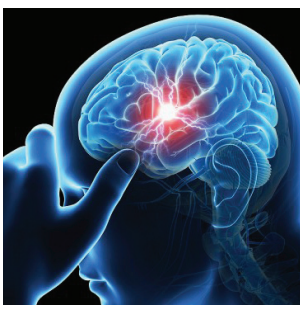
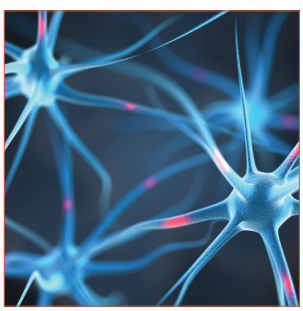
Keynote Forum
July 01, 2019

Brain Disorder 2019




6th International Conference on

Brain Disorders and Therapeutics

July 01-02, 2019 | Paris, France

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Theodore A Henderson

Neuro-Laser Foundation, USA

Functional Neuroimaging in the early diagnosis of Dementia and Mild Cognitive Impairment

As the world population ages, the incidence of dementing illnesses will dramatically increase. The number of people afflicted with dementia is expected to quadruple in the next 50 years. Repeated attempts at pharmacological interventions have failed largely because treatment is reserved for those already diagnosed with dementia. It is now clear that methods to diagnose early are critical to the success of treating dementia. Early detection and intervention also are key to mitigating the progression from Mild Cognitive Impairment to full-blown dementia. Anatomical imaging, cerebrospinal fluid markers, functional neuroimaging, such as positron emission tomography and single photon emission computed tomography, and molecular imaging, such as amyloid marker imaging, will be assessed in terms of sensitivity and specificity. Particular emphasis will be placed on the diagnosis of Mild Cognitive Impairment – the precursor to dementia. Cost will also be considered as the growing population afflicted with dementia represents an increasingly large financial encumbrance to the health care systems of every nation. Recent meta-analysis data of single photon emission computed tomography will be reviewed relative to sensitivity and specificity data of fluorodeoxyglucose and amyloid marker positron emission tomography.

Speaker Biography

Theodore A Henderson is founder of Neuro-Luminance Brain Health Centers, Inc. and director of the Synaptic Space. He has extensive training and experience to the practice of Psychiatry and brain sciences. He trained in Psychiatry at the prestigious Barnes/Jewish Hospitals at Washington University/St. Louis and Child & Adolescent Psychiatry at the University of Colorado. He also has training in Radiology, Nuclear Medicine, and the Genetics of Psychiatry. He established his private practice in Centennial Colorado in July of 2000. He has achieved an advanced level of understanding of Psychopharmacology, referred to as Master Psychopharmacologist. He lectures regularly and has written or published on unique treatment approaches to depression, chronic fatigue, ADHD, and anxiety. He also has taught courses on neuroimaging. He is a guest editor for a number of journals, including JAMA, Journal of Neuropsychiatry, and the Journal of Nuclear Medicine.

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Gabriele Saretzki

Newcastle University, UK

Telomerase activators improve motor function and protein degradation in a mouse model of Parkinson's Disease (PD)

While telomerase maintains telomeres in dividing cells, its protein component TERT (Telomerase reverse transcriptase) has various non-canonical functions such as localisation to mitochondria resulting in decreased oxidative stress, apoptosis and DNA damage. The TERT protein persists in adult neurons while telomerase activity is downregulated early during development (Ishaq et al., 2016). We recently demonstrated increased mitochondrial TERT protein in hippocampal neurons from Alzheimer's disease (AD) brains and mutual exclusion of pathological tau and TERT protein. Transduction of mutated tau into cultivated neurons confirmed that TERT decreases mitochondrial oxidative stress and lipid oxidation (Spilsbury et al., 2015). Mitochondrial dysfunction is also involved in the development of other neurodegenerative diseases. Treatment of PD model mice (Masliah et al., 2000) overexpressing human wild-type alpha-synuclein with 2 telomerase activators (TA Science Inc., USA) resulted in increased TERT expression in brain and amelioration of PD symptoms by significantly improving balance, gait and motor function as well as mitochondrial function. Analysing levels of total, phosphorylated and aggregated alpha-synuclein we found a substantial decrease of all these protein forms in the hippocampus and neocortex suggesting a better protein degradation after telomerase activator treatment. Interaction of TERT with proteasomal and autophagy pathways has been described recently. Accordingly, we have preliminary data showing a decrease in poly-ubiquitinated proteins and the autophagy receptor p62 and analyse the involvement of these degradation pathways currently. Thus, our results suggest that telomerase activators might form a novel treatment option for better degradation of toxic proteins in neurodegenerative diseases such as PD and AD.

