UK	
18:10-18:25	No: **Laszlo Vecsei**, Hungary	
18:25-18:30	Discussion and rebuttals	

18:30-19:05	**In MS patients with significant cognitive decline, drug treatment should be enhanced.**
	Capsule: Approximately 50% of people with MS become unemployed with a median EDSS of 3.0-3.5. They usually have acquired hidden disabilities related to cognitive impairment. Should MS specific drug treatment be modified in patients with cognitive decline whose EDSS is otherwise unchanged?
18:30-18:45	Switch to a newer agent: **Ron Milo**, Israel
18:45-19:00	Not so fast: **Amos Korczyn**, Israel
19:00-19:05	Discussion and rebuttals
19:05-19:40	**Should new therapies for MS be used even with poor scientific support?**
	Capsule: Over the past three decades, numerous drugs were approved for MS, following rigorous and expensive studies. In this session, the debaters will outline the pros and cons of using interventions based on poor scientific evidence, such as high dose vitamin D, fish oils, magnetic field therapy or hyperbaric oxygen (HBO).
19:05-19:20	Yes: **Gavin Giovannoni**, UK (Invited)
19:20-19:35	No: **Mark Freedman**, Canada
19:35-19:40	Discussion and rebuttals

SATURDAY, OCTOBER 31, 2020		
15:00-17:00	**STROKE I**	**HALL A**
Chair:	**Natan Bornstein**, Israel	
15:00-15:40	**In the presence of cerebral microbleeds (CMBs), antithrombotic therapy should be avoided.**	
	Capsule: The presence of microbleeds (detected only with MRI) is associated with increased risk of hemorrhagic and perhaps of ischemic stroke. The risk depends on the location and number of microbleeds. How dangerous is antithrombotic therapy in patients with microbleeds? The session provides an overview about the pros and cons.	
15:00-15:15	Yes: **Mahmut Edip Gurol**, USA	
15:15-15:30	No: **David Werring**, UK	
15:30-15:40	Discussion and rebuttals	
15:40-16:20	**Is left atrial appendage closure underutilized for stroke prevention in atrial fibrillation?** *Supported by an unrestricted educational grant by Boston Scientific*	
	Capsule: The majority of embolic stroke patients with nonvalvular atrial fibrillation are associated with left atrial thrombi, and left atrial appendage closure may be a suitable alterative to chronic anticoagulation.	
15:40-15:55	Yes: **George Chrysant**, USA	
15:55-16:10	No: **Roni Eichel**, Israel	
16:10-16:20	Discussion and rebuttals	
16:20-17:00	**Does the main benefit of AIS treatment come from tPA or stroke unit care?**	
	Capsule: The presence of a dedicated stroke unit allows for the management of all patients with suspected AIS. Treatment with tPA can only be offered to a smaller subset of AIS patients but the improvement in some treated patients can be very significant. In an era of limited resources, should we focus on ensuring that all AIS patients be admitted to a stroke unit or recommend fast triage methods for timely thrombolysis?	
16:20-16:35	tPA: **Gary Ford**, UK	
16:35-16:50	Stroke unit: **Milija Mijajlovic**, Serbia	
16:50-17:00	Discussion and rebuttals	

