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Matteo Santoro, J Alzheimers Dis Parkinsonism 2016, 6:6(Suppl)
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Activation of NLRP3-inflammasome in the MPTP mouse model of Parkinson`s disease might be triggered by HMGB1-MAC-1 axis

Matteo Santoro
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Mounting evidence suggests the involvement of the immune system in neurodegenerative disorders including Parkinson`s disease (PD). We recently reported increased levels of HMGB1 in PD patients as well as in the MPTP animal model of PD. In the present study we explored whether the release of HMGB1 in our mouse model of PD caused the activation of the NLRP3 (NOD-like Receptor Protein 3) positive inflammasome. NLRP3-inflammasome is a multiprotein complex, and part of the innate immune system that is activated in aseptic conditions such as tissue damage or metabolic impairment. Its activation leads ultimately to both formation and release of the proinflammatory cytokine IL-1β. C57BL6J mice were injected with the sub-acute regimen (30 mg/kg/day for five consecutive days i.p., control animals were injected with equivalent volume of saline solution) of the neurotoxin 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). Brain tissue was harvested 1-2 days post-injection. Tissue was then prepared for double immunofluorescent staining of three different cell types: dopaminergic neurons, astrocytes and microglia, performed on midbrain sections inclusive of substantia nigra, or for western blotting experiments conducted on protein lysate from ventral midbrain. Our confocal microscopy analysis confirmed an increase in NLRP3 protein levels in the cytoplasm of microglia one day after MPTP injections. In parallel, heightened levels of the microglial MAC-1 protein were confirmed histologically at the level of the substantia nigra and by western blotting. This up-regulation of MAC-l, a surface receptor for HMGB1, may therefore constitute a critical link in the activation of cytoplasmic pathways leading to activation of the NLRP3-inflammasome in Parkinsonism.

Biography

Matteo Santoro successfully graduated in Chemistry and Pharmaceutical Technology at the University of Calabria, Italy in the year 2012. He is currently working on Parkinson's disease and investigating the role or a protein called HMGB1 in the pathophysiology of the disease. During his PhD Matteo Santoro has received four different prizes for best PhD student poster presentation and talks at different conferences within and outside the University of Aberdeen. He successfully co-authored two peer reviewed research articles on Parkinson's disease. The latest publication has seen him the main contributor of the study. He is presently a PhD student on the behavioral characterization of three different MPTP mouse models of Parkinson's disease and investigating the role of acquired and innate immune system in Parkinson's disease.

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Notes:

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Grape skin powder mediates mitochondria function by autophagy activation and exhibits potential protective benefit in a *Drosophila* Parkinson's Disease Model

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Recent studies suggest that moderate red wine consumption may confer several health benefits: longer lifespan, protection against heart diseases, certain cancers and age-related neurological diseases. These health benefits are believed to come from a compound called resveratrol. Here we investigate the potential effect of grape skin from pure merlot on Parkinson's disease by incorporating grape skin powder into the daily food intake of *Drosophila melanogaster* with *PINK1* loss-of-function. The benefits of consuming this grape skin powder have featured not only the improvement of indirect flight muscle functions, as shown in the rescue of abnormal wing posture in *PINK1* mutant flies, but also prolonged the lifespan. The effect on WT flies' life span is not significant. Mitochondrial dysfunction is linked to the pathogenesis of Parkinson's disease, in particular, *PINK1* has been suggested to interact with mitochondrial fusion/fission machinery and the autophagy pathway. To underscore the beneficial qualities of the grape skin, we further showed that consumption of the grape skin powder demonstrated a rescue of mitochondria aggregation phenotype in the muscle of *PINK1* mutants. Moreover; results from western blots exhibited significantly elevated levels of LC3-II in the muscles of grape powder fed flies, indicating increased mitochondria autophagy. This effect is more obvious in flies fed with grape skin than the pure resveratrol compound. In addition, mutant flies appeared to be more sensitive than wild type flies. Our study suggested grape skin powder can induce autophagy activation, mediate the mitochondria function, and has potential protective benefit in a Parkinson's disease model.

Biography

Alan Wu is an intern student working in Dr. Bingwei Lu's lab in Stanford University School of Medicine. He is currently a Junior in the Crystal Springs Uplands High School. This study is done with the instruction and guidance from his mentor, Dr. Zhihao Wu, who is a post-doc in Dr. Lu's lab.

