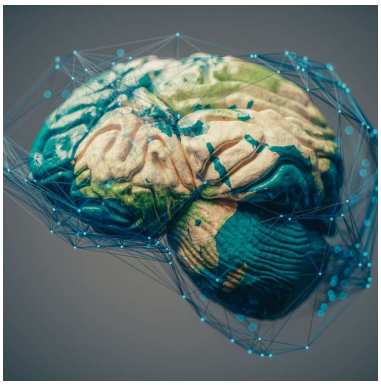
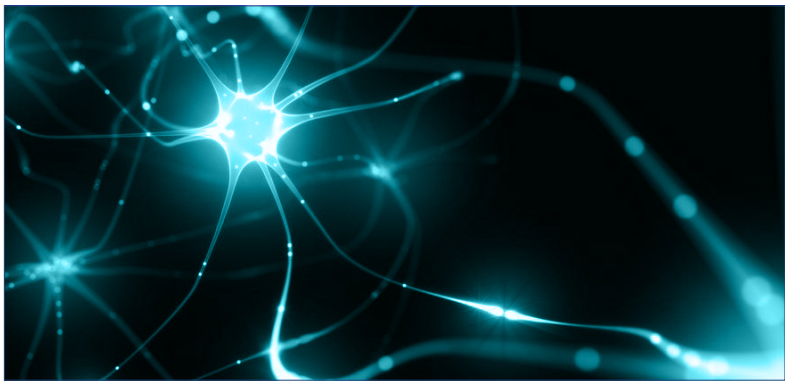
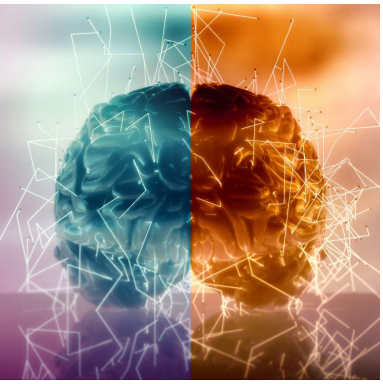
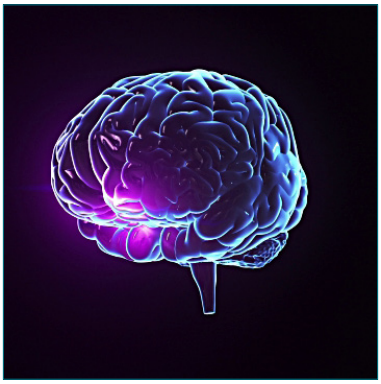
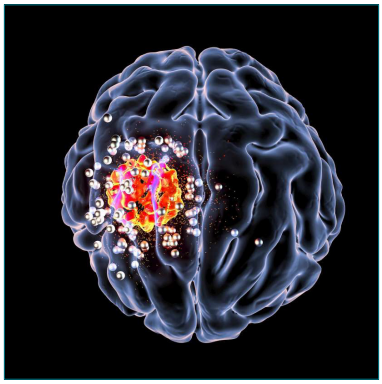
Scientific Tracks & Sessions
August 23, 2018

Neurology 2018

18th International Conference on

Neurology and Neurological Disorders

August 23-24, 2018 | Paris, France

Wharton's jelly mesenchymal stem cells and Insulin effect on BDNF expression in CA1 and CA3 regions of rats' hippocampus after Chronic Hypoxia

Simin Mahakizadeha, Mohammad Akbaria, Mohammad Sharifzadehb, Tayebeh Rastegara, Farid Abolhassania and **Gholamreza Hassanzadeha**
Tehran University of Medical Sciences, Iran

Objectives: Brain is vulnerable to deprivation of oxygen supply during hypoxia, and therefore undergoes neurodegeneration and cognitive dysfunction. Regarded to Regenerative capacities of Wharton's jelly -MSCs and insulin at the site of injury, we were aimed to evaluate the effect of Wharton's jelly -MSCs and insulin on degenerative consequences induced by chronic hypoxia.

Methods: 36 male rats were randomly divided in 6groups: Control(C), Sham1-saline (Sh1), Sham2-surgery (Sh2), Hypoxia (H), Hypoxia+ Insulin (HI), Hypoxia+ MSCs (HCs). Animals were exposed to hypoxic chamber (8% O2, 92% N2) for 30 days (4hours/day) in H, HI and HCs groups. Intranasal insulin and stereotaxical MSCs in HI and HCs was used, respectively. Spatial learning and memory were analyzed using the Morris water maze task. BDNF gene expression was studied in the hippocampus by real time-PCR.

