Keynote Forum
September 18, 2017

Neuroimmunology 2017

4th International Conference on

NEUROLOGY AND NEUROIMMUNOLOGY

September 18-19, 2017 | Embassy Suites by Hilton,
Dallas Park Central Area, Dallas, USA

Joel Brandon Brock, J Neurol Neurosci, 8:5
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Joel Brandon Brock

Cerebrum Health Centers, USA

Autoimmune encephalitis: The neuroimmunology of pediatric movement disorders and cognitive changes post infection: A functional integrative neurology approach

Autoimmune Encephalitis or pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (or PANDAS) is a disease that commonly has an epitope association with Streptococcus pyogenes (group A streptococci). The molecular memory between strep surface proteins and lysogangliosides, tubulin, pyruvate kinase, enolase, basal ganglia, dopamine D1, D2, Rapid strep or throat culture, ASO titers, DNase antibodies, Myelin basic protein antibodies, Alpha and beta Tubulin, Asiaganglioside , Cerebellar antibodies, Synapsin antibodies, GAD-65 antibodies, Dopamine D1 receptor antibodies, Dopamine D2 receptor antibodies, Lysogangliosides antibodies and CaM Kinase II antibodies are an important immunological component of understanding this disease. Looking at the interaction between the immune system and the brain and the basal ganglionic circuitry is imperative when discussing hyper and hypokinetic disorders. Diagnosis, management and outcomes of beta retrospective early analyses is will be discussed while looking at the exploration or two arms trials that are blinded, placeboed and controlled. These antibodies in the brain create a probability of generating tics, obsessive compulsive disorder (OCD) and its relationship to Sydenham chorea (SC), which is the neurologic manifestation of acute rheumatic fever. Other cognitive and movement manifestations may develop in someone afflicted with the condition.

Speaker Biography

Dr. Joel Brandon Brock is a Certified doctorate level trained Family Nurse Practitioner and a Diplomate in Functional Neurology, integrated medicine, and nutrition as well. In Dallas, Texas he serves as a clinician at Foundation Physicians Group. Dr. Brock has a passion for treating patients from pediatric to geriatric age groups as well as lecturing and giving learners didactic and academic skills in a way that is easy to digest, comprehend and utilize in a clinical setting. He has developed thousands of multidisciplinary hours of curriculum pertaining to neurology, nutrition, physical diagnosis, pharmacology, immunology, endocrinology and this has impacted students of multiple educational backgrounds, including medical doctors, nurse practitioners, chiropractors and more. He enjoys teaching and providing education and support to facilitate learning for multiple groups and agencies. This includes state association meetings to 2 governmental panels, and for his own lecture companies. Dr. Brock enjoys the development and administration of lectures and seminars of all types. Dr. Brock received the most outstanding functional neurology teacher of the year from the ACA council of Neurology four years straight and two times from IAFNR (International Association of Functional Neurology and Rehabilitation). Dr. Brock received the humanitarian award from IAFNR. Dr. Brock and is also the honorable recipient of the prestigious Living Legacy Award from Samford Universities Ida Moffett School Nursing in 2015. Currently Dr. Brock is a Doctorate of Nursing Practice from Duke University and a global clinical research scholar from Harvard University. Dr. Brock's unique blend of clinical and teaching experience along with a background in medicine, chiropractic, neurology and nutrition has created a very unique and integrated clinical background that has helped him treat difficult cases and offers comprehensive and multi-perspective angles on education and clinical presentations. His greatest desire in life is to help those with chronic health problems.

