TENTATIVE PROGRAM

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS

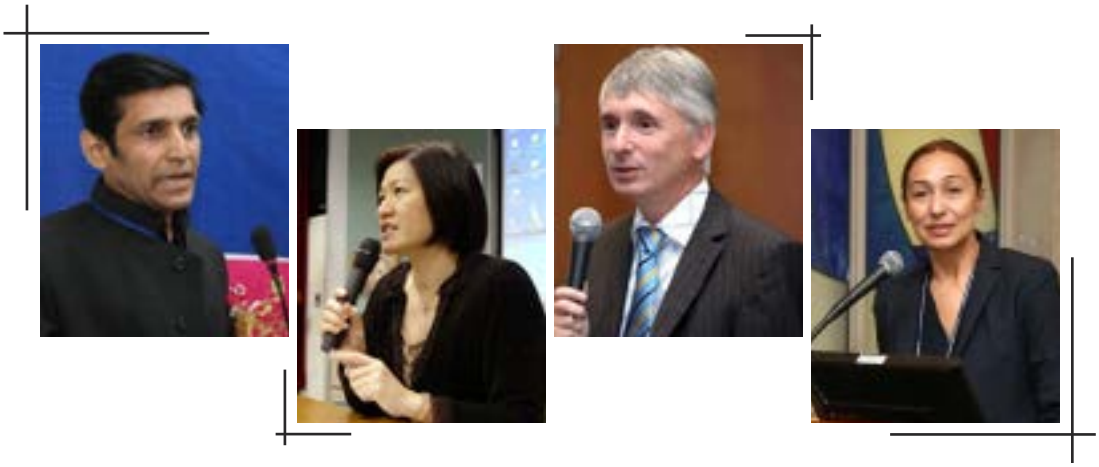
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Allied Academies
85 Great Portland St, Marylebone
London W1W 7LT, UK
http://www.alliedacademies.com/



International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

KEYNOTE FORUM

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

SANJOY MUKERJI
India

MIND-BODY MEDICINE

BIOGRAPHY

Dr. Sanjoy Mukerji is a Gold Medalist plus National and International Award-Winning Psychologist in Mumbai. He has done his Post Graduate Diploma in Psychological Counselling from the Institute for Behavioral and Management Sciences, India. Moreover, he has completed his Degree of Doctorate in Philosophy (Alternative Medicines) from the Indian Board of Alternative Medicines, established under the World Health Organization (WHO). In the field of alternative medicines, he has researched and specialized in mind-body medicine.

His counselling and therapies are based on the principle that our Mind affects our 3 Bs:

1. Brain (mental health)
2. Body (physical health)
3. Behavior (social health)

In his around 20 years of experience and practice, he has been instrumental in healing and helping thousands of people across the World through his counselling, therapy, talks, lectures, seminars, workshops, articles and books; and has received fantastic feedback, praises and blessings from them. He has been interviewed on various TV channels, and covered by almost all major newspapers and magazines.

Mind-body medicine explores the interconnection between the mind and body, under the premise that the mind affects "bodily functions and symptoms." As per the University of Maryland Medical Center, mind-body medicine uses the power of thoughts and emotions to influence physical health. As Hippocrates once wrote, "The natural healing force within each one of us is the greatest force in getting well." This is mind-body medicine in a nutshell.

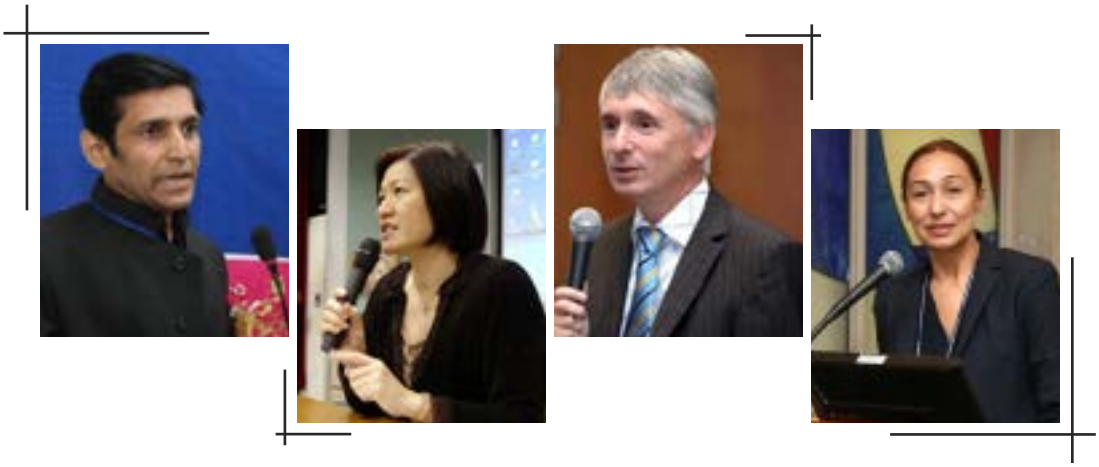
The term "psychosomatic disease/disorder/illness" is mainly used to mean "a physical disease that is caused, or made worse, by mental factors." The term is also used when mental factors cause physical symptoms but where there is no physical disease. For example, chest pain may be caused by stress and no physical disease can be found.

Some physical diseases are prone to be made worse by mental factors such as stress and anxiety. At any given time, a person's mental state can affect the degree of severity of a physical disease. Physical symptoms that are caused by mental factors are also called somatization or somatoform disorders. These symptoms are due to increased activity of nervous impulses sent from the brain to various parts of the body. There is a deep connection between the mind (beliefs, thoughts and emotions) and the different parts of the body and physical problems.

A number of factors may play a role in psychosomatic disorders, such as personality traits; genetic or environmental family influences; biological factors; learned behavior and more.

When one is not at ease, that means there is some kind of dis-ease; and disease can be reversed (completely or to a great extent) by simply reversing or changing mental/thought patterns, and at times by adding some physical exercises and changing some food habits.

According to Dr. J. A. Winter, M.D., the psychosomatic illness is one of function, rather than of structure, although structural changes may occur later. It is based on some past experience, usually painful. This illness seems to arise from problem situations and from words (reflection of thoughts), rather than from actual injuries, or infection.



International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

SCIENTIFIC TRACKS & ABSTRACTS

SESSIONS

June 21-22, 2018

Alzheimer's Disease | Dementia Care | Neurological Infections| Neuroimmunology |Parkinson's Disease| Movement Disorders | Genetics and Epigenetics in Neurodegenerative Disorders| Neuro-Degenerative Disorders | Central Nervous System | Neurological Disorders and Stroke |Paediatric Neuropharmacology |Psychiatric Disorders or Psychological Syndromes | Cerebrovascular Diseases

Session Introduction

Session Chair
Chan Kam Tim Michael
China

Session Co-chair
Sanjoy Mukerji
India

Title: **Itch-scratch cycle is a chronic cognitive addictive behaviour in our mind**
Chan Kam Tim Michael, China

Title: **Are inflammatory cytokines associated with mood symptoms among patients with bipolar disorder?**
Esther Lin, Taiwan

Title: **Pharmacogenetics of extrapyramidal syndromes associated with coadministration of opioids and antidepressants**
Helena Sarac, Croatia

Title: **Skin prion and its implications in prion diseases and other neurodegenerative diseases**
Zou WQ, USA

Title: **Candiate Biomarkers and CSF Profiles for Alzheimer's disease and CADASIL (Vascular dementia VS.Alzhemer's disease)**
Bowirrat Abdalla, Israel

Title: **The Biomechanical Aetiology of the so-called Idiopathic Scoliosis. The Role of Gait and Standing at "Ease" on the Right leg in the Development of the Deformity**
Tomasz Karski, Poland

Title: **Clinical trials of cell therapies in neuromuscular diseases**
Daniel Skuk,Canada

Title: **The Mechanisms of Neuroprotection of exosomes in ischemic brain disease**
Qiang Zhang, China

Title: **Brainstem Dysfunction in Neuropsychiatric Disorders- AD/PD/Depression**
Harry W.M.Steinbusch, Netherland

Title: **Effects of EEG-based Active Assisted Neurofeedback Therapy on Hemiplegic Upper Extremity Motor Function**
Joo-Hee, Park, South Korea

Title: **Mind-Body Medicine**
Sanjoy Mukerji, India

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

ITCH-SCRATCH CYCLE IS A CHRONIC COGNITIVE ADDICTIVE BEHAVIOUR IN OUR MIND

Name & Country

Chan Kam Tim Michael

China

Abstract

Scratching is a distinguishing feature of many resistant dermatosis like chronic atopic dermatitis. Recently, discovery in neuroendocrinology, immunology and MRI studies suggest itch – scratch cycle may be an addictive neuroendocrine mediated pathological movement pathway with an aberrant and imbalance of neurotransmitters in Central Nervous system (CNS).

