Poster Presentations

Dementia 2018




10th World congress on

Dementia and Alzheimer's Disease

August 16-17, 2018 | Copenhagen | Denmark

Interactions of mitochondrial matrix proteins 17β-hydroxysteroid dehydrogenase type 10 and cyclophilin D in people with Alzheimer disease and multiple sclerosis

Zdenka Kristofikova, Hofmannova A, Hromadkova L and **Bartos A**
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The nucleus-encoded mitochondrial matrix protein 17β-hydroxysteroid dehydrogenase type 10 (17β-HSD10) operates via multiple enzymatic as well as non-enzymatic functions. Its overexpression or deficiency is associated with various pathologies. Increased levels of 17β-HSD10 in cerebrospinal fluid (reflecting probably its brain overexpression) were found in patients with Alzheimer disease (AD) or multiple sclerosis (MS). Both neurodegenerative diseases are accompanied by mitochondrial dysfunction. Cytosolic 17β-HSD10 is imported into the mitochondrial matrix via PINK1-Parkin-TOM/TIM pathway. Here, it binds to cyclophilin D (cypD) and, by preventing its translocation to the inner mitochondrial membrane, can regulate the opening of the mitochondrial permeability transition pore mediated by cypD. Under conditions of increased accumulation of mitochondrial amyloid β (Aβ), observed especially in AD, interactions of 17β-HSD10 and cypD could be eliminated which may lead to apoptosis and mitochondrial dysfunction. Using cerebrospinal fluid samples of people with AD or MS and mitochondria isolated from double transgenic McGill-R-Thy1-APP rats (one of the best animal models of AD with intracelular accumulation of Aβ), we estimated levels of 17β-HSD10, cypD, Aβ 1-42, total Aβ and of various complexes (17β-HSD10 – Parkin, 17β-HSD10 – total Aβ, 17β-HSD10 – cypD). In AD, our results indicate that up-regulation of 17β-HSD10 does not have to be followed by increased levels in mitochondrial matrix and that the ability of the protein to regulate cypD is weakened. In MS, on the contrary, it seems that up-regulation can lead to increased PINK1-Parkin-TOM/TIM transport and that 17β-HSD10 in mitochondrial matrix is fully functional. Supported by GA CR (P304-12-G069) and AZV CR (16-27611A) projects.

Speaker Biography

Zdenka Kristofikova studied at Czech Technical Univerzity in Prague (Ing., Department of Nuclear Chemistry) and at Univerzity of Defence, Faculty of Military Health Sciences in Hradec Kralove (PhD, Department of Toxicology), both in the Czech Republic. She works at National Institute of Mental Health as a senior researcher and a head of working group. She is interested in Alzheimer disease for a long time (Web of Sciences: 122 results, 594 sum of times cited, h-index 15).

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Notes:

Alzheimer's disease and its Prevention: Epidemiology

Seth Omari Mensah
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Alzheimer's disease which is a form of dementia has been one of the deadly disease since 2001. There is no cure and no effective treatment. Alzheimer's disease presents policy-makers with many challenges, including, the cost pressures on long-term medical care worldwide. Alzheimer's disease is a progressive disease of the human brain that is characterized by impairment of memory and a disturbance in at least one other thinking function. When we hear about Alzheimer's disease we automatically think of older people. This is because this disease most often occurs in adults after the age of 65. Statistics show that one in eight individuals will have Alzheimer's after they reach age 65. The purpose of this study is to create awareness of Alzheimer's and how it can be prevented through research that has been made that plenty of omega-3 fats which Evidence suggests that the DHA found in these healthy fats may help prevent Alzheimer's disease and dementia by reducing beta-amyloid plaques and such omega-3 fats can be found mostly in sea food such as salmon, tuna etc. Methodological and theoretical orientation: Alzheimer's disease has always been diagnosed by immunochemical assay directly or indirectly detecting the presence or absence of an apolipoprotein E type 4 (ApoE4) isoform or DNA encoding ApoE4 in the subject. Findings: Initial studies indicated that repetition priming was immune to the effects of aging and greatly reduced in Alzheimer's disease (AD). As more studies have been performed, however, these initial conclusions appear less clear than before and, in the case of AD, actually misleading. Conclusion: Due to my vivid research since AD cannot be cured or treated but rather prevented awareness should be created and people should encouraged to take more omega- 3 fats since that may prevent AD.

