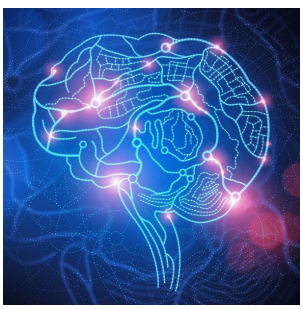
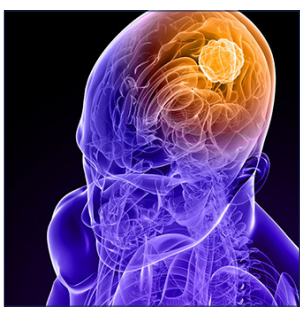
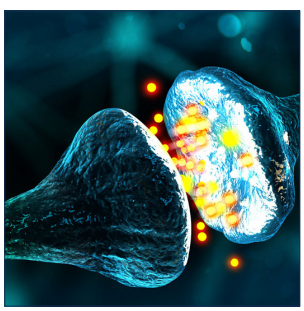
Poster

Parkinson's 2019



International Conference on

Parkinson's, Huntington's & Movement Disorders

April 17-18, 2019 | Frankfurt, Germany

Effects of cyclic AMP on the differentiation and bioenergetics of rat C6 glioma cells

Ahmed Hisab
South Denmark University, Denmark

Elevation in the level of intracellular cAMP is known to induce astrocytic differentiation of C6 glioma cells by unknown mechanisms. Therefore, cytoskeletal protein genes (phalloidin) fluorescents to investigate morphological changes, cell proliferation assay, MTT assay, flow cytometry, western blotting, in-cell western, immune-cytochemical (protein expression and localization), and oxygen electrodes (oxygen consumption rate) after a treatment with 0.25 mM dbcAMP were conducted. Undifferentiated cells (media without dbcAMP) showed a flat polygonal appearance, whereas those cultured in the presence of 0.25 mM dbcAMP exhibited a more differentiated astrocytic morphology. They had more numerous neurite-like thin processes. The cell proliferation of differentiated c6 glioma reduced at day 2 and then started to increase at day 3 till day 5 compared to undifferentiated c6 glioma cells. In terms of flow-cytometry data, dbcAMP had no apoptotic effect on the C6 glioma cells. There was an increase in the protein expression GFAP (specific marker for astrocytes). There was no significant effect between undifferentiated and 5-day differentiation regarding their response to glucose 10 mM. In addition, there were no significant effects of glucose on the basal of 5-day differentiation of C6 glioma cells. However, there was a significant correlation between the concentration of glucose and inhibition of the basal oxygen consumption. Finally, glucose 10 mM did not stimulate NAD (P)H levels of C6 glioma cells. The above results showed that cAMP induce C6 glioma cells differentiation without affecting its bioenergetics. Therefore cAMP is considered to be the best differentiating agent.

Speaker Biography

Ahmed Hisab have completed his MPhil in Pharmacology and cell Physiology at the age of 32 years from Nottingham University, UK. He is a master student in computational biomedicine of South Denmark University, Denmark. He has published over 6 publications in very scientific journals.

e: memeassd40@gmail.com

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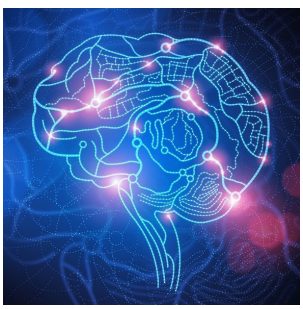
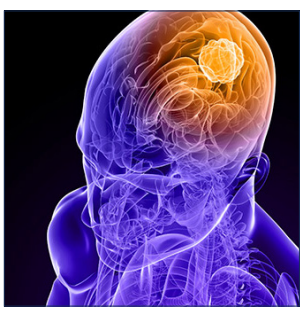
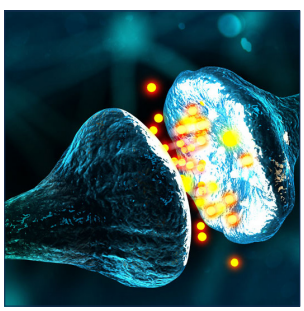
Video Presentation

