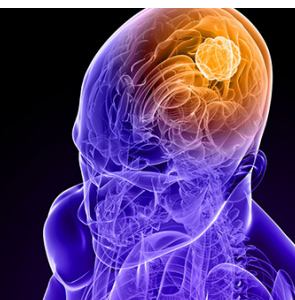

Video Presentation

Health 2019 ***Neuroscience 2019***



Joint Event on
3rd International Conference on
Health Care and Health Management
&
6th International Conference on
Neuroscience and Neurological Disorders

November 04-05, 2019 | Prague, Czech Republic

Inhibitory vs excitatory neuron factors - How the balance of conductive forces needs to be seen at a base level

Paul Lang

Epilepsy Connection Affiliated, Australia

Introduction: This presentation will be focussing on how the core differences between our 2 major neuron types – Inhibitory & Excitatory – play a major role in the balance which controls seizures. I will be discussing the original medical views on the matter, then how views changed once technology gave us greater insight into electrical activity within the brain – and finalise by relating current research to a very understandable analogy such as taste and sound to give the presentation a much wider audience scope.

Description: “Humans have between 90 & 170 different “types” of Neurons (depending on how you classify sub-categories) but they all actually fall into 2 categories – Inhibitory vs Excitatory. This effectively means the signal that neuron sends out either generates more (excitatory) or less (inhibitory) reactionary signals from the surrounding neurons. So basically, some neurons will generate more activity from the neurons around them – whereas others will cause the neurons around them to generate less activity. Prior Neurological studies into Epilepsy focussed mainly on the accelerant (Excitatory) factor – that flaring was only caused by an increase in electrical activity. But we have found this is not correct. It is actually a balancing act that involves the complex processes of ion gating channels combined MRNA


signals vs enzyme and protein regulation and charged ion volumes staying at a steady rate that does not activate the flaring process. And that’s just a simplified view of the issue to say the least.

A key factor we will be looking at is the role genetic signals play in ion gating channels that effect electrical conduction. Studies into SCN2A sodium regulation gene, CaMk11 enzyme, CNTN2 potassium regulators, PKD2 regulators, CRAC regulators, EAG2 potassium channels, MRNA Sodium channel regulators and Reelin gene will be discussed as part of the presentation.

Speaker Biography

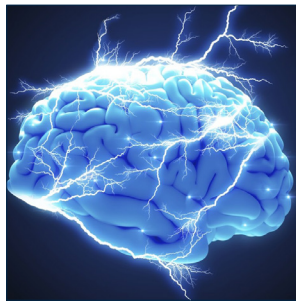
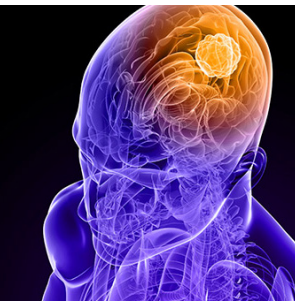
Paul Lang is the Founder and CEO of ECA. Having been diagnosed with a rare form of Epilepsy himself at age 19. He took a lifelong interest in the condition and Neurology itself. After spending much of his adult life researching his own condition, he founded the charity ECA to help spread awareness about Epilepsy - but also support the work being done by charities & research organisations worldwide. Currently his foundation works with organisations from all over the world and ECA is the largest free global affiliate for Epilepsy charity services currently operating. He works constantly as an advocate for research and the expansion of other charities worldwide – collaborating constantly with other global organisations on worldwide campaigns & international conferences.

e: info@epcona.org

 Notes:

Accepted Abstracts

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Deciphering the molecular basis of neuronal development deficits in the recurrent genomic disorder**Derek JC Tai**

Center for Genomic, USA

Reciprocal copy number variant (CNV) of chromosomes 16p11.2 (OMIM 611913), 15q13.3 (OMIM 612001), and 15q11.2-13.3 [Prader-Willi syndrome (PWS), OMIM 176270] are the highly significant recurrent genomic disorders (RGDs) associated with intellectual disability and autism spectrum disorder. The non-allelic homologous recombination (NAHR)-mediated CNV results from mispairing of the flanking segmental duplications, which can result in either loss or gain of the unique genic segment (600 kb in 16p11.2 RGD; 1.5 Mb in 15q13.3 RGD; 5.3 Mb in PWS RGD). However, the pathogenic mechanism and the functional relevance of individual genes within RGDs and the combined contributions of multiple genes are not known. To interrogate the region against an isogenic background, we developed a novel CRISPR/Cas9 genome engineering approach to efficiently generate reciprocal CNV that mimics NAHR. With the comprehensive cell models and the integrated molecular and computational approaches, we attempt to uncover the molecular basis for abnormal neurodevelopment in