17:00-19:40	**STROKE II**	**HALL A**
Chair:	**Ashfaq Shuaib**, Canada	
17:00-17:40	**Do diffusion weighted imaging (DWI) negative strokes exist?**	
	Capsule: Stroke is a clinical entity. Its exact identification is crucial as therapeutic options nowadays are associated with some risks. DWI MRI is considered the best imaging technique for the confirmation of acute ischemic stroke (AIS). Sensitivity, however, is not perfect, with debatable underlying reasons, raising the question: Do AIS with negative DWI imaging really exist?	
17:00-17:15	Yes: **Derk W. Krieger**, United Arab Emirates	
17:15-17:30	No: **Andrew Demchuk**, Canada	
17:30-17:40	Discussion and rebuttals	
17:40-18:20	**Should statins be given to people over age 80 for stroke prevention?**	
	Capsule: There is considerable evidence that the use of statins results in reduction of cardiovascular morbidity and mortality. Long-term treatment with statins can lead to side effects including muscle and liver damage. Clinical trials evaluating the efficacy of statins have mostly enrolled subjects younger than 75 years of age. Can we extrapolate the evidence to older individuals in whom the risk of side-effects may be higher?	
17:40-17:55	Yes: **Daniel Bereczki**, Hungary	
17:55-18:10	No: **Vida Demarin**, Croatia	
18:10-18:20	Discussion and rebuttals	
18:20-19:00	**Should symptomatic extracranial vertebral artery stenosis be stented?**	
	Capsule: Stenosis in the vertebro-basilar system accounts for about one quarter of all posterior circulation strokes. The risk profile is similar to that seen for carotid stenosis. Recent phase 2 trials have shown that extracranial vertebral stenosis can be stented with low risk but whether this reduces recurrent stroke risk compared with best medical therapy alone remains controversial. The debate will consider whether based on current evidence stenting should be recommended for recently symptomatic extracranial vertebral artery stenosis.	
18:20-18:35	Yes: **Laszlo Csiba**, Hungary	
18:35-18:50	No: **Hrvoje Budincevic**, Croatia	
18:50-19:00	Discussion and rebuttals	

19:00-19:40	**Is penumbral imaging mandatory for potential thrombectomy in patients arriving beyond six hours?**
	Capsule: There is general agreement amongst stroke experts that patient selection is essential for successful thrombectomy. The introduction of penumbral imaging may allow for improved patient evaluation but comes at a higher cost. Is there sufficient evidence that such imaging is made mandatory prior to initiation of treatment?
19:00-19:15	Yes: **Mayank Goyal**, Canada
19:15-19:30	No: **Ashfaq Shuaib**, Canada
19:30-19:40	Discussion and rebuttals

20:30-21:00	**PLENARY SESSION 5**	**HALL A**
Chair:	**Olaf Stüve,** USA	
	Multiple sclerosis is one disease; why definitions matter to patients and to how we treat them **Gavin Giovannoni**, UK (Invited)	

15:00-17:00	**MULTIPLE SCLEROSIS II**	**HALL B**
Chair:	**Ralf Linker**, Germany (Invited)	
15:00-15:40	**Are the 2017 MS McDonald criteria too liberal and should be more restrictive?**	
	Capsule: The 2017 revisions of the McDonald criteria for the diagnosis of MS were mainly designed to facilitate an earlier MS diagnosis and the clinically isolated syndrome. While the criteria are easy to use and highly sensitive, they lack specificity and may bear the risk of MS over diagnosis, exposing patients to unnecessary, expensive and sometimes dangerous therapy.	
15:00-15:15	Yes: **Brian Weinshenker**, USA	
15:15-15:30	No: **Christopher Hawkes**, UK	
15:30-15:40	Discussion and rebuttals	
15:40-16:20	**We are well enough equipped to identify fake news in MS therapy before they can cause harm.**	
	Capsule: Fake news are news stories or hoaxes created to deliberately misinform or deceive readers. Information that patients with MS read online, and especially in their social media feeds is often inaccurate or untrue. Misinformation about MS therapies have also been disseminated to care providers.	
15:40-15:55	Yes: **Nikos Evangelou**, UK	

15:55-16:10	No: **Radu Tanasescu**, UK
16:10-16:20	Discussion and rebuttals
16:20-17:00	**Newly diagnosed MS patients should be started on aggressive therapy.**
	Capsule: Early treatment is claimed to improve long-term prognosis in MS. Recent studies also suggest that early aggressive therapy with potent immunosuppressive drugs ("induction therapy") may improve long-term outcomes and perhaps lower the risk of conversion to secondary-progressive MS. Should newly diagnosed MS patients be started on such aggressive therapies? Do the potential benefits always outweigh their risks?
16:20-16:35	Yes:
16:35-16:50	No: **Uros Rot**, Slovenia
16:50-17:00	Discussion and rebuttals