Parkinson's 2019



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Telomerase activators improve motor function and protein degradation in a mouse model of Parkinson's disease

Gabriele Saretzki and **Tengfei Wan**
Newcastle University, UK

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. Analyzing levels of total, phosphorylated and aggregated alpha synuclein 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 found a decrease in poly-ubiquitinated proteins and the autophagy receptor p62 and analyze 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 has completed her PhD 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 87 papers in peer-reviewed journals and is an editorial board member of BMC Biology, PloS One and Oxidative Medicine and longevity.

e: gabriele.saretzki@ncl.ac.uk

Notes:

MicroRNAs modulation-A potential Neuroprotective strategy for Parkinson's therapy

Camila Hillesheim Horst
University of Brasília, Brazil

Parkinson's disease is the second most common neurodegenerative disorder and is characterized by the loss of nigrostriatal neurons. MicroRNAs (miRNAs) are noncoding RNAs that regulate cellular mRNA content. Considering that one single miRNA regulates hundreds of target genes by RNA interference (RNAi), a dysregulated expression of miRNAs may cause critical pathological consequences. Indeed, aberrantly expressed miRNAs can play a critical role in neurological disorders, as well as in Parkinson's disease. Therefore, controlling microRNA levels with synthetic oligonucleotides might protect dopaminergic neurons in models of Parkinson's disease. In this regard, our previous work revealed that microRNA-targeted oligonucleotides induced significant improvement in the viability of SH-SY5Y cells exposed to rotenone, suggesting a neuroprotective role to miRNA modulation. In the present study, we investigated if controlling microRNA expression would protect striatal neurons of rotenone-injured rats. We injected oligonucleotides structured in Neuromag® nanoparticles by stereotaxic surgery in the right lateral ventricle. Two days after surgery, animals received ten daily I.P. injections of rotenone. Fluorescence microscopy showed that nanoparticles successfully transfected the oligonucleotides into striatal neurons. Furthermore, the transfection produced a significant silencing in microRNA's expression in the area. Altogether, our results suggest that microRNAs are a potential target for Parkinson's therapy and therefore miRNAs modulation is a promising strategy to reduce the vulnerability of dopaminergic neurons to rotenone insult.

Speaker Biography

Camila H.H. is a biologist who graduated in the University of Brasília and is currently concluding her Master's degree in the same university. She has been studying Parkinson's disease for more than six years and has already contributed to Parkinson's research with her published articles. She has also worked with international research groups and attended meetings in the area of study. Finally, she intends to continue in this field in her Ph.D.

e: horstcamila@gmail.com

Notes:

Tourette Syndrome at school in Argentina

Andrea S Bonzini
Argentine Association for Tourette Syndrome, Argentina

Tourette Syndrome (TS) is a neurologic disorder that becomes in early childhood or adolescence. is characterized by motor and vocal tics. Some Tic Disorders may be transient, while others will persist into adolescence and adulthood. It is common for people with Tourette Syndrome to be affected by another co-occurring condition like Attention Deficit Hyperactivity Disorder (ADHD), Attention Deficit Disorder (ADD), Obsessive Compulsive Disorder (OCD), Anxiety, There is no cure for Tourette Syndrome, but there are various treatment options

I would like to tell you the story of AAST (Argentine Association for Tourette Syndrome).Our Conferences are aimed to health and education professionals and students in related careers, as well as to the families of children and/or adults suffering Tourette:I will briefly explain my own story about how I created the Association. When my daughter was 8 years old-at present she is 22-she was diagnosed with TS It was a long 2years journey until a diagnose was reached. When my daughter was 6 year old I began to notice several tics. During a routine control with her pediatrician, who knows her since she was born, I told him about certain sounds she made and I insisted on these tics such as winks and short head movements. Of course I received the same answer : "you are an overprotective mother, there is absolutely nothing wrong with your daughter, it will all go away". As the year went by and the situation worsened for new problems appeared such as anger and some phobia, the environment at home was one of constant fighting, screaming and swearing unimaginable for an 8-year-old. Years spent explaining that she was able to do the same things like any other girl her age first, then as a teenager, years sending medication to camp trips, to a graduate trip, with the look of apprehension for the responsibility and especially the fear of people who did not understand that it was neither dangerous nor contagious, that they just had to make sure she took her medication and played, enjoyed, danced, sang, etc.

