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Parkinson disease | Movement Disorders | Phytopharmaceuticals for Parkinson's disease

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Session Introduction

Title: **Muscle alterations in a Parkinson's disease animal model**

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Sagittal plane displacements of the cervico-thoracic region and their relationship to pain, disability, athletic performance and neurophysiological measures: Implications towards patients with movement disorders

Deed E Harrison
Chiropractic BioPhysics®, United States

Background: High quality case control and randomized clinical trials (RCTs) evaluating sagittal plane alignment rehabilitation methods and their correlation to improved disability, performance, and neurophysiological measures are lacking. Given the significant posture abnormalities in patients diagnosed with movement disorders (Parkinson's, Tourette's, etc.), it is prudent to conduct and review such studies as they may provide insights and innovations into non-pharmacological treatment for these and other patient populations.

Methods: Narrative literature review and discussion of our teams' recent publications. We present three case-control investigations looking at the relationship between forward head posture (FHP) magnitude relative to: 1) sensorimotor control variables and autonomic nervous system (ANS) function; 2) sensorimotor integration variables; and 3) athletic performance measurements. Additionally, we present 5 RCTs on the effect of sagittal spine correction on a variety of outcomes including: pain, disability, sensorimotor control, and neurophysiology.

Results: The case control investigations identified statistically significant differences between the FHP groups and control groups for sensorimotor measured variables ($p < 0.001$); for ANS measures ($p < 0.001$); for athletic performance measurements ($p < 0.005$), and for sensorimotor integration measurements ($p < 0.005$). The RCTs with long-term follow-up identified that patients receiving sagittal spine correction towards normal alignment improved statistically and clinically more in the following outcomes: pain and disability; sensorimotor control; ANS; and somatosensory evoked potentials and sensorimotor integration; all variables ($p < 0.005$).

Conclusions: Participants with FHP, abnormal thoracic and cervical sagittal curvature exhibited abnormal sensorimotor control, ANS dysfunction and athletic performance compared to those with normal posture alignment. Correction of sagittal displacements results in improved pain, disability, function, and neurophysiology. The implications of these findings relative to specific patients with Parkinson's disease and Tourette's syndrome are discussed.

Recent publications:

Ibrahim M Moustafa, Aliaa A Diab, Fatma Hegazy, Deed E Harrison(2021).Demonstration of central conduction time and neuroplastic changes after cervical lordosis rehabilitation in asymptomatic subjects: a randomized, placebo-controlled trial Nature communications doi: 10.1038/s41598-021-94548-z.

Biography

Deed E. Harrison, D.C., graduated from Life-West Chiropractic College in 1996. He has developed and researched original spinal rehabilitation procedures and has lectured at nearly 1000 educational conferences around the world. He has authored approximately 200 peer-reviewed spine related publications, 7 spine textbooks, and numerous conference proceedings. He is a highly respected chiropractic researcher and authority in today's profession. He is also a manuscript reviewer for approximately 20-different peer-reviewed spine and rehabilitative journals. Additionally, Harrison is a former member of the International Society for the Study of the Lumbar Spine (ISSLS), a former International Chiropractors Association's (ICA) Nevada State Assembly Representative member, and is the Chair of the PCCRP Chiropractic Radiography Guidelines. He formerly held a position in the Chiropractic Physicians Board of Nevada. Currently, Harrison is the President / CEO of Chiropractic BioPhysics® (CBP®) Technique & Seminars and is the President of CBP Non-Profit, Inc. – a spinal research foundation.

drdeedharrison@gmail.com

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Pilot study assessing the effect of fascial manipulation on fascial densifications and associated pain

Kena McDermott
University of Bridgeport, USA

Previous studies have demonstrated a connection between pain reduction and improved function after treatments targeting the heavily innervated fascial network. This study analyzed changes in fascial thickness at points identified by participants to be painful following a brief treatment consistent with the mechanical treatment associated with Fascial Manipulation (FM). Pathological fascial densifications within various somatic regions were self-identified by participants as painful prior to confirmation and measurement of fascial thickness utilizing diagnostic ultrasound. Densifications are defined as palpable thickenings of the loose connective tissue that are thought to consist of polymerized hyaluronan (HA) between the organized layers of collagen fibers of the epimysial deep fascia. After identification of the densification and measurement of thickness, 30-45 seconds of mechanical treatment consisting of deep, oscillating manual pressure was applied directly over the identified densification. Measurement was immediately taken using diagnostic ultrasound. In addition perceived pain was recorded using a 15cm visual analog scale (VAS) immediately pre and post treatment. The study found that even brief treatment using mechanical methods of FM led to reduced fascial thickness and statistically significant positive correlation between densification thickness and pain ratings pre and post treatment in females. No statistically significant differences were found in densification thickness or pain ratings between males and females. This study also demonstrated that densifications are measurable using diagnostic ultrasound. The findings of this study suggest that even brief FM treatment is effective at reducing pain and reducing fascial thickness and that diagnostic ultrasound may be useful as a pre and post treatment diagnostic tool.