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Michael Paul kilgard, J Neurol Neurosci, 8:5
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Michael Paul kilgard

University of Texas at Dallas, USA

Directing neural plasticity to understand and treat neurological disease

Neurological disorders are characterized by dysfunction across multiple brain networks. Effective treatments will require tools capable of modifying activity across these networks. During normal learning the timing of neuromodulator release regulates network plasticity. Brief bursts of vagus nerve stimulation can safely trigger release of these same plasticity-promoting neuromodulators in patients to repair damaged networks. We have shown that pairing VNS with specific experiences causes highly-specific and long-lasting changes in sensory, motor, or emotional networks. Pairing VNS with a specific movement reorganizes motor cortex. Pairing VNS with a specific tone frequency reorganizes auditory cortex. Pairing VNS with speech selectively enhances the cortical response to specific words. Pairing VNS with emotionally salient cues directs plasticity in the amygdala. Animal models of chronic stroke, traumatic brain injury, spinal cord injury, nerve damage, PTSD, and tinnitus all make substantially greater gains when VNS is timed to coincide with specific rehabilitation events compared to identical rehabilitation without VNS. The four clinical trials of this approach have all been successful. Pairing VNS with physical therapy in chronic stroke patients tripled the functional gains compared to controls who received identical physical therapy without VNS. Pairing VNS with tones in chronic tinnitus patients reduced tinnitus severity and decreased hypersynchronous gamma activity in auditory cortex, as in earlier animal studies. These results demonstrate that pairing VNS with rehabilitation generates highly specific network changes that treat the underlying problem. Targeted Plasticity Therapy will be an important addition to the growing toolbox of technology to facilitate study and repair of the human brain. Millions of lives were saved once scientists developed effective adjuvants (aluminum salts) that made it possible to direct plasticity in the immune system. Like vaccine technology, Targeted Plasticity Therapy is a platform technology that can be applied to many conditions.

Speaker Biography

Michael Paul kilgard has his training in Biochemistry and Genetics at UC Berkely and in Neuroscience at UC San Francisco. He is the Margaret Fonde Jonnson Professor and the Associate Director of the Texas Biomedical Device Center. He has published more than 90 papers in peer reviewed journals, including *Nature, Science, Neuron*, and *Stroke*. He holds 23 US patents and regularly reviews for the NIH. His work is currently supported by DARPA, NINDS, NIDCD, Wings for Life Spinal Cord Research Foundation, and the W W Caruth, Jr. Foundation Fund at Communities Foundation of Texas.

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Robet M Herndon, J Neurol Neurosci, 8:5
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Robert M Herndon

Mississippi State University, USA

The Role of Herpes Viruses in Autoimmune Diseases

The Herpes viruses are a group of species specific viruses with very little ability to cross species. Humans are the only known hosts for these viruses and they survive through latency and periodic recrudescence. There are nine known human Herpes viruses. Of these, 4, 6 and possibly 7 (EBV and Roseola viruses) appear to be involved in autoimmune disease. EBV has been found in the joint lesions or rheumatoid arthritis and, though controversial, has been reported in multiple sclerosis (MS) plaques. Also, Herpes 4 has been found in MS plaques. These Herpes viruses have the ability to immortalize lymphocytes (6 B-cells, 6 and 7 CD4 + T-cells and provide them resistance to elimination by apoptosis. Thus, the thymus may be unable to eliminate them in the thymus which is important in eliminating cells directed against self thus permitting autoimmunity. In rheumatoid arthritis, EBV is found in synovial tissues and is presumed to contribute to the inflammatory response and destroying the lymphocytes in the bone marrow and synovial tissues is usually effective in controlling the disease. In this lecture we will discuss the evidence for Herpes viral involvement in autoimmune diseases and possible mechanisms by which they could induce autoimmunity. In MS, a similar mechanism is likely to be in play and anti-CD20 monoclonal antibodies (rituximab and ocrelizumab) are effective therapies.

Speaker Biography

Dr. Robert M completed his MD degree at the age of 23 years from the University of Tennessee. His internship and neurology residency were at Wayne State University followed by fellowships at the Montral Neurological institute and Harvard University. He has serrve on the faculty at Stanford Medica School, Johns Hopkins Medical School, served as director of the Center for Brain Research, university of Rochester and at Oregon health and Science University.and is now Professor Emeritus at Univeristy of Mississippi.. He has published more than 100 papers in reputed journals and is the founding editor of the International Journal of MS Care.