Results: BDNF had the significant depletion in HI group and magnification in HI and HCs groups comparing with C and Sh groups ($p < 0.05$). Insulin and MSCs improve Hypoxia's signs such as BDNF gene expression fallen and memory impairment.

Conclusions: In conclusion, we indicated that use of insulin hormone and MSCs as neuroprotective and stimulating factors for neurogenesis, could be beneficial in neurodegenerative damage induced by hypoxia.

Speaker Biography

Simin Mahakizadeha has completed her PhD from Tehran University of Medical Sciences, Iran and received her master's degree in Anatomy from Golestan University of Medical Science, Iran. Her Bachelor's degree was received from Shiraz University of Medical Sciences in the field of physiotherapy. She has published her papers and participated in many national and international conferences.

e: si.mahakizadeh@gmail.com

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Clinico-Epidemiological determinants of hospital stay and ambulation in patients with Traumatic Spinal Cord Injury by survival analysis

Mohit K Srivastava
King George's Medical University, India

Traumatic spinal cord injury (TSCI) is one of the most devastating injury, and results in different neurological deficits. A long hospital stay occupies medical and financial resources which leads to substantial social loss and economic burden. To optimize resource utilization for rehabilitation care centers treating TSCI patients, it is important to evaluate the determinants of hospitalization length as well as their ambulation. A retrospective cohort study was conducted to identify the associated epidemiological and clinical factors affecting length of stay and ambulation in TSCI, utilizing a quantitative approach. The medical records of 108 patients with TSCI, who fulfilled the inclusion criteria and discharged from the hospital between 1st January 2015 and 30th June 2017 were reviewed using data collection tool. Survival Analysis was done to estimate the probability of ambulation in the TSCI patients with respect to their length of stay. The mean duration of hospital stay was 29.37±17.44 days. 63.6% of age group 15-30 years had hospital stay of ≥30 days. Age (AOR 9.88; 95% C.I. [2.33 – 41.81]; 0.002), employment status (AOR 5.57; 95% C.I. [1.09 – 28.37];0.039, location of residence (AOR 0.14 95%C.I. [0.03-0.63];0.01), Pressure Ulcer (AOR 5.81; 95% C.I. [1.77 – 19.06]; 0.004) and history of treatment (AOR 1.98; 95%C.I. [1.76– 14.16]; 0.002) were significant predictors of length of hospital stay in patients with TSCI. The probability of ambulation was better in females as compared to males on survival analysis (p<0.001). Age (≥35years), gender(males), location of residence (in same city) and neurological category (A) had hazard of non-ambulation.

Speaker Biography

Mohit K Srivastava has completed his M.B.B.S at the age of 25 years from B.R.D Medical College, Gorakhpur, India and is third year post graduate (MD) resident in Department of Physical Medicine and Rehabilitation in King George's Medical University U.P., Lucknow. He has also done fellowship in pain medicine and intervention. He has published 2 case reports and 3 original research papers and has presented paper in national conferences three times.

e: rohitsriv14elec@gmail.com

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Anti-AQP4 and anti-MOG positive Neuromyelitis Optica: Clinical experience with the novel diagnostic criteria and disease management

Jameelah Saeedi

King Abdullah Bin Abdulaziz University Hospital, Saudi Arabia

Neuromyelitis optica (NMO), also known as Devic's disease, is an autoimmune disease of central nervous system (CNS), in which immune cells and auto-antibodies primarily attack optic nerves and spinal cord, and also the brain. Autoimmune attack of optic nerves causes swelling and inflammation with recurrent optic neuritis and/or transverse myelitis, pain and loss of vision. Spinal cord damage leads to paralysis, loss of sensation, and other problems. Despite the initial mistaken belief that NMO is a variant of multiple sclerosis (MS), these two are distinct diseases with some similar clinical and radiological features. Autoimmune attack on aquaporin-4 (AQP4) water channels, located in optic nerves and spinal cord, probably causes NMO. NMO was likely misdiagnosed as MS in 30-40% of cases, prior to the availability of diagnostic test for anti-AQP4 antibodies. Since the identification of anti-AQP4 antibodies (NMO-IgG) in NMO patients in 2004, patients without the typical spinal cord and optic nerve manifestations have also been diagnosed with NMO. This led to the new diagnostic criteria defining anti-AQP4 positive and negative disease with a new unified term, NMO spectrum disorder (NMOSD) to describe the disease. Thus approximately 80% of the NMO patients display circulating NMO-IgG, whereas the others might have antibodies targeting myelin oligodendrocyte glycoprotein (MOG)—a protein expressed on the surface of oligodendrocytes in the CNS. In my presentation, I will discuss the differences between anti-MOG NMO and anti–AQP-4 NMO, the new diagnostic criteria and their radiological features and my experience in the management of NMO patients.