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Disorder of movement preparation in schizophrenia

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The beforehand preparation of movement is a highly important process, which increases the efficiency of movement execution. The disorder of movement preparation in schizophrenia patients was previously detected only with the help of electroencephalographic parameters, but not with the assessing of movement speed used in behavioral studies. It was recently found that appropriate preparation not only speeds up the movement, but also increases movement stability, which is measured with the intra-individual reaction time variability. Some studies also revealed that assessing of movement stability could detect the dysfunction of schizophrenia better than classical behavioral parameters. Hence, the main goal of this study was to verify if the parameter of movement stability could detect the impairment of movement preparation in schizophrenia patients. In order to achieve the main purpose, we carried out a study with 14 schizophrenia patients and 14 control group subjects. We used precueing task in our research, in which participants had to employ information about movement probability for its proper preparation. The main results showed that the impairment of movement preparation in schizophrenia patients was detected only with the movement stability measurement, although the movement speed failed to do so. Therefore, it was found that movement stability parameter has an appropriate sensitivity for detecting impaired movement preparation and could be employed in clinical studies.

Biography

Denisas Dankinas is a PhD student of Neurobiology and Biophysics Department of Vilnius University, Vilnius, Lithuania. He had participated in the number of international scientific conferences and had published 3 proceedings and 2 in a peer reviewed journal. At this moment he has 1 paper under review in reputed journal and one paper is also prepared for the publishing.

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Role of Therapeutic Touch in the management of responsive behavior in patients with dementia.

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Introduction: There was an estimated 36.5 million individuals worldwide living with dementia in 2010 as per World Health Organization. In addition to deficits in cognitive domains, responsive behaviours in dementia (RBD) greatly impact the quality-of-life of individuals with dementia and greatly increase caregiver burden. Current treatment modalities are not always effective, and thus non-pharmacological approaches along with careful use of pharmacological therapies should be considered in the management of RBD. Therapeutic Touch (TT) is a simple procedure that only requires a pair of hands and a compassionate mind. TT allows for clear and respectful communication with the patient and helps to avoid confrontation by providing stimulation and structure.

Objective: To review the publications that evaluate the use of TT in the management of RBD.

Methods: We searched PubMed for 'Therapeutic Touch' and "Dementia'. We limited our inclusion to reviews and studies published in the last 10 years. We excluded articles in languages other than English and studies for which no outcomes were reported.

Results: Four of the five examined studies suggest that Therapeutic Touch reduces restless behaviours found in dementia. However, there are limitations to these studies including methodological variability and small sample sizes.

Conclusions: TT is garnering attention for its potential role in ameliorating RBD in patients suffering from different stages of dementia and many are looking into using TT in palliative care settings. It can be used in inpatient and outpatient settings. However, at this time, there is insufficient data and further studies need to be done before definite conclusions can be drawn.

Biography

Helen Senderovich is a physician at Baycrest Health Science System with practice focused on Palliative Care, Pain Medicine and Geriatrics. She is an Assistant professor at the Department of Family and Community Medicine, and Division of Palliative Care at the University of Toronto who actively involved teaching medical students and residents. She has a broad international experience and a solid research background. Her research was accepted nationally and internationally. She is an author of multiple manuscripts focused on geriatrics, patient's centered care, ethical and legal aspect of doctor patient relationship, palliative and end-of-life care.

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Radek Vodicka et al., J Alzheimers Dis Parkinsonism 2016, 6:6(Suppl)
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Next generation sequencing data analysis evaluation in patients with Parkinsonism from genetically isolated population

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Parkinson's disease (PD) can be caused by genetic changes in a lot of genes. The effect of these changes is determined by the nature of the mutation and ranges from weak associations to pathogenic mutation which leads to loss of protein function. Our study is based on epidemiological data which show significantly increased prevalence of PD (2.9%) in an isolated population of south-eastern Moravia in the Czech Republic. We compared two different Next Generation Sequencing (NGS) data analysis approaches in DNA from 28 PD patients in the genes responsible for Parkinsonism (*ADH1C, ATP13A2, EIF4G1, FBXO7, GBA + GBAP1, GIGYF2, HTRA2, LRRK2, MAPT, PARK2, PARK7, PINK1, PLA2G6, SNCA, UCHL1* and *VPS35*) using: 1) Already described missense rare variants or pathogenic mutations and 2) Twelve control DNA samples from the same isolated population. Ion Torrent NGS data processing and trimming from Fastaq through "bam" to "vcf" files was done parallel by Torrent Suite/Ion Reporter and NextGene software. Variants were than filtered using following parameters: AQ>20; Read coverage >20; MAF<0,01; SIFT: 0 - 0,05 and/or PolyPhen-2: 0,2 -1. After filtering out, three missense mutations were found in *LRRK2* gene: rs33995883 in 6/0 patients/control (p/c), rs33958906 in 1/1p/c, rs781737269 in 3/0p/c, one missense mutation in *MAPT* gene rs63750072 in 6/1p/c and one mutation in *HTRA2* gene rs72470545 in 3/1p/c. Both the results from NextGene with Ion Torrent adaptation and from Ion Reporter significantly correlated in variant calling. Our study may contribute to further explanation of genetic background of Parkinsonism.