That's why is so important the awareness.

Speaker Biography

Andrea S.Bonzini, Founder & President for the Argentine Association for Tourette Syndrome since September 19th, 2012. The association was created, primarily to inform and educate teachers that our children would not have a bad experience throughout their school years, the most important experience of their growth. AATS is a non-profit association where all is done with the heart and with one's own efforts.

e: astourettearg@gmail.com

Notes:

E-BABE: The hidden relation, clues of Autism, ADHD and Depression which reveals the effective cause and cure

Van Duy Dao

Awaken You Wonderful We, Vietnam

Observing the facts: they lack social skills, they cannot talk: language is the product of living environment - native language; we speak it naturally without thinking at all. We are not born with our native language, so I doubt their connection with their living environment and the state of mind that they cannot learn. Testing them with Aesop fables, metaphors, pretending game - they do not understand these as normal kids; and they are poor in interacting, communicating or persuading. They are in the low level of this. For official test: you can test them with EQ test. All these low-level vital skills make them never feel safe, connection to the environment: it makes them stress. Over time, it makes the downward spirals that make them more and more lack of social skills and suffer more stress.

Poor in EQ: autistic kids do not understand fable and metaphors. Genes cannot make them poor in EQ, and cognitive thinking. Trainers know any skill can be learned with just basic supports.

ALL IN ONE, ONE IN ALL: As therapists: Neurologist, psychiatrist, sociologist, gastroenterologist, urologist, educators, sleep therapists, cardiologist, language therapists, educators, trainers and teachers, we should remember there is no separation in the health of heart, stomach, muscle, cognitive thinking, sleeping, hormone system: all are interdependent and under the state of mind. Characters of the mind: irrational mind, the giant brain evolved for millions of years, illogical mind and Placebo effects, neuro-plasticity, Mirror neurons, self-affirmation, self-talk, nocebo effects, T1/2 of all substances, taboos, rituals, religious belief, compound effects, conditioned responses, flexible adaptability, illusive mind, self-healing or self-destroying, irrational thinking, Subliminal message, Marketing of luxury brand, and Hysteria. What do people feed the mind of the kids every day? And what if all of these lead to negativity or positivity?

Speaker Biography

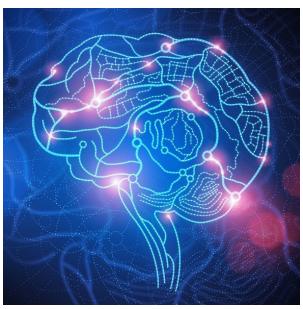
Van Duy Dao, born 1986, had completed Pharmacist Bachelor degree in Hanoi University of Pharmacy. He is a pharmacist, personal development lover, and meditation practitioner. Proud to be a pharmacist, but he is ashamed of the failure of knowledge and the medication in healing many unknown Causes. Most of the medications only make temporal relief and many medications chronic patients have to take in their lifetime. Worst of all: mental illness, depression, ADHD, Autism, suicide, and gunfire, social problems seem to go against all theadvancement in economic, standard living and Giant leap in technology. He keeps on searching. Advantage of the general knowledge of medicine, physiology, psychology, pharmacology, management, marketing, selling, persuading, motivating; and the mixing of Eastern Philosophies: Buddhism, Taoism, Confucianism which full of paradoxical subtle profound knowledge in the art of living, virtues, purposeless, pace of nature to gain success, health, happiness, and inner-peace in daily life.

