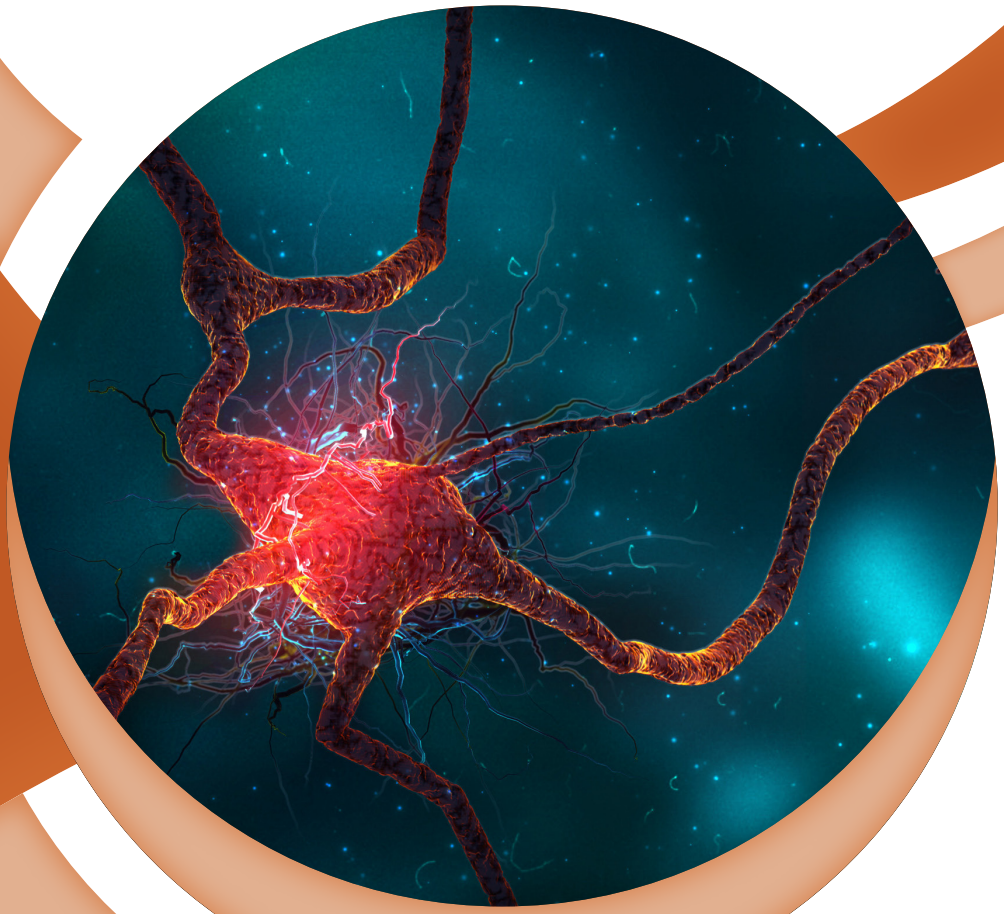

10th World Congress on

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Posters

Jae-Kyung Lee, J Alzheimers Dis Parkinsonism 2018, Volume 8
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Regulator of G-protein signaling 10 modulates neuroinflammation and metabolic homeostasis: A potential role in alzheimer's diseases

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Insulin resistance and aging-related metabolic disorders constitute serious threats to human health as risk factors for Alzheimer's Disease (AD); especially impaired brain glucose homeostasis was related to the severity of the AD pathology. Regulator of G-protein Signaling proteins (RGSs) are a family of proteins that negatively regulate G-Protein Coupled Receptors (GPCR) through their GTPase Accelerating Protein (GAP) activity. RGS10 is one of the smallest RGS family proteins which we have shown to negatively regulate microglia activation and the level of RGS10 in microglia significantly decreased within the microglia by age. RGS10-deficient microglia displayed impaired phagocytic activity to amyloid-beta fibrils (fAβ). Interestingly, RGS10-deficient mice spontaneously gained weight with age (>15 months) and the level of RGS10 protein was decreased in postmortem brains of the AD and Frontal Temporal Dementia (FTD). Our data demonstrate that RGS10-deficient mice display impaired glucose tolerance, the high level of triglycerides (TG) in plasma. RGS10-deficient mice spontaneously gained weight with age (>15 months). We also tested whether RGS10 plays a role in high-fat-induced chronic inflammation and glucose metabolism as a risk factor for metabolic disorder in the periphery and the CNS. Indeed, HFD-fed RGS10-deficient mice gained significantly more weight compared to HFD-fed wild-type (WT) mice. Importantly, HFD-fed RGS10-deficient mice displayed an insulin resistance phenotype and impaired Long-Term Potentiation (LTP). These data implicate RGS10 may play a critical role of in insulin sensitivity during metabolic disorders in the periphery and the CNS. Importantly, peripheral metabolic disorders, including obesity and insulin resistance along with chronic inflammation have been shown to contribute to development and progression of cognitive impairment and alzheimer's disease through multiple mechanisms. Our data strongly implicate the role of RGS10 in modulating metabolic homeostatsis related to its role in neuroinflammation. Elucidating RGS10 function in maintaining metabolic homeostasis in the CNS and periphery may provide the mechanism to link aging associated chronic inflammation and metabolic disorders, which could be a potential therapeutic target for alzheimer's diseases with dual effects on both inflammation and metabolic disruption. Overall, our study produced highly novel data delineating potential mechanisms of RGS10 function in metabolic homeostasis in the brain.

Biography

Jae-Kyung Lee has completed her PhD in UNT Health Science Center and Postdoctoral studies from UT Southwestern Medical Center at Dallas. She had worked as an Assiatant Professor at Emory University until 2015. Currently, she is an Assistant Professor in University of Georgia, USA. She has published more than 24 papers in reputed journals. Her research focused on understanding how inflammation influences neurodegenerative diseases.