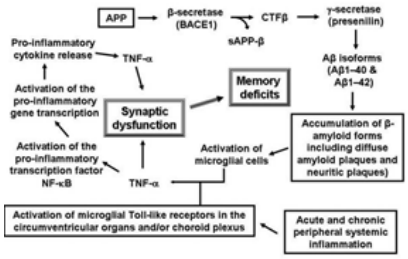

Fig. 1: Showing the mechanism of DHA found in Omega 3 fats reducing beta-amyloid plaques

Speaker Biography

Seth Omari Mensah is a 4th year student of the Kharkov National Medical University of Ghanaian nationality. He has attended and presented on schistosomiasis with Sustainable Medical Missions (May 2016), and attended numerous conferences held in Ukraine regarding various topics of healthcare. He is researching on tropical diseases to assist Sustainable Medical Missions with their conferences within the continent of Africa.

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Notes:

Accepted Abstracts

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Classification of Behaviors in Dementias Based on Principles of Compliance and Aggression

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Objectives: There is vastheterogeneityin use of terminology and classification of behaviors in dementia with no universallyaccepted classification system.

Methods: Criteria proposed by Davis, Buckwalter and Burgio(1997) were identified as the basis for classification ofbehaviors in dementia. A review of literature was done with a view to Identify the "Specification of the Theoretical Construct" (STC) to justify aggregation of similar Behavioral symptoms into clinically meaningful categories.

Results: STC identified for these behavioral categories are theories on compliance and aggression. Behavioral categories emanating from this construct are; Oppositional Behaviors (OB) and Physically Aggressive Behaviors(PAB).

Discussion: OB is the result of non-compliance to the directions being given bythe care provider. Thetypes of OBaredetermined bythe level of developmental sophistication or converselybythe degreeof cognitive impairment in patients with dementia. PABarethe result ofperceived impediment bythe patient in goal attainment. This results in the emergenceof negative emotions. These emotions are 'out of proportion' to the stimulus. The purpose of this behaviour is to warn the care provider of the noxious nature of their involvement in the present situation.

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Efficacy and safety of MMFS-01, a synapse density enhancer, for reversing age-related cognitive decline: a randomized, double-blind, placebo-controlled trial

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Neuropsychological evaluation identifies neurocognitive deficits to aid in the diagnosis of the specific type of dementia that carries a different intervention and/or treatment plan. Predominantly frontal (executive reasoning) deficits will tend to be more suggestive of frontal lobe dementia or disease; frontal and temporal deficits (memory and the impact of executive reasoning deficits) of cardiovascular disease; visual perceptual, executive reasoning deficits and psychiatric signs of Lewy body dementia; while rather clear signs of highly impaired memory (for retrieval and recognition) as well as visual spatial issues will tend to reflect the true Alzheimer's dementia. Cognitive decline associated with neurological diseases depends upon the brain areas affected; for example, Multiple Sclerosis (impacts memory and attention, processing information quickly and efficiently) Parkinson's disease (executive reasoning, as well as attention and memory) while Huntington's has a predilection for executive reasoning deficits and motoric response. rnUse of neuropsychological evaluation and diagnosing the specific deficit areas has allowed us to develop a very specific cognitive training regimen which has shown positive findings when comparing testing prior to and following treatment intervention. Specificity of the cognitive training has been a primary variable for improved functioning following treatment. At our facility all of the brain enhancing activities have been systematically studied and labeled for the effect they are expected to have in remediating brain function; memory (short and long term, retrieval and recognition, visual and verbal) executive reasoning processes (selective attention, integration, perseveration, sequential analysis, cognitive flexibility) language (word retrieval) and visual perceptual. The key to the most effective and efficacious outcome in our research has been early diagnosis and treatment. rnWe are on an outreach effort for neurocognitive evaluation of individuals with any type of illness (physical or psychiatric) in their sixties and everyone in their seventies. Outreach is currently ongoing to the primary care physician to query about memory difficulties and executive reasoning symptoms. In the USA, dementia is known as one of the most expensive medical conditions; costs are currently in the billions and projected to the trillions for the future. rnThe answer is early diagnosis and education of the general population of the increased risk of dementia when there is avoidance of memory problems. Fear and avoidance of dementia is enormous and pervasive; affecting the caregiver, spouse, as well as the children. Waiting until a basic dementia assessment or mini-mental status is failed or for the problem to be exacerbated and outwardly obvious, results in more severe diagnosis and complications. Our research and work with the aging population over the last ten years reveals the benefit of early diagnosis and the intervention of cognitive training/rehabilitation upon brain function in helping to remediate the effects of dementia. Case studies will be provided to illustrate the significant neurocognitive changes that occurred from cognitive rehabilitation which transferred to improved emotional and daily living skills.