17:00-19:30	**NMOSD**	**HALL B**
Chair:	**Hans Peter Hartung**, Germany	
17:00-17:50	**New regulatory-approved medications should be used exclusively for prevention of attacks of NMOSD over currently used non-regulatory approved medications.**	
	Capsule: It is likely that 3 immunomodulatory treatments, a C5 complement inhibitor, an anti-CD19 monoclonal antibody and an inhibitor of IL6 receptor, will all receive regulatory approval for treatment of NMOSD. Do these drugs offer sufficient advantages that they should replace currently used immunotherapies that are widely regarded as effective and are less expensive?	
17:00-17:20	Yes: **Orhan Aktas**, Germany	
17:20-17:40	No: **Andrzej Glabinski**, Poland	
17:40-17:50	Discussion and rebuttals	
17:50-18:40	**NMOSD attacks should be treated with apheresis/plasma exchange at first presentation, either with or without corticosteroids.**	
	Capsule: Recent experience suggests that concomitant or first line treatment with plasma exchange may be superior to treatment with corticosteroids. Is a change in the standard approach of using corticosteroids first appropriate, given the current state of knowledge?	
17:50-18:10	Yes:	
18:10-18:30	Steroids still should be used first: **Maria Isabel Leite**, UK	
18:30-18:40	Discussion and rebuttals	

18:40-19:30	**The 2015 International Panel criteria for NMOSD are outdated and should be replaced with a diagnostic classification based on autoantibody status rather than clinical presentation (*i.e.* AQP4 disease, MOG disease).**
	Capsule: We now know the molecular target of the autoimmune insult in the majority of patients with NMOSD, and molecular classification based on the target of the antibody can be used in lieu of clinical criteria for diagnosis of what we currently refer to as NMOSD. Are we ready for a molecular classification of NMOSD in 2020?
18:40-19:00	Yes: **Angela Vincent**, UK
19:00-19:20	No:
19:20-19:30	Discussion and rebuttals

15:00-19:00	**PARKINSON'S DISEASE II**	**HALL C**
Chair:	**Mike Samuel**, UK	
15:00-15:40	**Botulium toxin is first line treatment for sialorrhea in PD** *Supported by an unrestricted educational grant from Merz*	
	Capsule: Sialorrhea is an inconvenient and embarrassing symptom which requires treatment. Is botulinum toxin the best approach?	
15:00-15:15	Yes: **Fernando Pagan**, USA	
15:15-15:30	No: **Eugenia Yiannakopoulou,** Greece	
15:30-15:40	Discussion and rebuttals	
15:40-16:20	**When is the right time to refer PD patients for deep brain stimulation (DBS)?**	
	Capsule: DBS is effective in treating medication-refractory symptoms (motor and non-motor) and improves patients' quality of life in advanced PD. Currently, it is usually performed in late stage of PD. Advances in our understanding of the natural history of the disease, improved surgical techniques, brain imaging and device design require us to re-evaluate whether the right time for DBS is in earlier stages of the disease.	
15:40-15:55	Late: **Patricia Limousin**, UK	
15:55-16:10	Early: **Vladimira Vuletic**, Croatia	
16:10-16:20	Discussion and rebuttals	

16:20-17:00	**DBS effectiveness against nonmotor features in PD is similar to those of infusion therapies.**
	Capsule: DBS and infusion therapies are both recognized as therapeutic approaches in the treatment of motor symptoms in advanced stage PD when patients develop "wearing off" and/or dyskinesias with oral dopaminergic medication. However, the effects of these two therapeutic approaches on nonmotor features are still under debate.
16:20-16:35	Yes: **Abdelhamid Benazzouz**, France
16:35-16:50	No: **Keyoumars Ashkan**, UK
16:50-17:00	Discussion and rebuttals
17:00-17:40	**Are there important environmental factors for PD?**
	Capsule: The pathogenesis of the neurodegenerative processes in PD are not well understood. Although several genes were found to be associated with the development of PD, the causative agents for a great percent of cases remain unclear. Various factors were incriminated to increase or reduce the risk of PD development yet their contribution as well as interactions with genetic factors are unknown.
17:00-17:15	Yes: **Cristian Falup-Pecurariu**, Romania
17:15-17:30	No: **Zvezdan Pirtosek**, Slovenia
17:30-17:40	Discussion and rebuttals
17:40-18:20	**Is CSF alpha-synuclein a useful biomarker for PD?**
	Capsule: A change in the content of α-synuclein (α-syn) in CSF is considered as a promising biomarker of PD. Indeed, the total α-syn content in CSF decreases, and the fractions of phosphorylated and oligomeric α-syn increase in PD. However, attempts to use α-syn as a biomarker for differential diagnosis or prognosis were unsuccessful so far. Can CSF α-syn be used as a biomarker?
17:40-17:55	Yes:
17:55-18:10	No:
18:10-18:20	Discussion and rebuttals
18:20-19:00	**What should be done when OFF periods develop: Increase dopaminergic drugs or introduce non-dopaminergic agents**
18:20-18:35	Yes:
18:35-18:50	No:
18:50-19:00	Discussion and Rebuttals