Biography

Radek Vodicka obtained his degree in Biology from the Faculty of Science, Masaryk University, Brno, Czech Republic, specializing in Genetics and Molecular Biology. He joined the DNA laboratory of the Department of Medical Genetics and Foetal Medicine, the University Hospital, Olomouc, where he obtained a post-graduate qualification in the Laboratory Method of Medical Genetics (2002). He defended his PhD thesis 'Y-chromosomal sequences in Turner syndrome patients' in 2003 and habilitated in 2013. His main research and clinical interest is a quantitative analysis of Human DNA sequences in relation to genetic diseases, infertility and cancerogensis.

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Junxia Xie et al., J Alzheimers Dis Parkinsonism 2016, 6:6(Suppl)
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Differences in vulnerability of neurons and astrocytes to hemeoxygenase-1 modulation: Implications for mitochondrial ferritin

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Induction of the antioxidant enzyme hemeoxygenase-1 (HO-1) was observed in both astrocytes and neurons in the substantianigra of patients with Parkinson's disease (PD). In the current study, we investigated whether HO-1 behaves differently between neurons and astrocytes under the condition of neurotoxicity related to PD. The results showed a time-dependent HO-1 upregulation in primary cultured ventral mesencephalon (VM) neurons and astrocytes treated with the mitochondria complex I inhibitor 1-methyl-4-phenylpyridinium (MPP+) or recombinant α-synuclein. However, HO-1 upregulation appeared much later in neurons than in astrocytes. The HO-1 inhibitor zinc protoporphyrin (ZnPP) aggravated MPP+- or α-synuclein-induced oxidative damage in both astrocytes and neurons, indicating that this HO-1 response was cytoprotective. For neurons, the HO-1 activator cobalt protoporphyrin IX (CoPPIX) exerted protective effects against MPP+ or α-synuclein during moderate HO-1 upregulation, but it aggravated damage at the peak of the HO-1 response. For astrocytes, CoPPIX always showed protective effects. Higher basal and CoPPIX-induced mitochondrial ferritin (MtFt) levels were detected in astrocytes. Lentivirus-mediated MtFt overexpression rescued the neuronal damage induced by CoPPIX, indicating that a large MtFt buffering capacity contributes to pronounced HO-1 tolerance in astrocytes. Such findings suggest that astrocyte-targeted HO-1 interventions have potential as a novel pharmacological treatment strategy in PD.

Biography

Junxia Xie is Taishan scholar Distinguished Professor. She is currently the Director of Shandong Provincial Collaborative Innovation Center for Neurodegenerative Disorders and Excellent Innovative Team of Shandong Province. She is the Vice-President of Qingdao University, Chinese Association of Physiological Sciences as well as Chinese Neuroscience Society. She has 7 projects from the NSFC, including 2 key projects and participates in National 973 Project. She has published 150 articles up to now.

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Bioinorganic Implications and Strategies in Parkinson's Disease

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Brain metal compositions change with aging and with the progression of neurodegenerative diseases. Under oxidatively stressed conditions, redox metals can promote the generation of free radicals. Alpha-synuclein (αS), the structurally dynamic protein implicated in Parkinson's disease (PD) pathology, has shown variable changes in its native biophysical properties in the presence of redox metals. Such metal promoted αS misfolding and/or post-translational modifications can have detrimental effects on normal function. The presented research will highlight metal-induced changes in the folding pattern of αS and the distinguishable differences observed between various metal oxidation states. The aggregation propensity of αS has been examined by circular dichroism analysis, dynamic light scattering, FT-IR, and thioflavin T fluorescence assays, as well as through immunoblotting techniques. Another avenue of this research capitalizes on these innate bioinorganic characteristics to target the dissociation of aggregation prone αS. Macrocyclic metallospecies are being utilized to proteolytically cleave αS β-sheet fibrils. Thus, the same characteristics that can make aberrant metal-promoted chemistry detrimental, have also been utilized to affect αS aggregates, as are found in PD Lewy bodies. Through this work, new therapeutic strategies for the treatment of PD and related disorders may become apparent.

Biography

Heather R. Lucas obtained her Ph.D. in 2009 at the Johns Hopkins University and subsequently carried out research at Osaka University in Japan with the support of a fellowship through the Global Center of Excellence. She was additionally a Lenfant Biomedical Fellow at the National Heart, Lung and Blood Institute and an NIH postdoctoral fellow at the National Institute on Aging. Dr. Lucas then joined the faculty within the Virginia Commonwealth University Department of Chemistry and Chemical Biology Program as an Assistant Professor for the Spring of 2014, where her research group encompasses inorganic biochemistry, biophysics, as well as inorganic and organic syntheses.