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Adapting the Tinetti tool for balance and gait for person with dementia

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Background: A recent review of the literature reveals an absence of standardized measures to assess mobility in persons with advanced dementia. Persons with moderate to severe dementia have significant difficulty adhering to instructions. The aim of the study was to develop a standardized measure of gait and balance for use with persons with dementia. We chose to modify the 'Tinetti Assessment Tool for Balance and Gait' because many of the items are based on observation. Modification of test items requires analysis of reliability prior to establishing validity.

Objective: To determine the inter-rater and test-retest reliability of 'Tinetti Assessment Tool for Balance and Gait-Dementia'.

Methods: Inter-rater reliability: Participants were observed and scored simultaneously by two raters familiar with the written instructions (physiotherapy, occupational therapy or nursing staff) Test-retest.

Reliability: The test was re-administered after 10 to 30 minutes. Results: A total of n=20 participants were recruited and included. The mean age of participants was 75 with the majority being female (n=11, 55%). All were diagnosed with dementia or cognitive impairment. Secondary diagnoses include heart disease, diabetes and Parkinson's disease. The mean cognitive assessment (SMMSE) score was 8.5/30 (n=12). Inter-rater reliability of the total score was high (r=0.90) as was test-retest reliability (r=0.92).

Discussion: These results are comparable to established reliability of the original Tinetti tool (r>0.8). The results indicate that the modified measure has sufficient reliability to commence validity testing. Developing a measure that assesses functional changes in this population is important for determining the impact of mobilization and least restraint programs for people with dementia in long term care. Further study will establish validity of the cut score for predicting falls risk.

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Alzheimer's cognitive impairment can be recovered by decreasing homocysteic acid in blood

Caron Leid
Aspen University, Canada

Although more and more people are being affected by Alzheimer's disease, there is not enough information surrounding the true daily struggle of the disease. Due to that reason, I felt there was a huge gap for those in the sandwich generation. My mother was diagnosed with Atypical Alzheimer's disease at the age of 57. The book which I wrote is a journey to assist others with resources and the struggle of living with the disease, from a raw perspective.

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Alzheimer disease research in the 21st century: the shift towards a new paradigm

Francesca Pistollato
Physicians Committee for Responsible Medicine, USA

Animal models of Alzheimer disease (AD) have been extensively utilized in the last few decades in an effort to elucidate the pathophysiological mechanisms of this disease and to test novel therapeutic approaches. However, research success has not effectively translated into therapeutic success for human patients. We investigated the reasons for this translational discrepancy. Our analysis revealed that translational failure is due – at least in part – to the overuse of animal models that cannot accurately recapitulate human AD etiopathogenesis or drug responses and the inadequate use of human-based investigational methods. Here we present the challenges and opportunities in AD research and propose how we can mitigate this translational barrier by employing human-based methods to elucidate disease processes occurring at multiple levels of complexity (from gene expression to protein, cellular, tissue/organ to individual and population level). Novel human-based cellular and computational models are already being applied in toxicology and regulatory testing, and the adoption and the widespread implementation of such tools in AD research will undoubtedly facilitate human-relevant data acquisition. Additionally, clinical studies focused on nutritional and lifestyle intervention strategies to reduce and/or prevent early symptoms of AD represent another relevant and important way to elucidate AD pathogenesis and treatment options in a human-based setting. Taken together, it is clear that a paradigm shift towards human-based research is the best way to tackle the ever-increasing prevalence of AD in the 21st century.