Speaker Biography

Jameelah Saeedi is a certified Saudi Neurologist who specializes in Multiple Sclerosis and Neuroimmunological Diseases. She received her medical qualification from King Abdulaziz University in Saudi Arabia in 2001 followed by two-boards in Neurology from Saudi Commission for Health Specialties and the Jordanian Medical Council in 2007. Dr. Saeedi is alumni of University of British Columbia where she pursued her fellowship and training in Neuroimmunology and Multiple Sclerosis with Prof. Peter Rieckman in 2009. In 2010 she received two more fellowships in Electromyography and Boutlinum Toxin Injection treatment from the University of Toronto. She is one of few leading pioneers who holds vast knowledge, experience, sub-specializes and practices Multiple Sclerosis and Neuroimmunological Diseases in Saudi Arabia. She has been working at King Fahad Medical City as a Subspecialty Consultant and KFMC Comprehensive Neuroimmunology Program Director. She is currently working at King Abdullah Bin Abdulaziz university hospital in Saudi Arabia.

e: jamysa@hotmail.com

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Relation of serum levels of homocysteine, vitamin B12 and folate to cognitive functions in Egyptian Multiple Sclerosis patients

Haidy Elshebawy
Cairo University, Egypt

Background: Vitamin(B12) and folate have a role in normal methylation through (folate-vitamin B12-methylation) pathway which needed for myelin regeneration. Hyperhomocysteinaemia, vitamin(B12) and folate deficiency have been linked to cognitive dysfunction in multiple sclerosis (MS) patients.

Aim: This study aimed to examine the relation between serum levels of homocysteine, vitamin(B12), folate and cognitive functions in Egyptian MS patients.

Methods: Forty-five clinically definite MS patients and twenty matched healthy controls were included in the study. Cognitive assessment done for all participants using Addenbrooke's Cognitive Examination III (ACE-III) and trail making test. Serum levels of homocysteine, vitamin(B12) and folate were estimated using ELISA technique.

Results: MS patients showed significant worse performance in ACE-III and trail making tests compared to controls (P≤0.001). Serum levels of homocysteine, vitamin(B12) and folate showed no significant difference between patients and controls. ACE-III total score showed a significant negative correlation with homocysteine level (r = -0.692, P ≤ 0.001) and a significant positive correlation with Vitamin(B12)(r = 0.480, P= 0.001) and folate levels (r = 0.312, P= 0.037).Trail making test showed a significant positive correlation with homocysteine level (r = 0.394, P =0.007), and a significant negative correlation with Vitamin(B12) level (r = -0.345, P=0.20).By using regression analysis, Homocysteine was found to be the only significant predictor for cognitive impairment in MS patients.

Conclusion: Hyperhomocysteinaemia, vitamin(B12) and folate deficiency were associated with cognitive impairment in MS patients. Homocysteine was an independent risk factor and predictor for cognitive impairment in MS patients.

Speaker Biography

Haidy Elshebawy has completed her PhD at the age of 21 years from faculty of medicine, Alkasr Alainy Hospitals, Cairo University and received her master's degree in neuropsychiatry in May 2014 with excellent degree. She has published her papers and participated in many national and international conferences.

e: haidyshebawy@gmail.com

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A case report on Ischaemic Stroke due to Hyperthyroidism

Bashir Adam Yakasai
Kaduna State University, Nigeria

A 39yr old primary school teacher presented with fever, headache, and palpitation. The fever was described as high grade, intermittent, with a generalized throbbing headache that was temporarily relieved with simple analgesic. While waiting for a medical attention at the emergency unit, he collapsed and developed a generalized tonic-clonic convulsion that lasted about 3 minutes. After the seizures he was noticed to have developed a left hemiplegia. Prior to this admission, he was said to have had a goiter 5 years ago which was poorly managed. On examination, his Thyroid Function Tests (TFT) was abnormal and consistent with that of hyperthyroidism.