Mas-related G protein-coupled receptor A3 (MrgprA3) and MrgprC11 expressed afferent neurons penetrated in the epidermis together with Transient Receptor Potential (TRP) receptors like TRPV (vanilloid) 1, TRPV 3, TRPV4, TRPA (ankyrin) 1 together with Serotonin receptors relay itch signals from the periphery synapses to the dorsal horn of spinal cord. Pruritogenic signals via the afferent neurones synapse with gastrin -releasing peptide receptors(GRPR) in the spinal cord. GRPR activation released substance P, Calcitonin G Releasing Peptide, Vasoactive Intestinal Peptide including Pituitary adenylate cyclase activating peptides which was distributed in the CNS. Endothelin-1, tachykinin through neurogenic inflammation increased levels of Th2 cytokines and interleukin – 31 also mediate itch. The central station of itch transmission in our brain is the Thalamus. Hedonic scratch activated the primary somatosensory S1 areas gave the perception of comfort in the cingulate cortex decided the planned motor response of scratching. The midbrain, striatum, ventral tegmental area, caudate nucleus and ventromedial prefrontal cortex as shown by MRI studies are activated in this pleasant circuitry. If this endogenous neuroendocrine circuitry become uncontrolled; harmful cravings behaviour superseded. Insula cortex and Claustrum of the brain play a prominent role in interoception including addiction. They are highly activated when itch is intensified. The adverse pruritic experience is represented in amygdala, subcallosal gray matter and nucleus accumbens. The miswiring and imbalance of 5 Hydroxytryptamine and its multiple receptors are involved.

Besides pharmacological intervention, cognitive behavioural therapy including education, refocusing attention strategy; virtual reality immersion; audio visual distraction techniques; habit reversal training; arousal reduction and cognitive restructuring are helpful.

Biography

Chan Kam Tim Michael is a practicing private Dermatologist in Hong Kong. He received Dermatology Specialist Fellowship in Hong Kong Academy of Medicine in 1998. In the same year, he was granted a Government of Hong Kong scholarship for post graduate training in UCLA, USA. He is now the Vice President of the Association of Integrative Aesthetic Medicine in Hong Kong. He was editor of the Hong Kong Journal of Dermatology and Venerology from 2002 to 2007. He acts as Editorial Board Members of the following international journals since 2017: Research Journal of Nervous System; The Cognitive Neuroscience Journal, Medical Reports and Case Studies and Advances in Neurology and Neuroscience. He has been working in the University of Hong Kong as Honorary Clinical Assistant Professor from 2007 to 2009. He is now a part time lecturer in the Baptist University of Hong Kong for teaching Master Course in Public Health.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

ARE INFLAMMATORY CYTOKINES ASSOCIATED WITH MOOD SYMPTOMS AMONG PATIENTS WITH BIPOLAR DISORDER?

Name & Country

Esther Ching-Lan Lin

Taiwan

Abstract

Bipolar disorder (BD) is a severe mental illness characterized by chronic course, pervasive instability, and higher recurrence and suicide rate. Evidence supports the associations of instable social rhythm and increased inflammatory cytokines and symptom severity in BD. This cross-sectional study examined the relationships among inflammatory cytokines and mood symptoms. One-hundred and twenty-one individuals with a DSM-IV diagnosis of BD were recruited from the psychiatric ward and outpatient department of a southern medical center in Taiwan. Most participants were female, unmarried, unemployed, diagnosed as bipolar II, and outpatients. There were no significant associations between inflammatory cytokines and mood symptoms. Relative lower level of inflammatory cytokines in these stabilized patients cannot reflect from different mood states. Future studies should compare the inflammatory cytokines of patients who were at different mood states.

Keywords: bipolar disorder, inflammatory cytokines, mood symptoms

Biography

Esther Ching-Lan Lin is an Associate Professor in the Department of Nursing, College of Medicine, National Cheng Kung University, and adjunct Head Nurse of the Department of Psychiatry, National Cheng Kung University Hospital, Tainan, Taiwan. She has been a nurse, a manager, a teacher, and an advisor for 18 years. After completing her PhD at National Taiwan University. She continued her academic career in nursing education and has focused on improving the quality of care for patients with severe mental illness. She has published 30 papers in English-language and Chinese-language journals—most recently on developing and evaluating psychosocial treatments for patients with schizophrenia and bipolar disorder—and has been an editorial board member for the past 3 years of a national nursing journal in Taiwan.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

PHARMACOGENETICS OF EXTRAPYRAMIDAL SYNDROMES ASSOCIATED WITH COADMINISTRATION OF OPIOIDS AND ANTIDEPRESSANTS

Name & Country

Helena Sarac

Croatia

Abstract

Opioid analgesics are widely used for the pain relief. More than 0.8% of the global population between 15 and 65 used opioid analgesics, in last years. The currently marketed alkaloid opiates are codeine, hydrocodone, oxycodone, methadone, tramadol, fentanyl, morphine, hydromorphone and oxymorphone. Opioids have a narrow therapeutic index, and can be associated with severe adverse reaction, addiction, dependance, tolerance and fatal overdose. Opioid's adverse effects have been shown to increase the risk of seizures and serotonin syndrome characterized as a triad of neuro-excitatoty features; altered mental sttaus (e.g. sedation or agitation), autonomic hyperactivity (e.g. diaphoresis, mydriasis, tachycardia, nausea, urinary retention, diarrhea) and neuromuscular hyperactivity (tremor, myoclonus, hyper-reflexia, pyramidal rigidity). Although the development of extrapyramidal symptoms is under-recognized in clinical practice, with the widespread use of opioid analgesics, increasing numbers of patients with movement disorders following exposure to these drugs have been reported. Chronic pain syndromes are commonly associated with depression and clinicians simultaneously treat both of these conditions prescribing opioids for pain while also administer a selective serotonin reuptake inhibitor (SSRI) for depression. Although there are much efforts has been directed to prevention of misuse, the importance of pharmacokinetic drug-drug interactions related to opioids has received little attention. Drug-drug-interactions-induced serotonin syndrome caused by treatment with oxycodone and SSRI antidepressants is widely known.2,3,4 Herein, we report a cases of extrapyramidal syndromes induced by coadministration of antidepressants and opioids, caused by cytochrome 450 polymorphisms and drug-drug interactions.

Biography

Helena Sarac was born in 1968 in Zagreb. She graduated from Medical School in 1992 and attained her PhD from Medical School University of Zagreb in 2013. She was a visiting research scientist at the Mount Sinai Hospital,New York. Since 1999 she had headed the Diagnostic Center Neuron at the Croatian Institute for Brain Research, Medical School University of Zagreb. She is neurologist at the Depratment of neurology, University Hospital Centre Zagreb, Croatia and scientist at the Centre of Research Excellence for Clinical and Translational Neuroscience. Her research topics are movement disorders, neurodegeneration, and pharmacogenetic of extrapyramidal syndromes. Her significant contribution to the development of science in the neuroimmunology. Dr Sarac has long been interested in how serotonergic system is influenced by autoimmune disorders. She authored multiple scientific publications that have been cited, and has been serving as an editorial board member of reputed Journals and has been serving as an editorial board member of reputed Journals. Dr Sarac has been guest speaker at the international conferences worldwide.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

SKIN PRION AND ITS IMPLICATIONS IN PRION DISEASES AND OTHER NEURODEGENERATIVE DISEASES

Name & Country

Wenquan Zou

USA

Abstract

Prions (or PrPSc) are associated with a group of fatal transmissible prion diseases including sporadic Creutzfeldt-Jakob disease (sCJD, the most common human prion disease) in humans as well as scrapie, mad cow disease, and chronic wasting disease in animals. The currently incurable sCJD is transmissible, due to the contamination of abundant infectious prions in the brain through medical or surgical procedures. Some epidemiological studies have also associated sCJD risk with non-neurosurgeries, suggesting that prions may be present in other tissues such as skin. In addition, once disease onset has occurred, the brain becomes inevitably damaged. So, preclinical detection is key to providing the critical window for early treatments before irreversible brain damages occur once cures become available. Our recent study using the highly sensitive real-time quaking-induced conversion (RT-QuIC) assay and humanized transgenic (Tg) mice-based bioassay revealed that the skin of sCJD patients harbors infectious prions (Orrú et al., 2017). Moreover, our new preliminary results further indicate that skin PrPSc is detectable by RT-QuIC and serial protein misfolding cyclic amplification assays far ahead of neuropathological changes in prion-infected animal models. Our findings not only raise concerns about the potential for iatrogenic sCJD transmission via skin but also provide a basis for establishing alternative premortem and postmortem diagnostic assays for prion diseases. Moreover, they may improve our understanding of the role of other skin misfolded proteins in the diagnosis and pathogenesis of neurodegenerative diseases such as Alzheimer's and Parkinson's diseases in which disease-specific misfolded proteins have been detected in the skin of patients with these diseases. [Supported by the CJD Foundation, NIH (NS062787, NS087588 and NS096626), and CDC].