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Alzheimer's cognitive impairment can be recovered by decreasing homocysteic acid in blood

Tohru Hasegawa
Saga Woman Junior College, Japan

Recent big two clinical trials of amyloid beta treatment for Alzheimer's disease are all failed to recover the cognitive impairment, it has forced us to reconsider the central hypothesis of amyloid pathogen for Alzheimer's disease. We recently published that human Alzheimer's patients showed the strong relationship between Mini Mental State Examination (MMSE) scores and blood homocysteic acid (HA) level. 6 AD patients (all female: age 77, 82, 86, 87, 91, 91) were given green tea powder 1g at every meal for 2 months. Their blood HA level and MMSE score were measured before and after taking green tea powder. The relationship between blood HA level change and MMSE score change was investigated. The strong statistically significant negative relationship between blood HA level change and MMSE score change: r=-0.96, p=0.00018, n=6. From our observation, it showed that blood HA level change induced MMSE score change, that is, Alzheimer's cognitive ability was controlled by blood HA level. Now we can present that some healthy food, that is named HBF, can recovered 100% Alzheimer's cognitive impairment by the decreasing the homocysteic acid in a peripheral blood. Now we have made a relative large open trial of AD patients. 91 patients were enrolled. Their cognitive recovery was measured by NM scale (New Clinical Scale for Rating of Mental States). All patients who took HBF showed the recovery of their behaviors. From this open-trial of HBF, (1) Alzheimer's cognitive impairment could be recovered at even end stage. (2) Alzheimer's disease is induced by homocysteic acid.

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The TTAP Method; A Proven Structured Non-pharmaceutical Approach to Enhancing Cognition and Socialization in Mild-Moderate Stages of Alzheimer's disease

Linda Levine Madori
St.Thomas Aquinas College, USA

This Paper will establish through an examination of neurobiology the how and why the creative art therapies most effectively be utilized in the treatment with those individuals afflicted with early to middle stages of dementia, specifically Alzheimer's Disease, which currently comprises approximately 80% of all dementia's. An in-depth overview on how Therapeutic Thematic Arts Programming© (TTAP Method) stimulates both right and left brain functioning in the early stages to middle stages of Alzheimer's disease will be analyzed from various clinical studies done in 2011, 2012, 2013. This paper will cover the most recent and basic functional organization of the brain, neuroplasticity, including neurons, neurotransmitters and areas of the brain involved in transforming perceptual inputs into physiological responses and behaviors (Damasio, 1998, 1999; Golomb, J.,1996, Grober, E., 1999; Kandel, Schwartz & Jessel, 2000; LeDoux, 2000; Levine Madori, 2007-2014). A review the innovative new methodology, the TTAP Method© which utilizes person centered themes within the therapeutic process to engaged participants in a twelve step process that incorporates mediation & mindfulness, drawing, sculpture, movement, phototherapy and other forms of the creative arts into an ongoing enriching non-pharmaceutical approach for this special and rapidly growing population. This method substantiates how art therapy is quickly becoming a powerful window into brain functioning and self-discovery (Cozolino, 2012, Luzebrink, 2013, Hass-Cohen, 2014).

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Exploring the kaleiodoscopic oasis of epigenetics-based diet, brain games and physical exercises in cognitive aging and Alzheimer dementia: Evidence, promises and challenges

