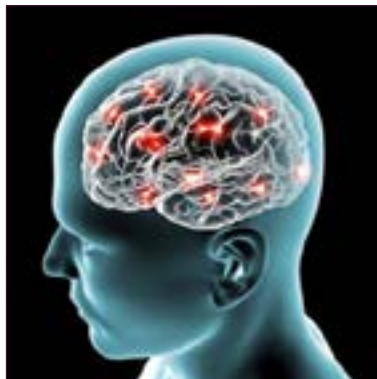

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DNA nanoprobe for real-time imaging and simultaneous quantification of mitochondrial Ca²⁺ and pH in neurons induced by superoxide anion and aggregated amyloid beta

Zhichao Liu and Yang Tian
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Mitochondria play vital roles in cellular energy production, signal transduction and Ca²⁺ homeostasis, as well as the cell death. Besides, mitochondrial pH and Ca²⁺ are closely associated with cellular functions and diseases. Thus, simultaneous imaging and biosensing are essential for understanding inter-relationship between Ca²⁺ and pH in physiological and pathological processes. Herein, we created a highly selective DNA nanoprobe for real-time imaging and simultaneous quantification of pH and Ca²⁺ in mitochondria, in which a new Ca²⁺ fluorescent probe was synthesized and assembled onto a DNA nanostructure together with pH-responsive, inner-reference, and mitochondria-targeted molecules. This new nanoprobe powerfully tracked pH and Ca²⁺ dynamics at the same localization in response to superoxide anion (O^{2•-})-induced oxidative stress and aggregated amyloid beta (Aβ) stimulation with a temporal resolution of milliseconds. Using this new tool, we discovered that acid-sensing ion channel 1a (ASIC1a) channel plays a vital role in O^{2•-} and Aβ-induced mitochondrial Ca²⁺ burst, which may contribute to neuron death. Moreover, psalmotoxin 1 (PcTX1) effectively protects against neuron injury, providing a potential drug for O^{2•-} and/or Aβ-induced neuronal death. Using the DNA-assembled nanosensor for determination of pH and Ca²⁺ at the same localization, we demonstrated that mitochondrial Ca²⁺ is increased ~4-fold in neurons compared with HeLa cells, whereas mitochondrial pH exhibits no obvious difference between the two types of cells. Furthermore, experimental results demonstrated diverse mitochondrial Ca²⁺ and pH values in different regions of neurons. The close relationship between Ca²⁺

and pH in mitochondria was discovered. Mitochondrial pH value in neurons obviously increased with increasing Ca²⁺ concentration, which may be attributed to the function of the Ca²⁺/H⁺ antiporter in mitochondria. On the other hand, the mitochondrial Ca²⁺ burst can be adjusted by the ASIC1a channel during cytoplasmic acidosis. O₂•⁻ induces transitory cytoplasmic acidosis, which may activate the ASIC1a channel in the mitochondrial membrane, resulting in alkalization and Ca²⁺ overload in mitochondria. Mitochondrial Ca²⁺ overload is possibly one of the important factors in O^{2•-}-induced neuronal death. These results offer a new view for understanding the signaling pathway of ROS-induced oxidative stress and neuron injury. Aggregated Aβ is highly toxic to neurons. After stimulation by Aβ₂₅₋₃₅, the pH value in the cytoplasm clearly decreased together with the Ca²⁺ burst, leading to acidification and Ca²⁺ overload in mitochondria through ASIC1a. PcTX1 protein protect neurons from death by preventing mitochondrial Ca²⁺ overload stimulated by O^{2•-} and aggregated Aβ, suggesting that PcTX1 is a potential drug for O^{2•-} and/or Aβ-induced neuronal death.

Speaker Biography

Zhichao Liu, PhD is a student of Analytical Chemistry under the supervision of Prof. Tian in East China Normal University. He received his MS degree in Analytical Chemistry from Nanchang University in 2015. His doctoral research now focuses on the design, synthesis, characterization, and application of fluorescent nanomaterial for sensing and imaging in biological applications.