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Dystonia profile in a tertiary care hospital from southern India

Prabaharan Chellamuthu, Lakshmi Narasimhan R, Gobinathan S and Bhanu K
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Dystonia is a movement disorder characterized by sustained or intermittent muscle contractions causing abnormal, often repetitive, movements, postures, or both. The aim of the study is to present the profile of patients presenting with dystonia in a tertiary care center in southern India. The study period is from January, 2015 to June, 2016. Data of patients admitted in neurology ward or attending movement disorder clinic of Madras Medical College, Chennai, India were collected retrospectively and were analyzed. A total of 30 patients with dystonia were included in the study of which 43% (n=13) were males and the rest were females; 40% (n=12) patients had isolated dystonia while the remaining 60% (n=18) had combined dystonia syndromes. Based on the age of onset, 30% (n=9) patients had adolescent onset (13-20 yrs), 43% (n=13) had early adulthood onset (21-40 yrs) and 27% (n=8) had late adulthood onset (>40 yrs). Based on body distribution, 33% (n=10) patients had focal dystonia, 7% (n=2) had segmental, 47% (n=14) had multifocal dystonia, 7% (n=2) had hemidystonia and 7% (n=2) had generalized dystonia. Based on the etiology 47% (n=14) patients had degenerative cause [43% (n=13) cases had Wilson and 3% (n=1) had neuro acanthocytosis], 13% (n=4) patients had acquired cause [10% (n=3) cases were drug induced and 3% (n=1) due to hypoxic brain injury and in the remaining 40% (n=12) patients, no cause could be found. Based on the progression, 47% (n=14) patients had progressive disease and in the remaining patients the disease was static or recovered. Wilson's disease is by far the common degenerative disease presenting with dystonia in our center & cervical dystonia is the most common idiopathic cause.

Biography

Prabaharan Chellamuthu is currently doing his Neurology residency in Institute of Neurology, Madras Medical College, Chennai, India. He has done his under graduation from 2004-2010 in Stanley Medical college, Chennai. He has done MD Paediatrics from 2011-2014 at Lady Hardinge Medical College, New Delhi, India. He has received Dr. C.B. Rama Rao prize and N. Radhakrishna Iyer prize in Stanley Medical College. He has published paper on infantile spasms and presented posters in both national and international conferences. His areas of interest are paediatric epilepsies and movement disorders.

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Muhammad Faizan Sadaqat et al., J Alzheimers Dis Parkinsonism 2016, 6:6(Suppl)
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TREQUANT- Tremor quantifying wearable for movement disorder patients

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[2]CTO & cofounder of Trequant

Trequant is a bio-informatics wearable designed specifically for diagnosing and classifying tremor based movement disorders including Parkinson's, essential tremor, dystonia and others. Tremors affects 221 million people worldwide including 15 million from USA and it is one of the most prevalent and incurable neurological disorder. The complete end-to-end solution we are providing includes a wearable device, an app on a mobile device, and our data analytics platform in the cloud. Currently Trequant device is being used by more than 50 patients and 2 hospitals. With the results and data recorded from our device, doctors are better able to understand their patient's condition and with the help of our remote monitoring portal and they can see the effect of the prescribed medicine and can customize the type and dosage of the medicine according to every individual patient. Mobile app also helps patients in understanding what type of activities can make their tremors condition worse or good. At Trequant, we are all about having people that are afflicted with tremors live a more independent life and be more in control of their condition and actionable data is the answer to this.

Biography

Muhammad Faizan Sadaqat has completed his Electrical Computer Engineering from COMSATS Institute of Information Technology, Pakistan. He has built sound technical base by working on diverse projects related to bio-medical, automotive, big data analysis, extensive computation, electrical and electronics. In the field of MRI, he has earned name by accelerating MR image reconstruction time from minutes to msec. Implementation of MRI reconstruction algorithm on GPU is one of his biggest achievement. He has published research paper in reputed journal and presented in several prestigious conferences.

Usman Shabbir, the CTO & cofounder of Trequant, is an Electrical Engineer and Electronics Expert by profession. He has interest in wearable gadgets, embedded systems and miniaturization of circuits. A passionate person about robotics and artificial intelligence has done different projects on automation of things and robots. He is the hardware geek for Trequant and currently working on hardware design and engineering. He has won fifteen national and international awards presenting his work at different stages.