Biography

Wenquan Zou, MD/PhD, is Associate Professor at the Departments of Pathology and Neurology and Associate Director of the National Prion Disease Pathology Surveillance Center at the Case Western Reserve University School of Medicine in Cleveland, Ohio, USA. Dr. Zou received his medical degree from Jiangxi Medical College, his MSc from Tongji Medical University, and his PhD from Shanghai Medical University. He has practiced Internal Medicine and Nephrology for years in Nanchang and Shanghai, China. Dr. Zou's current research focus is in the areas of the formation and inhibition of misfolded protein aggregates in the conformational diseases especially on the physiological and pathologic prion proteins in prion diseases as well as on neurotoxic amyloid β and α-synuclein in Alzheimer's disease and Parkinson's disease, respectively.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

DEMENTIA OF ALZHEIMER'S TYPE AMONG ARAB POPULATIONS: GENETICS AND EPIDEMIOLOGICAL STUDIES

Name & Country

Bowirrat Abdalla

Israel

Abstract

Introduction: Neurodegenerative disorders, Primarily, are multifactorial diseases characterized by chronic and progressive loss of neurons in discrete areas of the brain, causing debilitating symptoms and globally decreasing cognitive function such as dementia, loss of memory, loss of sensory or motor capability, decreased overall quality of life and well-being, disability, and eventually, premature death.

Objective: To study the genetic and environmental risk factors and the prevalence of dementia of the Alzheimer type (DAT) among the elderly in an Arab community in Israel.

Material and Methods: Epidemiological and genetic studies of dementia have rarely been reported in an Arab population. Alzheimer disease (AD [MIM #104300]) is a progressive, neurodegenerative disease characterized clinically by gradual loss of memory and pathologically by neurofibrillary tangles and amyloid plaques in the brain. We have observed an unusually high prevalence of dementia of the Alzheimer type (DAT) in Wadi Ara, an inbred Arab community in northern Israel comprising ~850 persons over the age of 60 years. Apolipoprotein E (APOE- ε4), has been established as a strong susceptibility marker that accounts for nearly 30% of the risk in late-onset AD.

Results: Remarkably, in our study DAT is not associated with APOE because the frequency of the ε4 allele is very low in both nondemented (2.4%) and demented elders (3.6%). We also map chromosomal loci contributing to DAT susceptibility; we conducted a 10 cM scan in a series of twenty cases and twenty controls selected from one hamula. Markers from 18 chromosomal regions showed significant allelic association with DAT ($P<0.05$). Locations on chromosomes 2, 9 and 10 remained significant after testing additional affected and non-demented individuals. Significant associations were also observed for markers on chromosome 12, which overlap with a locus implicated in previous genome scans. Additionally, several lines of evidence support for a role of angiotensin converting enzyme (ACE) in Alzheimer disease (AD). Most genetic studies have focused on an Alu insertion/deletion (I/D) polymorphism in the ACE gene (DCP1) and have yielded conflicting results. We evaluated the association between 15 (SNPs) in DCP1, including the I/D variant, and AD in a sample of 92 patients with AD and 166 non-demented controls from an inbred Israeli Arab community.

Although there was no evidence for association between AD and I/D, we observed significant association with SNPs rs4343 (P= .00001) and rs4351 (P=.01). Conclusion: In Wadi Ara the high prevalence may be due to a founder effect enhanced by consanguinity, which make this population attractive for investigating DAT susceptibility recessive genes; thus, a specific disease susceptibility allele may be overrepresented in cognitively impaired subjects compared with cognitively healthy residents. Other two main conclusions can be drawn from the genome-wide linkage and linkage disequilibrium (LD) studies. Firstly, multiple genes are involved in DAT. Secondly, there is a high level of consistency among linkage and association studies regarding the general location of putative AD genes. However, the general location of putative AD genes on a given chromosome covers a broad region, which may contain several genes.

Biography

Bowirrat Abdalla M.D., Ph.D. has completed his MD from Rome University, his residency in Clinical Neurology from London University, UK, his PhD from Tel-Aviv University, Israel and postdoctoral studies from Boston University, USA. He received his Professorship from Boston University. He has published more than 105 manuscripts and 6 books in reputed journals and has been serving as an editorial board member of repute. Furthermore, he received many international awards including the Bruce S. Schoenberg international award in Neuroepidemiology of AD from the American Academy of Neurology.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

THE BIOMECHANICAL AETIOLOGY OF THE SO-CALLED IDIOPATHIC SCOLIOSIS. THE ROLE OF GAIT AND STANDING AT "EASE"ON THE RIGHT LEG IN THE DEVELOPMENT OF THE DEFORMITY

Name & Country

Tomasz Karski

Poland

Abstract

The development of scoliosis is connected with the asymmetry of hip movement in gait and with habitual standing "at ease" only or mostly on the right leg. During the gait,due to the restriction of movement of the right hip, a compensatory movement is transmitted to the pelvis and to the spine causing scoliosis. Another influence is connected with permanent standing "at easy" on the right leg. Other influence are connected with abnormalities of central Nerve system (CNS) and they are extension contracture of spine, anterior tilt of pelvis and laxity of joints.

Biography

Tomasz Karski MD PhD studied at Medical University in Lublin has completed his Medical Doctor degree and PhD. He served as a Head of Chair and Department of Pediatric Orthopedics and Rehabilitation of Medical University in Lublin/Poland. He is a Member of Polish Orthopedic and Traumatology Association, Société Internationale de Chirurgie Orthopédique et de Traumatologie (SICOT) from 2002 and also honorary member of Hungarian, Slovak and Czech Orthopedic and Traumatology Association. His research lies in orthopedics: spine, hips, knee, feet, CP and others. He has published 479 articles and 5 books.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

CLINICAL TRIALS OF CELL THERAPIES IN NEUROMUSCULAR DISEASES

Name & Country

Daniel Skuk

Canada

Abstract

Cell therapy is an experimental strategy for a potential treatment of genetic and / or degenerative muscular disorders, among which the most important target is Duchenne muscular dystrophy (DMD). To be used in this approach, the cells must be myogenic, that is, they must have at least one of the following properties: (1) fuse with the patient's myofibers to induce the expression of therapeutic proteins in them, (2) form new myofibers, and / or (3) give rise to new muscle specific stem cells (satellite cells). Considering reports of experiments conducted on mice and dogs, the repertoire of cells exhibiting some of the myogenic capacities seems to have expanded in recent years. Among these cells we have: CD56+ muscle-derived cells, muscle-derived stem cells, CD133+ cells, mesoangioblasts / pericytes, myoendothelial cells, ALDH+ cells, PW1+/Pax7– interstitial cells, and ß-4-integrin+ cells. The clinical studies of cell therapy conducted so far showed that, of the four cell types transplanted in patients with DMD, namely CD56+ muscle-derived cells, bone marrow derived cells, CD133+ cells and mesoangioblasts, the only for which there were observed myogenic properties in the clinics were CD56+ muscle-derived cells, that is, satellite cell derived myoblasts. In a clinical trial, we allotransplanted CD56+ muscle-derived cells in 1 cm3 of muscle in 9 patients with DMD immunosuppressed with tacrolimus. Four weeks later, we observed restoration of the therapeutic protein (dystrophin) in 3.5% to 26% myofibers. Evidences of small myofiber neoformation and of potentially graft-derived satellite cells were also observed. A 26-years old DMD patient also received cell allotransplantations under tacrolimus immunosuppression in different muscles, restoring dytrophin in 27.5% of myofibers at 1 month and in 34.5% at 18 months. This patient evidenced that our protocol was feasible in large muscles of humans and that long-term expression of donor-derived dystrophin can be obtained under proper immunosuppression. Further improvements are desirable for efficient clinical applications of this strategy and we are currently working on it.

Biography

Daniel Skuk is Associated Researcher at the Research Center of the University Hospital Center of Quebec, Canada. He studied medicine at the University of the Republic of Uruguay, a country where he worked as neuromuscular pathologist, collaborating in the creation of the Laboratory of Neuromuscular Pathology at the Institute of Neurology of the University Hospital. In 1996 he moved to Quebec, working since then in research in cell therapy and regenerative medicine applied to the treatment of skeletal muscle disorders. He was responsible for studies in nonhuman primates since 1997, being the only researcher worldwide to use primates for studies of cell therapy in myopathies. Using this animal model, he improved the cell transplantation protocols in the muscle, creating the method of "matrices of high density injections" and finding new instruments for this application. He quantitatively defined the most important parameters for the intramuscular transplantation of muscle precursor cells, and obtained the best results observed so far in this animal model. His research was the basis of improved clinical trials of cell therapy in Duchenne muscular dystrophy patients, in which Dr. Skuk tested his method of transplantation on humans. The results of these clinical trials are the most significant so far achieved with a cell therapy strategy in myopathic patients. Among othee things, he developed the first prototype of a device for the repetitive intramuscular injection of cells in clinical conditions, defined the histopathological features to diagnose acute rejection of muscle fibers expressing allogeneic proteins, publish the first morphological evidence of the mechanism by which T lymphocytes remove the target muscle fibers and defined the way in which the grafted cells interact with the recipient tissue to give rise to the final graft ourcome.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on
NEUROLOGY AND BRAIN DISORDERS
June 21-22, 2018 | Osaka, Japan

Title

THE MECHANISMS OF NEUROPROTECTION OF EXOSOMES IN ISCHEMIC BRAIN DISEASE

Name & Country

Qiang Zhang

Chian

Abstract

Purpose: Exosomes, an important mediator of intercellular communication by transferring signals to their target cell via surface ligands and delivering receptors and functional molecules, are secreted by various mammalian cells under physiological conditions and various disease states. In the brain, exosomes play an important role in neurons communication, which can contribute to a range of neurobiological functions. In this review, we summarized the role and potential mechanisms of exosomes in ischemic brain disease.