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 Notes:

Neurorehabilitation of disordered sleep, chronic headache and processing speed after traumatic brain injury

Dafna Paltin, Yuri Danilov and Mitch Tyler
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Forty-four subjects were enrolled into a seven-month-long FDA, double-blind clinical trial designed to evaluate the efficiency of our unique, multi-modal approach to neurorehabilitation for chronic symptoms associated with mild-to-moderate traumatic brain injury (TBI). Our intervention combines the use of targeted training, breathing and awareness exercise, and non-invasive neurostimulation delivered transcutaneously through the tongue. While results are preliminary, effects of the intervention on disordered sleep, chronic headache, and processing speed are clear. At the time of enrollment, 36 participants had negatively impacted sleep quality, 37 experienced chronic headache or migraine, and 17 had below average processing speed. Between two weeks and three months of intervention the number of positive responders to treatment for sleep, headache, and processing speed was 22, 27, and 39 respectively. Already, we can see that the range of improvement includes several significant outcomes. These results are highly encouraging regarding the applicability of our therapy to rehabilitate several chronic conditions that often result from TBI. Disordered sleep, chronic headache,

and decreased cognitive processing speed are common and often untreatable manifestations of traumatic brain injury that can devastate an individual's quality of life. Our results demonstrate the recoverability of chronic symptoms beyond what was previously thought possible. These findings have important applications in the fields of applied neuroscience and rehabilitation.

Speaker Biography

Dafna Paltin was an Executive Member of her university's Neuroscience Honor Society (Nu Rho Psi) and a recipient of the Diamond Peer Scholar Award. She got her start in behavioral neuroscience research at the Center for Neural Decision Making (CNDM) in Philadelphia, where she assisted in the study of neuro-economics. After her independent research on neuroplasticity and sensory substitution captured the attention of scientific director, Dr. Danilov and then she was invited to join him in Madison at the Tactile Communications and Neurorehabilitation Laboratory (TCNL). There she performs all the neuropsychological and cognitive testing with clinical research participants. Her recent achievements include scientific publication in peer-reviewed journals as well as poster presentations at conferences. She will continue to seek out diverse opportunities that relate to her interests in Neuroscience and Psychology as she progresses towards her goal of enrolling into a graduate program for the study of Clinical Psychology.

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 Notes:

Coenzyme-Q10 deficiency and stress oxidative in children with autism spectrum diseases

Elham Mousavinejad
University of Medical Sciences, Iran


Assume that serum concentration of total CoQ¹⁰ and stress oxidative factors could be used as important biomarkers of therapy. One hundred and eighty children (aged 3-12 years), one group consisted of children with autism (n=90) and other consisted of health children (n=90). Children with autism according to the DSM-IV criteria and using CARS were included in the study. All the subjects were Iranian, born and living in the state Khuzestan. This was an original study. The present study aimed to analyze the serum levels of concentration of CoQ¹⁰-TOTAL, in the children. In total, patient group and health group, including boys and girls, were matched for age, gender, and body mass index (BMI). Serum levels of CoQ¹⁰-TOTAL in children with ASDs were significantly lower than that in the healthy children. We propose that serum concentration of CoQ¹⁰-TOTAL could be

used as relevant biomarkers of CoQ¹⁰ supportive therapy. Overall supplementation with Co-Q¹⁰ provides promising alternatives to current therapies for neurodevelopmental disorders. CoQ¹⁰ is a naturally occurring flavonoid with potent antioxidant, properties that are found in green plants. This study with a larger number of patients is confirmed these previous research.

Speaker Biography

Elham Mousavinejad has completed her MSc in the Department of Biochemistry, School of Medical Sciences, Ahvaz Jundishapur University of Medical Sciences, in 2016, and BSc Degree in Community Nutrition, School of Nutritional Sciences and Dietetics, Jundishapur University of Medical Sciences, Ahvaz, Iran in 2006. Her research area involved Nutritional Neuroscience and various nutritional deficiencies described in children with ASDs.