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Notes:

Jun-Ah Song et.al., J Alzheimers Dis Parkinsonism 2018, Volume 8
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Exploring the concept of family caregiver's competence for managing behavioral and psychological symptoms of dementia

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It is important for health professionals to teach family caregivers of persons with dementia about how to deal with Behavioral Psychological Symptoms of Dementia (BPSD) well. However, little information is available about what aspects exactly they need to learn to be competent in managing BPSD. The purpose of this study was to explore attributes of the concept of family caregiver's competence for managing BPSD (CM-BPSD). A rapid realist review was conducted to synthesize the complex range of concepts and define attributes of the family caregiver's CM-BPSD from the comprehensive literature. Three databases were used including MEDLINE, Embase and CINAHL. The search terms used were dementia, caregiver, coping (managing) and competence. The selection criteria were articles being published in English between 1990 and 2017 and considered appropriate for the topic. Among the 235 articles, 89 were excluded for duplication and six for not being written in English. From the concept synthesis of competence for managing, four dimensions were defined: Judging, setting the direction, adjusting and reflecting. For the concept of family caregiver's CM-BPSD, eleven attributes were derived. Findings of this study suggest that the concept of family caregiver's CM-BPSD should reflect the procedural aspect. Reflecting can be considered as a meta-competence because it may influence the other three phases of managing BPSD. A field study would be of great value to clarify the attributes of the concept.

Biography

Jun-Ah Song has completed her PhD from University of Michigan, School of Nursing and Postdoctoral studies from Oregon Health and Sciences University. She is a Professor of Korea University College of Nursing and an Expert Committee of the National Institute of Dementia in Korea. She has been serving as a Chief Editor of the *Journal of Korean Gerontological Nursing* and an Editorial Board Member of other professional journals. She has published more than 30 papers about caregiving issues related to dementia in reputed national and international journals.

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Different influences of CADASIL pathologies on cognitive function and quality of life

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The purpose of this study is to investigate the effects of three major neuroimaging markers of Cerebral Autosomal Dominant Arteriopathy with subcortical infarcts and Leukoencephalophathy (CADASIL) on cognition and Quality of Life (QoL). 84 participants with CADASIL completed the comprehensive clinical evaluation including 3T MRI and genotyping of NOTCH3. WMH volume was calculated using volumes were measure by a fully automated monospectral segmentation method using FLAIR MRIs. Neurocognitive function was evaluated using CERAD-K neuropsychological assessment battery (CERAD-NP) and quality of life was measured using the Short Form 36 Health Survey Questionnaire (SF-36). Greater WMH volume had a negative impact on 8 neurocognitive tests ($p<0.05$; t-test) in CERAD-NP except constructional praxis. The number of lacunar infarctions was associated with poor performance of MMSE-KC ($p<0.05$; t-test) only and the number of CMBs was not related to any neurocognitive test scores. WMH volume was negatively associated with Physical Function (PF), role limitations, Vitality (VT), Mental Health (MH), Physical Component Score (PCS) and the mental component score. The number of lacunar infarction was only related to poor PF ($p<0.05$; t-test). The number of CMB was associated with the lower scores of quality of life, especially in general health, VT, MH and PCS. WMH volume [Odds Ratio (OR): 1.03; 95% Confidence Interval (CI): 1.007-1.060] in patients with CADASIL was associated with dementia, indicating that for every 1 ml of WMH volume, the risk of depressive disorder increased by 3%. WMH volume has the most significant effect on both cognitive function and quality of life.

Biography

Joon Hyuk Park has completed his PhD from Seoul National University, Republic of Korea. He is an Assoicate Professor at Jeju National University, School of Medicine and the Director of Jeju Province Dementia Center. His research interests include epidemiology of dementia, BPSD and vascular depression and has published more than 50 papers in reputed journals.

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The role of AMP-activated protein kinase as a novel therapeutic target for alzheimer's disease

Hyunji Jo, Kyu-Yeon, Jun and Youngjoo Kwon
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Alzheimer 's Disease (AD) is an aging-associated neurodegenerative disease. AD patients suffer from behavioral deficits, mental distress and progressive memory impairments. Currently existing AD drugs are merely symptomatic treatments. Therefore, we need to find a novel therapeutic strategy for AD. Amyloid beta (Aβ), known as a marker of AD, is generated by cleaving Amyloid-Precursor Protein (APP) with β-secretase (BACE1) and γ-secretase. Identifying effective methods to suppress the Aβ accumulation has long been of great interest. Recently, AMP-Activated Protein Kinase (AMPK), a serine/threonine protein kinase, began to be focused as a novel therapeutic target since it has been reported to regulate formation of Aβ. Thus, in this study, 100 compounds were selected from screening a chemical library containing one million compounds by in silico study. We finally found YE-06 through chemical modifications and various bioassays. In accordance with the docking study, YE-06 potentially bound to the AMP binding site of AMPK. Compared to Metformin, which is a well-known AMPK activator, YE-06 significantly activated AMPK and consequently down-regulated the protein level of BACE1. The mRNA level of BACE1 was significantly reduced. We showed improvements in the cognition and movement coordination of AD rat model in YE-06 treated group through water maze test, probe test, passive avoidance test, rotarod test and vertical pole test. YE-06 efficiently increased ACh and decreased the AChE activity. Also, YE-06 significantly reduced neuronal cell death of AD rat models. Therefore, our results suggest that YE-06 is a potential compound for AD treatment.

Biography

Hyunji Jo has graduated from Konyang University in 2016. She is currently pursuing Doctoral studies in Pharmacy at Ewha Womans University. Her research focuses on studying the role of AMPK as a novel therapeutic target for Alzheimer's disease.