Results: The neurons are very sensitive to ischemia stress. Many brain diseases, such as stroke and other cerebrovascular diseases are often in the ischemic state that can cause perfusion defects and neurons injury. In ischemic brain disease, extracellular vesicles derived different cells can protect neurons to reduce ischemic injury. The possible mechanisms of neuroprotection of exosomes in ischemic brain disease mainly include the following aspects: 1. Exosomes ameliorate inflammation that causes neuronal cell death, short-term myelination deficits and long-term microstructural abnormalities of the white matter. 2. The release of exosomes carrying prion protein and other molecules reduce neuronal cell death by relieving oxidative stress under hypoxic and ischemic conditions. 3. The exosomes play a protective role in ischemic brain disease by promoting neurite outgrowth, neural plasticity and functional recovery. The exosomes enriched with the miR-17-92 cluster enhance neural plasticity and functional recovery, possibly via targeting phosphatase and tensin homolog to activate the PI3K/protein kinase B/mechanistic target of rapamycin/glycogen synthase kinase 3β signaling pathway.

Conclusion: The exosomes play neuroprotection role in ischemic brain disease. The mechanisms are mainly included inhibition of inflammatory response, reduction of oxidative stress, and enhancing neural plasticity and functional recovery.

Keywords: exosomes; ischemic brain disease; neuroprotection

Biography

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

BRAINSTEM DYSFUNCTION IN NEUROPSYCHIATRIC DISORDERS – AD/PD/DEPRESSION

Name & Country

Harry W.M. Steinbusch

Netherland

Abstract

Despite the fundamental role of the brainstem in regulating vital functional abilities such as arousal, breathing, autonomic nervous system activity as well as regulating all higher cerebral functions via neurotransmitter projections systems originating in the brainstem, the role of the brainstem has received relatively little attention in most neuropsychiatric disorders. Besides the dorsal and median raphe nuclei complex comprising mainly serotonin-producing neurons, the brainstem also contains noradrenalin, dopamine and histamine-producing nuclei, i.e. resp. the locus coeruleus, the substantia nigra and the mamillary bodies. The brainstem is furthermore the relay station of afferent and efferent projections between the autonomic nervous system in the peripheral body and higher cerebral brain regions. The current presentation aims to review the neuroanatomy of the brainstem as well as the current status on findings, derived from a wide range of studies using molecular, cellular and imaging technologies, of brainstem involvement in neurodevelopmental (i.e. autism, schizophrenia) and neurodegenerative disorders (Alzheimer's and Parkinson's disease). Over the past decades, the incidence of age-related, neurological and psychiatric disorders such as Alzheimer's disease (AD), Parkinson's disease (PD), but also depression has considerably increased. Mood disorders are strongly related to the exposure to stress. The hippocampus and other forebrain structures are the apex of the stress hormone control mechanism and damage to them may be one way in which stress hormone secretion escapes from inhibitory control in depression. In turn, stress, probably through toxic effects of glucocorticoids, decreases neurogenesis and cell survival while antidepressants enhance these processes in experimental animals. Therefore, since treatment strategies are not yet available, primary prevention in these age-related and stress related neurological disorders is of importance. As mentioned before most of the focus on neurobiological questions on above mentioned disease are related to forebrain structures since they are often associated with cognitive dysfunction. The brainstem is a highly neglected brain area in neurodegenerative diseases, including Alzheimer's (AD) and Parkinson's (PD) disease and frontotemporal lobar degeneration. Likewise, despite a long-standing recognition of brainstem involvement, relatively few studies have addressed the exact mechanisms that underlie brainstem autonomic dysfunction. Improved insight in the cellular and molecular characteristics of brainstem function is pivotal to study the developmental origins. As brainstem dysfunction also poses health issues in several other, neurodegenerative, disorders (like AD and PD), progress in these neurological fields will benefit from scientific advancement in the current proposal as well. In the area of depression, several observations have been made in relation to changes in one particular brain structure: The Dorsal Raphe Nucleus (DRN). In addition dysfunction of the cerebellum is also observed in AD and associated with pulmonary deregulation. The DRN is also related in the circuit of stress regulated processes and cognitive events. In order to gain more information about the underlying mechanisms that may govern the neurodegeneration, e.g. amyloid plaques, neurofibrillary tangles, and impaired synaptic transmission in AD, a rat dissociation culture model was established that allows mimicking certain aspects of our autopsy findings. We observed a similar phenomenon in brains from patients suffering from neurodegenerative disease since this also related to changes in BDNF levels. The ascending projections and multitransmitter nature of the DRN in particular and the brainstem in general stress its role as a key target for AD/PD research and autonomic dysfunction. It also points towards the increased importance and focus of the brainstem as key area in various neurodevelopmental and age-related diseases.

Biography

Harry W.M.Steinbusch is a Full Professor in Cellular Neuroscience & Director of the European Graduate School of Neuroscience, since 1996. He is Director for Institute Brain & Behavior & Mental Health and Neuroscience. He received his PhD from the Faculty of Medicine of the Catholic University, Nijmegen entitled: "Serotoninergic Neurons in the Central Nervous System of the Rat" in 1982.

His Research is focussing on the neuroanatomical, pharmacological, physiological and behavioral aspects of development and aging. Our general working hypothesis is that pre/ peri or postnatal stress can lead to depression and this by itself can be an early initiator of neurodegeneration. In addition, neurodegeneration and functional repair are studied in animal models and in human material obtained from patients. Topics are development, plasticity, brain aging and dementia, movement disorders, learning and memory. Research questions have primarily to do with the mechanism of changes in the nervous system in diseases and in development and aging. Participating disciplines are: Animal neuropsychology, genetics, neuroanatomy, neuropathology, neurochemistry, neuroimmunology, animal neuropsychology, molecular cell biology, neurophysiology, developmental neurobiology, neuropharmacology.

He is an Editorial Board member & Reviewer for many Journals.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

EFFECTS OF EEG-BASED ACTIVE ASSISTED NEUROFEEDBACK THERAPY ON HEMIPLEGIC UPPER EXTREMITY MOTOR FUNCTION

Name & Country

Joo-Hee Park

South Korea

Abstract

The purpose of this thesis was to investigate the effect of EEG-based active assisted neurofeedback therapy (AANT) on stroke patients to improve both their upper extremity functionality and brain activity. Twenty chronic hemiplegic patients were included in this study. The subjects were assigned to two groups (10 per group), the control, which received only physical therapy and the experimental, which additionally received AANT. Subjects in both groups underwent routine physical rehabilitation, involving 30 minutes of exercise, 3 times/week for 4 weeks. Subjects in the experimental group performed an active assisted wrist extension exercise, which was combined with EEG neurofeedback. AANT was performed for 1 hour, 3 times/week for 4 weeks. Specifically, the subjects were asked to try extending their wrist and finger while looking at a monitor, which depicted the magnitude of real-time mu rhythm from the EEG. After an obvious voluntary suppression of the mu rhythm was achieved with the initiation of the wrist/finger extension, a physical therapist assisted the participant to attain full wrist and finger extension. The outcome variables of pre- and post- treatment evaluation included the EEG mu rhythm. We found that the electromyogram (EMG) activity and upper extremity Fugl-Meyer Assessment (FMA) score were significantly increased in patients of the experimental than in those of the control group. In addition, there was a significant increase in brain activity of the affected (contralateral) sensorimotor area (SMA) in the experimental, but not in the control group. Spasticity, on the other hand, was significantly decreased in the experimental, but not in the control group. According to the results of this experiment, AANT improved brain activity in the affected SMA as well as upper extremity functionality in stroke patients. Therefore, we suggest neurofeedback therapy combined with proper physical guidance, as a promising treatment option for chronic stroke patients.

Biography

Joo-Hee Park has completed her doctoral degree at the age of 31 years from Yonsei University and post-doctoral course from Yonsei University School of Physical Therapy. HyeSeon Jeon is professor of Yonsei University.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

MIND-BODY MEDICINE

Name & Country

Sanjoy Mukerji

India

Abstract

Mind-body medicine explores the interconnection between the mind and body, under the premise that the mind affects "bodily functions and symptoms." As per the University of Maryland Medical Center, mind-body medicine uses the power of thoughts and emotions to influence physical health. As Hippocrates once wrote, "The natural healing force within each one of us is the greatest force in getting well." This is mind-body medicine in a nutshell.