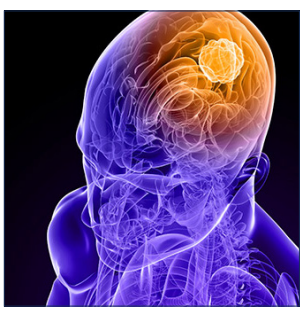
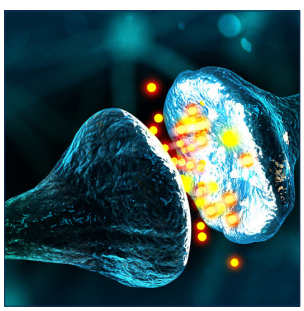
e: duyvan64@gmail.com

Notes:

E-Poster

Parkinson's 2019



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Verification of skill improvement of Dementia prevention supporters

Kazue Sawami[1], Mitsuo Kimura[1], Tetsuro Kitamura[1], Mihoko Furusumi[1], Masahiko Kawaguchi[1],Chizuko Suishu[2], Naoko Morisaki[3] and **Sonomi Hattori[4]**

[1]Nara Medical University, Japan
[2]Shubun University, Japan
[3]Himeji University, Japan
[4]Wakayama Medical University, Japan

Background and purpose of the study: Nowadays, one out of four elderly people suffer from dementia. To establish the prevention for dementia is one of our international goals. With this background, we conducted a so-called "Step-up Training" comprised of 12 sessions for caregivers who possess certification for recreational therapy. The intended effect is to for them to learn techniques for dementia prevention and practice these techniques to elderly people.

Methods: We conducted surveys once at the beginning of the training and one more time at the session number six, which serves as the interim assessment. We compared these results to see how their responses changed after going through 6 sessions of the trainings.

Results: This survey presents the results of the 6th interim evaluation. The data for 46 valid respondents out of the 69 registrants were analyzed. The average age was 60.1 ± 9.5, with 6 males and 40 females. The results showed that they did stepped up from just knowing recreational therapy to knowing various methods and evidence behind these methods regarding dementia prevention. Their self-assessment of techniques increased from 2.26 to 4.02 in average with 5 being the maximum (paired t-test: $p<0.01$).

Conclusion: Practical dementia prevention requires the combined method of aerobic exercises and cognitive trainings and also they have to be enjoyable so that the elderly people can enjoy these activities every day. To develop the talents who can extensively practice this is an urgent matter in the field. Also it is an effective support for increasing number of the preventable people of dementia. We were able to confirm the improved skills among the trainees in this study. In the future, we will present the results of their actual practices and application of their skills. This research funding is scientific research expenses of the Japanese Ministry of Education, Culture, Sports, Science and Technology.

Speaker Biography

Kazue Sawami is a professor at Nara Medical University and completed her PhD at health science. Her research is about the cognitive abilities of elderly people. Current clinical trials below. UMIN000029749, 000025484.

e: sawami@naramed-u.ac.jp

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Pharmacological correction of Mitochondrial Dysfunction in rotenone model of Parkinson's disease: potential participation of P53, NF κB and Nrf2

Olga Gonchar and **Iryna Mankovska**

Bogomoletz Institute of Physiology National Academy of Sciences of Ukraine, Ukraine

Mitochondrial dysfunction has been widely implicated in the neuronal degeneration in Parkinson's disease (PD). Mitochondria-targeted protective compounds that prevent or minimize a wide range of mitochondrial defects constitute new therapeutic strategies in the prevention and treatment of such degeneration. The antioxidant mexidol (2-ethyl-6-methyl-3-hydroxypyridine succinate) is used in clinical practice as a neuroprotector due to its positive influence on the brain energetic metabolism and free radical processes. However, its application as mitochondria-targeted agent to prevent or treat of PD had not been studied yet. We have used rotenone long-term administration as a rat model of PD to investigate in brain mitochondrial oxidative stress intensity, protein expression/activity of antioxidant enzymes-manganese superoxide dismutase (MnSOD), glutathione peroxidase (GPx), and antiapoptotic Bcl-2 as well as protein expression of their upstream regulators: P53, Nrf2 and NF-kB. Rotenone intoxication induced an increase in ROS formation, lipid peroxidation, H2O2 production and a decrease in GSH/GSSG ratio, mitochondrial aconitase activity as well as disorders in mitochondrial antioxidant status (reduced MnSOD, GPx activities/protein content and mRNA expression). In parallel with P53 mitochondrial translocation, we found a decrease in Bcl-2 protein level, an enhance in nuclear accumulation of the phosphorylated NF κB p65 protein. Under the action of rotenone with mexidol, there was demonstrated a reduction in oxidative stress biomarkers, elevation of antioxidant capacity by an increase in protein expression of Nrf2 and its targets (MnSOD and GPx). In brain mitochondria Mexidol interrupted apoptotic cascade by lowering of P53 protein accumulation as well as increasing Bcl-2 protein content. Simultaneously we registered some decline in NF κB p65 protein level in nuclear extracts of brain cells. The efficacy of mexidol determined in the rotenone model of PD may be explain by its ability to influence on mitochondrial redox status and in that way modulate many signaling pathways in brain cells.