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Chronic microvascular ischemia is associated with cerebral amyloid burden in patients with cognitive impairment

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Background & Purpose: White Matter Lesions (WML), detected as hyperintensities on T2-weighted magnetic resonance imaging, represent chronic microvascular ischemia in the brain and are considered potential risk factors for memory and cognitive impairment in the elderly. The purpose of this study is to evaluate the association between WML and the cerebral β-Amyloid (Aβ) burden in patients with cognitive impairment.

Method: 19, 30 and 34 patients with subjective cognitive impairment, mild cognitive impairment and alzheimer's disease, respectively, who underwent brain MRI and F-18 florbetaben PET, were included. The Fazekas scale was used to quantify WML on brain T2-weighted images. The cerebral Aβ burden was quantitatively estimated using volume-of-interest analysis. The difference in Fazekas scale was evaluated between the Aβ positive and negative groups. The relationship between the Fazekas scale and the cerebral Aβ burden was evaluated using linear regression analysis after adjustment for age and sex.

Result: There were no differences in age and sex among the patients with subjective cognitive impairment, mild cognitive impairment and alzheimer's disease. In the overall cohort and mild cognitive impairment group, Aβ positive patients exhibited significantly higher Fazekas scale compared with Aβ negative patients (0.8 vs. 1.3; P=0.024 and 0.5 vs. 1.4; P=0.022). In addition, the cerebral Aβ burden was positively correlated with the Fazekas scale (β=0.299; P=0.006 and β=0.517; P=0.003).

Conclusion: WML are associated with the cerebral Aβ burden in patients with cognitive impairment. This suggests that chronic microvascular ischemia contributes to the development of alzheimer's disease.

Biography

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Yung-Chih Kuo et.al., J Alzheimers Dis Parkinsonism 2018, Volume 8
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Rosmarinic acid and curcumin-loaded polyacrylamide-cardiolipin-poly(lactide-co-glycolide) nanoparticles with conjugated 83-14 monoclonal antibody to protect β-amyloid-insulted neurons

Yung-Chih Kuo and He-Cheng Tsai
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Polymeric nanoparticles (NPs) combined with lipids can have profound effects on treatment efficacy in patients with neurological disorders such as Alzheimer's Disease (AD). We developed polyacrylamide (PAAM)-cardiolipin (CL)-poly(lactide-co-glycolide) (PLGA) NPs grafted with surface 83-14 Monoclonal Antibody (MAb) to carry Rosmarinic Acid (RA) and Curcumin (CUR). This drug delivery system was used to cross the Blood-Brain Barrier (BBB) and enhance the viability of SK-N-MC cells insulted with β-Amyloid (Aβ) deposits. Experimental evidence revealed that an increase in the concentration of 83-14 MAb enhanced the permeability coefficient of RA and CUR using the nanocarriers. The levels of phosphorylated p38 and phosphorylated τ-protein at serine 202 in degenerated SK-N-MC cells were in the order: Aβ>(Aβ+RA-CUR)>(Aβ+83-14 MAb-RA-CUR-PAAM-PLGA NPs)>(Aβ+83-14 MAb-RA-CUR-PAAM-CL-PLGA NPs)≈control. The viability of SK-N-MC cells reduced with time and CL in 83-14 MAb-RA-CUR-PAAM-CL-PLGA NPs advantaged Aβ-targeted delivery of RA-CUR. These results evidenced that the current 83-14 MAb-RA-CUR-PAAM-CL-PLGA NPs can be a promising pharmacotherapy to permeate the BBB and reduce the fibrillar Aβ-induced neurotoxicity.

Biography

Yung-Chih Kuo is a Professor at National Chung Cheng University, Taiwan. His research interests are focused on biomaterials, nanomedicine, tissue engineering, blood-brain barrier, cancer therapy, nerve regeneration, spinal cord injury and stroke treatment and Alzheimer's and Parkinson's disease therapy. He has authored over 140 SCI journal papers. He is a Fellow of Royal Society of Chemistry, UK and an Honor Member of Phi Tau Phi Society. He has also won Prof. Yen-Ping Shih Award in 2017; Best Paper Award in 2016 and 2008; Prof. Tsai-Teh Lai Award in 2015; Special and Talented Scholar Award in 2013 and Outstanding Research Award in 2013.

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e-Poster

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Accelerated brain aging with relevance to type 3 diabetes and alzheimer's disease

Ian James Martins
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The main constituent of plaques in the brain of Alzheimer's Disease (AD) individuals namely Amyloid beta (Aβ) is a proteolytic product of a larger protein the Amyloid Precursor Protein (APP) protein. Carriers of the apo E4 allele are at greater risk of developing AD with increased deposition of amyloid-beta plaques in western countries. Protein and Aβ homeostasis is now crucial to the lifespan of organisms and is an important feature that determines the aging process in obesity, diabetes and neurodegenerative diseases. The scientific understanding of the maintenance of peripheral blood plasma Aβ and caffeine metabolism has now become essential to prevent neurodegeneration that is linked to type-3 diabetes. The concentration of Aβ within the brain is determined by hepatic Aβ clearance and interest in the liver has increased markedly since in western countries the incidence of Non-Alcoholic Fatty Liver Disease (NAFLD) and insulin resistance has reached approx. 20% of the developed world. Induction of type-3 diabetes is related to delayed hepatic caffeine metabolism (NAFLD) with circadian dysynchrony (type-3 diabetes) connected to defective peripheral hepatic caffeine and Aβ metabolism. Healthy diets stabilize type-3 diabetes and maintain the circadian rhythm with relevance to brain insulin resistance and alzheimer's disease.