these disorders by recapitulating neuropathology of RGD in derivative neuron models. Our preliminary data and several recent studies have strongly suggested KCTD13 and CHRNA7 might be one of the drivers of 16p11.2 and 15q13.3 RGDs respectively. We then defined cellular phenotypes, transcriptional signatures, and co-expression modules that are differentially altered by RGDs. Our transcriptome profiling and analyses showed that genes regulating cytoskeleton (GO:0005856) and translational initiation (GO:0006413) were significantly altered in the neurons with 16p11.2 CNV, and the genes involving axon guidance (GO:0007411) and Wnt signaling pathway (KEGG:04310) aberrantly expressed due to 15q13.3 perturbation. The neuron phenotyping experiments revealed aberrant neurite length, branch points, and electrophysiological features in the RGD neuron models. These studies will allow us to gain more insights into the relationship of gene expression to phenotype and the pathogenic mechanism underlying the disease.

e: jctai@mgh.harvard.edu

Anatomy of spinal dorsal rami and its implications in back pain

Linqiu Zhou

Thomas Jefferson University, USA

The diagnosis and treatment of back pain are challenging. Knowledge of neuroanatomy and biomechanics of the spinal dorsal rami are crucial. The spinal dorsal rami are the posterior branches of the spinal nerve. They innervate back from the occiput to the sacrum. The dorsal rami of C2 and C3 become greater, lesser and third occipital nerves. Dysfunction of these nerves can cause cervicogenic headache. In the low cervical and lumbar spine, each spinal dorsal ramus divides into medial and lateral branches. The medial (small) branch supplies the tissues from the midline to the zygapophysial joint line and innervates two to three adjacent zygapophysial joints and soft tissues. The lateral (large) branch innervates the tissues lateral to the zygapophysial joint line. The dermatome of the dorsal ramus is completely different from the dermatome described in the text book. Clinically, the pain presentations follow these anatomic distributions, which can be used for localization of the disordered dorsal ramus. The

diagnosis can be confirmed by single dorsal ramus block by resulting in relief of pain and muscle spasm. Anatomically, the common dorsal ramus and its medial and lateral branches surround the zygapophysial joint, which serves as a pivot of the spinal functional unit. Any abnormal movement or pathology of the zygapophysial joint can cause stress or tension to the dorsal rami and induce back pain. Clinically, in the low back, L1 and L2 are the most common sites of dorsal rami disorder, because of their anatomical and biomechanical factors. Treatments of back pain include spinal dorsal ramus injection, percutaneous neurotomy and core muscle strengthening exercise. Summarily, disorder of the spinal dorsal ramus is an important source of back pain. Based on the anatomy and clinical presentation, the disordered spinal dorsal ramus can be localized and treated.

e: linzhoumd@yahoo.com

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Attention, memories, and behavioral data-driven study

Milan Jovovic

The New York Academy of Science, USA

Statement of the Problem: Circular search patterns, encoding information in atomic structures have been described. This multidimensional scaling property of the atomic structure has been derived by the Theory of Stochastic Resonance Synergies. In this research, internal states of networked neural system is compared by its interaction with the environment via coupled information propagation.

Methodology & Theoretical Orientation: Scaling multidimensional information with stochastic resonances gives in theory an answer to the emergence of periodic tables of the atomic elements. The clusters of information arranged by the nucleons derives a modeling approach to attention and memory, within a behavioral experiment. This modeling

approach applied to the neuroimaging data is used as a basis for building a datamining analysis tool.

Findings: Initial findings have been reported. We have shown that the intensity-independent auditory distance feature detectors, along with the tonotopic map of the auditory cortex, result from the information flow in the brain.

Conclusion & Significance: We propose a genotype information flow processing in brain scans that do not directly apply temporal dynamics. Neuroimaging and dynamical brain maps source localization have shown potential in clinical applications that in can be used in diagnosis, analysis and monitoring, as well as treatment of disordered neural states.

e: jovovic.milan@gmail.com

Beyond diagnosis: A longitudinal, case control study predicting use of case management services for adolescents and adults living in community following brain injury, with implications for long-term support access**Barbara Baptiste**