The term "psychosomatic disease/disorder/illness" is mainly used to mean "a physical disease that is caused, or made worse, by mental factors." The term is also used when mental factors cause physical symptoms but where there is no physical disease. For example, chest pain may be caused by stress and no physical disease can be found.

Some physical diseases are prone to be made worse by mental factors such as stress and anxiety. At any given time, a person's mental state can affect the degree of severity of a physical disease. Physical symptoms that are caused by mental factors are also called somatization or somatoform disorders. These symptoms are due to increased activity of nervous impulses sent from the brain to various parts of the body. There is a deep connection between the mind (beliefs, thoughts and emotions) and the different parts of the body and physical problems.

A number of factors may play a role in psychosomatic disorders, such as personality traits; genetic or environmental family influences; biological factors; learned behavior and more. When one is not at ease, that means there is some kind of dis-ease; and disease can be reversed (completely or to a great extent) by simply reversing or changing mental/thought patterns, and at times by adding some physical exercises and changing some food habits. According to Dr. J. A. Winter, M.D., the psychosomatic illness is one of function, rather than of structure, although structural changes may occur later. It is based on some past experience, usually painful. This illness seems to arise from problem situations and from words (reflection of thoughts), rather than from actual injuries, or infection.

Biography

Sanjoy Mukerji is a Gold Medalist plus National and International Award-Winning Psychologist in Mumbai. He has done his Post Graduate Diploma in Psychological Counselling from the Institute for Behavioral and Management Sciences, India. Moreover, he has completed his Degree of Doctorate in Philosophy (Alternative Medicines) from the Indian Board of Alternative Medicines, established under the World Health Organization (WHO). In the field of alternative medicines, he has researched and specialized in mind-body medicine.

His counselling and therapies are based on the principle that our Mind affects our 3 Bs:

1. Brain (mental health)
2. Body (physical health)
3. Behavior (social health)

In his around 20 years of experience and practice, he has been instrumental in healing and helping thousands of people across the World through his counselling, therapy, talks, lectures, seminars, workshops, articles and books; and has received fantastic feedback, praises and blessings from them. He has been interviewed on various TV channels, and covered by almost all major newspapers and magazines.

SESSIONS

June 21-22, 2018

Alzheimer's Disease | Dementia Care | Neurological Infections| Neuroimmunology |Parkinson's Disease| Movement Disorders | Genetics and Epigenetics in Neurodegenerative Disorders| Neuro-Degenerative Disorders | Central Nervous System | Neurological Disorders and Stroke |Paediatric Neuropharmacology |Psychiatric Disorders or Psychological Syndromes | Cerebrovascular Diseases

Session Introduction

Session Chair
Chan Kam Tim Michael
China

Session Co-chair
Sanjoy Mukerji
India

Title: **The Integral Role of Neurosurgery in Managing Rare Craniofacial Anomalies**
Hazem A. Mostafa, Egypt

Title: **Evidence synthesis on traumatic brain injury as a precursor for neurodegeneration, cognitive decline, and development of Alzheimer's disease**
Tatyana Mollayeva, Canada

Title: **Autonomic Function Assessment in Parkinson's patients with Yoga practicing Using Kernel Method And Entrainment Techniques**
Kamal A, USA

Title: **Protective and Retroactive roles of a 24-amino acid peptide (p5) derived from Cdk5, neuron specific kinase activator protein p35**
Harish C. Pant, USA

Title: **Efficacy of microvascular decompression for spasmodic torticollis**
Li Xinyuan, China

Title: **Epitope Fingerprinting for Recognition of the Polyclonal Serum Autoantibodies of Alzheimer's Disease**
Lourena Costa, Brazil

Title: **Neural correlates of care setting in a sample of Chinese children double orphaned by AIDS.**
Michael E. Behen, USA

Title: **RegenerAge System: Therapeutic effects of combinatorial biologics (m RNA and allogenics MSCs) with a spinal cord stimulation system on a patient with spinal cord section**
Joel I.Osorio, Mexico

Title: **Dementia of Alzheimer's Type among Arab Populations: Genetics and Epidemiological Studies.**
Bowirrat Abdalla, Israel

Title: **Electron microscopic studies of brain tissue in fetuses from schizophrenic mothers**
Segundo Mesa, Cuba

Title: **Attentional deficots affect activities of daily living (ADLs) in dementia of Alzheimer type**
Pooja Rai, India

Title: **Stroke in Asia: Geographical variations and temporal trends**
Man Mohan Mehndiratta, India

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

THE INTEGRAL ROLE OF NEUROSURGERY IN MANAGING RARE CRANIOFACIAL ANOMALIES

Name & Country

Hazem A. Mostafa

Egypt

Abstract

Craniofacial anomalies are rare complex pathologies which needs a craniofacial team composed of neurosurgeon, a craniofacial plastic surgeon, and an ophthalmologist. Anomalies at craniofacial region either due to developmental malformation of the brain (neural tube defects) or premature closure of cranial or skull base sutures resulting in skull deformities and problems in normal physiological neurological development. Each of pathologies needs special neurological surgery management, sometimes the management is multi-staged. Neurosurgical management varied from diagnosis, the surgical procedures and long-term follow up. Hence, we describe the pathology of craniofacial anomalies and its associated syndromes in addition to the proper investigation needed for diagnoses and predict possible short and long-term complication. Also, what craniofacial anomalies care giver should be focusing on regarding neurological issues such as intra-cranial pressure early detection and treatment if high and optic nerve problems. Also dural repair, dealing with brain parenchyma and its vasculature, and better cosmetic outcome according to craniofacial metrics.

Keywords: encephalocele, craniosynostosis, sutures, craniofacial, neural tube defect.

Biography

Experienced professor Medical Doctor with a demonstrated history of working in the hospital & health care industry. Skilled in Surgical Navigation, Interventional Spine, Spine, Emergency Medicine, and Trauma Surgery. Strong healthcare services professional with a Doctor of Medicine (M.D.) focused in Neurosurgery from Ain Shams University Hospitals.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

EVIDENCE SYNTHESIS ON TRAUMATIC BRAIN INJURY AS A PRECURSOR FOR NEURODEGENERATION, COGNITIVE DECLINE, AND DEVELOPMENT OF ALZHEIMER'S DISEASE

Name & Country

Tatyana Mollayeva

Canada

Abstract

Despite indications that TBI may be a precursor for cognitive decline and subsequent development of Alzheimer's disease (AD), little is known about the time course of this relationship and the factors that contribute to synaptic dysfunction and neurodegeneration. We summarized the current evidence on the course and prognostic factors of cognitive outcome in adults with TBI. Our results highlighted that as time since injury progresses, performance of measures of recent memory, executive function, language, and information processing speed tend to improve or remain stable from baseline in non-sports-related mild TBI samples and mixed severity samples, indicating potential neurogenesis or practice effect. Severe injury results depict mostly improvements or stability with respect to cognitive performance, and as last follow up time progresses it appears that improvements are abated and reports of no change dominate. Although several mechanisms were found to modulate the risk of cognitive decline in persons with TBI, the evidence in the longitudinal studies published to date suggests the ability of the brain to compensate and naturally recover after injury is associated with genetic makeup, injury severity and count, age, and sex. The evidence taken together, however, is not strong and, as such, not convincing of the presence and strength of a relationship between TBI and cognitive decline, and subsequent risk for development of neurodegenerative disorders. Some of the issues in the studies published to date are attributable to the limited information present on evaluative properties of the outcome measures used to assess cognition, unknown sensitivity to changes over time and practice effect. Future work must apply these considerations in their design process and execution to circumvent issues present in current studies, providing a more concrete understanding of the relationship in question and factors that modulate it. Acknowledgements: This research program is supported by the Alzheimer's Association grant (AARF-16-442937).