Speaker Biography

Gabriele Saretzki has completed her PhD in 1990 at Humboldt University Berlin and performed most of her postdoctoral studies at the Institute for Ageing and Health in Newcastle upon Tyne (UK) where she is a Lecturer in Ageing Research since 2002. Her main interests are telomeres, telomerase, senescence, ageing, oxidative stress, mitochondria, stem cells and brain. She has pioneered work on non-canonical functions of the telomerase protein TERT shifting her focus recently to brain ageing and neurodegenerative diseases. She has published more than 88 papers in peer-reviewed journals and is an editorial board member of BMC Biology, PloS One and Oxidative Medicine and longevity.

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Theodore A Henderson

Neuro-Laser Foundation, USA

The viral etiology of Chronic Fatigue Syndrome: Proof for low-grade CNS viral infections

Chronic Fatigue Syndrome (CFS) is a medically unexplained disorder which presents with severe fatigue, flu-like symptoms, and neuropsychological impairments, which can include mental "fogginess", decreased concentration, poor memory, low motivation, diminished mood, and increased duration of sleep. The Centers for Disease Control and Prevention estimate that between 1 and 4 million patients exist in the United States alone, although less than 20% have been so diagnosed. Although CFS was initially thought to be a psychosomatic illness, research has recently shifted to determining the biological basis to the disorder. In recent years, numerous studies have demonstrated a link between chronic viral infections and CFS. Viruses such as Epstein-Barr Virus (EBV), Cytomegalovirus (CMV), and several herpes viruses (e.g., Herpes 1, 6, 7 – HSV-1, HHV-6, HHV-7) cause or contribute to the symptoms of a large percentage of patient with CFS. These infections are generally not acute, but represent intracellular reactivation of an old infection; hence, an elevation of IgM antibodies is typically not seen with reactivated infections of EBV, CMV, or HHV-6. Furthermore, a recent study demonstrated conclusively that HHV-6 can invade and replicate within human brain cells based on the finding of HHV-6 virus in Cerebellar Purkinje cells of a large number of human brains. These data will be reviewed and the treatment of CFS as a viral illness will be outlined. Clinical data from over 150 patients with CFS will be presented. The distribution of active viral infections, treatment protocols, response rates, and stepped therapy to more aggressive antiviral therapies will be described.

Speaker Biography

Theodore A Henderson is founder of Neuro-Luminance Brain Health Centers, Inc. and director of The Synaptic Space. He has extensive training and experience to the practice of Psychiatry and brain sciences. He trained in Psychiatry at the prestigious Barnes/Jewish Hospitals at Washington University/St. Louis and Child & Adolescent Psychiatry at the University of Colorado. He also has training in Radiology, Nuclear Medicine, and the genetics of psychiatry. He established his private practice in Centennial Colorado in July of 2000. He has achieved an advanced level of understanding of psychopharmacology, referred to as Master Psychopharmacologist. He lectures regularly and has written or published on unique treatment approaches to depression, chronic fatigue, ADHD, and anxiety. He also has taught courses on neuroimaging. He is a guest editor for a number of journals, including JAMA, Journal of Neuropsychiatry, and the Journal of Nuclear Medicine.

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Louise Olivier

Psychological Society of South Africa, South Africa

The role of the hidden markers (Depression, Sexual Dysfunction and Personality Change) in Traumatic Brain Injury

Many professionals especially Clinical- and Neuro-Psychologists when doing a medico-legal evaluation for purposes of medical negligence or compensation in Traumatic Brain Injury tend to focus only on the neurocognitive fall outs as a result of the injury and how this would impact on the ability of the patient to carry on with an occupation and quality of life. It is the experience of the presenter that the personality functioning-, emotional-functioning and sexual functioning is equally important to be focused on during a comprehensive medico-legal evaluation for the following reasons:

(a) Research indicates that injury to the brain can cause personality disorder. Rucco (2009) found that damage to the frontal and temporal cortex, diffuse axonal injury and disruptive neurotransmitter systems can cause personality disorders and other psychiatric disorders. Nichol as early as 2009 also stated that damage to the medial frontal area of the brain can cause personality disorders while damage to the limbic system, orbitofrontal cortex and left anteromedial frontal lobe can cause aggressive disorders. Personality disorders and psychiatric problems impact directly on the functioning of a person in the work situation and their quality of life. If psychiatric problems, personality problems or aggressive disorders are found which was not applicable prior to the injury this can directly validate a Traumatic Brain Injury.

(b) A Traumatic Brain Injury can directly impact on the sexual functioning of the patient – either causing inhibition or causing disinhibition. This then also impacts directly on the work performance of the patient as it impacts on his or her personal life.

During the presentation some strategies for measuring personality functioning- emotional functioning- and sexual functioning as hidden markers for Traumatic Brain Injury will be discussed.

Speaker Biography

Louise Olivier is a registered Clinical- and Counselling Psychologist. She has been President of the Psychological Society of South Africa twice. She is at present Chairperson of the Division for Neuropsychology and Forensic Psychology of the Psychological Society of South Africa. She has also been elected to the Professional Board of Psychology of the Health Professions Council and has been a council member for many years. As such she was on the committee of the Health Professions Council of South Africa to determine the criteria for the registration of Neuropsychologists. She has written several books in collaboration with colleagues all over the world regarding forensic psychology and the importance of neuropsychology in forensic work. She has been invited as key note speaker for several conferences in the United States, Australia, and several other countries. She does extensive work both therapeutically and for forensic purposes with Traumatic Brain Injury patients.