SUNDAY, NOVEMBER 1, 2020		
15:00-18:20	**ALZHEIMER'S DISEASE AND DEMENTIA II**	**HALL A**
Chair:	**Robert Perneczky**, Germany	
15:00-15:40	**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 or perhaps that hearing loss may merely be an early symptom of neurodegenerative changes.	
15:00-15:15	Yes: **Sergi Costafreda**, UK	
15:15-15:30	No: **Lev Kruglov**, Russia	
15:30-15:40	Discussion and rebuttals	
15:40-16:20	**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 by itself or whether 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.	
15:40-15:55	Yes: **Dag Aarsland**, UK	
15:55-16:10	No: **Naji Tabet**, UK	
16:10-16:20	Discussion and rebuttals	
16:20-17:00	**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?	
16:20-16:35	Yes: **Babak Tousi**, USA	
16:35-16:50	No: **Panteleimon Giannakopoulos**, Switzerland	
16:50-17:00	Discussion and rebuttals	

17:00-17:40	**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.	
17:00-17:15	Yes: **Panteleimon Giannakopoulos**, Switzerland	
17:15-17:30	No: **Irena Rektorova**, Czech Republic	
17:30-17:40	Discussion and rebuttals	
17:40-18:20	**Can stress can cause dementia?**	
	Capsule: In the last decades AD research has focused on possible preventable risk factors including mood and anxiety disorders studies in animals have shown that chronic stress exacerbates the deposition of proteins involved in AD in particular tau pathology. In humans, psychological stress has been associated with higher risk of AD clinical syndrome. Stress can have a damaging effect on brain health. One of the possible therapeutic targets should be to mitigate the extensive negative effects of stress.	
17:40-17:55	Yes: **Luiza Spiru**, Romania	
17:55-18:10	No: **Bogdan O. Popescu**, Romania	
18:10-18:20	Discussion and rebuttals	
18:20-19:30	**NEURODEGENERATIVE DISEASES**	**HALL A**
Chair:	**Marios Politis**, UK (Invited)	
18:20-19:00	**Do infectious agents trigger and influence neurodegeneration?**	
	Capsule: In the last decades, infectious origin of neurodegenerative diseases, such as Parkinson's or Alzheimer's, was hypothesized. Viruses, bacteria and subtle changes in gut microbiota were incriminated. However, all these infections are still of uncertain significance in the complex cascade of pathogenic mechanisms of neurodegeneration and are denied by others. Are anti-infectious interventions worthy of clinical trial development?	
18:20-18:40	Yes: **Bogdan O. Popescu**, Romania	
18:40-18:50	No: **Peter Jenner**, UK	
18:50-19:00	Discussion and Rebuttals	

19:00-19:30	**Development of precise preclinical diagnosis of neurodegenerative diseases – illusion or reality?**	
19:00-19:10	Illusion: **Giancarlo Logroscino**, Italy	
19:10-19:20	Reality: **Tamas Revesz**, UK	
19:20-19:30	Discussion and rebuttals	
20:30-21:00	**PLENARY SESSION 6**	**HALL A**
Chair:	**Amos Korczyn**, Israel	
	Parkinson's disease: From mitochondria to lysosomes and on to therapy **Anthony Schapira**, UK	