Speaker Biography

Olga Gonchar is a senior researcher, Department of Hypoxia, Bogomoletz Institute of Physiology National Academy of Sciences of Ukraine, and she studied mechanisms of correction of tissue hypoxia by common use of biomembrane stabilizators and antioxidants; studied the methods of adaptation to hypoxia (high altitude stay and intermittent hypoxia) and their use in medicine and sports; studied the genetic and epigenetic mechanisms of the oxygen-dependent cell processes regulation under adaptation to hypoxia and oxidative stress; experimental and clinical (Parkinson's disease and Diabetes mellitus) investigation of mitochondrial dysfunction development under hypoxia and oxidative stress.

e: olga.gonchar@i.ua

Notes:

Accepted Abstracts

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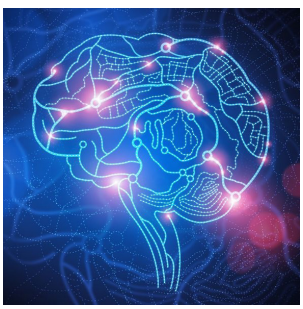


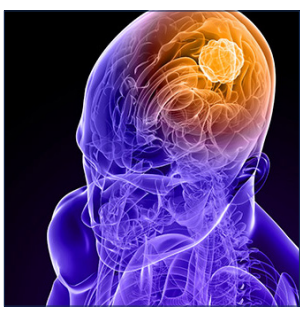
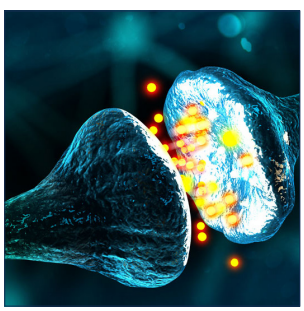

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Management of the behavioral aspects of Parkinson's Disease

Leora L Borek
MedOptions, USA

Parkinson's disease is a progressive and debilitating movement disorder that is diagnosed by its motor signs. The behavioral manifestations of Parkinson's disease are prevalent and frequently complicate the course of the disease. These may be due to the illness itself or its treatment and are often more disabling than the motor symptoms. This review focuses on the management of the most common behavioral symptoms of Parkinson's disease, including depression, anxiety, psychosis, dementia, and sleep disorders.

e: leolisa@yahoo.com

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Effects of biofeedback postural training on pre-existing low back pain in static-posture workers

Sehun Park
Mercy Sports Medicine, USA

Many studies report a relationship between poor static posture (SP) and low back pain (LBP). The study examines the effects of a vibrational feedback postural training program on pre-existing LBP, spinal health benefits, level of physical activity, workability. The researchers want to know if static posture training with biofeedback can alleviate LBP. Control group wore a pedometer and experimental group wore a postural training device for three weeks. Participant answered a pain scale survey and had side-view pictures before and after the intervention. LBP discomfort decreased between the two groups. There was no statistical difference in average steps between the two groups. Postural training with biofeedback reduced the frequency of having LBP, discomfort level of the pain, and lost work time due to LBP. The study concludes that the vibrational feedback from the device helps correct poor SP. Also, increasing levels of physical activity by registering more daily steps provided a positive effect for decreasing frequency of LBP. However, increased level of physical activity worsened discomfort level of LBP and increased lost work time in the control group.

e: sehun.park2@mercy.net

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Using *Drosophila* to define the role of glia in alpha-Synucleinopathies