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[1]George N Shilau and [2]Oleg N Bubel

[1]Republican Clinic Medical Center, Belarus

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View at the anticonvulsive and Nootropic effects from position of new imagination on understanding of the structure and function GABA-Benzodiazepine receptor complex on the basis of the investigation of the molecular geometry and quantum-chemical characteristics main group anticonvulsants, inhibitor amino acids and some convulsive agent

Statement of the Problem: Until these days, searching of endogen agonists of benzodiazepine receptors is actual task, because a lot of problems clinical medicine neurology, Epileptology, Narcology deepened the understanding mechanism of action and function of GABA benzodiazepine receptor complex to elaborate new perspective anticonvulsive and nootropic compounds.

Purpose: Investigate quantum mechanics characteristics and molecular geometry has three conformational states GABA: linear (GABA-1 conformer), bucket-like (GABA-3 conformer) agonists of which is bucket-like conformer of GABA and isoguvacine, but antagonists are picrotoxin and bicuculline; cyclic (GABA-2 conformer) agonists of which are cyclic conformer of GABA, glycine and β-alanine,but antagonists are bemegride, pentilentetrazol and strychnine; and GABA-3 receptors; maine anticonvulsant's groups. Investigate nootropic's and anticonvulsants effects one-valence salts of glycine and GABA.

Method: Molecular geometry of the benzodiazepine's pharmacophores, main GABA conformers and glycine where studied in the approximation of molecular mechanics with the use of the MM2 force field. Influence introperitonial injection different one-valence salts of glycine and GABA on the cerebral neurophisiological activity in white rats (taking of EEG) and their anticonvulsant activity using strychnine, picrotoxin, pentylenetetrazol and maximal electro seizure models.

Results: It was show, that anticonvulsive and other behavioral effects of derivatives of barbituric acid, benzazepine, benzodiazepine, gidantoine, succinimide and oxasolidindione are realized probably via GABA-2 receptors to switch on them the following functional centers of their structure are nessesary: α, and [δ-ε] for barbitirates; β, [δ-ε] and γ for carbamazepine; β and [δ-ε] for benzodiazepine derivatives, gabapentine and vigabatrine; α, β, γ and [δ-ε] for gidantoine and oxasolidindione derivatives; α, β, γ for succinimide derivatives. The expression of any (including nootropic) behavioral effects of anticonvulsants and inhibitory amino acids depends on power, location and numbers of hydrogen bounds developed between active centers of pharmacophore of anticonvulsant or inhibitory amino acids and active centers of functional skeleton of GABA-2 receptorcomplex.

Conclusion: 1. The more stronger the charge on the atoms of the pharmacophore of the GABA agonist, the more expressive its anticonvulsant effect and Vice versa, the weaker the charge on the atoms, the more expressive the nootropic effects appear 2. There are perspectives of synthesis of compounds, pharmacophore of which should be like as cyclic conformer of GABA, glycine and β-alanine on their quantum mechanics characteristics and molecular geometry.

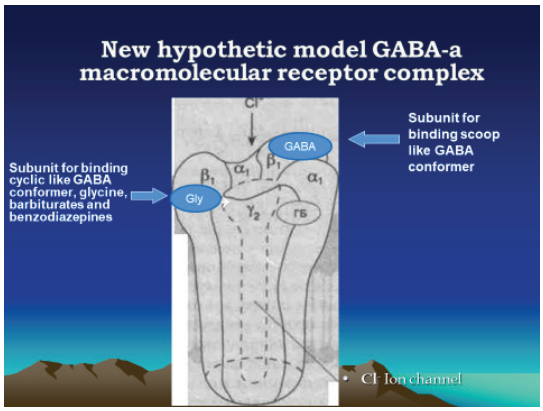

Figure 1. New hypothetic model GABA-a receptor complex

Speaker Biography

George N Shilau has completed his PhD at age of 29 years old from Byelorussian State Medical University. He long time work as senior scientific worker in the laboratory of the biochemistry of neurohormones and neurosurgery over mention University and then as leading scientific worker central scientific-investigating laboratory of Byelorussian Medical Postgraduate Academia and neurologist practitioner, and also as MRY diagnostician. Then he worked as deputy Director of the center of Medical Information "EOCEN" and continues his scientific work in close cooperation with Laboratory of free-radical process chemistry of the Research Institute of Physical Chemical Problems of the Belarusian State University. Now he works as MRY diagnostician of the Republican Clinic Medical Center of the Presidential Administration. He has published more than 40 papers in reputed journals. His interests include neuropharmacology (GABA-benzodiazepine and glycine receptors and their interactions), the clinic of epilepsy.

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