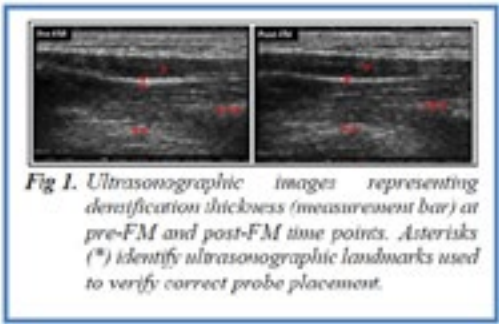

Fig 1. Ultrasonographic images representing densification thickness (measurement bar) at pre-FM and post-FM time points. Asterisks (*) identify ultrasonographic landmarks used to verify correct probe placement.

Recent Publications:

Hughes EJ, McDermott K, Funk MF. Evaluation of hyaluronan content in areas of densification compared to adjacent areas of fascia. J Bodyw Mov Ther. 2019 Apr;23(2):324-328. doi: 10.1016/j.jbmt.2019.01.017. Epub 2019 Feb 5. PMID: 31103115.

Morley J, Fan C, McDermott K, Fede C, Hughes E, Stecco, C (2019) The crural interosseous membrane re-visited: a histological and microscopic study. European journal of translational myology 29(3).

Hughes E, Koenig J, Lee R, McDermott K, Freilicher T, Pitcher M (2022) Pilot study assessing the effect of Fascial Manipulation on fascial densifications and associated pain. Eur J Transl Myol 32(1): 10369,2022, doi: 10.4081/ejtm..2022.10369.

Biography

Prior to obtaining her doctor of chiropractic degree and master's in clinical nutrition, Kena McDermott worked and taught histology within pathology laboratories. She also obtained clinical research experience working on breast cancer trials and currently works as a research associate for Yale School of Medicine. She is an active researcher with focus areas in chronic pain, genetic polymorphisms, nutrition and general chiropractic. She is an internationally, known certified fascial manipulation specialist and has a private practice in West Hartford, CT where she focuses on treating chronic pain and practicing functional nutrition.

mcdermottkena@gmail.com

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AUTOTAC is a novel Parkinson's disease therapeutic agent targeting alpha-synuclein aggregates to autophagic-lysosomal degradation

Yong Tae Kwon, Jihoon Lee and **Ki Woon Sung**
AUTOTAC Bio Inc., South Korea

Currently, there is no disease-modifying therapeutics for treating Parkinson's disease (PD). Though many efforts were undertaken to develop therapeutic approaches aiming to lead to transient symptomatic relief of PD, the presence of untreated α-synuclein aggregates stimulate recurrence of symptoms and even further progression of PD. Here, we propose a promising disease-modifying therapeutic agent targeting PD, Autophagy Targeting Chimera (AUTOTAC), which is comprised of its α-syn aggregate-binding ligand (TBL) linked to autophagy targeting ligand (ATL) that binds to ZZ domain of autophagy receptor p62/SQSTM1. This chemical platform provides a basis for targeting α-syn aggregates for autophagic-lysosomal degradation. We employed α-syn implicated PD experimental models using α-syn preformed fibrils (PFFs) in order to explicitly study α-syn aggregate-induced PD pathology. We witnessed that PD-AUTOTAC selectively targets α-syn aggregates through its TBL and sequestration mediated through p62 oligomerization, and this enables activation of downstream autophagic machinery for further lysosomal degradation in concentration-dependent manner (Fig. 1A). We also show that PD-AUTOTAC induces alleviation of synucleopathy-associated genotoxicity and mitotoxicity (Fig. 2A and B). The degradation of α-syn aggregates is expected to fundamentally suppress the progression of PD by attenuating α-syn aggregate-induced cytotoxicity, as the PO administration of PD-AUTOTAC into PFF-stereotaxic surgery mouse model mitigated progression of behavioral deficits (Fig 3A and B). As there has been little to no efforts in developing therapeutics degrading the fundamental causative agents of PD, PD-AUTOTAC will provide a distinct paradigm of therapeutic strategy for targeting PD.

