12th International Conference on

Alzheimer's Disease & Dementia

October 29-31, 2018 | Valencia, Spain

Special Session

Dementia 2018

T Y Chang et al., J Alzheimers Dis Parkinsonism 2018, Volume 8
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T Y Chang & Catherine Chang

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Mobilizing cholesterol in the brain to treat Niemann-Pick type C disease and Alzheimer's disease

All cells require cholesterol and a portion of cellular cholesterol is stored as cholesterol esters; this process is catalyzed by the storage enzyme acyl-CoA:cholesterol acyltransferase 1 (ACAT1). Once formed, cholesterol esters cannot substitute the functions of cholesterol. In certain neurodegenerative dieases, needs for additional cholesterol in the brain arise. Inhibiting ACAT1 may benefit these diseases, by preventing cholesterol from being stored, thus providing the additional cholesterol that the diseased cells need. We tested this idea in two neurodegenerative dieases i.e., Niemann-Pick type C disease (NPCD) and Alzheimer's disease (AD). NPCD is a rare and genetic neurodegenerative disease. The NPCD patients almost invariably die before reaching teenage. In mutant NPC cells, malfunction in endosomal cholesterol egress occurs causing chronic functional cholesterol deficiency in the plasma membrane and golgi membrane. We show that ACAT1 KO or ACAT1 inhibitors provide more cholesterol to golgi membranes of mutant NPC1 cells. In vivo, ACAT1 KO increases the life span and improves several other distinguishing features of the mutant NPC1 mouse. We next show that in a mouse model for AD, inhibiting ACAT1 provides more cholesterol to cell membranes of neuronal cells and produces multiple benefits that includes reducing Abeta production, increasing oligomeric Abeta degradation and increasing unhyperphosphorylated mutant human tau degradation. ACAT1 blockage also ameliorates the cognitive deficits of the AD in mouse. In summary, we show that inhibiting ACAT1 mobilizes a specific cholesterol pool in the brain to benefit two different neurodegenerative diseases i.e., NPCD and AD.

Biography

TY Chang and Catherine Chang are a husband/wife team, and share the same laboratory, with TY as the PI and Cathy as the co-PI, at Geisel School of Medicine at Dartmouth. They have been working on cholesterol metabolism research for more than four decades. The Changs and their colleagues did ground breaking work by identifying the gene that encodes the cholesterol storage enzyme acyl-CoA:cholesterol acyltransferase 1 (ACAT1/SOAT1). Subsequently, they performed extensive functional analysis of the enzyme. More recently, they demonstrated ACAT1 as a potential target for treating several human diseases, including Alzheimer's disease and Niemann-Pick type C disease.

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Notes:

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Scientific Tracks & Abstracts

Day 1

Dementia 2018

Day-1

SESSIONS

Therapeutic Targets and Mechanisms for Treatment | Animal Models and Translational Medicine | Alzheimer's disease Pathophysiology and Disease Mechanisms | Dementia with Lewy Bodies | Managing Dementia | Care Practice and Awareness

Chair: Magdalena Turek, Care Homes Zapiecek, Poland

SESSION INTRODUCTION

Title: Early brain connectivity alterations before amyloid deposition in a rat model of Alzheimer's disease
Guadalupe Soria, Institut d'Investigacions Biomèdiques August Pi i Sunyer (IDIBAPS), Spain

Title: A case study on the successful transition for seniors living with Alzheimer's disease from an aged care residential facility to a domestic environment
Emily Gillett, The CareSide, Australia

Title: Yolkin polypeptide complex from hen egg yolk – isolation, characterization and neuroprotective activity
W Kazana, Hirszfeld Institute of Immunology and Experimental Therapy-Polish Academy of Sciences, Poland

Title: Boosting brain insulin signaling to combat neurotoxicity arising in type 2 diabetes
Shaymaa Abdulghaffar Abdulmalek, Alexandria University, Egypt

Title: *In silico* identification of novel ApoE4 inhibitor for Alzheimer's disease therapy
Muhammad Asif Rasheed, COMSATS University Islamabad, Pakistan

Title: Carbamylated erythropoietin-FC protects hippocampus against Aβ induced memory deterioration in a rat model of Aβ toxicity: Considering Akt/GSK-3β, MAPKs and MMP-2
Etrat Hooshmandi, Shahid Beheshti University of Medical Sciences, Iran

Guadalupe Soria, J Alzheimers Dis Parkinsonism 2018, Volume 8
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Early brain connectivity alterations before amyloid deposition in a rat model of Alzheimer's disease

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Animal models of Alzheimer's disease (AD) are essential in understanding the disease progression and development of early biomarkers. In this study, a transgenic rat model of AD (TgF344-AD) was longitudinally analyzed to describe both cognitive performance and brain connectivity. Cognitive abilities were assessed longitudinally by a delayed non match-to-sample (DNMS). Every three months after DNMS, MRI acquisition was performed including diffusion-weighted MRI and resting state functional MRI, which were processed to obtain the structural and functional connectomes, respectively. Global and regional graph metrics were computed to evaluate network organization in both transgenic and control rats. Less efficient organization of the structural brain networks of the transgenic rats with respect to controls was observed at five months of age, before a significant concentration of β-amyloid plaques is present. Specific regional differences in connectivity were identified in both structural and functional networks. A strong correlation was observed between cognitive performance and brain networks, including whole brain structural connectivity as well as functional and structural network metrics of regions related to reward, memory and sensory performance processes. Despite, the fact that the Tg animals showed a smaller number of responses in the first phase, the hit rate was very similar in both groups throughout the study. The AD also is a connectopathy in the TgF344AD rat model and this integrative approach is without a doubt, a track that promises results much more efficient from the point of view of translational research.