Speaker Biography

Bashir Adam Yakasai, MNIM, Fss, mss, DSS, psc, Cert. Av.Medicine, DC Neurol, DPM, PGCert Neuroimaging, PGCert Clinical pharmacology and Pharmacotherapy, MSc in Addiction Studies, FMCPsych. He is a Chief Consultant Neuropsychiatrist in the Nigerian Air Force and a specialist in Clinical Neurology and Aviation Medicine.

e: ybashiradam@gmail.com

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August 24, 2018

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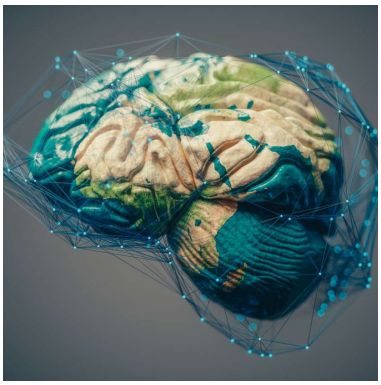
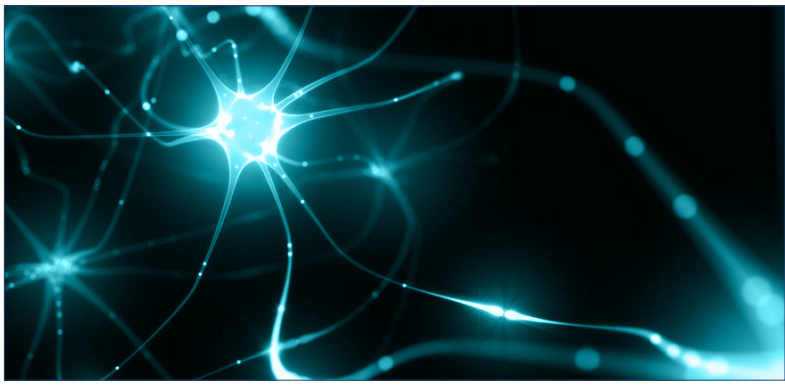
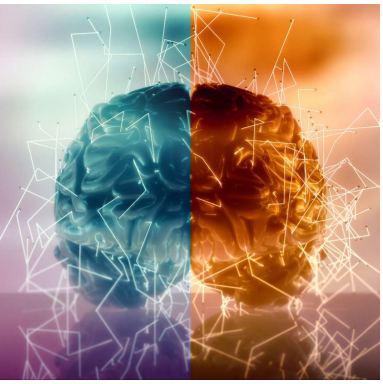
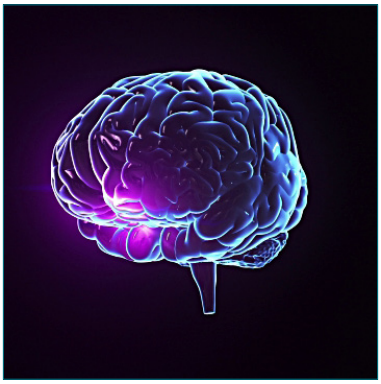
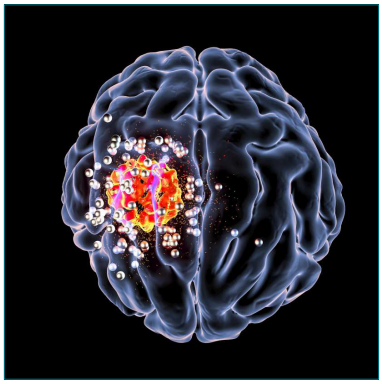

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Parietal-thalamic dysconnectivity during sustained attention processing in young adults with Traumatic Brain Injury

Xiaobo Li and **Ziyan Wu**
New Jersey Institute of Technology, USA

Traumatic brain injury (TBI) is a major public health problem with potentially serious long-term neurobehavioral sequelae. Attention deficits occur in approximately 15-20% of TBI survivors and are the most common persistent cognitive impairments post TBI. The consensus regarding appropriate evaluation of attention deficits in adults with TBI is rather limited due to lack of understanding of the neurobiological substrate associated with this syndrome.