A

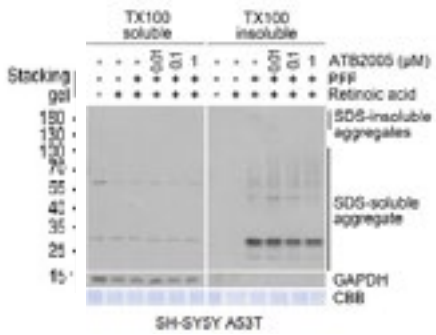
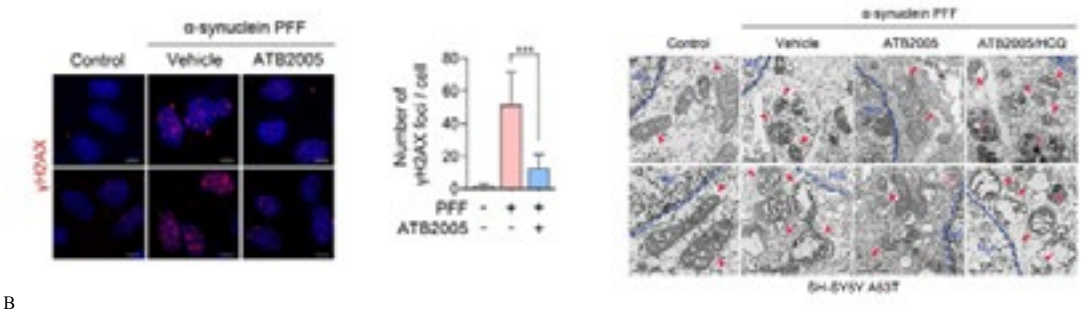


Figure 1: A, SH-SY5Y cells overexpressing A53T mutant α-syn were subjected to retinoic acid differentiation and subsequently transduced with α-syn PFFs for 48 h for α-syn aggregate generation. ATB2005 was then treated for 24 h, and the cell lysates were fractionated with triton x-100 for western blot analysis.



B

Figure 2: A, transmission electron microscopy analysis. α-Syn PFFs were transduced into SH-SY5Y A53T mutant cells, and the cells were administrated with PD-AUTOTAC (1 uM, 24 h) or HCQ (an autophagy inhibitor, 25 uM, 24 h). B, immunocytochemistry analysis and its quantification in HEK293A cells transduced with α-syn PFFs. PD-AUTOTAC was treated at 0.1 uM for 48 h (24 h * 2).

A B

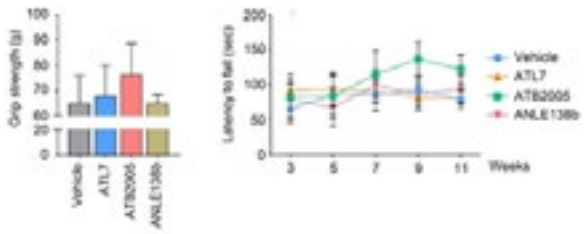


Figure 3: A, 6-week-old C57BL6/J male mice were subjected to stereotaxic surgery with α-syn PFFs into brain striata, and after 12 weeks, grip strength was measured. B, the mice were subjected to rotarod test

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1. Cha-Molstad, H., Yu, J. E., Feng, Z., Lee, S. H., Kim, J. G., Yang, P., Han, B., Sung, K. W., Yoo, Y. D., Hwang, J., McGuire, T., Shim, S. M., Song, H. D., Ganipisetti, S., Wang, N., Jang, J. M., Lee, M. J., Kim, S. J., Lee, K. H., Hong, J. T., … Kim, B. Y. (2017). p62/SQSTM1/Sequestosome-1 is an N-recognin of the N-end rule pathway which modulates autophagosome biogenesis. *Nature communications*, 8(1), 102. https://doi.org/10.1038/s41467-017-00085-7

2. Ji, C. H., Kim, H. Y., Lee, M. J., Heo, A. J., Park, D. Y., Lim, S., Shin, S., Yang, W. S., Jung, C. A., Kim, K. Y., Jeong, E. H., Park, S. H., Bin Kim, S., Lee, S. J., Na, J. E., Kang, J. I., Chi, H. M., Kim, H. T., Kim, Y. K., Kim, B. Y., … Kwon, Y. T. (2022). The AUTOTAC chemical biology platform for targeted protein degradation via the autophagy-lysosome system. *Nature communications*, 13(1), 904. https://doi.org/10.1038/s41467-022-28520-4