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Homozygosity mapping in an Iranian pedigree affected with early onset Parkinson's disease (EOPD) and linkage to chromosome 6

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Parkinson's disease (PD) is a common neurodegenerative disease. Its prevalence is estimated 2% among individuals older than 65 years. Both environmental and genetic factors contribute to the etiology of PD. Five (5) principal PD causing genes were identified: *α-synuclein* (PARK1), *LRRK2* (Leucine-rich repeat kinase 2; PARK8), *PRKN* (parkin; PARK2), *PINK1* (PTEN-induced putative kinase 1; PARK5), and *DJ-1* (PARK7). Mutations in these genes account for disease in a few percent of the patient, suggesting other PD causing genes remain to be identified. The combination of homozygosity mapping and exome sequencing is a powerful and efficient gene finding method applicable to recessive disorders in inbred populations, and the finding of new genes will enhance understandings of diseases pathogenesis. In our study, genome-wide single nucleotide polymorphisms (SNP) genotyping was carried out in an Iranian EOPD family using high density SNP chips. Two (2) affected siblings, 2 unaffected siblings and unaffected parents were genotyped. Homozygous regions common to all affected individuals and absent in non-affected individuals were sought. Disease status in the family is linked to a large homozygous region of 15Mb on chromosome 6. The linked region included 130 genes. Mutation screening of these genes is difficult and costly, so exome sequencing on 2 affected and 2 unaffected siblings was performed and candidate genes in the linked region were analyzed then. Age at onset of symptoms was in the second decade of life, and the mode of inheritance was autosomal recessive. Bradykinesia and rigidity, tremor, eye movement abnormalities including supranuclear gaze palsy, dystonia and bulbar anomalies were reported in 2 affected siblings.

Biography

Afagh Alavi received his BSc in Biology from Shahid Chamran University of Ahvaz, Iran (1998) and MSc and PhD in Molecular Biology from the University of Tehran, Iran (respectively, 2007 & 2013). He spent more than 15 years as a teacher of Biology (1998-2013), and then moved to University of Social Welfare and Rehabilitation Sciences, Tehran, Iran (2013). Since 2013, he serves as an Assistant Professor in the Genetics Research Center in this university. His main areas of research interest lie in the area of genetics and molecular analysis of neuromuscular and muscular disorders.

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Art therapy: The healing powers within the Parkinson's brain

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This paper tries to delineate novel thinking in the field of neuroscience that points to the fact that the brain is not just a static organ as once believed; but actually a thriving moldable, pliable neuroplastic puddy that given the right circumstances can re–learn old behavior and even learn new skills. This thinking is exemplified by as new scientific breakthrough in the field of neuroscience. The cerebellum was discovered to have a role in higher cortical function regulating thought and movement said Dr. Schmahmann, Professor of Neurology at Harvard medical School and Director of the Mass General Hospital Ataxia Unit and the Laboratory for Neuroanatomy and Cerebellar Neurobiology. Previously by "*de novo* artistic expression" in people with severe head injuries which has been also reported in Parkinson's patients which appears to be enhanced by dopamine replacement. Therefore it would not be inconceivable that these same patients when exposed to various forms of art therapy including painting, sculpting and the like, as well as artistic vocal expression singing in a choir for instance can also bring forth new motor functions or redevelopment of old functions. As Parkinson's patients are turning more and more to these types of therapies for the soothing, calming effect as well as the sense of having control over something tangible in their lives I wonder if there is more to doing art therapy. There is abundant literature regarding people with severe chronic illnesses like Van Gogh and other great artists like Salvador Dali and Horst Aschermann both believed to have suffered from PD in which these artists used art to help not only cope with their disease but also help them heal physically, mentally and spiritually. This form of healing has dated back to ancient times for the same purposes. However, there is a scarcity of literature delineating any motor or non-motor effects on the Parkinson's individual despite the many self-reports and accounts of increased artistic expression with PD. I have witnessed this phenomenon of improved motor skills first hand in my grandfather with dementia due to strokes and in my own Parkinson's patients; so I have set about to investigate further the relationship of art therapy and effect on the Parkinson's brain. I asked a number of patients who have been involved in art therapy and in choir (artistic voice therapy) to fill out a questionnaire to see if there was actual improvement of motor symptoms vs. only a sense of overall wellbeing from participation in these activities.

Biography

Maria L De Leon is Neurologist, who completed her Post-graduate Movement Disorder Fellowship Training at Baylor College of Medicine in 1999. Recently, she published her first book- "*Parkinson's Diva: A Woman's Guide to Parkinson's Disease*". Currently, she is working on her second book. She is a Consultant and frequent Guest Lecturer for the School of Social Work at Stephen F. Austin University and for the Muhammad Ali Center of Parkinson's, Hispanic Outreach Program Maria established "defeatparkinsons". She is also currently involved in the Hispanic Subcommittees of the upcoming World Parkinson Congress. She is former recipient of the Association of American University Women Award for her work in the field of medicine.