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 Notes:

Analysis of differentially expressed genes in iron-induced prion protein conversion

Hee-Jong Woo

Seoul National University, South Korea


The conversion of the cellular prion protein (PrPC) to the protease-resistant isoform is the key event in chronic neurodegenerative diseases, including transmissible spongiform encephalopathies (TSEs). Increased iron in prion-related disease has been observed due to the prion protein-ferritin complex. Additionally, the accumulation and conversion of recombinant PrP (rPrP) is specifically derived from Fe(III) but not Fe(II). Fe(III)-mediated PK-resistant PrP (PrPres) conversion occurs within a complex cellular environment rather than via direct contact between rPrP and Fe(III). In this study, differentially expressed genes correlated with prion degeneration by Fe(III) were identified using Affymetrix microarrays. Following Fe(III) treatment, 97 genes were differentially expressed, including 85 upregulated genes and 12 downregulated genes (≥ 1.5 -fold change in expression). However, Fe(II) treatment produced moderate alterations in gene expression without inducing dramatic alterations in gene expression profiles. Moreover, functional

grouping of identified genes indicated that the differentially regulated genes were highly associated with cell growth, cell maintenance, and intra- and extracellular transport. These findings showed that Fe(III) may influence the expression of genes involved in PrP folding by redox mechanisms. The identification of genes with altered expression patterns in neural cells may provide insights into PrP conversion mechanisms during the development and progression of prion-related diseases.

Speaker Biography

Hee-Jong Woo Immunology VMD, Ph.D professor of faculty of veterinary medicine, Seoul National University since 1992. Has completed his Ph.D of Immunology at the University of Tokyo, Japan in 1987. His postdoctoral training was at the Division of molecular diseases, Department of Pediatrics, School of medicine, University of Pennsylvania, and was an instructor of Laboratory of cancer biology, Department of surgery, School of Medicine at the Harvard University. He provides presentations on topics related to the neurodegenerative diseases in brain. Expertise in all immunological field and special interest in prion biology and the inflammation of brain.

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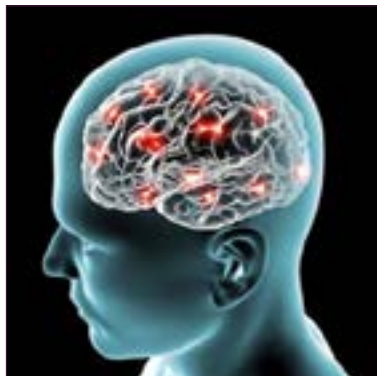
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Evaluation of chronic tramadol administration on memory of rat

Leila Kanaani
Azad University, Iran


The impairment of memory functions is very common in patients with chronic pain, particularly in patients with existing cognitive disorders. In this study, we investigated the effects of chronic exposure of tramadol, which could impair the memory evaluated in the ORT. For this purpose, this study was carried out on 15 (3×5 group) male Wistar rats (weighing 220±20 g). The animals had free access to food and water before the experiment. They were kept at a constant room temperature (22±1C) under a 12-12 h light/dark cycle, while using an apparatus consisting of a circular arena, Then, TRM was dissolved freshly in distilled water. The animal was received gavage 50 mg/kg daily for 30 days according to the respective chronic treatment groups. Each respective control group took distilled water in the same manner. The administration of drug was done between 8-22 am every day. To check the memory in the scheme of Task Recognition Object, a test was employed to detect objects based on the animal's natural desire to explore new object in front of a familiar object. The results showed that the physiological function of GABA and inhibitory effect

of ACh release of TRM in cholinergic activity can indicate some negative behavioral effects of TRM. In summary, our research confirms that the low doses in chronic exposure of tramadol could impair the memory evaluated in the ORT. The agonistic property of Tramadol for GABA receptor, disruption of normal GABA physiological function and the inhibitory effect of Tramadol on the ACh release and the cholinergic activity could be supposed as some possible mechanism of negative behavioral effect of Tramadol. However, more molecular studies are needed for the declaration of the exact mechanism of the nervous system in the future.

Speaker Biography

Leila Kanaani has completed her MSc of Medical Toxicology at Azad University, Shahreza, Iran in 2016. Her specialist training involved study, research and teaching of Nutrition, Diet Therapy and Medical Toxicology. She has authored numerous public international and national works and provides presentations on topics related to the Nanodrug Delivery. Her expertise is in Medical Toxicology field and special interest in Nanodrug Delivery. She has experience of work in the Nutrition and Diet Therapy in Iran.