Biography

Jihoon Lee studied human biology and cell and molecular biology at the University of Toronto. After finishing his study in Canada, he moved to Korea and joined the research team in Cellular Degradation Biology Center, College of Medicine, Seoul National University. He is also co-affiliated with AUTOTAC Bio Inc., Seoul, South Korea, and has been participating as a researcher to study the utilization of AUTOTAC compounds in proteinopathies for targeted degradation. Collaborating with researchers at Seoul National University and research team members at AUTOTAC Bio Inc., he is now focusing his project on targeting α-syn aggregates as a therapeutic approach to treat Parkinson's disease.

jihoon.lee@mail.utoronto.ca

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The Q58L mutation of the androgen receptor (AR) gene as possible cause of early Kennedy disease

Marco Cassone
Hospital Major of Modica Nino Baglieri, Italy

Introduction: In this work a severe neuromuscular disease unexplained for years due to a missense mutation in the AR gene, found in an Italian male patient of 28 years with Sardinian origin. This case would broaden the genotype-phenotype correlation of the AR gene mutations, furthermore it would change the opinion that only expansion mutations can cause Kennedy's disease.

Materials and Methods: Detailed clinical history (table 1), whole exome and whole genome sequencing of proband and parents DNA. Skin biopsy in the proband and the unaffected father to obtain and isolate fibroblasts. Fibroblasts were cultured and used as model to discriminate cells with the mutation from those without mutation. To evaluate AR mRNA expression, total RNA was extracted and analyzed by Real Time RT-PCR. AR protein was detected by a specific antibody to study the immunolocalization in the patient fibroblasts and in control fibroblasts.

Results: Genome sequencing detected pathogenic mutations c.173 A>T, p.Q58L within AR gene, already described and associated with androgen insensitivity syndrome, which potentially explains the patient's clinical features. The expression analysis conducted using fibroblast from proband and father on total RNA extracts for Real Time RT PCR, shows a lower expression of the AR transcript in the patient's cells compared to the wild type father's cells, the same result was found by immunolocalization of the AR protein, in fact the proband's fibroblasts showed a very low presence of the AR protein compared with normal presence in the father's cells.

Conclusions: The association between phenotype and genotype was hypothesized on the basis of the symptomatology, which appeared to be a severe and earlier form of Kennedy's disease, on the basis of the mutation, which had already been described in literature and associated with androgen insensitivity syndrome and at the end on the basis of expression analysis..

Recent Publications:

Cassone M, Fiorillo C, Zara F, Vitali C. New phenotype caused by POMGNT2 mutations. BMJ Case Rep. 2021 Jul 22;14(7):e242358. doi: 10.1136/bcr-2021-242358. PMID: 34301702; PMCID: PMC8728375

Biography

Marco Cassone is a Clinical Geneticist who focused his studies on the molecular basis of rare diseases, especially neuromuscular disorder. Oncogenetics, prenatal diagnosis, dysmorphology, neurogenetics are the fields in which he spent most time and efforts. In the last years he assisted about 2000 new-borns after birth and performed hundred paediatric and genetics consultations. He is a clinical geneticist with an excellent experience in molecular biology; moreover he has a lot of skills regarding paediatric and neonatology. Currently his dream is to develop personalized medicine for clinical use. Currently he works in a public hospital in the south of Sicily and collaborates with an important genetic laboratory in America.

marcocassone@yahoo.it

Liliana Bernardino, J Neurol Clin Neurosci
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MicroRNA-based therapeutic approaches for Parkinson's disease