Abby L Olsen
Harvard Medical School, USA

α-synucleinopathies are neurodegenerative diseases that are characterized pathologically by α- synuclein inclusions in neurons and glia. In spite of this, the role of glial α-synuclein and even glia more broadly in these diseases is not well understood. Glial α-synuclein may be of particular importance in multiple system atrophy (MSA), which is defined pathologically by glial cytoplasmic α-synuclein inclusions. We have previously described *Drosophila* models of neuronal α-synucleinopathy, which recapitulate key features of the human disorders. We have now expanded our model to express human α-synuclein in glia. We demonstrate that expression of α-synuclein in glia alone results in α-synuclein aggregation, death of dopaminergic neurons, impaired locomotor function, and autonomic dysfunction. Furthermore, co-expression of α- synuclein in both neurons and glia worsens these phenotypes as compared to expression of α- synuclein in neurons alone. We identify unique transcriptomic signatures induced by glial as opposed to neuronal α-synuclein. These results suggest that glial α-synuclein may contribute to the burden of pathology in the α-synucleinopathies through a cell type specific transcriptional program. This new *Drosophila* model system enables further mechanistic studies dissecting the contribution of glial and neuronal α-synuclein in vivo, potentially shedding light on mechanisms of disease that are especially relevant in MSA but also the α-synucleinopathies more broadly. Indeed, beyond glial α-synuclein, we identify additional novel glial modifiers of neuronal α- synuclein toxicity in the hopes of eventually turning these modifiers into glial-based therapeutics for Parkinson's disease

e: alolsen@partners.org

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Managing life with Parkinson's disease

Irene Treacy
Smovey Health, Ireland

Abstract in dealing with Parkinson's Disease including taking medications correctly, huge selection of different types of exercise available, correct nutrition and various diet options, mindset, motivation and selection of holistic treatments available. Everybody knows the mainstream way to deal with Parkinson's Disease but very few contemplate or are conscious of alternative therapies like Smovey, Tapping, Yoga or Neuro Linguistic Programming to help people with Parkinson's. I want to talk about the hundreds of clients I have that are maintaining their condition and many improving on their circumstances through mindset and motivation. As a certified practitioner of Neuro Linguistic Programming, I believe that we can achieve much more that we are aware of and I want to show the audience some basic principles of NLP and show how it can improve their quality of life. I would also like to give a free on line course to all that sign up.

e: irene@smoveyhealth.com

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Non- Pharmacological interventions for Lateral Axial Dystonia (Pisa syndrome) in Parkinson's diseases: A review

Mohammad Etoom
Al-isra University, Jordan

Postural deformities are frequent and disabling complication of Parkinson's diseases. Pisa syndrome (PS), or lateral axial dystonia is a postural deformity characterized by a marked lateral flexion of trunk. PS studies showed patterns of trunk muscles hyperactivity ipsilateral or contralateral to bending side. Recent evidences found that PS is correlated with more impairments vestibulospinal tract, upper limb functions, spine pain, and quality of life. The aim of this abstract to review the non-pharmacological interventions for PS in Parkinson's diseases through MEDLINE database. The current evidence shows a set of non-pharmacological interventions address PS as: exercise interventions, Botulinum toxin, spine surgery, spinal cord stimulation, and subthalamic deep brain stimulation. The studies show positive effects for exercise interventions, Botulinum toxin, spinal cord stimulation, and deep brain stimulation on lateral trunk deformity, spine pain, and motor impairments. Spine surgery did not improve spine deformities or motor impairments. Exercise and Botulinium toxin recommended considering the hyperactive muscles regardless the bending side. Observational studies found that the more impaired muscles are paraspinal, abdominal oblique, rectus femoris, rectus abdominis, external oblique, and quadratus lumborm. According to the previous findings, non-pharmacological interventions except spine surgery are beneficial option for PS. More consideration for the impaired muscle, dystonia characteristics, and trunk proprioception is required to treat and rehab patients with PS.

e: mohammed.etoom2016@gmail.com

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Increase or alleviate weight in individuals with SCA? Two lines of evidence for the physiotherapeutic approach