Biography

Ian James Martins is an Editor and Reveiwer for Open Acess Pub/MDPI journals. He has appointed as the Chief Editor for *International Journal of Diabetes Research* (2014-2018); *Research and Reviews: Neuroscience* (2016-2018) and *Journal of Diabetes and Clinical* Studies (2017-2018); Scientist for Science Advisory Board (USA) and Academic with Academia.edu. He also has Lifetime Membership by International Agency for Standards and Ratings as Fellow; Winner of World Academic Championship-2017 in Diabetes and Medical Science (Nutrition).

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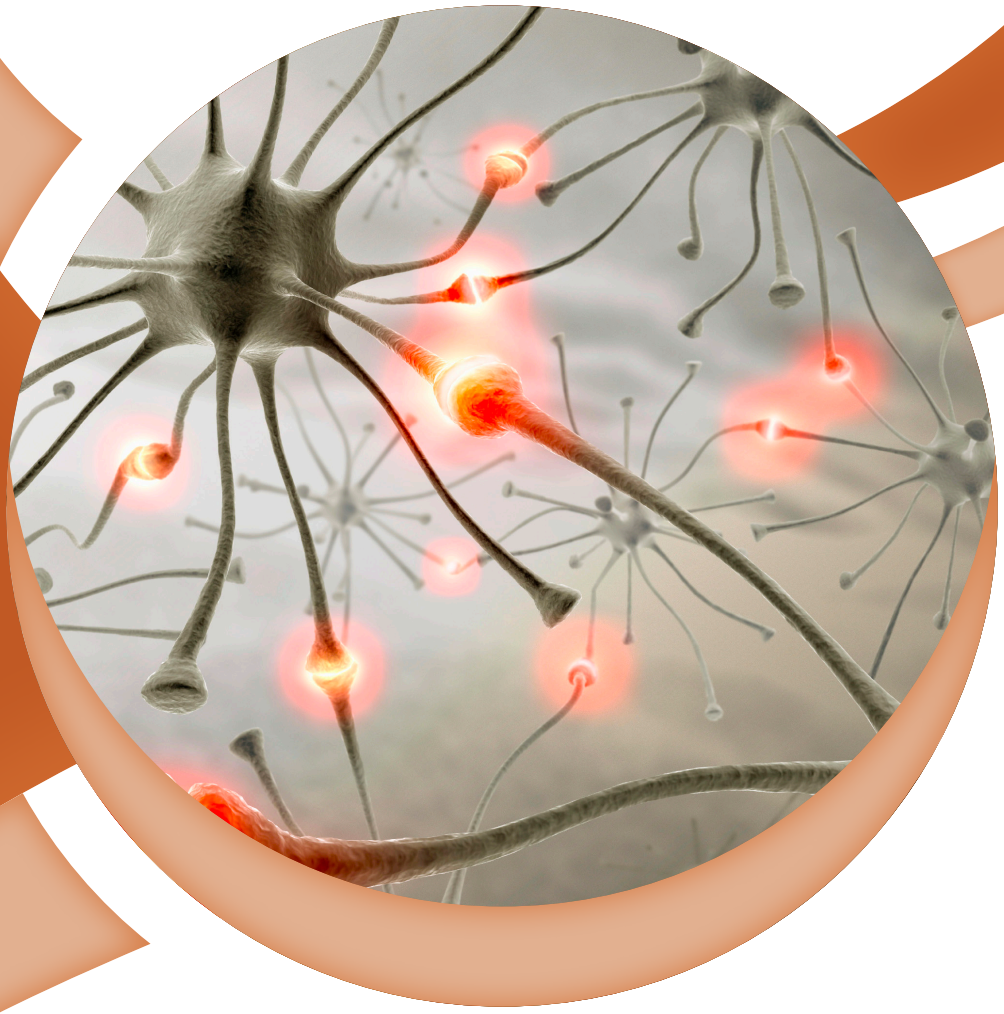

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Genetics and epidemiological studies of dementia of alzheimer's type among Arab populations

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Objective: To study the genetic and environmental risk factors and the prevalence of Dementia of the Alzheimer Type (DAT) among the elderly in an Arab community in Israel.

Material & Method: Epidemiological and genetic studies of dementia have rarely been reported in an Arab population. Alzheimer disease (AD [MIM #104300]) is a progressive, neurodegenerative disease characterized clinically by gradual loss of memory and pathologically by neurofibrillary tangles and amyloid plaques in the brain. We have observed an unusually high prevalence of dementia of the Alzheimer type in Wadi Ara, an inbred Arab community in northern Israel comprising <850 persons over the age of 60 years. Apolipoprotein E (APOE- ε4), has been established as a strong susceptibility marker that accounts for nearly 30% of the risk in late-onset AD.

Result: Remarkably, in our study DAT is not associated with APOE, because the frequency of the ε4 allele is very low in both nondemented (2.4%) and demented elders (3.6%). We also map chromosomal loci contributing to DAT susceptibility; we conducted a 10 cM scan in a series of twenty cases and twenty controls selected from one hamula. Markers from 18 chromosomal regions showed significant allelic association with DAT ($P<0.05$). Locations on chromosomes 2, 9 and 10 remained significant after testing additional affected and non-demented individuals. Significant associations were also observed for markers on chromosome 12 which overlap with a locus implicated in previous genome scans. Additionally, several lines of evidence support for a role of angiotensin converting enzyme (ACE) in Alzheimer Disease (AD). Most genetic studies have focused on an Alu insertion/deletion (I/D) polymorphism in the ACE gene (DCP1) and have yielded conflicting results. We evaluated the association between 15 (SNPs) in DCP1, including the I/D variant and AD in a sample of 92 patients with AD and 166 non-demented controls from an inbred Israeli Arab community. Although there was no evidence for association between AD and I/D, we observed significant association with SNPs rs4343 ($P=0.00001$) and rs4351 ($P=0.01$).