Liliana Bernardino
University of Beira Interior, Portugal

Parkinson's disease (PD) is the second most prevalent neurodegenerative disorder, and its incidence is rising, representing a substantial socioeconomic impact. The major neuropathological hallmarks are the degeneration of dopaminergic neurons in the nigrostriatal pathway and alpha-Synuclein inclusions known as Lewy bodies and Lewy neurites. Clinically, PD is defined by motor (e.g., rigidity, tremor, bradykinesia) and non-motor symptoms (e.g., depression, dementia). Although some treatments can reduce motor symptoms, no effective prevention and curative therapies have been developed yet. MicroRNAs (miR), small non-coding RNAs that post-transcriptionally regulate gene expression, are promising candidates for PD therapy, as they regulate several pathophysiological aspects of this multifactorial disease. miR have been used for stem cell-based therapies, inducing the differentiation into a specific cell phenotype, and as targets to modulate pathological mechanisms. However, effective clinical translation critically depends on developing efficient delivery systems. We first developed polymeric nanoparticles (NP) that release miR-124 to boost neurogenesis in the context of PD. miR-124 NP induced neurogenesis by targeting the stemness-related genes Sox9 and Jagged1 in vitro while increasing the number of new neurons in lesioned striatum and ameliorating motor symptoms in the 6-hydroxydopamine (6-OHDA) mouse model of PD in vivo. However, NP delivery has several challenges, such as degradation, bioaccumulation, retention in the basal lamina, and toxicity. Recently, we developed an innovative and novel delivery system based on small extracellular vesicles (sEV) as disease-targeted, biological delivery vectors for miR-124. In vitro, miR-124 sEV induced neurogenesis and protected N27 cells against 6-OHDA-induced toxicity. In vivo, although miR-124 sEV did not increase the number of new neurons in the lesioned striatum, our formulation protected dopaminergic neurons, which ameliorated motor symptoms. Thus, our findings support the therapeutic value of miR in the context of PD and the relevance of drug delivery systems to target distinct biological responses and enhance therapeutic effects.

Recent Publications:

1. Saraiva C, Talhada D, Rai A, Ferreira R, Ferreira L, Bernardino L, Ruscher K. MicroRNA-124-loaded nanoparticles increase survival and neuronal differentiation of neural stem cells in vitro but do not contribute to stroke outcome in vivo. PLoS One. 2018 Mar 1;13(3):e0193609. doi: 10.1371/journal.pone.0193609. PMID: 29494665; PMCID: PMC5832317.

2. Cláudia Saraiva, Marta Esteves, Liliana Bernardino, MicroRNA: Basic concepts and implications for regeneration and repair of neurodegenerative diseases, Biochemical Pharmacology, Volume 141, 2017, Pages 118-131, ISSN 0006-2952, https://doi.org/10.1016/j.bcp.2017.07.008.

3. Saraiva C, Ferreira L, Bernardino L. Traceable microRNA-124 loaded nanoparticles as a new promising therapeutic tool for Parkinson's disease. Neurogenesis (Austin). 2016 Nov 14;3(1):e1256855. doi: 10.1080/23262133.2016.1256855.

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5. Esteves M, Cristóvão AC, Saraiva T, Rocha SM, Baltazar G, Ferreira L, Bernardino L. Retinoic acid-loaded polymeric nanoparticles induce neuroprotection in a mouse model for Parkinson's disease. Front Aging Neurosci. 2015 Mar 6;7:20. doi: 10.3389/fnagi.2015.00020. PMID: 25798108; PMCID: PMC4351630

Biography

In 2003, Liliana Bernardino obtained her BSc in Biology and her PhD in Molecular Biology at the University of Coimbra, in collaboration with the Mario Negri Institute for Pharmacological Research, Milan, and the University of Southern Denmark, Denmark in 2008. During her career, she disclosed the effects of several molecules (e.g., histamine, retinoic acid, microRNA) on microglia activity and the impact on neuronal differentiation, function and survival. She also developed novel drug delivery systems, aiming to boost their therapeutic effects in the context of Parkinson's disease and ischemic stroke. Liliana Bernardino co-authored 58 publications, some in renowned journals (e.g., Nature Communications, Stem Cells, Journal of Neuroscience, Journal of Controlled Release, Journal of Neuroinflammation). Currently, she is an Assistant Professor with Habilitation at the University of Beira Interior. The main scientific interests of Liliana Bernardino's research group are to identify and develop novel brain repair therapies for Parkinson's disease.

libernardino@fcsaude.ubi.pt

Ana L Albarracín et al., J Neurol Clin Neurosci
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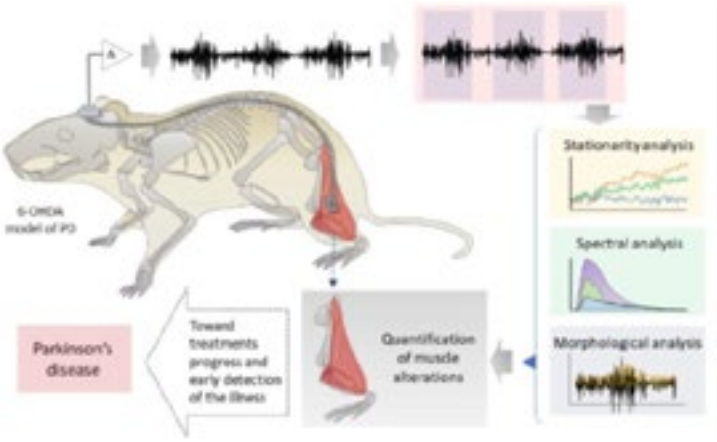
Muscle alterations in a Parkinson's disease animal model