Biography

Tatyana Mollayeva received her medical degree from the Moscow Medical Academy and worked for the Ministry of Health in Central Asia. Upon immigrating to Canada, she was trained in sleep medicine and completed her PhD on the topic of sleep dysfunction in traumatic brain injury. She is currently a postdoctoral research fellow working with Professor Angela Colantonio on the topics related to injury prevention, cognitive outcome assessment, sex and gender, and brain injury comorbidity. Since her entry into research in 2010, Dr. Mollayeva has authored more than thirty peer reviewed papers, three book chapters, and a book on polysomnography. Her contributions have been recognized internationally by the early career Award from the American Academy of Rehabilitation Medicine and the Elio Lugaresi Award for Education from the World Sleep Congress.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

AUTONOMIC FUNCTION ASSESSMENT IN PARKINSON'S PATIENTS WITH YOGA PRACTICING USING KERNEL METHOD AND ENTRAINMENT TECHNIQUES

Name & Country

Kamal Ahmed

USA

Abstract

The experimental procedure of lowering and raising a leg Figure 1, while the subject in supine position is considered to stimulate and entrain the autonomic nervous system of fifteen patients with Parkinson's disease practicing Yoga and fifteen age and sex matched control Parkinson's disease non Yoga practicing patients . The assessment of autonomic function for each group is achieved using an algorithm based on Volterra kernel estimation. By applying this algorithm and considering the process of lowering and raising a leg as stimulus input and the Heart Rate Variability signal (HRV) as output for system identification, a mathematical model is expressed as integral equations. The integral equations are considered and fixed for Parkinson's Patients without Yoga practicing and Parkinson's Yoga practicing patients so that the identification method reduced to the determination of the values within the integral called kernels, resulting in an integral equations whose input-output behavior is nearly identical to that of the system in both Control Parkinson's without yoga practicing patients and Parkinson's Yoga practicing patients. The model for each group contains the linear part (first order kernel) and quadratic part (second order kernel). A difference equation model was employed to represent the system for both control Parkinson's patients without Yoga practicing and Parkinson's Yoga practicing patients. The results show significant difference in first order kernel(impulse response) and second order kernel (mesh diagram) for each group. Using first order kernel and second order kernel, it is possible to assess autonomic function qualitatively and quantitatively in both groups.

Biography

Kamal was appointed as Faculty at different university around the world including England .Kuwait, Bahrain, Saudi Arabia and USA. He worked at Boston University and Johns Hopkins University as research professor in Biomedical Engineering. He published more than 100 publications. He has been serving as an editorial board member of reputed journal. He is now professor of Bio manufacturing at College of Engineering, Tennessee Tech University.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

PROTECTIVE AND RETROACTIVE ROLES OF A 24-AMINO ACID PEPTIDE (P5) DERIVED FROM CDK5, NEURON SPECIFIC KINASE ACTIVATOR PROTEIN P35

Name & Country

Harish C. Pant

USA

Abstract

Cdk5 is a multifunctional protein kinase important in neuronal development, physiological function and survival. Its activity is tightly regulated under physiological conditions. However, upon neuronal insults Cdk5 is deregulated and hyper activated and produces a number of neurodegenerative diseases and may be in part responsible for the hall mark pathology of amyloid plaques and neurofibrillary tangles (NFTs). The etiology of neurodegenerative diseases including Alzheimer's Disease is very complex, and arises as a result of kinase dysfunction and other accumulated insults to many cellular, environmental & interacting metabolic pathways during aging. It has been proposed that deregulated and hyperactive Cdk5 results from the over expression of p25, (a truncated fragment of p35, the normal Cdk5 regulator). p25 has a higher affinity with Cdk5 compared to p35 which when complexes to Cdk5, causes hyper activation and deregulation, neurofilament and Tau hyperphosphorylation, tangles (NFTs) formation and neuronal death.

Thus Cdk5/p25 becomes pathological target. The objective of our studies has been to inhibit selectively pathological Cdk5/p25 but not Cdk5/p35, the physiological target (Cdk5/p35) in situ / in vivo. During the course of these studies we found a small 24 amino acid peptide (p5) derived from p35, Cdk5 activator selectively inhibited the deregulated, Cdk5/p25 but not physiological (Cdk5/p35) activity. Intraperitoneal (i.p.) injections of p5- modified peptide (TFP5), penetrated the blood-brain barrier and significantly rescued AD-like pathology in 5XFAD model mice. As a proof of concept, it is essential to demonstrate the peptide's efficacy in a mouse model expressing high levels of p25, such as the inducible CamK-inducible CK-p25Tg, AD model mouse that overexpresses p25 in CamK positive neurons. Using TFP5 treatment, we show that peptide i.p.

Injections in these mice decrease Cdk5 hyperactivity, au, neurofilament-M/H hyper phosphorylation, and restore synaptic function (LTP) and behavior (i.e., spatial working memory). It is noteworthy that TFP5 does not inhibit endogenous Cdk5/p35 activity, or other Cdks in vivo suggesting it might have no toxic side effects, and may serve as an excellent therapeutic candidate for neurodegenerative disorders expressing abnormally high brain levels of p25 and hyperactive Cdks in neurodegenerative diseases. We have demonstrated that the peptide, injected into an AD model mouse overexpressing p25 (P25Tg) and 5XFAD, specifically targets the hyperactive kinase, reduces or eliminates familial and sporadic AD phenotypes and restores normal behavior. We suggest that the peptide may serve as a potential therapeutic candidate.Recently, we have extended these neuroprotective and restroactive effects of Peptide TFP5/TP5 in Parkinson's disease, MPTP PD models.

A manuscript on this subject is published in Molecular Biology of Cell (2016). The editor of the journal made the following comments. "This is a major advancement that should be considered for clinical applications in light of these studies presented in this manuscript. I recommend publication as is, no revisions required". Current studies related to PD, using different PD models will be discussed.

Biography

Pant received his M.A. and Ph.D. degrees in Physics from Agra University, Agra, India. His postdoctoral studies were conducted on the mechanisms of electron and ion transport in model membrane systems at the Department of Biophysics at Michigan State University. He joined the Laboratory of Neurobiology in the NIMH as a senior staff fellow in 1974 with Dr. Ichiji Tasaki where he studied the function of the axonal cytoskeleton in the squid giant axon. In 1979 he moved to the NIAAA extending his studies on the neuronal cytoskeleton and the effects of alcohol on its regulation. Dr. Pant moved to the NINDS, Laboratory of Neurochemistry in 1987 where he is presently chief of the section on Cytoskeleton Regulation. His laboratory is studying the mechanisms of topographic regulation of neuronal cytoskeleton proteins by post-translational modification, including the role of kinase cascades in normal brain and during neurodegeneration.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on
NEUROLOGY AND BRAIN DISORDERS
June 21-22, 2018 | Osaka, Japan

Title

EFFICACY OF MICROVASCULAR DECOMPRESSION FOR SPASMODIC TORTICOLLIS

Name & Country

Li Xinyuan

China

Abstract

Objective To access the clinical efficacy of microvascular decompression(MVD) for spasmodic torticollis(ST).

Methods From January 2014 to Janurary 2017, a total of 117 patients with spasmodic torticollis treated with microvascular decompression at Shanghai Tongren Hospital were enrolled retrospectively. In this study, 110 cases underwent MVD with retrosigmoid approach and 7 cases underwent MVD with paramedian suboccipital approach. Intraoperative findings and follow-up results were analyzed.

Results The mean follow-up was 18.7 months. Six months after the MVD surgery, 33 cases(28.21%) were cured, 50 cases（42.74%) improved significantly, 21 cases （17.95%）improved moderately, and 13 cases（11.11%） improved minimally or unchanged. The total efficiency was 88.89%. Vertebral artery was the most common offending vessel in 104 cases（88.89%） and posterior inferior cerebellar artery (PICA) was offending vessel in 13 cases（11.11%）. In the 110 cases with retrosigmoid approach, the neurovascular conflict was spinal accessory nerve(SAN) in 78 cases (70.91%), C1C2 in 37 cases(33.64%), and the junctional area of the brainstem and spinal cord in 21 cases(19.09%). In total, 2 or 3 conflict sides were involved in 15 cases (13.64%). Of the 7 cases with paramedian suboccipital approach, the conflict site was found in bilateral SAN in 2, in the C1-C4 in 3 cases, and in the spinal cord in 2. Postoperative complications, which including shoulder numbness in 3 and hoarseness in 1, were completely relieved during the follow-up period. There was no limb weakness, cerebrospinal fluid leakage, infection, cerebral hemorrhage and other complications.

Conclusions Vascular compression may be one of the major causes of ST. MVD is a safe and effective method for the treatment of ST. With the study of etiology and pathogenesis, MVD might be one of the most effective methods to treat ST in the future.

[Key words] spasmodic torticollis; microvascular decompression; neurovascular compression; accessory nerve; cervical nerve

Fund Program: Shanghai Municipal Commission of Health and Family Planning (201540299)

Biography

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

EPITOPE FINGERPRINTING FOR RECOGNITION OF THE POLYCLONAL SERUM AUTOANTIBODIES OF ALZHEIMER'S DISEASE

Name & Country

Lourena Costa

Brazil

Abstract

Autoantibodies (aAb) associated with Alzheimer's disease (AD) .ave not been sufficiently characterized and their exact involvement is undefined.The use of information technology and computerized analysis with phage display technology was used, in the present research, to map the epitope of putative self-antigens in AD patients. A 12-mer random peptide library, displayed on M13 phages, was screened using IgG from AD patients with two repetitions. Seventy-one peptides were isolated; however, only 10 were positive using the Elisa assay technique (Elisa Index >1). The results showed that the epitope regions of the immunoreactive peptides, identified by phage display analysis, were on the exposed surfaces of the proteins. The putative antigens MAST1, Enah, MAO-A, X11/MINT1, HGF, SNX14, ARHGAP 11A, APC, and CENTG3, which have been associated with AD or have functions in neural tissue, may indicate possible therapeutic targets.