Conclusion: In Wadi Ara, the high prevalence may be due to a founder effect enhanced by consanguinity which make this population attractive for investigating DAT susceptibility recessive genes. Thus, a specific disease susceptibility allele may be overrepresented in cognitively impaired subjects compared with cognitively healthy residents. Other two main conclusions can be drawn from the genome-wide linkage and Linkage Disequilibrium (LD) studies. Firstly, multiple genes are involved in DAT. Secondly, there is a high level of consistency among linkage and association studies regarding the general location of putative AD genes. However, the general location of putative AD genes on a given chromosome covers a broad region which may contain several genes.

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The relationship between social isolation and dementia: A behavioral rodent study

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Some risk factors that can be detected early in adulthood might render a person more susceptible to cognitive decline. It is becoming increasingly clear that social isolation could be such a risk factor for age-related cognitive decline and dementia. Social isolation has been linked to a higher risk in developing dementia in elderly persons. But in our modern society, loneliness is also highly prevalent among adults. In the US range from 25 to 60%, could possibly result in social isolation and thus an increased risk of developing, accelerating or exacerbating dementia pathology and symptoms. Moreover, changes in social interaction and electronic communication are placing an increasing proportion of the adult demographic at risk for loneliness. Given the high prevalence of social isolation, studying the effects of this on mental health is much timely than ever. We found that rodents that were isolated for 4 weeks during adolescence developed a specific dementia-like phenotype compromising both cognitive and non-cognitive domains, compared to socially housed animals. This behavioral phenotype was expressed as reduced investigation of social stimuli and intense aversive responses toward them, such as freezing. Animals also displayed a long-lasting impairment in a memory task. This phenomenon was found to be dependent on proper ventral and dorsal hippocampal synaptic plasticity. Implications for the clinic and therapeutic strategies and interventions are discussed.

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Family carers of people with young-onset dementia: Their experiences with the supporter service

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Background: Family carers and people with Young-Onset Dementia (YOD) require tailored assistance as dementia progresses. A variety of health care services is needed, including supporter services. To our knowledge, research focusing on experiences with the supporter service is scarce.

Aim: To evaluate the supporter service by examining how primary family carers experience the assistance provided.

Method: Qualitative interviews with 16 primary family carers of people with YOD were performed from 2014 to 2015. Content analysis was used to analyze the data.

Result: Three main themes emerged from the interviews. First, a good match focused on the carers' experiences of the relationship between the supporter and the person with YOD and included three subthemes: A nice, empathetic personality, a friendship-like relationship and the content of the meetings. The second theme, relief, addressed the carers' experiences with the service. The third, coordination, concerned the carers' relationship with the health care service.

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Tumor microenvironment associated immune suppression mediated disease progression

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Tumor microenvironment consists a lot of various cell types and plays a key role for tumor survival. These different cells, such as Myeloid-Derived Suppressor Cells (MDSCs), Tumor-Associated Macrophages (TAMs), Cancer-Associated Fibroblasts (CAFs), express different function-associated molecules which are involving in mediating tumor progression. CD39/CD73-adenosine pathway has been recently defined as an important tumor-induced immunosuppressive mechanism. We here documented a fraction of CD11b+CD33+ MDSCs in peripheral blood and tumor tissues from Non-Small Cell Lung cancer (NSCLC) patients expressed surface ectonucleotidase CD39 and CD73. Tumor TGF-β stimulated CD39 and CD73 expression, thereby inhibited T cell and NK cell activity and protected tumor cells from the cytotoxic effect of chemotherapy through ectonucleotidase activity. Moreover, CD39+CD73+ MDSCs expressed higher levels of typical MDSC-associated suppressive factors and were significantly associated with disease progression and the poor response to chemotherapy. Our further studies for the reduction of CD39 and CD73 expression by Metformin could block the suppressive function. CD39 and CD73 on MDSCs, therefore, link their immunosuppressive and chemo-protective effects to cancer progression, providing novel targets for chemo-immunotherapeutic intervention.

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Candidate biomarkers and CSF profiles for alzheimer's disease and CADASIL

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The differential diagnosis between Alzheimer's Disease (AD) and Vascular Dementia (VaD) are still roughly problematic in clinical practice, despite the widely used diagnostic criteria to differentiate between the two disorders. There is an increasing evidence that cerebrovascular dysfunction plays a role not only in vascular causes of cognitive alterations but also in AD. Cognitively patients, with AD, show sometimes-mixed degrees of associated vascular lesions in 30-60% of AD cases. In opposition, AD pathology may be present in 40%-80% of VaD patients, thus impeding diagnosis accuracy. Therefore, to eliminate this bewilderment and discrepancies in the diagnosis between the AD and VaD, it is worthy to shed light firstly on a disease that is a microangiopathy and represents VaD with clear milestones and features, as is the case of Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL). Studying CADASIL CSF biomarkers profile will help in the differential diagnosis between both diseases sharing the coexisting neurodegeneration, furthermore, CADASIL is a dominantly inherited mid-adult life disorder causing ischemic strokes, which belongs to vasculopathies and symbolizes a genuine prototype of VaD that provides a valuable opportunity for studying its CSF biomarkers. Secondly, examining and evaluating the CSF biomarkers of AD compared to that of CADASIL. The pathogenesis similarities between CADASIL and early onset AD affecting the small vessels of the brain have suggested plausible molecular mechanisms involved in vascular damage and their impact on brain function and come from the fact that in both diseases genetic mutations occur. CADASIL mutations in NOTCH3 gene generate toxic protein aggregates (Granular Osmiophilic Material-GOM) in the vicinity of Vascular Smooth Muscle Cells (VSMCs) causing degeneration and loss of VSMCs in small arteries and arterioles of white matter regions of the brain that lead to dementia, similar to those attributed to mutant forms of the Amyloid Precursor Proteins (APP) and presenilins genes who cause overproduction and accumulations of the toxic Aβ42 protein in the brain and collapse of Aβ42 clearance mechanisms in AD. Despite the presumed pathological similarities, substantial differences between the two phenomena may exist especially in the CSF neurochemical phenotypes.