Ana L Albarracín and **Fernando F Farfán**
CONICET, Argentina

The most common Parkinson's disease animal model induces massive nigrostriatal degeneration by intracerebral infusion of 6-hydroxydopamine (6-OHDA). Motor deficits in rat models of Parkinson's disease were previously addressed in other works. However, an accurate quantification of muscle function in freely moving PD-lesioned rats over time has not been described until now. In this work we address the muscular activity characterization of a 6-OHDA-lesion model of PD along six weeks post-lesion based on spectral and morphological analysis of the signals. Using chronic implanted EMG electrodes in a hindlimb muscle of freely moving rats, we have evaluated the effect of the PD neurotoxic model in the muscular activity during locomotion. EMG signals obtained from animals with different time post-injury were analyzed. Power spectral densities were characterized by the mean and median frequency and the EMG burst stationarity was previously verified for all animals. Our results show that as the time post-lesion increases both frequency parameters decrease. Probability distribution function analysis was also performed. The results suggest that contractile dynamics of the biceps femoris muscle change with time post-lesion. We have also demonstrated here the usefulness of frequency parameters as biomarkers for monitoring the muscular function changes that could be used for early detection of motor dysfunction.



Recent publications:

Ana L. Albarracín, Fernando D. Farfán, Muscle function alterations in a Parkinson's disease animal model: Electromyographic recordings dataset, Data in Brief, Volume 40, 2022, 107712, ISSN 2352-3409.

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Biography

Albarracín is specialized in neurophysiology. Her initial scientific research focused on the peripheral nervous system of the vibrissal system. She has extensive experience in extracellular electrophysiological and patch-clamp techniques in anesthetized and in live animals. Her most important contributions in the sensorial vibrissal system are related to neuronal coding in the vibrissal system, specifically in the texture discrimination code. However, her most recent research is carried out in the neurodegenerative diseases field; specifically she is working on motor deficits and biomechanical aspects in an animal model of Parkinson's disease. These studies include gait studies and analysis of muscular and cortical electrical activity in live animals in order to establish biomarkers related to the pathology.

aalbarracin@herrera.unt.edu.ar

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Assessment of the memory impairment among young and middle age COVID-19 convalescent persons

Y Hovhannisyan[1], S Symonyan[2], H Torosyan[2], K Yenkoyan[2]
[1]Heratsi N1 University Hospital Complex, Armenia
[2]YSMU, Armenia

Background: The coronavirus disease 2019 (COVID-19) pandemic emphasized the occurrence of neurological manifestations associated with SARS-CoV-2 infection and highlighted the question of the neuro-pathogenicity of coronaviruses. Studies have shown a direct influence of viral infections on cognition, especially in the development of MCI and dementia. (Damiano, R.F., 2022). It has been demonstrated that many people infected with the SARS-CoV-2 virus experience short- and/or long-term neuropsychiatric symptoms, including cognitive and attention deficits, known as "brain fog" (Marcia N., 2021). Additionally, probable short- and long-term COVID-19 impacts in cognition, even in asymptomatic individuals have been discussed which could be accounted for by direct and indirect pathways to brain dysfunction (Miners, S., 2020). Patients with SARS-CoV-2 infection appear to experience global cognitive impairment, impairment in memory, attention and executive function, and in particular verbal fluency (Daroissce,2021). Nevertheless, understanding of the correlation of SARS-CoV-2 virus with cognitive impairment remains unclear.

Aim: Our study aims to find out memory disturbances among young and middle-aged (20-65 years)

COVID-19 survivors (convalescent patients) and clarify the direct connection with the depression.

Materials and methods: On an outpatient basis, we plan to examine around 130 COVID-19 convalescent subjects, including females and males of different ages. Different age groups (20-65 y.o.), with and without subjective memory impairment will undergo several examinations, including general neurological examination, test for cognitive function (RBANS, MOCA), and depression (PHQ-9).

Hypothesis: We hypothesize that CIVID-19 convalescent subjects will demonstrate significant disturbances in their memory and are more correlated to existing depression. We also don't exclude different levels of impairment in all age and sex groups.