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 Notes:

The frequency and patterns of Nabilone prescribing and administration among hospitalized oncology patients

Sardar Alam¹, Muhammad Safdar¹, Muhammad Tariq², Abdul Zahir³ and Waqar Alam⁴

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Background: Of all the side effects of chemotherapy, CINV remains one of the most dreaded by patients. Evidence based guidelines recommend nabilone for prevention of anticipatory chemotherapy-induced nausea/vomiting (ACINV) but not for rescue nausea/vomiting (RNV). Nabilone may increase the risk for over sedation and falls. The purpose of the study was to characterize the frequency and patterns of Nabilone prescribing and administration as well as to compare the rate of falls in patients prescribed vs non-prescribed nabilone for nausea/ vomiting among hospitalized oncology patients.

Methods: A retrospective study was conducted by reviewing the medical charts of 300 oncology patients admitted in months of June-August 2016 in tertiary care hospitals in KPK. Prescribed indications and actual administration of Nabilone as well as documented patient falls were recorded. Potentially inappropriate prescriptions were defined as frequency <8 hours, dose >2mg, multiple concurrent as needed prescriptions. Inappropriate administrations were not given in the prescribed 1st/2nd/3rd line sequence. Nabilone prescriptions for neuropathic analgesia were excluded.

Result: Out of 300 patients, 61% (n=183) patients with mean


age 51±19 years were prescribed nabilone. The length of stay was 14±11 (p-value=0.0001). The results showed that Nabilone was prescribed for RNV was 49% (n=91) while for ACINV, it was only 21% (n=38). Inappropriate dosing frequency was 9%, concurrent prescriptions were 17% and inappropriate administration was 19% (n=53) patients. Interestingly fall rate in nabilone prescribed patient's p value was 0.7 and among non-prescribed patient's p value was 1.5.

Conclusion: Potentially inappropriate prescribing and administrations of Nabilone for rescue nausea/vomiting were common. Estimated fall rate was not significantly different between patients prescribed and not prescribed Nabilone in this small pilot study. Informed consent was obtained from all patients..

Speaker Biography

Sardar Alam, Pharm.D, now a pre-doctoral student in the school of medicine, University of Crete, Greece. He is a registered pharmacist. His research interest includes oncology, CAM therapies and drug delivery systems. His research articles and reviews have been published in international peer-reviewed journals as well as in conferences. His research expertise includes prescriptions interventions, dose adjustment. He has experience in hospital as well as pharmaceutical industry where he works as a pharmacist as well as R & D officer respectively.

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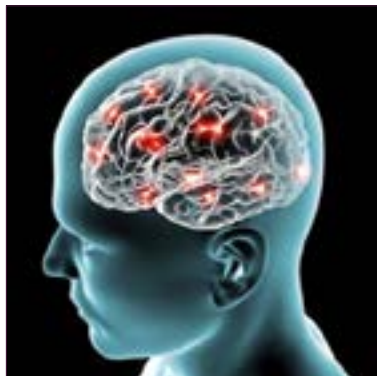
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Polyfunctional chromen-4-ones based anti-Alzheimer's agents: Design, synthesis and biological evaluation

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Chromen-4-ones has emerged as a 'master key' due to its presence in wide range of activities related to Alzheimer's disease (AD), a multi-factorial cognitive disorder. Polyfunctional compounds comprise a novel class of therapeutic agents for the treatment of multi-factorial disease like AD. In present study, total 33 chromen-4-ones were designed and synthesized by making modifications at different positions using Baker-Venkatraman rearrangement. These compounds were primarily evaluated for in vitro acetylcholinesterase (AChE) inhibitory, advanced glycation end products (AGEs) inhibition and antioxidant activity and showed that most derivatives inhibited AChE with IC₅₀ values in the nanomolar range with additional AGEs inhibitory and radical scavenging activities. The most active compounds FLV-16, FLV-31 and FLV-32 (IC₅₀=6.33, 6.48 and 5.83 nM, respectively) were also ameliorated scopolamine-

induced amnesia in mice model using moris water maze test and also reversed the changes of various oxidative stress markers (GSH and TBARS). The docking study revealed the binding pattern of compounds simultaneously bind with catalytic active site (CAS) and peripheral anionic site (PAS) of AChE. Moreover, the in silico pharmacokinetic profiles were predicted and revealed the drug-like properties with good penetration in brain and good oral absorption of compounds. After MD simulations, RMSD plots showed that the docked complexes were quite stable for the specified time of 10 ns with minor fluctuations. Thus, newly designed chromen-4-ones can act on different targets related to AD and the poly-functional attribute of these compounds make them potential candidates for the development of drugs for AD.