Simon Chiu
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Background: Recently, increased interest has been expressed in developing diverse strategies to optimize cognitive aging and to modify the onset and course of Alzheimer's dementia (AD). Th e interaction of Gene and Environment in modulating cognitive decline is best understood within the framework of Epigenetics. Epigenetics refers to heritable changes in gene expression and remodeling of chromatins and dependent of alterations in DNA sequence, and comprise of three key components; DNA methylation, histone modifi cations (acetylation and deacetylation) and non-coding microRNA. Epigenetics targets play major role in reprogramming of neural networks and neural repair. Epigenetics can turn genes "On" and "Off " depending upon the milieu. There is emerging evidence supports the model of dysregulation in epigenomics in age-related cognitive decline and AD. A large number of studies have shown that nutrition factors: diets, dietary and herbal supplements, functional foods, are capable of regulating the epigenetic states and targets in reversing abnormal gene activation or silencing. Physical exercises and e-delivered brain games likewise can change various domains in aging and in AD through the epigenetics signatures. We review the translational and clinical evidence in support of the benefi cial eff ects of dietary phytochemicals from diverse dietary sources; grapes, chocolates, green and black coff ee, soya beans and fava beans, curry extract, peanuts, garlic and ginger and seafood products have positive impact on epigenomics in facilitating translational and transcriptional events involved in memory, attention and executive functions. The findings from the studies on DASH and Mediterranean diets reinforce the relevance of epigenetic diet menu, along with the proposed Epigenetics diet for cognitive aging platform. We will also discuss the multi-faceted actions of herbal supplements: Panax Ginseng and Curcumin from Curry extract, and Zembrin extract from South African plant Sceletiumtortuosum. and diet menu in enhancing vascularneuronal coupling and to reduce metabolic and vascular risks in aging. Epigenetics targets are also sensitive to environmental stimuli and processing. Hence physical exercises and e-delivered cognitive challenge tasks like puzzles, video games. The evidence is mounting in terms of the putative positive eff ects in reprogramming neural circuitry for cognition and reactivating neurogenesis in the hippocampus. We conclude that epigenomics-driven lifestyle measures and diet interventionsare promising in cognitive aging on the progression of AD. We anticipate in the near future we will have epigenetics-based dietary and exercise and e-stimulation can prevent AD and optimize cognitive aging and will be translated intoevidence-based practice guidelines forgeriatric care. We believe that biotechnology caneventuallytransform bioactive factors to CNS drug candidates for AD treatment and prevention.

Objectives: At conclusion of this session, the participant should be able to understand how cognition can be regulated through dietary interventions targeting Epigenomics in aging & Alzheimer dementia (AD). To evaluate the benefi t-to-risk ratio and evidence of clinical cognitive studies of specialized diets, dietary supplements and functional foods in preventing cognitive decline in aging and in AD. To gain insights into the benefi ts of aerobic exercise and e-delivered video games in reprogramming gene expression and neural circuits involved in cognitive aging and in modifying the course of AD. To identify fi scal and systemic issues involved in translating new research fi ndings on brain foods, exercise and e-delivered brain exercise to evidence-based practice in geriatric are to understand how epigenomics may shed light on the link of nutrition, cognition and AD and has the potential to transform bioactive phytochemicals to promising drugs for treating and preventing AD.

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Tohru Hasegawa
Saga Woman Junior College, Japan

Recent big two clinical trials of amyloid beta treatment for Alzheimer's disease are all failed to recover the cognitive impairment, it has forced us to reconsider the central hypothesis of amyloid pathogen for Alzheimer's disease. We recently published that human Alzheimer's patients showed the strong relationship between Mini Mental State Examination (MMSE) scores and blood homocysteic acid (HA) level. 6 AD patients (all female: age 77, 82, 86, 87, 91, 91) were given green tea powder 1g at every meal for 2 months. Their blood HA level and MMSE score were measured before and after taking green tea powder. The relationship between blood HA level change and MMSE score change was investigated. The strong statistically significant negative relationship between blood HA level change and MMSE score change: r=-0.96, p=0.00018, n=6. From our observation, it showed that blood HA level change induced MMSE score change, that is, Alzheimer's cognitive ability was controlled by blood HA level. Now we can present that some healthy food, that is named HBF, can recovered 100% Alzheimer's cognitive impairment by the decreasing the homocysteic acid in a peripheral blood. Now we have made a relative large open trial of AD patients. 91 patients were enrolled. Their cognitive recovery was measured by NM scale (New Clinical Scale for Rating of Mental States). All patients who took HBF showed the recovery of their behaviors. From this open-trial of HBF, (1) Alzheimer's cognitive impairment could be recovered at even end stage. (2) Alzheimer's disease is induced by homocysteic acid.

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Lifestyle interventions for Dementia using the internet

Veronika Vd Wardt
University of Nottingham, UK

Phytoestrogens found in soy products such as tofu have been shown to exert protective eff ects on brain function. However, in this talk we review the data from observational and treatment studies which show discrepant results. Many studies have either shown no associations or even negative associations. Th e importance of age and estrogen status, duration of treatment and ethnicity will be discussed.

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