Conclusions: A wide range of neurological manifestations, including cognitive impairments, has been associated with SARS-CoV-2 infection. There is no definite evidence to support the direct correlation of SARS-CoV-2 with cognitive impairment and further research is necessary to confirm this correlation, including direct neuropathogenicity of SARS-CoV-2 infection.

Biography

Yekaterina Hovhannisyan, head of Neurology Service in Heratsi N1 University Hospital Complex,YSMU, Armenia, PhD-fellow at Neuroscience Laboratory, YSMU. After graduating Medical University and clinical residency program in clinical Neurology she made Stroke Fellowship at USC, Los Angeles, USA, "Primary stroke prevention" joint research program with Austria (Christian-Doppler Klinik, Paracelsus Medical University, Salzburg, Austria) and USA (New York Presbyterian Hospital/Weill Cornell Medical Center). Currently, she is mainly focused on Stroke, Dementia, Neurodegenerative Disorders in clinical practice.

katyaarmdoc@yahoo.com

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The over-expression of survivin could prevent the oxidative stress and toxicity of rotenone in SH-SY5Y cells

Arman Rahimmi
Kurdistan University of Medical Sciences, Iran

Background and aim: It is important to find novel therapeutic molecular targets for curing Parkinson's disease (PD). Accordingly, this study aimed to evaluate the effect of the over-expression of survivin gene, as a gene frequently reported as neuroprotective, on the in vitro model of PD.

Methods: Survivin was over-expressed in SH-SY5Y cells. Next, the cells were treated with rotenone (500 nM) for 24 hours. Then, the viability and the total antioxidant capacity were assessed. The expression levels of 15 important genes of key cellular processes (oxidative stress, apoptosis, cell cycle, and autophagy) were assessed. The studied genes included survivin, superoxide dismutase, catalase, BAX, bcl2, caspase 3, caspase 8, caspase 9, p53, SMAC, β-catenin, mTOR, AMPK, ATG7, RPS18. The apoptosis level and the frequency of cell cycle stages were assessed by flow-cytometry. For analyzing the data, ANOVA test followed by Tukey's test was used to evaluate the significant differences between the experimental groups. $P < 0.05$ was considered as significant.

Results: Survivin could significantly decrease the rotenone-induced apoptosis in SH-SY5Y cells. The rotenone treatment leaded to down-regulation of catalase and upregulation of bax, bcl2, caspase 3, caspase 8, P53, β-catenin, and ATG7. Survivin could significantly neutralize the effect of rotenone in most of the genes. Survivin could also increase the total antioxidant capacity of SH-SY5Y cells.

Conclusion: Survivin could prevent the toxic effect of rotenone on SH-SY5Y cells during the development of in vitro PD model via regulating the genes of key cellular processes, including anti-oxidation, apoptosis, cell cycle, and autophagy.

Keywords: Parkinson's disease, survivin, Apoptosis, Autophagy, Oxidative stress.

Recent Publications:

Arman Rahimmi, Sima Tozandehjani, Mona Daraei, The neuroprotective roles of Dietary Micronutrients on Parkinson's disease: a review May 2022, Molecular Biology Reports, DOI:10.1007/s11033-022-07345-w

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Arman Rahimmi Effect of lobeglitazone on motor function in rat model of Parkinson's disease with diabetes co-morbidity May 2021, Brain Research Bulletin 173:184-192.

Biography

Arman is Ph.D. of Molecular Medicine from Kurdistan University of Medical Sciences, Sanandaj, Iran. Arman does his researches exclusively in the field of neurodegenerative diseases, especially Parkinson's disease since 2012. He has performed several research projects about pathophysiology of PD, novel therapies, optimizing animal models of PD, etc. He is well-experienced in several cellular and molecular techniques, designing projects, and scientific writing. He is going to continue his scientific career as a postdoctoral researcher abroad.

a.rahimmi@yahoo.com

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The effects of transcranial direct current stimulation on gait in patients with Parkinson's disease: a systematic review

Hamzeh Baharlouei[1], Fateme Pol[1], Mohammad Ali Salehinejad[2] and **Michael A Nitsche[1]**
[1] Isfahan University of Medical Sciences, Iran
[2]Leibniz Research Centre for Working Environment and Human Factors, Germany

Background: Gait problems are an important symptom in Parkinson's disease (PD), a progressive neurodegenerative disease. Transcranial direct current stimulation (tDCS) is a neuro-modulatory intervention that can modulate cortical excitability of gait-related regions. Despite an increasing number of gait-related tDCS studies in PD, the efficacy of this technique for improving gait has not been systematically investigated yet. Here, we aimed to systematically explore the effects of tDCS on gait in PD, based on available experimental studies.