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Multi-muscle synergies: A sensitive tool for Parkinson's disease

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Postural instability is one of the cardinal signs of Parkinson's disease (PD). Quantifying postural stability is commonly used to measure PD severity. We hypothesized that postural synergy indices in the space of activation of muscle groups (M-modes) may be used to measure changes in motor coordination due to PD and dopamine-replacement therapy. Synergy indices stabilizing the center of pressure (COP) were compared between 11 patients without clinical symptoms of postural instability (Hoehn-Yahr stage-II) and 11 age-matched controls, and between 10 patients (stage II and III) tested off-drug and on-drug. Electromyographic signals from 13 leg and trunk muscles, recorded during cyclic body sway and releasing a load from extended arms, were used to quantify synergy indices by comparing the variance that had no effect on the COP coordinate and the variance that changed COP coordinate. Since this analysis needs multiple trials to identify the variance structure, we also quantified components of motion in the space of M-modes that had (non-motor equivalent) and did not have effect (motor equivalent) on COP coordinate using individual sway cycles. PD patients showed significantly lower synergy indices, and reduced ability to adjust these indices in preparation to an action. Motor equivalence analysis confirmed these differences. Impaired ability to adjust synergy indices in preparation to an action may contribute to rigidity and episodes of freezing. We conclude that analysis of motor equivalence in muscle activation space, using a few trials, can be used as a clinical measure for early diagnosis of PD and tracking disease progression.

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Identifying Park-weight phenotype in Parkinson's disease: Implications on disease management

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A number of phenotypes are being identified in the neurodegenerative Parkinson's disease, mostly in reference to the non-motor symptoms. One purpose of identifying phenotypes is to manage disease process more effectively. PD patients have variable impairment of olfaction; high proportions develop body-weight change (gain or loss) as the disease advances. PD patients have a lower body weight as compared to non-PD controls. Weight loss in PD is not a benign phenomenon. Lower initial body weight and weight loss during the course of the disease predispose to the risk of dyskinesia; there being a relationship between body weight and levodopa dose per kilogram for dyskinesia. Additionally weight loss increases the risk of under-nutrition, frailty, poor quality of life and mortality. Patients at the risk of weight loss may be identified by their severe olfactory loss (anosmia) at an early stage, since anosmia, as compared to hyposmia, seems to represent more severe neurodegenerative process predisposing to weight loss and dyskinesia describing the "olfaction-weight-dyskinesia" phenotype in Parkinson's disease. Weight loss is not due to higher energy expenditure or lower energy intake. The basis of severe neurodegenerative process and weight loss might be a longer pre-clinical phase in this phenotype. PD patients should be monitored for weight loss and the dose of levodopa adjusted accordingly as the disease advances. Measures should be taken to prevent weight loss in such patients to prevent the low body-weight related non-motor and motor adverse effects. This may result into better quality of life.

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The gait restorative effects of robotic-assisted gait training for multiple sclerosis, Parkinson's disease, and progressive supranuclear palsy

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Background: Neurodegenerative diseases and disorders present with a wide range of clinical and neuropathological symptoms caused by progressive neuronal dysfunction and eventual neuronal death. As individuals with neurodegenerative diseases experience gradual sensory, motor, and cognitive debilitation, the maintenance and recovery of a functional gait holds physiological, psychological and financial importance. Developments in robotically-aid therapies are becoming more commonly used as a therapeutic tool for the improvement of gait characteristics and overall motor function for individuals with various gait impairments. To date, studies examining the effects of robotic-assisted gait training (RAGT) as treatment for neurodegenerative diseases, have only been performed in individuals with multiple sclerosis (MS), Parkinson's disease (PD) and progressive supranuclear palsy (PSP).

Purpose: The purpose of this review is to summarize and show trends to the efficacy of RAGT as a gait restorative and preservative modality for individuals with these neurodegenerative diseases including MS, PD, and PSP.

Results: The overall trends reported by these reviewed studies show that RAGT may be an effective therapy for producing significant improvements in multiple gait characteristics including balance, walking speed, endurance, leg strength, gait safety, and motor function for individuals with neurodegenerative disease.

Conclusion: The studies in this review suggest that RAGT therapies may be an effective substitute for, or addition to, present conventional therapies for individuals with neurodegenerative disease, however the long-term effects of this therapy is still not known for these individuals.

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Glutathione deficiency as a cause and consequence of Parkinson's disease

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Parkinson's disease (PD) is one of several prevalent neurodegenerative diseases plaguing the aging population. To date, no biological therapies have been shown to slow, stop, or reverse PD progression; the disease is considered irreversible and progressive. The hypothesis that deficiency of reduced glutathione (GSH) contributes to PD degeneration was proposed over thirty years ago. Advances in neuroimaging and pharmaceutical science now permit quantification of brain GSH concentrations and novel methods of delivery, respectively. The goal of this lecture will be to present the data in evaluation of this hypothesis and identify gaps in knowledge. Post mortem brain from individuals with premotor PD shows a deficiency of GSH and it has been hypothesized that deficiency of GSH contributes to PD neurodegeneration. The role of GSH in the healthy brain will be described, and evidence of GSH deficiency in PD will be reviewed. The pros and cons of various augmentation strategies will be discussed, e.g. oral, intravenous and intranasal. All four clinical trials of GSH in PD have demonstrated a mild symptomatic improvement. In a cross-sectional analysis of 58 individuals with PD, low blood GSH was associated with greater disease severity. Taken together, these data support the hypothesis that GSH depletion contributes to PD and that intranasally-administered GSH has therapeutic potential as both a symptomatic treatment and a disease modification strategy.