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Neuritin attenuates early brain injury in rats after experimental subarachnoid hemorrhage

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Objectives: Early Brain Injury (EBI) is central to the pathological progress of Subarachnoid Hemorrhage (SAH). In this study, we determined if neuritin protects the brain against EBI in rats and discussed the role of apoptosis pathway mediated by Endoplasmic Reticulum (ER) stress in this neuroprotective route.

Methods: A total of 96 male Sprague Dawley rats were divided into control, sham, SAH and SAH+neuritin groups. The rat SAH model was induced by injection 0.3 mL of nonheparinized arterial blood into the prechiasmatic cistern. Mortality assay, neurological scores, brain water content measurement, Evans blue dye assay, TUNEL stain assay, and Western blot analysis were performed.

Results: Neuritin significantly improved the neurological scores, brain water content, Blood Brain Barrier (BBB), and apoptosis compared with the control and sham groups within 24 hours after SAH. TUNEL staining assay results demonstrated that apoptosis was ameliorated, MMP-9 expression was reduced, whereas GRP78, CHOP, caspase-12, and ASK1 levels were markedly preserved after neuritin application.

Conclusion: Our study demonstrated that neuritin plays a neuroprotective role on EBI after SAH by attenuating BBB disruption, brain edema and apoptosis.

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Diagnosing dementia across cultures

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The number of people with dementia is growing worldwide. Early and accurate diagnosis is important. Growth will occur particularly in developing countries which often do not have the resources (staff, financial, equipment) to do an extensive dementia assessment. In this talk, we will explore the different methods used to do a stepwise low cost and cross culturally applicable screening for dementia. Data from China, India, Indonesia, Australia, UK and Singapore are presented. We will show different methods including paper and pencil tasks and computerized assessments which have high validity and reliability. These tests were originally developed at Oxford University and are now used as a gold standard in several settings.

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Rab21, a novel PS1 interactor, regulates γ-secretase activity via PS1 subcellular distribution

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The γ-secretase has been a therapeutically target for its key role in cleaving APP to generate β-Amyloid (Aβ), the primary constituents of senile plaques and a hallmark of Alzheimer's Disease (AD) pathology. Recently, γ-secretase associating proteins showed promising role in specifically modulating APP processing while sparing Notch signaling; however, the underlying mechanism is still unclear. A Co-Immunoprecipitation (Co-IP) coupled with mass spectrometry proteomic assay for Presenilin1 (PS1, the catalytic subunit of γ-secretase) was firstly conducted to find more γ-secretase associating proteins. Gene ontology analysis of these results identified Rab21 as a potential PS1 interacting protein, and the interaction between them was validated by reciprocal Co-IP and immunofluorescence assay. Then, molecular and biochemical methods were used to investigate the effect of Rab21 on APP processing. Results showed that overexpression of Rab21 enhanced Aβ generation, while silencing of Rab21 reduced the accumulation of Aβ, which resulted due to change in γ- secretase activity rather than α- or β-secretase. Finally, we demonstrated that Rab21 had no effect on γ-secretase complex synthesis or metabolism but enhanced PS1 endocytosis and translocation to late endosome/lysosome. In conclusion, we identified a novel γ-secretase-associating protein Rab21 and illustrate that Rab21 promotes γ-secretase internalization and translocation to late endosome/lysosome. Moreover, silencing of Rab21 decreases the γ-secretase activity in APP processing thus production of Aβ. All these results open new gateways towards the understanding of γ-secretase-associating proteins in APP processing and make inhibition of Rab21 a promising strategy for AD therapy.

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Perspective on neurobiological and clinical early indicators of mild cognitive decline and alzheimer's disease

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There is a need for early diagnosis, monitoring and treatment of Alzheimer's Disease (AD) and Mild Cognitive Impairment (MCI). Traditional assessments of cognitive decline have been found to lack sensitivity and accuracy in differentiating varying stages of Dementia and cognitive decline as well as being time consuming in their administration. Key components of cognition namely memory and executive function have been identified as most predicative of AD status. Brief cognitive screening tools such as the Montreal cognitive assessment have been recommended both as a primary clinical and research assessment offering more sensitivity in differentiating AD and MCI. However, overlapping clinical features and impairments in cognitive processing suggest a need for biological risk factors. Neurobiological indicators of cognitive deterioration have been identified implicating measures of cerebrospinal fluid and temporal lobe atrophy as potential biomarkers of early clinical phases of AD and predictors of cognitive decline. Evidence shows the utility of automated classification methods in processing and analyzing multivariate neuroimaging data which improves our accuracy for the prediction of conversion of MCI to AD. In this review, we discuss the clinical usefulness of such approaches and the need for big data and multi-site studies in improving our understanding of AD neuropathology and confirming pathophysiological mechanisms that can reliably be used to differentiate MCI and AD and predict disease progression and cognitive decline.

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Proprioceptive neuromuscular facilitation increases alpha absolute power in the dorsolateral refrontal cortex and superior parietal cortex

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The clinical practice of physiotherapists includes Proprioceptive Neuromuscular Facilitation (PNF), which is a treatment concept that accelerates the response of neuromuscular mechanisms through spiral and diagonal movements. The adaptations that occur in the nervous system following PNF are still poorly described in the literature. Thus, the aim of this study was to investigate the electrophysiological changes in the fronto-parietal circuit during PNF and movement in the sagittal and diagonal patterns. This study included 30 female participants, who were divided into 3 groups (control, PNF and flexion groups). Electroencephalogram measurements were determined before and after tasks were performed by each group. For the statistical analysis, a two-way ANOVA was performed for the factors, group and time. Interactions between the two factors were investigated using a one-way ANOVA. $P<0.004$ was considered significant. The results showed an increase in alpha absolute power in the left dorsolateral prefrontal cortex and upper left parietal cortex of the PNF group, suggesting these areas work together to execute a motor action. The PNF group showed a greater alpha absolute power compared with the other groups, indicating a specific cortical demand for planning and attention, reinforcing its use for the rehabilitation of individuals.