In this study, functional magnetic resonance imaging data during a visual sustained attention task were obtained from 14 young adults who had history of one or multiple diffuse axonal TBIs which were clinically confirmed at least 6 months prior the study and 15 demographically matched normal controls. Task responsive brain activation map was constructed for each participant using FEAT/FSL (www.fmrib.ox.ac.uk/fsl). Between-group comparisons of whole brain voxel-based functional activations were conducted using unpaired two-sample t-test. Relative to controls, subjects with TBI showed decreased activations in frontal and parietal cortices and increased activations in bilateral thalami (Figure 1A). Based on these results, four regions of interest (ROIs) from the right middle frontal cortex, left inferior parietal cortex and bilateral thalami were located. The average time series inside each ROI was calculated. Functional connectivity between each pair of the ROIs was examined by calculating the Pearson's correlation coefficient of the average time series of the two ROIs. Between-group comparisons of the functional connectivity measures were carried out using unpaired two-sample t-test. Multiple comparisons were corrected using the FDR at $\alpha = 0.05$. Relative to controls, subjects with TBI showed significantly decreased functional connectivity between the left inferior parietal cortex and right thalamus.

Parietal cortex and thalamus are key components in attention and cognitive processing pathways. The results of decreased functional activations in parietal region, increased functional activations in thalamic area and reduced interactions between these two areas during visual attention processing in patients with TBI, thus suggest that functional alterations in parietal cortex and thalamus may significantly contribute to TBI induced attention deficits. Further study can focus on investigating associations between brain imaging and attention-related behavioral measures in TBI patients in a larger study sample.

Speaker Biography

Xiaobo Li is an Associate Professor and Director of the Computational Neuroanatomy and Neuroinformatics lab (CNN lab) in the Department of Biomedical Engineering at New Jersey Institute of Technology (NJIT). Dr. Li received her Ph.D. in 2004 from the University of Birmingham, UK, on geometrical modeling in digitized data. She has extensive research experience in developing and translating mathematical techniques to quantitatively evaluate the structural and functional organization in the human brain using structural MRI/fMRI/DTI data, and extensive clinical application experience in brain development and disorders such as Attention Deficit/Hyperactivity Disorder (ADHD), Autism Spectrum Disorder (ASD), schizophrenia, etc.

e: xli.aecom@gmail.com

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Chronic stress and moderate exercise prompt widespread common activation and limited differential activation in specific brain regions

Tae-Kyung Kim[1,2], **Seung-Jae Lee**[1], and **Pyung-Lim Han**[2]

[1]Seoul National University College of Medicine, South Korea

[2]Ewha Woman's University, South Korea

Chronic stress in rodents produces depressive behaviors, whereas moderate exercise counteracts stress-induced depressive behaviors. Stress and exercise appear to produce such opposing effects by changing the neural activity of specific brain regions. However, the detailed mechanisms through which the two different types of stimuli regulate brain function in opposite directions are not clearly understood. In the present study, we attempted to explore the neuroanatomical substrates mediating stress-induced depressive behavioral changes and anti-depressant effects of exercise by examining stimulus-dependent c-Fos induction in the brains of mice that were exposed to repeated stress or exercise in a scheduled manner. Systematic and integrated analyses of c-Fos expression profiles indicated that various brain areas, including the prelimbic cortex (PrL), parietal cortex (PaC), lateral septal nucleus (LS), and paraventricular nuclei of hypothalamus (PVN) were commonly and strongly activated by both stress and exercise, while the habenula (HB) and hippocampus (HP) were identified as being preferentially activated by stress and exercise, respectively. Exercise-dependent c-Fos expression in all regions examined in the brain occurred in both glutamatergic and GABAergic neurons. These results suggest that chronic stress and moderate exercise produce counteractive effects on mood behaviors, along with prompting widespread common activation and limited differential activation in specific brain regions.