Methods: Using the PRISMA approach, PubMed, Web of Science, Scopus, and PEDro databases were searched for randomized clinical trials assessing the effect of tDCS on gait in patients with PD.

Results: 18 studies were included in this review. Overall, tDCS targeting the motor cortex and supplementary motor area bilaterally seems to be promising for gait rehabilitation in PD. Studies targeting the dorsolateral prefrontal cortex or cerebellum showed more heterogeneous results. More studies are needed to systematically compare the efficacy of different tDCS protocols, including protocols applying tDCS alone and/or in combination with conventional gait rehabilitation treatment in PD.

Conclusions: tDCS is a promising intervention to improve gait in PD. Anodal tDCS over motor areas showed a positive effect on gait, but stimulation of other areas was less promising. However, heterogeneities of methods and results make it difficult to draw firm conclusions and require systematic exploration of tDCS protocols to optimize efficacy.

Keywords: Transcranial direct current stimulation, gait, Parkinson's disease.

Recent Publications:

1. Pol, Fateme & Baharlouei, Hamzeh & Taheri, Alireza & Menz, Hylton & Forghany, Saeed. (2021). Foot and ankle biomechanics during walking in older adults: A systematic review and meta-analysis of observational studies. Gait & Posture. 89. 10.1016/j.gaitpost.2021.06.018.

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Biography

Hamzeh Baharlouei finished his PhD in Physiotherapy in 2020. His PhD project was about the effect of transcranial direct current stimulation (tDCS) on balance in older adults. He published some studies about the effect of tDCS on balance and gait in young adults, older adults and patients with Parkinson's disease. Beside working as a researcher, he teaches physiotherapy in neurological disease at Isfahan University of Medical Sciences since 2012. His research field is tDCS, older adults, balance, and gait.

hamzehbaharlouei@gmail.com

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The association between animal protein sources and risk of Parkinson's disease: a systematic review and dose-response meta-analysis

Hamed Mohammadi and **Sepide Talebi**
Tehran University of Medical Sciences, Iran

We aimed to investigate the associations between dietary intake of animal protein sources and the risk of developing Parkinson's disease (PD). These animal protein sources included total dairy, milk, yogurt, cheese, total meat, red meat, processed meat, poultry, fish, and egg. PubMed, Scopus, Web of Science, and Google Scholar were searched until October 2021. Prospective cohort study designs that investigated the association between dietary animal protein sources and PD risk were included. Relative risks (RR) were pooled using a random-effects model. In addition, a dose-response relationship was examined between dietary animal protein source intake and PD risk. The Grading of Recommendations Assessment, Development, and Evaluation (GRADE) was used to rate the certainty of the evidence. Eight prospective cohort studies were eligible. The risk for developing Parkinson's disease was significantly higher in those with the highest compared to the lowest intake categories of total dairy (RR: 1.49, 95% CI: 1.06, 2.10; n = 5) and milk (RR: 1.40, 95% CI: 1.13, 1.73; n = 6). A linear dose-response meta-analysis revealed that each additional 200g/d of total dairy consumption was associated with an 11% higher risk of PD (RR: 1.11, 95% CI: 1.02, 1.20; n = 4). There was evidence of departure from linearity between total dairy intake and risk of PD (P non-linearity= 0.31, P dose-response= 0.01; n = 6). Overall, a higher intake of dairy consumption is associated with an increased risk of Parkinson's disease. Future, well-designed prospective studies, incorporating well-controlled randomized controlled trials are needed to validate the present findings.

Recent Publications:

1.Nikoo Hossein-Khannazer, Gholamreza Azizi, Solat Eslami, Hussaini Alhassan Mohammed, Farimah Fayyaz, Ramin Hosseinzadeh, Abubakar B Usman, Ali N Kamali, Hamed Mohammadi, Farhad Jadidi-Niaragh, Emad Dehghanifard, Mohammad Noorisepehr(2020). The effects of cadmium exposure in the induction of inflammation

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Biography

Hamed Mohammadi has completed his PhD at the age of 28 from Isfahan University of Medical Sciences, Isfahan, Iran. He is currently an assistant professor at Tehran University of Medical Sciences, Tehran, Iran. He has published more than 100 papers in reputed journals and has been serving as an editorial board member of repute.

mohamadihd@gmail.com