Laura Oliveira
IFRJ/UNISUAM, Brazil

Spinocerebellar ataxia (SCA) comprises a family of autosomal dominant inherited disorders that result from progressive degeneration of the cerebellum and its associated systems. Besides cerebellar deterioration, SCA is accompanied by degeneration of other nervous system sites. It leads to non-cerebellar signs such as pyramidal and extrapyramidal losses, uncommon in ataxia of other etiologies that can worsen the impairments of people with SCA. The most common motor deficiencies in SCA are related to gait and body balance, with increased risk of falls and predisposition to physical inactivity, followed by cardiorespiratory capacity limitation. Despite the huge recent advances in neurogenetic research, an effective pharmacological approach to face this condition is still unknown. In this context, rehabilitation strategies could represent an alternative to improve the physical condition and to reduce the impairments of these individuals. In this lecture I will present two lines of intervention. First, the results of a study in which gait, cardiopulmonary capacity, and balance were challenged during gait using a partial body weight support (BWS) and a treadmill will be discussed. The effects of this training over functionality and quality of life will be also presented. In few words, gait training using partial BWS significantly increased gait performance, treadmill inclination, duration of exercise, and cardiopulmonary capacity in individuals with SCA. Balance improvements were also found. In the second part, I will show an opposite line of intervention: the effects of progressive addition of external loads over the performance in tests of postural stability and gait, and the comparison of load addition over different body parts (waist girdle, pelvic girdle and lower limbs). The evaluation instruments used in this part were a posturography examination, kinematic gait analysis, Scale for the Assessment and Rating of Ataxia, Modified Dynamic Gait Index, Berg balance scale, Inventory of Non-Ataxia Signs and SCA Functional Index.

e: lauraoliveira.ft@gmail.com

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Analysis of Sleep Macrostructure in patients diagnosed with Parkinson's disease

Justa Elizabeth González-Naranjo
International Center for Neurological Restoration, Cuba

Patients diagnosed with Parkinson's disease present sleep disorders with a higher frequency than the general population. The sleep architecture in these patients shows variations with respect to the normal population, so in this work it was decided to investigate the characteristics of the macroarchitecture of sleep in patients diagnosed with Parkinson's disease. A polysomnographic study was carried out on 77 patients diagnosed with Parkinson's disease. All the studies were processed according to the AASM Manual for the Scoring of Sleep and Associated Events v.2.2, and to the criteria of the International Classification of Sleep Disorders 3rd ed. (2014). Processing was carried out using descriptive statistics, as well as non-parametric analysis for comparison between cases and controls. The group of patients showed significant reductions of the N2, N3, and REM sleep stages when compared with a control group, as well as a significant increase in intra-sleep wakefulness. The number of REM–NoREM sleep cycles and sleep efficiency showed marked reduction compared to the control group. There was a statistically significant difference in the macroarchitecture of sleep between patients diagnosed with Parkinson's disease and healthy controls.

e: egonzalez@neuro.ciren.cu

Comprehensive approach for genomic characterization of individuals with Parkinson's disease

Bashayer Al Mubarak
King Faisal Specialist Hospital and Research Centre, Saudi Arabia

The majority of available genetic tests for Parkinson's disease are focused on a limited number of genes. These tests may not be informative particularly with the increasingly recognized genetic heterogeneity of PD. As more than 40 candidate genes have been discovered through whole-exome sequencing (WES) oriented studies, in addition to a number of genes previously identified by linkage and candidate gene approaches. Therefore, implementing a genetic assessment workflow that can capture the genetic lesions typically found in PD (copy number variations (CNVs) and single nucleotide variants (SNVs)), is essential for a comprehensive molecular characterization. In this study, we devised an integrative genomic evaluation workflow that can capture both SNVs and CNVs. The multistage strategy that we have employed involves screening samples for CNVs in known PD genes, followed by WES and variant prioritization. By applying this approach we have successfully identified 125 novel rare variants in 85% of our cases including 22 high confidence ones. All the discovered variants were present in new candidates genes that have not been previously reported in PD except for two (EIF4G1 and ATP13A2). Our combined molecular approach provides a comprehensive strategy applicable for complex genetic disorders.

e: bai-mubarak@kfshrc.edu.sa

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