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Tau-BiFC platform to investigate pathological tau aggregation *in vitro* and *in vivo*

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Abnormal tau aggregation is a pathological hallmark in multiple neurodegenerative diseases collectively called tauopathies. Mounting evidences suggest that tau aggregates are not only neurotoxic, but also propagate in neurons acting as a seed for native tau aggregation. Accordingly, prevention of prion-like tau aggregation becomes an important therapeutic strategy to cure the disease. However, progress has been slow due to the lack of reliable methods to investigate tau pathology. In this regard, we developed a cell-based sensor that could monitor and quantify tau aggregation in neurons. By introducing bimolecular fluorescence complementation (BiFC) technique to tau, we could achieve spatial and temporal resolution

of tau-tau interactions in a range of states, from soluble dimers to large aggregates. Furthermore, by generating tau-BiFC mouse model that expresses neuron-specific tau-BiFC expression in the brain, we could visualize tau aggregation occurs in the diverse brain regions in the brain. Our tau-BiFC mouse started to present increased BiFC fluorescence indicating abnormal tau aggregation in the hippocampus from 6-month-old, and in the cortices from 12-month-old. Tau-BiFC responses were significantly increased in diverse brain regions in an age-dependent manner, demonstrating progression of tau pathology in the brain.

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New drug treatment and mechanism of the central anticholinergic drug trihexylphenidyl in reducing posttraumatic nightmares in patients with PTSD

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Objective: The central anticholinergic drug trihexylphenidyl (TP) was previously reported to show remarkable effectiveness against posttraumatic stress disorder (PTSD) flashbacks (FB) as well as the associated onset mechanism at WFSBP, 2015, Athens) and (CINP, 2016, Seoul). The objective of the current study was to assess the efficacy of TP in reducing the nightmares associated with PTSD and elucidate the underlying mechanism.

Methods: An open-label trial was conducted between 2009 and 2017 in outpatients who received a diagnosis of PTSD in accordance with DSM-5. TP (2 mg 1T-3T) was administered to 29 outpatients with PTSD depending on their condition. This study targeted refractory patients who had experienced no therapeutic benefit from any psychotropic drug over a number of years. The primary outcome variable was the change from baseline to endpoint in global Clinician-Administered PTSD Scale (CAPS-5) score and memory-related items B2 (nightmares) and B3 (flashbacks) for PTSD memory-related assessment. Secondary efficacy measures were the impact of event scale-revised (IES-R), which presents the overall clinical profile. Informed consent was obtained

from all patients. This study was approved by the Ethical Committee of Warakukai. UMIN trial ID: UMIN000028461.

Result: The therapeutic outcome in 29 patients demonstrated an extremely high efficacy rate, with 70.3% achieving complete remission (CR), indicating CR+ partial remission (PR: 29.7%=100% (flashbacks CR was 66.5%).

Conclusion: This study is the first pharmacological report on the novel use of TP against nightmares in PTSD. TP was markedly effective in the treatment of both nightmares and flashbacks. The nightmares onset mechanism is more closely linked to ACh transmission, and the nightmares is different to regular dream caused by Ch5 (PPN) and Ch6 (LDT) in the brain stem. Posttraumatic nightmares are flashbacks in dreams. The state in which neurotransmission of ACh-memory-related-Circuit (comprised by Ch4/Meynert-Amygdala, Ch1/medial septal nucleus, Ch2/Broca's diagonal band-hippocampus) generating PTSD-flashbacks is added to regular REM is considered posttraumatic nightmare (author's hypothesis).