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Aage R Møller, J Neurol Neurosci, 8:5
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Aage R Møller

The University of Texas at Dallas, USA

What is new in Neuroscience?

New techniques make it possible to study morphology and function of the nervous system in healthy humans and individuals with diseases. Other technological developments make it possible to determine the strength of connections in the brain and the spinal cord. Symptoms of disease were earlier believed to be caused by pathology of specific parts of the nervous system, but it has become evident that many parts of the brain may be involved in causing the symptoms of each one of many diseases. Symptoms of diseases were earlier believed to be related to detectable morphological changes. It is now known that symptoms of many neurological diseases may be caused by changes in connections between parts of the central nervous system, changes that do not have morphological correlates that can be detected by available clinical diagnostic methods. Neuroplasticity that makes it possible to learn new skills and adapt to changing demands also has dark sides; maladaptive plasticity plays an important role in many common diseases such as chronic neuropathic pain, tinnitus, spasticity, and probably also fibromyalgia and the chronic fatigue syndrome. There is now evidence that the role of harmful (maladaptive) neuroplasticity is greater than earlier presumed. Altered functional connections in the brain are related to the pathology of diseases such as chronic neuropathic pain and severe tinnitus, age-related symptoms and signs. The cholinergic system of the forebrain (the nucleus of Meynert) that promotes activation of neuroplasticity can be activated through the vagus nerve. Electrical stimulation of the vagus nerve may thereby promote plastic changes, and it may reverse the symptoms and sign of some plasticity diseases when paired with appropriate sensory stimuli. There is recent evidence that both the innate and the adaptive immune systems can influence neural functions and that the nervous system can affect the immune system. The vagal immune reflex is an example. The immune system can modulate many forms of pain and immunoglobulin is now being considered for treatment of certain pain conditions. Stress can suppress the immune system. Signals from the gut affect many neural functions, for example, receptors in the distal portion of the small intestine can affect pain circuits in the brain. Epidemiological studies have shown that the risk of giving birth to a child with autism spectrum disorder or spina bifida (neural tube defects) can be lowered significantly if the mother takes a B-vitamin (folic acid) before and during pregnancy, indicating that the root cause of these diseases are errors in the early development of the brain that occurs in early stage of pregnancy. This is an example of how the occurrence of serious diseases that have no known cure can be reduced by administration of a harmless supplement. Unfortunately, only a few people take advantage of that.

Speaker Biography

Aage R Møller is known internationally for his innovative research on sensory systems and neural plasticity and for developing methods for reducing the risk of neurological deficits in neurosurgical operations. His work has helped establish UT Dallas as a leader in tinnitus-related research. His lengthy research career has focused on four primary areas: The basic function of the ear, sound transmission in the middle ear and cochlea, the neural code of complex sounds and neural plasticity. He eventually moved on to research in humans aimed at studying disorders of the year and the nervous system, such as tinnitus. He began his research career at the famed Karolinska Institut in Sweden. In 1978, he was invited to join the University of Pittsburgh. There he did innovative research in the area of neurosurgery and intraoperative neurophysiology; he developed methods for reducing the risks of serious neurological deficits after neurosurgical operations. He was one of the founders of a new specialty; intraoperative neurophysiological monitoring and he did innovative research that lead to better understanding of several neurological diseases. When he joined UT Dallas in 1997, he became interested in abnormalities in the nervous system function among individuals with autism. He developed teaching programs in the biology of pain; sensory systems, neuroplasticity and he established the first university program in teaching IONM in a graduate program. During his time at UT Dallas, he was named the university's "President's Teaching Excellence Award," won Teacher of the Year for the School of Behavioral and Brain Sciences, and was named distinguished Lecturer in Cognition and Neuroscience. He earned his PhD in Medical Science at the Karolinska Institut in Stockholm, Sweden.