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Differential diagnosis of alzheimer's disease

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Alzheimer's disease is a most common dementia in the world. Approximately 30% of the population over the age of 80's will present it. Although it is a disease with the well-defined diagnostic criteria, the exclusion of other diseases is very important. The physician should present in his mind the arsenal of differential diagnoses of main clinical features of each disease that potentially mimics an alzheimer's disease, thus facilitating the identification of them. In this heterogeneous group of dementias, curiously found some of them that are potentially reversible. In the group of dementias, we found primary and secondary diseases as cause of dementia process: Deficiency of vitamin B12, B1, tertiary syphilis, normal pressure hydrocephalus, brain tumors, vascular dementia, delirium, depression, Lewy body disease, Central Nervous System (CNS) vasculitis, fronto-temporal dementia, drug intoxication and Parkinson's disease, although the technology evolution, many of the aforementioned diagnosis may be performed based on the patient's medical history and physical examination. Patients with reports of sadness and loss of energy may suggest the diagnosis of depression in an elderly patient with cognitive impairment. Cognitive changes initiated abruptly suggest more the diagnosis of delirium than dementia. In these cases, a secondary etiology needs more investigation. The history of daily alcohol consumption may suggest deficiency of vitamin B1 (Korsakoff syndrome). Young patients with arthralgia, weight loss and rash are associated with CNS vasculitis. Changes in gait, urinary incontinence are common findings in normal pressure hydrocephalus. Patients with reports of recurrent strokes may have vascular dementia. Motor changes such as spasticity is associated to dementia by Lewy bodies and Parkinson's disease in a late stage. It is concluded that anamnesis and physical examination is considered the initial step in the investigation of a patient who comes at the office with a complaint of memory problems.

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Effects of mesenchymal stem cells transplantation on cognitive deficits in animal models of alzheimer's disease

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Introduction & Purpose: Alzheimer's Disease (AD) is a globally prevalent neurodegenerative disease, clinically characterized by progressive memory loss and gradual impairment of cognitive functions. Mesenchymal Stem Cells (MSCs) transplantation has been considered a possible therapeutic method for AD. However, no quantitative data synthesis of MSCs therapy for AD exists. We conducted a systematic review and meta-analysis to study the effects of MSCs on cognitive deficits in animal models of AD.

Method: We identified eligible studies published from January 1980 to January 2017 by searching four electronic databases (PubMed, Medline, Embase, CNKI). The endpoint was the effects of MSCs on cognitive performance evaluated by the Morris Water Maze (MWM) test including escape latency and the number of platform crossing and time in the target quadrant.

Result: Nine preclinical studies incorporating 225 animals with AD were included for the meta-analysis. The studies indicated that MSCs based treatment significantly improved the learning function through measurements of the escape latency (SMD=-0.99; 95% CI=-1.33 to -0.64; P<0.00001). Additionally, we observed that transplantation of MSCs significantly increased the number of platform crossing in six experiments (SMD=0.78; 95% CI=0.43 to 1.13; P<0.0001). What's more, the times in the target quadrant were increased in five studies indicated that transplantation of MSCs could ameliorate the cognitive impairments (SMD=1.06; 95% CI=0.46 to 1.67; P=0.0005).

Conclusion: The current study showed that MSCs transplantation could reduce cognitive deficits in AD models. These findings support the further studies to translate MSCs in the treatment of AD in humans.

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Identification of novel ApoE4 inhibitor for alzheimer's disease therapy

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ApoE4 is a major genetic risk factor due to its increase incidence of developing alzheimer's disease. The study was designed to predict such compounds that may helpful in designing drug to suppress the over activity of apoE4 protein. 22 natural compounds (marine, microorganism and plant derivative) were used as inhibitors and docked with apoE4 (PDB id 1B68). 6 Synthetic compounds (in clinical trials) were docked with target protein to compare and analyze the docking results with natural compounds. Compounds S-allyl-L-cysteine, epicatechin gallate and fulvic acid shows high binding affinity i.e. -7.1, -7 and -7, respectively. Epicatechin gallate shows hydrogen bond with Gln156 and Asp35 and fulvic acid shows hydrogen bonding with Glu27. In case of synthetic compounds tideglusib did not show hydrogen bonding with any amino acid residue of ApoE4 but show high binding affinity of -7.2 same as of natural compound S-allyl-L-cysteine which show high binding affinity of -7.1 but did not show hydrogen bonding with any amino acid residue. Protein-protein interactions of ApoE4 show physical and functional interaction with related proteins. Our study predict a compound epicatechin gallate on the basis of binding affinity and hydrogen bonding with amino acid residue as a potential lead compound which may be used as an inhibitor.

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Use of mancala/sungka to reduce cognitive decline in institutionalized elderly in Metro Manila

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Introduction & Purpose: As people age, their mental and physical functions diminish due to their inactivity. The purpose of this study is to use sungka to reduce cognitive decline and enhance cognitive aspects (memory and retention, attention and concentration, executive function and mood) among institutionalized elderly in Metro Manila.

Method: The playing of sungka was incorporated into a structured routine program, entitled COMPLY: Communicate-Move-Play (which includes social, physical and cognitive activities). The subjects were 12 elderlies, who came from an elderly institution in Metro Manila, selected under purposive sampling. The study was conducted for 45 minutes to one hour, once a week for four weeks. Quasi-experimental design was utilized through a pre- and post-intervention test using Mini Mental State Exam (MMSE). Two researcher developed tools were used in determining the effect of the structured routine program. The consent of the institution and the subjects were obtained and was assured that their privacy, confidentiality and anonymity were secured. Measure of pre-and post MMSE scores of were analyzed through dependent T-test, while, cognitive aspects under the research developed tool were analyzed through repeated measures ANOVA.