Rehabilitation Sciences Institute (RSI), Canada

Statement of the Problem: Individuals diagnosed with brain injury (BI) experience a myriad of functional limitations throughout their lifespan. Case Management (CM) is a service that assists in coordinating care to ensure access and use of services to address both activity and participation limitations. These functional changes range from physical to cognitive and include mental health challenges. The impact on individuals, families and society is substantial. After diagnosis, the first question individuals and families will ask the physicians is on prognosis (their future). What will the future bring? How will I function? Can I work? Will I experience a downward spiral at some point? CM has been identified as a support service to assist people through the maze of lifelong changes. This research used a published model for service use to analyze a province-wide, longitudinal database of persons with BI, which included questions on service use. A case-control design was used to compare users and non-users of the CM service. CM use was considered a primary access point for other services in the community. The study sample came from questionnaires of 203 users of CM services and 273

non-users, complete for all outcome and predictor variables. These were individuals with BI, 15 years of age and older. Out of a dataset of 1,960 questionnaires, 476 met the inclusion criteria. There were eight predictor variables and one outcome variable (use or non-use of the service). Predictor variables considered the framework of the Behaviour Model of Health Service Use (BMHSU); specifically, pre-disposing, need and enabling factor groups as these relate to health outcomes and service use and access. Analyses revealed significant differences between users and non-users of CM services. In particular, users were significantly younger than non-users as the older the person the less likely to use the service. Also, users had less education and more severe activity limitations and lower community integration. Persons living alone are less likely to use case management. Funding groups also significantly impact users. Implications exist for future care need and service use and access, following BI and other neurotraumas and health conditions.

e: bbaptiste@rehabilitation.ca

**Therapeutic ketosis and the broad field of applications for the ketogenic diet:
Ketone ester applications & clinical updates****Raffaele Pilla**

St. John of God Hospital, Italy

It has been recently shown that nutritional ketosis is effective against seizure disorders and various acute/chronic neurological disorders. Physiologically, glucose is the primary metabolic fuel for cells. However, many neurodegenerative disorders have been associated with impaired glucose transport/metabolism and with mitochondrial dysfunction, such as Alzheimer's/Parkinson's disease, general seizure disorders, and traumatic brain injury. Ketone bodies and tricarboxylic acid cycle intermediates represent alternative fuels for the brain and can bypass the rate-limiting steps associated with impaired neuronal glucose metabolism. Therefore, therapeutic ketosis can be considered as a metabolic therapy by providing alternative energy substrates. It has been estimated that the brain derives over 60% of its total energy from ketones when glucose availability is limited. In fact, after prolonged periods of fasting or ketogenic diet (KD), the body utilizes energy obtained from free fatty acids (FFAs) released from adipose tissue. Because the brain is unable to derive significant energy from FFAs, hepatic ketogenesis

converts FFAs into ketone bodies-hydroxybutyrate (BHB) and acetoacetate (AcAc)-while a percentage of AcAc spontaneously decarboxylates to acetone. Large quantities of ketone bodies accumulate in the blood through this mechanism. This represents a state of normal physiological ketosis and can be therapeutic. Ketone bodies are transported across the blood-brain barrier by monocarboxylic acid transporters to fuel brain function. Starvation or nutritional ketosis is an essential survival mechanism that ensures metabolic flexibility during prolonged fasting or lack of carbohydrate ingestion. Therapeutic ketosis leads to metabolic adaptations that may improve brain metabolism, restore mitochondrial ATP production, decrease reactive oxygen species production, reduce inflammation, and increase neurotrophic factors' function. It has been shown that KD mimics the effects of fasting and the lack of glucose/insulin signaling, promoting a metabolic shift towards fatty acid utilization. In this work, the author reports a number of successful case reports treated through metabolic ketosis.

e: raf.pilla@gmail.com

*Notes:*

Spinal benign tumors treated By full endoscopy interlaminar approach

Márcio Robertti Ramalho Da Cunha

Freie Universitat, Germany

Percutaneous endoscopic technique has been used to treat disk herniation and spinal stenosis, so far we have very few reports to treat benign spinal tumors treated by this minimally invasive treatment. We would like to present some cases of lumbar benign spinal tumors removed by full endoscopic approach. We've described 04 cases of benign lumbar spinal tumors, that were treated by full endoscopy in a period of two years. The patients had no major neurological signals, only back pain associated or not with radicular symptoms. The M.R.I demonstrated small lesions in the lumbar field measuring from 1,0cm to 4,0cm. We've performed all the procedures with a single skin incision less than 08mm, placing the working canula between the interlaminar bone window, according to the level related to the lesion, making a enlargement under assistance of diamond burr to expose the ligamentum flavum from the base to the tip of the ascending facet to make a good

exposition of the surgical area. After opening the ligamentous flavum, the tumors were totally removed piecemeal under endoscopic guidance. The procedures lasted less than three hours with no support in intensive care unit and the pathological examination confirmed : 01 case of angioliopoma, 02 cases of Schwannoma and one case of neurinoma. All the patients had a hospital discharge less than twelve hours after the surgical procedure with complete relief of neurological symptoms and using minor pain killer to control the back pain. After one week he could return to his work under the support of physiotherapy rehabilitation. Even though we don't have so much papers about this surgical practice, we think that is a feasible treatment bringing all the benefits of the minimally invasive approach pointed in many papers. Of course is necessary to develop more proper endoscopic tools to accelerate the surgical time.