Biography

Lourena Costa has completed her PhD at the Federal University of Minas Gerais – Brazil in 2016. She is professor of the post-graduation program of Infectolgy and Tropical Medicine(UFMG). She has over 37 publications and she has been serving as an editorial board member of reputed Journals.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

NEURAL CORRELATES OF CARE SETTING IN A SAMPLE OF CHINESE CHILDREN DOUBLE ORPHANED BY AIDS

Name & Country

Michael E. Behen

USA

Abstract

Behen will present data from a set of studies focusing on the functional and structural neural correlates of differential care settings (orphanage, kinship care, community group homes) (NIH: 5R21HD087108-02) in children double orphaned by AIDS. The talk will highlight the behavioral and neural phenotypes associated with such early adversity and across care settings, and also predictors of such outcomes, especially focusing on timing and care setting parameters.

Objective: Studies investigating the effects of early social deprivation associated with institutional rearing reveal increased incidence of cognitive/behavioral problems and altered neural structure/function, raising concerns about the use of institutional settings (i.e., orphanages) in the care of orphaned children, and prompting study of alternative programs (i.e., foster care) for the care of such children. However, empirical scrutiny of neurodevelopmental outcomes across care settings (and timing/care setting parameters associated with outcomes) is critical before a global push to foster care is undertaken. Method: We applied neurocognitive/behavioral assessments, and structural/functional MR imaging in 124 Chinese children double-orphaned by HIV/AIDS (mean age=14.7+SD=1.5 years), across three care settings (orphanage, community group home, kinship care), and two age groups (onset of adversity <3years, >8 years of age). Data analyses included between-group comparisons on cognitive/behavioral outcomes and structural/functional neural connectivities. Regression analyses were used to identify/determine relationships between duration in care and outcomes across settings, and whether relationships are moderated by age of onset of adversity. Results: Analyses revealed increased incidence of cognitive/behavioral problems in children raised in orphanages and kinship care compared to those in community group homes. Further, orphanage rearing was associated with altered neural connectivities, especially involving frontal and temporal regions, compared to community group home. Outcomes were associated with duration in care (longer duration in orphanage was associated with poorer outcomes over time; care in group homes was associated with improved outcomes over time); findings were accenuated in children with onset of adversity before 3 years. Conclusions: Community group care was associated with improved neurodevelopmental outcomes compared to orphanage care. Such outcomes appear to be strengthened over time in such settings, particularly in children with early onset of adversity. Such data may have important implications for policy for how growing numbers of children, worldwide, can be best cared for following early adversity.

Biography

Michael E. Behen is an Assistant Professor of Pediatrics and Neurology at Wayne State University in Detroit, MI. Dr. Behen is a clinical psychologist and neuroscience researcher at the Children's Hospital of Michigan, Translational Imaging Center, and also is the executive director of a large private practice in the Detroit area.

Dr. Behen is an NIH funded researcher; his research interests revolve around the concepts of neuroplasticity and neurodevelopmental disorder. Specifically, Dr. Behen applies comprehensive neurocognitive and behavioral evaluations and advanced multimodal imaging to identify the functional and structural neural correlates of various developmental, neurologic, and psychiatric disorders.

Current projects include investigation of the functional and structural neural correlates of early severe social deprivation, neural correlates of differential care settings in Chinese children orphaned by AIDS, and neurocognitive and neural correlates of various developmental disorders (e.g., autism spectrum disorders, language disorder) and neurologic (e.g., Sturge Weber Syndrome) conditions.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

REGENERAGE SYSTEM: THERAPEUTIC EFFECTS OF COMBINATORIAL BIOLOGICS (MRNA AND ALLOGENIC MSCS) WITH A SPINAL CORD STIMULATION SYSTEM ON A PATIENT WITH SPINAL CORD SECTION

Name & Country

Joel I. Osorio

Mexico

Abstract

As it has been previously demonstrated that coelectroporation of Xenopus laevis frog oocytes with normal cells and cancerous cell lines induces the expression of pluripotency markers and in experimental murine model studies that mRNA extract (Bioquantine) purified from intra and extra-oocyte liquid phases of electroporated oocytes) showed potential as a treatment for a wide range of conditions, including Spinal Cord Injury (SCI) among others. The current study observed beneficial changes with Bioquantine administration in a patient with a severe SCI. Pluripotent stem cells have therapeutic and regenerative potential in clinical situations CNS disorders even cancer. One method of reprogramming somatic cells into pluripotent stem cells is to expose them to extracts prepared from Xenopus laevis oocytes. The positive human findings for spinal cord injury with the results from previous animal studies with experimental models of traumatic brain injury and SCI respectively as our evidence and due to ethical reasons, legal restrictions and a limited number of patients, we were able to treat only a very small number of patients, deciding to include in our protocol the RestoreSensor SureScan to complete it. Based on the electrical stimulation for rehabilitation and regeneration after spinal cord injury published by Hamid and MacEwan, we designed an improved delivery method for the in-situ application of MSCs and Bioquantine in combination with the RestoreSensor Sure Scan. To the present day the patient who suffered a complete section of spinal cord at T12-L1 shows an improvement in sensitivity, strength in striated muscle and smooth muscle connection, 13 months after the first treatment and 6 months after the placement of RestoreSensor® at the level of the lesion, showing an evident improvement on his therapy of physical rehabilitation (legs movement) on crawling forward and backwards and standing on his feet for the first time and showing a progressively important functionality on both limbs.

Biography

CEO and Founder of Biotechnology and Regenerative Medicine at RegenerAge International ™ (www.regenerage.clinic). Vice President of International Clinical Development for Bioquark, Inc. (www.bioquark.com) and Chief Clinical Officer at ReAnima™ Advanced Biosciences (www.reanima.tech). Advance Fellow by the American Board of Anti-Aging and Regenerative Medicine (A4M), Visiting Scholar at University of North Carolina at Chapel Hill (Dermatology). Fellow in Stem Cell Medicine by the American Academy of Anti-Aging Medicine and University of South Florida.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

DEMENTIA OF ALZHEIMER'S TYPE AMONG ARAB POPULATIONS: GENETICS AND EPIDEMIOLOGICAL STUDIES

Name & Country

Bowirrat Abdalla

Israel

Abstract

Introduction: Neurodegenerative disorders, Primarily, are multifactorial diseases characterized by chronic and progressive loss of neurons in discrete areas of the brain, causing debilitating symptoms and globally decreasing cognitive function such as dementia, loss of memory, loss of sensory or motor capability, decreased overall quality of life and well-being, disability, and eventually, premature death.

Objective: To study the genetic and environmental risk factors and the prevalence of dementia of the Alzheimer type (DAT) among the elderly in an Arab community in Israel.

Material and Methods: Epidemiological and genetic studies of dementia have rarely been reported in an Arab population. Alzheimer disease (AD [MIM #104300]) is a progressive, neurodegenerative disease characterized clinically by gradual loss of memory and pathologically by neurofibrillary tangles and amyloid plaques in the brain. We have observed an unusually high prevalence of dementia of the Alzheimer type (DAT) in Wadi Ara, an inbred Arab community in northern Israel comprising ~850 persons over the age of 60 years. Apolipoprotein E (APOE- ε4), has been established as a strong susceptibility marker that accounts for nearly 30% of the risk in late-onset AD.

Results: Remarkably, in our study DAT is not associated with APOE because the frequency of the ε4 allele is very low in both nondemented (2.4%) and demented elders (3.6%). We also map chromosomal loci contributing to DAT susceptibility; we conducted a 10 cM scan in a series of twenty cases and twenty controls selected from one hamula. Markers from 18 chromosomal regions showed significant allelic association with DAT ($P<0.05$). Locations on chromosomes 2, 9 and 10 remained significant after testing additional affected and non-demented individuals. Significant associations were also observed for markers on chromosome 12, which overlap with a locus implicated in previous genome scans. Additionally, several lines of evidence support for a role of angiotensin converting enzyme (ACE) in Alzheimer disease (AD). Most genetic studies have focused on an Alu insertion/deletion (I/D) polymorphism in the ACE gene (DCP1) and have yielded conflicting results. We evaluated the association between 15 (SNPs) in DCP1, including the I/D variant, and AD in a sample of 92 patients with AD and 166 non-demented controls from an inbred Israeli Arab community. Although there was no evidence for association between AD and I/D, we observed significant association with SNPs rs4343 ($P= .00001$) and rs4351 ($P=.01$). **Conclusion:** In Wadi Ara the high prevalence may be due to a founder effect enhanced by consanguinity, which make this population attractive for investigating DAT susceptibility recessive genes; thus, a specific disease susceptibility allele may be overrepresented in cognitively impaired subjects compared with cognitively healthy residents. Other two main conclusions can be drawn from the genome-wide linkage and linkage disequilibrium (LD) studies. Firstly, multiple genes are involved in DAT. Secondly, there is a high level of consistency among linkage and association studies regarding the general location of putative AD genes. However, the general location of putative AD genes on a given chromosome covers a broad region, which may contain several genes.