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Art is medicine: Combating Parkinson's disease with creativity, positivity and movement

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People with Parkinson's disease (PD) benefit from engaging in activities that promote creativity, relaxation and positivity. The Art Cart's Smile Trough Art Workshop is specifically designed to help those with PD explore their creativity by targeting areas that are unique to this population. Our program includes modified equipment (easels, paint brushes, palates, etc.) which has shown to meet the needs of PD participants more successfully than traditional art equipment. For the workshops we design activities that will help this population combat the symptoms of PD such as tremors, rigidity of limbs, micrographia and loss of fine motor control. While our paint dries, we encourage our participants to follow along and participate in exercises that encourage strengthening of fine motor movement. After the culmination of each workshop, participants have the opportunity to provide their feedback regarding the impact the art workshop had on them through a survey. As anticipated, those with PD who participated in our Smile Through Art Workshop left with a heightened level of mood and an increased interest in exploring their creativity. Thus far, 12 workshops have been programmed for the PD population in Massachusetts, targeting a total of 26 participants. Nine (9) of the 12 workshops were offered through a continuous weekend program. Thus, it is believed that engaging in art and promoting a healthy environment is beneficial and leaves those with PD and their caregivers with a heightened quality of life.

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Nilotinib effects in Parkinson's disease and dementia with Lewy body

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Cancer and neurodegeneration include a group of diseases that are mechanistically distinct but may share common therapeutic targets. Autophagy is a common quality control mechanism shared by mitotic and post-mitotic cells and it can be exploited to accelerate clearance of unwanted oncogenes and reduce accumulation of toxic proteins in cancer and neurodegeneration respectively. Tyrosine kinase inhibition is a therapeutically relevant strategy that can induce autophagy. Our laboratory investigates TKIs that activate autophagy and are FDA-approved for cancer, thus significantly reducing research and development efforts and cost by re-purposing. In neurodegeneration, the non- receptor tyrosine kinase ABL is activated. Nilotinib and bosutinib are second generation BCR-ABL and SRC (short for Sacoma)-ABL inhibitors, respectively, that are therapeutically used for individuals with leukemia. A fraction of nilotinib and bosutinib crosses the blood-brain barrier (BBB), inhibits ABL and facilitates autophagic misfolded protein clearance, leading to neuroprotection and improved cognition and motor behavior. Mice treated with a much lower dose of these drugs (< 25% of the typical leukemia dose) show significant motor and cognitive improvement and degradation of misfolded proteins, leading to normal cell survival. We evaluated the effects of low doses of Nilotinib, on safety and pharmacokinetics in Parkinson's disease dementia or dementia with Lewy body. Twelve subjects were randomized into 150 mg (N=5) or 300 mg (N=7) groups and received oral daily doses of nilotinib for 24 weeks. The primary objectives were safety and tolerability; pharmacokinetics and target engagement were secondary, while clinical outcomes were exploratory. This study shows that 150 mg and 300 mg daily doses of nilotinib are safe and well tolerated in advanced Parkinson's disease. Nilotinib is detected in the CSF and seems to engage the target via Abl inhibition. Parkinson-related CSF biomarker, including homovanillic acid is significantly increased, DJ-1 is reduced and α-synuclein is stable between baseline and 24-week nilotinib treatment. Exploratory cell death biomarkers including neuron specific enolase and tau are also reduced. Motor and cognitive performance suggests stabilization of clinical outcomes. These data support the potential of TKIs in the treatment of PD.

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Pramipexole combined with the BDNF gene transfection to surviving dopamine neurons rescues dendritic spines and motor behavior in the rat model of Parkinson´s disease

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For a treatment to be successful in treating Parkinson´s disease, it should control the atrophy of the dendrites and loss of spines of the striatal MSNs. The dendritic abnormalities are not only due to a diminished dopamine delivery, but also to a reduced BDNF delivery because of the degeneration of the dopamine nigral neurons. Both dopamine D3 receptors and BDNF are required for the survival, protection and proliferation of dopamine nigral neurons, and apparently, they act synergistically. We have been studying the effects of long-term activation of dopamine D3 receptors combined with the BDNF gene transfection to dopamine neurons surviving the 6-OHDA-induced degeneration. Here, we studied the effect of the long-term administration of oral Pramipexole combined with the non-viral BDNF gene transfection to dopamine nigral neurons surviving the 6-OHDA-induced degeneration. The combined treatment rescued the dendritic spines of the MSNs and the dopamine nigral neurons, which was associated with the full recovery of motor behavior and normal muscle tone (muscular rigidity abolished). The recovery apparently was permanent because it persisted 3 months after the end of the treatment, which is consistent with the recovery of the dendritic spines of the striatal neurons. Thus, the treatment appears to be a promising disease modifying treatment for Parkinson´s disease.