e: maramalho65@gmail.com

Cognitive flexibility predicts PTSD symptoms: Observational and interventional studies

Ziv Ben-Zion

Sagol Brain Institute Tel-Aviv, Israel

Introduction: Post-Traumatic Stress Disorder(PTSD) is a prevalent, severe and tenacious psychopathological consequence of traumatic events. Neurobehavioral mechanisms underlying PTSD pathogenesis have been identified and may serve as risk-resilience factors during the early aftermath of trauma exposure. Longitudinally documenting the neurobehavioral dimensions of early responses to trauma may help characterize survivors at risk and inform mechanism-based interventions. We present two independent longitudinal studies that repeatedly probed clinical symptoms and neurocognitive domains in recent trauma survivors. We hypothesized that better neurocognitive functioning shortly after trauma will be associated with less severe PTSD symptoms a year later, and that an early neurocognitive intervention will improve cognitive functioning and reduce PTSD symptoms.

Methods: Participants in both studies were adult survivors of traumatic events admitted to two general hospitals' emergency departments (EDs) in Israel. The studies used identical clinical and neurocognitive tools, which included assessment of PTSD symptoms and diagnosis, and a battery of neurocognitive tests. The first study evaluated 181 trauma-exposed individuals one-, six-, and fourteen months

following trauma exposure. The second study evaluated 97 trauma survivors one month after trauma exposure, randomly allocated to 30 days of web-based neurocognitive intervention (n=50) or control tasks (n=47) and re-evaluated all subjects three- and six months after trauma exposure.

Results: In the first study, individuals with better cognitive flexibility at one-month post-trauma showed significantly less severe PTSD symptoms after 13 months ($p=0.002$). In the second study, the neurocognitive training group showed more improvement in cognitive flexibility post-intervention ($p=0.019$), and lower PTSD symptoms six months post-trauma ($p=0.017$), compared with controls. Intervention-induced improvement in cognitive flexibility positively correlated with clinical improvement ($p=0.002$).

Discussion: Cognitive flexibility, shortly after trauma exposure, emerged as a significant predictor of PTSD symptom severity. It was also ameliorated by a neurocognitive intervention and associated with a better treatment outcome. These findings support further research into the implementation of mechanism-driven neurocognitive preventive interventions for PTSD.

e: zivbz1@gmail.com

Idiopathic bilateral simultaneous facial nerve palsy (B-Fnp)

Theresia Christin

Mohammad Hoesin Palembang, Indonesia

Background: Bilateral facial nerve palsy (B-FNP) is a rare clinical manifestation with incidence of 1 per 5 million people which representing less than 2 % of all cases of facial nerve palsy. Majority of the cases is due to serious underlying medical condition. However, this case report shows bilateral facial nerve palsy that could not be attributed to any particular etiology.

Case Report: A male 64-year-old with idiopathic bilateral simultaneous facial nerve palsy that occurred suddenly with difficulty closing both eyes and facial droop. Neurological examination shows bilateral peripheral facial nerve palsy. The right side is grade IV, while grade III in the left side based on House Brackman grading system. There is no other cranial nerve abnormality with normal motoric and sensory function in all extremities. There is no sign of central lesion such as vascular disease, tumor, autoimmune, intoxication or infection. Supporting examinations such as lumbar puncture,

thorax X-Ray, and head MRI with contrast shows normal result. Neurophysiology examination which is nerve conduction study (NCS) was done. There were normal results of NCS in all extremities, which is necessary to exclude Guillain-Barre Syndrome (GBS). NCS and EMG study in facial muscles shows abnormal results which supported by blink reflex testing that absent of all parameters. The patient then treated with prednisone 60 mg orally that was given with tapering off dosage for 10 days. Patient was hospitalized for 12 days and discharge with good clinical improvements marked by follow up grading scale of which grade II in right side and grade I for the left side.

Conclusion: Bilateral facial nerve paralysis is a rare condition and challenging in diagnosis. It is important to have appropriate diagnosis with supporting examination and comprehensive treatments for better prognosis.

e: dr.theresiachristinsp.s@gmail.com