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Fats and multiple sclerosis: association between fats/oils intake and disability in patients with MS

Mohammad Bagher Maljaei, Vahid Shaygannejad and Omid Mirmosayyeb
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Introduction: Multiple sclerosis (MS) is a chronic demyelinating disease of the nervous system which is the most common cause of neurological irreversible disability in young adults who are professionally and socially active persons. Due to the variable clinical course of MS, it is classified into relapsing and progressive phases and three phenotypes of relapsing remitting MS (RRMS), primary progressive MS (PPMS), and secondary progressive MS (SPMS). Assessment of dietary intakes of fats is an approach that has been used to evaluate diet-disease and diet-disability association.

Method: 126 patients with diagnosed MS (84 RRMS, 21 PPMS and 21 SPMS) with MRI assessment of brain and spinal cord were recruited from multiple sclerosis clinic in Kashani Hospital of Isfahan University of Medical Sciences, Isfahan, Iran include from present cross-sectional study. A 168-item semi-quantitative food frequency questionnaire was used for assessment of dietary intakes of fatty acids. Medical history questionnaire, Expanded Disability Status Scale (EDSS) and Fatigue questionnaire record from all participants.

Results: Mean±SD of EDSS and fatigue scale in SPMS and PPMS groups was significant higher than RRMS group. There

was a negative significant correlation between intakes of Poly Unsaturated Fatty Acids (PUFAs) including Linolenic Acid ($r=-0.418$, $p=0.018$), Linoleic Acid ($r=-0.312$, $p=0.031$) with EDSS in all participants. In addition, there was a negative significant correlation between intakes of Mono Unsaturated Fatty Acids (MUFAs) ($r=-0.348$, $p=0.028$) with EDSS in all participants. Correlation between Saturated Fatty Acids (SFAs) with EDSS ($r=0.465$, $p=0.009$) and fatigue scale ($r=0.298$, $p=0.043$) was significantly positive in all participants. Although correlation between total dietary fats with EDSS and fatigue scale in all participants and subgroups were positive, but was not significant. Age, gender and blood pressure were not confounder variables. In addition, we adjusted energy intakes in subgroups.

Conclusion: Our study demonstrated that there is a positive significant correlation between intakes of SFAs with EDSS and fatigue scale in all participants. In addition, dietary intakes PUFAs and MUFAs can decrease EDSS in all patients with MS. Further studies with larger sample sizes and other population needed to prove this correlation.

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Diet and neuromyelitis optica spectrum disorder; association between food group intakes and disability in patients with NMOSD

Mohammad Bagher Maljaei, Vahid Shaygannejad and Omid Mirmosayyeb
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Introduction: Neuromyelitis Optica Spectrum Disease (NMOSD) is an inflammatory disorder of the central nervous system (CNS) that presents typically with relapses of optic neuritis or transverse myelitis, in which IgG autoantibodies against aquaporin-4 water channel protein probably play a pathogenic role and IgG-NMO levels had correlation with disability in this patient. Assessment of dietary intakes of food groups is an approach that has been used to evaluate diet-disease and diet-disability association.

Method: 68 patients with diagnosed NMOSD with MRI assessment of brain and spinal cord, clinical symptoms and IgG-NMO test were recruited from multiple sclerosis clinic in Kashani Hospital of Isfahan University of Medical Sciences, Isfahan, Iran, include from present cross-sectional study. A 168-item, semi-quantitative food frequency questionnaire (FFQ) was used for assessment of dietary intakes of food groups. Medical history questionnaire, Expanded Disability Status Scale (EDSS) and Fatigue questionnaire record from all participants.

Results: Mean \pm SD of EDSS and fatigue scale in IgG-NMO positive group was significant higher than IgG-NMO negative group. There was a negative significant correlation between

intakes of whole grain ($r=-0.312$, $p=0.031$), fish ($r=-0.452$, $p=0.018$) and fresh fruits ($r=-0.365$, $p=0.026$) with EDSS in all participants and intakes of fresh vegetables ($r=-0.394$, $p=0.038$) and EDSS in IgG-NMO negative subgroup. In addition, there was a negative significant correlation between intakes fresh vegetables ($r=-0.302$, $p=0.034$) and fresh fruits ($r=-0.372$, $p=0.023$) with fatigue scale in all participants. Correlation between red and processed meats with EDSS ($r=0.512$, $p=0.002$) and fatigue scale ($r=0.439$, $p=0.020$) was significantly positive in all participants. Although correlation between dietary intakes of dairy and vegetable oil with EDSS and fatigue scale in all participants and subgroups were negative, but was not significant.