Speaker Biography

Tae-Kyung Kim graduated from Korea University in South Korea with a bachelor's degree in biology. During graduate study at Rutgers University (Robert Wood Johnson Medical School), he studied the molecular mechanisms and regulation of eukaryotic gene expression under the supervision of Dr. Danny Reinberg (HHMI, currently at NYU). After obtaining a PhD degree in Biochemistry, he continued his research career in the laboratory of Dr. Michael Greenberg (Harvard Medical School) as a postdoctoral fellow, studying how neuronal activity controls gene expression in neurons to mediate synapse remodeling and plasticity. He joined the faculty in the Department of Neuroscience at UT Southwestern in 2010.

e: spt21@hanmail.net

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Coenzyme Q10 supplementation reduces oxidative stress and decreases antioxidant enzyme activity in children with Autism Spectrum disorders

Elham Mousavinejad
Ahvaz Jundishapur University of Medical Sciences, Iran

Antioxidants and oxidative stress can participate in pathobiochemical mechanisms of autism spectrum disorders (ASDs). The aim was to identify the effects of early CoQ10 supplementation on oxidative stress in children with ASDs. Ninety children with ASDs were included in this study, based on DSM-IV criteria and using Childhood Autism Rating Scale (CARS) scores. Concentrations of CoQ10, MDA, total antioxidant status (TAS) assay, and antioxidant enzymes (superoxide dismutase or SOD and glutathione peroxidase or GPx) activity were determined in serum before and after 100 days of supportive therapy with CoQ10 at daily doses of 30 and 60 mg. Data on children's behavior were collected from parents and babysitters. CoQ10 supportive therapy was determined after three months with daily dose 2× 30 mg improved oxidative stress in the children with ASDs. A relation was seen between serum MDA (r2 = 0.668) and TAS (r2 = 0.007), and antioxidant enzymes (SOD [r2 = 0.01] and GPx [r2 = 0.001]) activity and CARS score. Based on the results, high doses of CoQ10 can improve gastrointestinal problems (P = 0.004) and sleep disorders (P = 0.005) in children with ASDs with an increase in the CoQ10 of the serum. We concluded that the serum concentration of CoQ10 and oxidative stress could be used as relevant biomarkers in helping the improvement of ASDs.

Speaker Biography

Elham Mousavinejad has completed her MSc in the Department of Biochemistry, School of Medical Sciences, Ahvaz Jundishapur University of Medical Sciences, in 2016, and BSc Degree in Community Nutrition, School of Nutritional Sciences and Dietetics, Jundishapur University of Medical Sciences, Ahvaz, Iran in 2006. Her research area involved Nutritional Neuroscience and various nutritional deficiencies described in children with ASDs.

e: mousavinejad.e@ajums.ac.ir

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The role of Hypoxia in periventricular white matter degeneration

Santiago Martinez Sosa
UCL Institute of Neurology, UK

The deep and periventricular white matter is preferentially affected in several neurological disorders, including cerebral small vessel disease (SVD) and multiple sclerosis (MS), suggesting that common pathogenic mechanisms may be involved in this injury. Here we consider the potential pathogenic role of tissue hypoxia in lesion development, arising partly from the vascular anatomy of the affected white matter. Specifically, these regions are supplied by a sparse vasculature fed by long, narrow end arteries/arterioles that are vulnerable to oxygen desaturation if perfusion is reduced (as in SVD, MS and diabetes) or if the surrounding tissue is hypoxic (as in MS, at least). The oxygen crisis is exacerbated by a local preponderance of veins, as these can become highly desaturated 'sinks' for oxygen that deplete it from surrounding tissues. Additional hemodynamic deficiencies, including sluggish flow and impaired vasomotor reactivity and vessel compliance, further exacerbate oxygen insufficiency. The cells most vulnerable to hypoxic damage, including oligodendrocytes, die first, resulting in demyelination. Indeed, in preclinical models, demyelination is prevented if adequate oxygenation is maintained by raising inspired oxygen concentrations. In agreement with this interpretation, there is a predilection of lesions for the anterior and occipital horns of the lateral ventricles, namely regions located at arterial watersheds, or border zones, known to be especially susceptible to hypoperfusion and hypoxia. Finally, mitochondrial dysfunction due to genetic causes, as occurs in leukodystrophies or due to free radical damage, as occurs in MS, will compound any energy insufficiency resulting from hypoxia. Viewing lesion formation from the standpoint of tissue oxygenation not only reveals that lesion distribution is partly predictable but may also inform new therapeutic strategies.

Speaker Biography

Santiago Martinez Sosa is a medical student at University College London, from where he also received his bachelor's in Neuroscience (Hons). His interests include hippocampal physiology, neuroinflammation, and neurodegenerative disease. He is the UCL Medical Society coordinator for the Neurology society, which serves to facilitate student involvement in all branches of the specialty. This is his first publication.

e: zchamar@ucl.ac.uk

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