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Denis Gris

Université de Sherbrooke, Canada

Computerized behavioral assessments of novel model of multiple sclerosis

Multiple sclerosis is an autoimmune disease of the central nervous system. Recently, we have discovered an endogenous pathway that limits inflammation multiple sclerosis-like disease in mice. One of the key molecules of these pathways is Nlrx1 that belongs to Nlr family of proteins. Nlrs bind multiple proteins inside cells thus redirecting molecular signaling. Using state-of-the-art automated behavioral platform, we demonstrate that Nlrx1 inhibit progression of the diseases in a mouse model of MS. Furthermore, we were able to construct mice with increased predisposition to MS. These mice demonstrate the spontaneous appearance of the disease without any immunization. This model helped us to dissociate sickness behavioral profile from the behavioral signature of neuroinflammation. In addition, our results suggest that predisposition may rise from the disturbed homeostasis in the central nervous system rather than the peripheral activation of immune system. We observed that the inflammatory effect of Nlrx1 at the mitochondrial level, in inflammatory cells such as microglia and astrocytes, results in inhibition of assembly of proinflammatory pathways including Type I interferon and NFkB. Accordingly, we observed the reduction in the expression of iNOS, cytokines including IL-1beta and TNF-alpha during microglial activation. In neurons, Nlrx1 effect results in inhibition of necrosis and increased viability. Using N2A cell line, we demonstrated that Nlrx1 protects cells from rotenone toxicity. We demonstrated that Nlrx1 overexpressing cells were more viable than Nlrx1 KO cells and the ratio of apoptosis to necrosis was shifted to necrosis in cells that lacked Nlrx1. In conclusion, our study demonstrates that targeting central nervous system innate immune responses presents promising novel strategy treatments of multiple sclerosis.

Speaker Biography

Denis Gris has started his scientific career with the Master's and PhD in Neuroscience at Dr. Lynn Weaver's laboratory at the University of Western Ontario. He studied the role of inflammation in spinal cord injury. He discovered that the influx of neutrophils is detrimental for recovering after spinal cord injury. Using anti CD11d antibody as a treatment, he demonstrated that animals recovered faster and better after the treatment. Also, he showed that sever spinal cord injury results in massive inflammatory reactions throughout the body leading to syndrome similar to multiple organ dysfunction syndrome. He continued his education in Dr. Jenny P-Y Ting's laboratory as a Post-doctoral fellow at the University of North Carolina at Chapel Hill. There, he studied in detail mechanism of activation of innate and adoptive immune responses. In collaboration with Dr. Wen, Dr. Eitas, Dr. Allen, and other members of the laboratory, he studied inflammation during obesity which leads to insulin resistance; innate and adoptive responses during multiple sclerosis. In summary, his role in this laboratory was to define the role of novel family of immuno regulatory proteins (NLRs) in different human diseases. Currently, he is a member of Immunology Program at the University of Sherbrooke and is studying neuro-immune interactions during healthy state and disease.

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John M Baumann, J Neurol Neurosci, 8:5
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John M Baumann

University of Louisville, USA

Discussing with your patients the importance of reclaiming posi-spective, uncovering your purpose and staying engaged

My target audience is all medical personnel. The purpose of this Presentation is to encourage medical personnel who treat those diagnosed with a life-changing condition (for example, Parkinson's disease) to go beyond the clinical role and discuss with their patients the importance of (a) keeping or, if the disease has caused them to take on a negative attitude, reclaim a positive perspective, (b) uncover their purpose and (c) stay engaged in whatever they can still do that they love to do using the twelve Decide Success principles. In today's world, medical personnel are under so much pressure to see as many patients as possible that there is no time for the medical personnel to address important, but not clinical information.

Speaker Biography

John M Baumann inspires and helps real people to live their lives to the fullest, and even embrace their life-changing event, with the goal of uncovering their life's purpose (JohnBaumann.com). He is an internationally-recognized inspiring success speaker. In 2002, at 41 years old, working as the top attorney for a public company, John was diagnosed with Parkinson's disease. From 2005 until 2014, John taught law at the College of Business at the University of Louisville to over 1,000 undergraduates. John was selected the Most Inspiring Professor John wrote a book entitled, "Decide Success - You Dead Yet.

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