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Estrogen affects iron metabolism in astroctyes and neurons

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Epidemiological studies have demonstrated that the postmenopausal women harbor a higher level of body iron than premenopausal women. Nigral iron accumulation is involved in the etiology of Parkinson's disease. Recent studies demonstrated that the women are on average 2.1 years older than the men at time of diagnosis. Moreover, medical conditions leading to estrogen depletion increase the risk of PD. The importance of estrogens and iron to physiology and disease has been known for decades, but we often overlook that these two factors interact. In this study, we investigated the effect of estrogen on the iron transport proteins as well as its mechanisms in midbrain. The results were as follows: Iron exporter ferroportin1 (FPN1) and iron importer divalent metal transporter 1 (DMT1) were up-regulated after estrogen was treated in primary cultured astrocytes, while hypoxia inducible factor-1alpha (HIF-1α) was up-regulated, but hypoxia inducible factor 2 alpha (HIF-2α) remained unchanged. In neurons, DMT1 was decreased but FPN1 was up-regulated after estrogen was treated. IRP1 was down-regulated while HIF-1α and HIF-2α remained unchanged after estrogen was treated in primary cultured neurons. The results suggest that the regulations for iron metabolisms of estrogen on astroctyes and neurons are different. Estrogen can increase FPN1 and DMT1 expressions by elevating HIF-1α in astrocytes. However, the decreased expression of IRP1 may account for the decreased DMT1 and increased FPN1expressions in neurons.

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Narrative therapy, visualization, and brain neurons in Parkinson's disease

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Narrative therapy (story telling) and expressive poetry (sensory words) can be used to engage the mirror neurons and motor neurons in the brain and decrease the symptoms in Parkinson's disease. The kind of stories we listen to and whether we identify with the narrator or not influence what parts of our brain "lights up," get more blood flow, more nutrients, and more stimulation causing it to better develop or heal. This means the kind of stories we tell in our families and communities, the kind of speakers we hear, the kind of music we listen to influences the ability of our cortex to function. Story telling provides another doorway to greater brain health. Mirror neurons cause us to feel the actions of others in our own body. Motor neurons can be engaged through seeing another person move or through guided visualizations. An engaging story about someone walking is a brisk and balanced way and can create an image in the mind of someone with Parkinson's disease. That image is then translated into a subtle contracting of the muscles needed to walk in that particular way. This stimulates the brain and nerve pathways to the muscles that are needed in order to do these actions. Research has also shown that injured athletes who visualize themselves doing their sport come back to the game with better skills than an athlete who doesn't do any visualization.

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Development of preclinical diagnostics of Parkinson's disease - strategy and recent progress

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At Parkinson's disease (PD) motor symptoms first appear many years after the onset of degeneration of nigrostriatal dopaminergic neurons that explains low efficiency of treatment. Therefore the development of preclinical diagnostics based on a search for biomarkers mainly as a change in the composition of plasma and expression of specific genes and phenotype of blood cells in drug-naive patients at the early clinical stage is of a high priority. Still, there is no guarantee that biomarkers, found at clinical stage are also a characteristic of preclinical stage. Therefore, we searched for biomarkers in experimental models of the earlier clinical and preclinical stages of PD (MPTP-treated mice) in addition to drug-naive patients shortly after the appearance of motor symptoms. According to our data, the concentration of some markers in plasma, e.g., L-DOPA, were modified in the same way in mice at the presymptomatic and symptomatic stages of Parkinsonism and patients. The concentration of others, e.g., DOPAC differed at the presymptomatic stage in mice from those in mice at the symptomatic stage and patients. Apparently, the former is more reliable than the latter. Moreover, we have developed at experimental models a novel complementary approach to the preclinical diagnosis of PD by using a pharmacological provocation test, which induces short-term increase of a failure of the nigrostriatal system and motor dysfunctions. Peripheral biomarkers and a positive provocation test, found at prophylactic examination of humans would allow including them in a risk group for the final diagnosis with positron emission tomography.

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Dystonia-Parkinsonism: The overlap

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Several movement disorders overlap with one another as part of the course of the syndrome or illness. The discourse attempts to help the clinician explore disorders with such overlap of dystonia and Parkinsonism and offer a way to enumerate clinical features in the context of Parkinson's disease (PD) and its therapy-related disorders. In the field of dystonia, the overlap is recognized in the classification of heredo-degenerative dystonia–Parkinsonism syndromes. The hereditary dystonia-plus (Parkinsonism) syndromes (with a special account on 'lubag'), neurochemical disorders and other characterized movement disorders in a mix with dystonia–parkinsonism will also be included in the discourse.

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