Conclusion: Our study demonstrated that there is a positive significant correlation between intakes of red and processed meat with EDSS and fatigue scale in all participants. In addition dietary intakes of whole grain, fish and fresh fruits can decrease EDSS in all patients with NMOSD. This is first report of dietary intakes of food groups in NMOSD patients. Further studies with larger sample sizes and other population needed to prove this correlation.

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Relationship between caffeine intake, EDSS and fatigue scale in patients with multiple sclerosis

Mohammad Bagher Maljaei, Vahid Shaygannejad and Omid Mirmosayyeb
Isfahan University, Iran

Introduction: Multiple sclerosis (MS) is a chronic inflammatory autoimmune demyelinating disorder of the central nervous system (CNS). Due to the variable clinical course of MS, it is classified into relapsing and progressive phases and three phenotypes of relapsing remitting MS (RRMS), primary progressive MS (PPMS), and secondary progressive MS (SPMS). Caffeine is a central nervous system stimulant of the methylxanthine class. It is the world's most widely consumed psychoactive drug. Recent scientific evidences showed that caffeine intake could be associated with decreased mortality from cardiovascular and neurological diseases, diabetes type II, as well as from endometrial and liver cancer. Previous studies on consumption of caffeine and (MS) have yielded inconclusive results. We aimed to investigate whether consumption of caffeine is associated with disability of MS.

Method: 126 patients with diagnosis of MS (42 RRMS, 42 PPMS and 42 SPMS) with MRI assessment of brain and spinal cord were recruited from multiple sclerosis clinics in Kashani Hospital of Isfahan University of Medical Sciences, Isfahan, Iran included in the present study. A 168-item semi-quantitative food frequency questionnaire (FFQ) was used for assessment of dietary intake of caffeine. Medical history

questionnaire, EDSS and Fatigue questionnaire recorded from all participants.

Results: Mean±SD of EDSS and fatigue scale in SPMS and PPMS groups was significantly higher than RRMS group. Dietary intakes of caffeine in RRMS was higher than two other subgroups but not significantly. There was a negative significant association between caffeine intake and EDSS in RRMS subgroup ($r=-0.556$, $p=0.031$). In addition, there was a significant negative correlation between caffeine intake and Fatigue scale in all participants ($r=-0.312$, $p=0.028$). Other correlations were not significant. Age, gender, energy intake and blood pressure were not confounder variables.

Conclusion: Our study demonstrated that there is a negative significant correlation between intakes of caffeine with fatigue scale in all participants. In addition dietary intakes of caffeine can decrease EDSS in RRMS patients and caffeine consumption may exert a protective role against multiple sclerosis. The concern of high caffeine intake is dehydration induced caffeine. Further studies with larger sample sizes and other population needed to prove this correlation.

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The impact of actuarial scales in predicting the risk of sexual recidivism and in the implementation of a specialized treatment program for sex offenders released from penitentiary

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Various methods based on scientific evidence (interviews, questionnaires, PPG) have been used to estimate sex offenders' potential for recidivism. This estimation is an essential component of sex offender risk management and treatment program. Risk assessment informs all participants in the criminal judiciary system, from judges to treatment providers to the police force, of the likelihood of certain types of sexual reoffending. It specifically enlightens the type and length of the sentence, the type and intensity of the treatment and the level of police supervision. Since the nineties, in order to improve the accuracy of the clinical judgement, actuarial risk assessment scales, involving historical events and treatment-related information, have been developed to evaluate sex offenders. Static and dynamic factors, statistically associated with increased risk of sexual reoffending, are scored jointly to produce a probability

estimate against a comparison group. Researchers of the world have established that these actuarial scales are consistently predicting relative risks of criminal behavior and are significantly more accurate than the unstructured clinical judgment. In the last 15 years, having been applying specific actuarial scales (Static 99, 99/R Stable and Acute 2007) with a large population of high-risk-and-need sex offenders recently released from penitentiary, in this presentation, the author proposes to establish their impact on the assessment procedure and treatment implementation of sex offenders recently released in the community. Their limitations and advantages will be presented. The common myths from the opponents to the use of actuarial scales will also be discussed.

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