Result: Findings revealed that, there is a significant difference between the pre- and post- MMSE ($p=0.001$). The results suggest that sungka is effective in reducing cognitive decline in geriatric subjects. Further, attention and concentration were shown to have a significant change ($F=4.600$; $p=0.030$) indicating that the subjects were being taught lesser every session has transpired. However, memory and retention ($F=1.882$; $p=0.169$), executive function ($F=0.792$; $p=0.502$) and mood ($F=1.0000$; $p=0.339$) had no significant change.

Conclusion: The significant difference in the pre- and post- MMSE shows that the use of mancala/sungka can reduce cognitive decline among institutionalized geriatric subjects in Metro Manila. Further research is needed to extend the length of application of the said structured program.

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Practical communication skills for dementia care partners

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The number of individuals around the world affected by dementia is growing. To address the looming crisis of dementia, there is a tremendous need to increase knowledge of dementia and awareness of early warning signs and help people learn skills to communicate and interact effectively with a person who has dementia. This workshop will increase awareness of dementia and its symptoms and will teach basic communication techniques to people who care for individuals living with dementia. Participants will have the opportunity to practice specific techniques on how to connect and approach a person with dementia using both verbal and non-verbal cues. Increasing basic knowledge and communication skills of care partners will lead to interactions with better outcomes, will improve quality of life for everyone involved, and will allow people living with dementia to continue to live full lives for as long as possible. By the end of this workshop participants will be able to: recognize the differences between normal and not normal aging, describe how dementia affects the brain, demonstrate specific communication, cueing and approach techniques and appreciate how it feels to be living with dementia.

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The language of food

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Food is a language that can unlock memories for a person living with dementia. This presentation will present how the language of food transcends race, sex, age, class and yes even dementia. For this reason, food is one of the three pillars of "NoosaCare's Dementia Living" module of care. In most residential care facilities, the focus is on meeting nutritional needs, being cost effective, but what if we moved beyond these boundaries and we looked at the passion food can evoke in people? Our people, our residents? Although we all do it in different ways, the one thing every culture in the world does is prepare food and eat it. In our memory support unit, we have planted verdant edible gardens that the residents tend to, harvest and then they use in the preparation of their meals. We also identified that the language of food needs to give dignity to the person living with dementia, so we have replaced the baby like term of pureed food with a sexier smooth food and the finger food menu has been replaced with a tapas menu to give back dignity to our residents who prefer to dine on the run.

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Neuropsychiatric and cognitive subtypes among community-dwelling older persons and the association with DSM-5 mild neurocognitive disorder: Latent class analysis

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Background: Neuro Psychiatric Symptoms (NPS) have been shown to increase the risk of Neurocognitive Disorders (NCD), leading to the recently-published criteria of Mild Behavioral Impairment (MBI) to identify pre-dementia using NPS alone. However, MBI drew concerns about over-diagnosing subclinical psychiatric disorders.

Objective: We hypothesized that the specificity of NPS in predicting NCD may be improved by considering NPS together with various domains of cognitive deficits. We tested this hypothesis by identifying subtypes based on the combination of NPS and cognitive deficits among community-dwelling older persons and evaluating how the identified subtypes were associated with mild NCD.

Method: Our participants were from a community-based cohort study. They completed assessments such as Geriatric Depression Scale (GDS), Geriatric Anxiety Inventory (GAI) and Montreal Cognitive Assessment (MoCA). Those with possible cognitive impairment underwent further evaluations for mild NCD. Latent class analysis was conducted using GDS, GAI and MoCA domains. Logistic regression was performed to investigate the association between the latent-classes and mild NCD.

Result: We included 825 participants and identified four distinct subtypes: Subtype-1 (no NPS or cognitive deficits); subtype-2 (NPS alone); subtype-3 (cognitive deficits alone) and subtype-4 (both NPS and cognitive deficits). Subtype-1 and 2 had low risk of prevalent mild NCD (OR 0.92-1.00), while subtype-3 conferred a moderate risk (OR 4.47-4.85) and subtype-4 had the highest risk (OR 7.95-8.63).

Conclusion: We demonstrated the benefits of combining NPS and cognitive deficits to predict those at highest risk of prevalent mild NCD. Our findings highlighted the relevance of subclinical psychiatric symptoms in predicting NCD and indirectly supported the need for longer durations of NPS to improve its specificity.

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Orthosiphon stamineus improves cognitive functions after ICV streptozotocin insult in alzheimer's disease model

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Alzheimer's disease is a chronic neurodegenerative disease that causes cognitive impairment like learning and memory. *Orthosiphon stamineus* (OS) is a medicinal herb that has been reported to exert various pharmacological activities. The objective of this study was to evaluate whether this Malaysian plant extract can reverse Streptozotocin (STZ) induced cognitive dysfunction in experimental animals. Rats were subjected to Intra Cerebro Ventricular (ICV) injections of STZ (3 mg/kg) bilaterally. The STZ-injected rats received oral treatment of OS (50,100 and 200 mg/kg) one day after the surgery, for 7 days before being subjected to behavioral analysis. The learning and memory performance was assessed using passive avoidance and elevated plus maze tests. It was found that the OS administration significantly attenuated learning and memory impairment induced by STZ-injection. The hippocampal tissues were extracted for gene expression analysis, known to be modulated in AD condition. All the three doses demonstrated effectiveness against Scopolamine-induced cognitive impairment when compared to the negative control group. Therefore, these results demonstrate the effectiveness of OS in averting cognitive deficits could serve as a potential therapeutic option for the treatment of neurodegenerative diseases like AD.

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