Biography

Bowirrat Abdalla M.D., Ph.D. has completed his MD from Rome University, his residency in Clinical Neurology from London University, UK, his PhD from Tel-Aviv University, Israel and postdoctoral studies from Boston University, USA. He received his Professorship from Boston University. He has published more than 105 manuscripts and 6 books in reputed journals and has been serving as an editorial board member of repute. Furthermore, he received many international awards including the Bruce S. Schoenberg international award in Neuroepidemiology of AD from the American Academy of Neurology.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

ELECTRON MICROSCOPIC STUDIES OF BRAIN TISSUE IN FETUSES FROM SCHIZOPHRENIC MOTHERS

Name & Country

Segundo Mesa Castillo

Cuba

Abstract

The neurodevelopmental theory in the aetiology of schizophrenia is considered one of the most consistent at present. Evidence from epidemiological and neuropathological studies indicates that the pathogenic process that culminate in the development of schizophrenia are initiated early in life and has been associated with a variety of prenatal environmental insults to the developing brain, including infection. Although the infectious agents have been proposed as one of the risk factors for schizophrenia the data on the association of a specific infectious agent with prenatal brain evidence is absent. Understanding of the structural abnormalities would allow a better identification of neurodevelopmental processes that contribute to risk for schizophrenia. We have hypothesized that at ultra high-risk fetuses would have alterations at cellular level that would let us differentiate them to the comparison subjects. A reappraisal of our ultrastructural studies carried out in samples of the left temporal lobe of foetuses at ultra high risk of developing schizophrenia is presented. The findings obtained are compatible with an active infection of the central nervous system by herpes simplex hominis type I [HSV1] virus. The present results are the first direct evidence that demonstrate the presence of this virus in the central nervous system of foetuses from schizophrenic mothers in the critical period of foetal development. The importance of this finding can have practical applications in the prevention of the illness keeping in mind its direct relation to the aetiology and physiopathology of schizophrenia.

Biography

Segundo Mesa Castillo. As Specialist in Neurology, he worked for 10 years in the Institute of Neurology of Havana, Cuba. He has worked in Electron Microscopic Studies on Schizophrenia for 32 years. He was awarded with the International Price of the Stanley Foundation Award Program and for the Professional Committee to work as a fellowship position in the Laboratory of the Central Nervous System Studies, National Institute of Neurological Diseases and Stroke under Dr. Joseph Gibbs for a period of 6 months, National Institute of Health, Bethesda, Maryland, Washington D.C. USA, June 5, 1990.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

ATTENTIONAL DEFICITS AFFECT ACTIVITIES OF DAILY LIVING (ADLS) IN DEMENTIA OF ALZHEIMER TYPE

Name & Country

Pooja Rai

India

Abstract

Attention has been found to be an important predictor of overall ADL scores in patients with Alzheimer's disease. Attentional deficit is progressively acknowledged as an important cognitive symptom in Dementia of Alzheimer type (DAT). DAT affects people's cognitive ability to perform activities of daily living is often referred to as functional ability and as a measurement of disability, initially in complex activities but progressing to more basic activities such as the ability to cope with dressing and toileting later in the disease course. The neuropathological basis of these attentional deficits remains unsettled, with two competing hypotheses: spread of pathology from the medial temporal to basal forebrain structures versus corticocortical tract disconnection. Moreover, it is still not clear whether improving attention could improve ADL performance, and the present study purports to assess the relationship between attentional dysfunction and the ability to perform everyday life activities. We used the Informant interview using the 11-item Everyday Abilities Scale for India (EASI) to assess evidence for functional limitation in activities of daily living (ADL). Pearson corrletion coefficient were calculated between activities of dily living and Mini-Mental State Examination (MMSE) scores. The result shows that among all the cognitive domain, attention domain of MMSE was the most predicative of EASI followed by visuospatial, language and delayed recall. So, in sum the study suggested that improving attention could improve ADL performances in MCI and DAT patients.

Biography

Pooja Rai is pursuing Ph.D. from Cognitive Science Laboratory, Department of Psychology, Banaras Hindu University, Varanasi, India, under the supervision of Prof. Indramani. L. Singh on the topic "Attentional deficits in persons with Alzheimer's disease (AD) and Healthy Aging". She has been working on dementia patients since 3 years and have seen approx. 50 patients of dementia during this course of study. She is working with several neurologists. She has published 4 of her papers in her Ph.D course. She has been awarded as B.H.U. Gold medalist for two times in her academic history for securing highest marks in Masters in Psychology and Bachelor's in Psychology respectively. Besides doing Ph.D. and a UGC JRF itself, She is also working on a project on Alzheimer's sponsored by Cognitive Science Research Initiative, Department of Science and Technology, Ministry of Science and Technology, New Delhi.

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on
NEUROLOGY AND BRAIN DISORDERS
June 21-22, 2018 | Osaka, Japan

Title

STROKE IN ASIA: GEOGRAPHICAL VARIATIONS AND TEMPORAL TRENDS

Name & Country

Man Mohan Mehndiratta

India

Abstract

Asian countries are in various stages of epidemiologic transition and therefore, exhibit a great diversity in disease patterns. Collectively, they comprise almost two third of the world's total mortality due to stroke. The purpose of this review is to highlight the temporal trends in stroke epidemiology in various regions of Asia and predict future patterns based on these observations.

Our search revealed that there is a lack of good epidemiologic data from most Asian countries. Estimates for stroke incidence vary from 67/100,000/year to 384/100,000/year. These estimates exhibit country to country variation and also within country variability. Ischemic strokes have been the predominant subtype and their proportion seems to be rising over the past decade. This is secondary to changing lifestyles as countries undergo development and epidemiological transition. Hemorrhagic strokes in Asia have been higher in proportion compared to Western, more developed countries but this has since declined somewhat due to better control of hypertension.

Among ischemic stroke subtypes, lacunar strokes were once the commonest variety. However, emerging data suggests that large artery atherosclerosis and in particular that of intracranial vessels is the predominant etiology in most Asian countries. Rheumatic valvular disease was also once fairly common, but now it mostly accounts for young strokes. Another important cause of stroke particularly in young Asian women is cortical venous thrombosis.

We call for collaborative efforts between various Asian countries to better understand this disease and undertake policy level interventions to stop this epidemic.

Biography

20 YEARS OF EXCELLENCE IN INTERNATIONAL MEETINGS
alliedacademies.com

International Conference on

NEUROLOGY AND BRAIN DISORDERS

June 21-22, 2018 | Osaka, Japan

Title

THE STORY OF GLATIRAMER ACETATE (COPAXONE) IN THE TREATMENT OF MULTIPLE SCLEROSIS - THE POTENTIAL FOR NEUROPROTECTION BY IMMUNOMODULATORY TREATMENT

Name & Country

Rina Aharoni

Israel

Abstract

Multiple sclerosis (MS) is currently recognized as complex diseases in which inflammatory autoimmune reactivity in the central nervous system (CNS) results in demyelination, axonal and neuronal pathology. Treatment strategies thus aim to reduce the detrimental inflammation and induce neuroprotective repair processes.

The synthetic copolymer Copaxone (glatiramer acetate, GA), an approved drug for the treatment of MS, is the first and so far the only therapeutic agent to have a copolymer as its active ingredient. Using the animal model of MS - experimental autoimmune encephalomyelitis (EAE), the mechanism of action of GA was elucidated.

These studies indicated that GA treatment generates immunomodulatory shift from the inflammatory towards the anti-inflammatory pathways, such as Th2-cells that cross the blood brain barrier (BBB) and secrete in situ anti-inflammatory cytokines, as well as T-regulatory cells (Tregs) that suppress the disease.

The consequences of GA treatment on the CNS injury inflicted by the disease were studied using immunohistochemistry, electron microscopy, and magnetic resonance imaging. These analyses revealed reduced demyelination and neuro-axonal damages, as well as neuroprotective repair processes such as neurotrophic factors secretion, remyelination and neurogenesis.

These combined findings indicate that immunomodulatory treatment can counteract the neurodegenerative disease course, supporting linkage between immunomodulation, neuroprotection and therapeutic activity in the CNS.

Biography

Currently - Senior Research Staff Scientist, Department of Immunology, The Weizmann Institute of Science, Israel. BSc in Biology in Biology, Hebrew University, Jerusalem, Israel. MSc and PhD in Life Sciences, The Weizmann Institute of Science, Rehovot, Israel. Postdoctoral research, Stanford University, CA, USA.Main research interests: Neuroimmunology; Autoimmunity; Pathology and therapy of multiple sclerosis (MS) and its model experimental autoimmune encephalomyelitis (EAE); immunomodulation, neuroprotection and repair processes in the Central Nervous System; Inflammatory bowel diseases (IBD). Published more than 70 papers and reviews on these subjects. Editorial board member of 20 journals.