Biography

Guadalupe Soria is Incharge of the Experimental MRI 7T Unit at IDIBAPS, Barcelona. She has specialization studying Neurodegeneration, MRI Biomarkers and Animal Models. Her areas of research interests are discovery of MRI biomarkers for early diagnosis of neurodegenerative disorders such as AD, shortening of the translational gap between preclinical and clinical research and cognitive enhancement as a potential intervention for AD.

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Emily Gillett, J Alzheimers Dis Parkinsonism 2018, Volume 8
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A case study on the successful transition for seniors living with Alzheimer's disease from an aged care residential facility to a domestic environment

Emily Gillett
The CareSide, Australia

Alzheimer's disease changes how seniors interpret their environment. Names, places and people they know become unfamiliar, leading to disorientation, stress and isolation. The CareSide has worked with a large number of clients and their families to ensure this disorientation is minimized, specifically by supporting ageing at home. I would like to present 2 case studies where we have moved seniors from the traditional nursing home back to their domestic home environment. I will outline the planning and transition stages and how ultimately this has had a positive outcome on the clients and their families.

Biography

Emily Gillett began her career as a Registered Nurse and then quickly moved into general management, followed by strategic consulting and Project Management in Health services. She has completed an Executive MBA at the University of New South Wales and a post grad in OccHealth. Inspired by personal experiences with family members living with Alzheimer's disease, she decided to use her diverse set of skills in Health Management to cofound The CareSide (www.thecareside.com.au) - a fast growing company that provides In Home Personal Care and Nursing Services for Seniors in Perth, Australia. Emily is the Director of Care Services at the CareSide. Her main responsibility in this role is to ensure quality care is provided to clients and their families living with physical and cognitive health challenges.

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Notes:

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Yolkin polypeptide complex from hen egg yolk—isolation, characterization and neuroprotective activity

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Age-related diseases are the most frequent ones among diseases diagnosed in the populations. Due to the increase of this phenomenon, the study on substances, which can inhibit neurodegenerative processes, is beginning to have special prominence. Hen eggs have been recognized as an excellent, natural and easily bio-renewable source of substances with a well-documented biological activity. One of them is yolkin, a group of peptides accompanying IgY in hen's egg yolk plasma, of a molecular weight from range 1 to 35 kDa. Yolkin possess immunoregulatory and neuroprotective activity, indicating its potential function in inhibition of the progression of dementia in the course of neurodegenerative disorders. Yolkin also mitigates behavioral symptoms of aging and improves cognitive learning and memory in young and old rats. Its neuroprotective effect was observed in PC12 cells treated with toxic amyloid protein Aβ1-42 and on the nerve fibers of NGF-differentiated PC12 cells treated with aggregated Aβ1-42. It was also shown that yolkin stimulates both PC12 neuron-like cells and human whole blood cells to release significant amounts of brain-derived neurotrophic factor (BDNF) in dose- and time-dependent manner. BDNF regulates neuronal survival and outgrowth, influences synaptic plasticity and is a key molecule in the maintenance of memory storage in the hippocampus. Moreover, level of BDNF mRNA or protein are dramatically decreased in parts of the brain affected by neurodegenerative processes. In conclusion, yolkin thanks to its beneficial properties, can be considered as a potential therapeutic agent in the treatment of neurodegenerative diseases.

Biography

W Kazana is a second-year PhD student at the Hirszfeld Institute of Immunology and Experimental Therapy, PAS in Wrocław. She graduated in biotechnology in Wrocław University of Science and Technology (MSc thesis awarded). Her research focuses on neuroprotective and immunoregulatory properties of yolkin, polypeptide complex isolated from hen egg yolk. She has already presented her preliminary results at NEURONUS conference (poster) and symposium opening Biotechnology: Research and Industrial Applications conference (oral presentation) during this year. She is also one of the co-authors of an article published in Neuropsychiatry (London) (2018) 8(3), 833-842, DOI:10.4172/Neuropsychiatry.1000410.

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Notes:

Shaymaa Abdulghaffar Abdulmalek, J Alzheimers Dis Parkinsonism 2018, Volume 8
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Boosting brain insulin signaling to combat neurotoxicity arising in type 2 diabetes

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Background: Insulin signaling reveals to be a very promising pathway for the prevention and treatment of Alzheimer's disease (AD). Available data have indicated that brain insulin resistance may contribute to neurodegenerative diseases.

Aim: The present work aimed to study the role of combined metformin with natural oil to enhance brain insulin signaling in type 2 diabetic (T2D) rats as well as study its role on the expression profile of AD related miRNA that is possibly related to AD pathology and its impact in the early diagnosis of AD in T2D.

Methods: After intraperitoneal injection of AG538, an insulin receptor substrate inhibitor, the induced rats were orally administrated metformin and oil for 21 days.

Results: We identified significant disturbances of insulin signaling in the brain of induced rats, including the inhibition of physiological, p-IRS1, p-AKT and p-GSK3β, as well as the enhancement of tau protein phosphorylation; these effects were clearly attenuated by treatment. Remarkably, AD associated pathological proteins, such as oxidative stress, inflammation, BACE-1, APP and Aβ42 were noticeably increased and these effects were significantly revoked after treatment. Interestingly, the expression profile of AD related miRNAs in sera and brain tissues displayed its neuro protection role.

Conclusion: These findings shed light on the specific roles of insulin signaling in T2D-mediated AD like neurotoxicity. Thus, an understanding of the regulation of brain insulin signaling may provide novel insights into potential therapeutic targets for T2D-mediated AD-like neurotoxicity.

Biography

Shaymaa Abdulghaffar Abdulmalek received her PhD in Alexandria University, Egypt in 2017. Currently, she is working as a Lecturer of Biochemistry in Alexandria University, Egypt. Her research includes molecular therapy of Alzheimer's disease, neuroinflammation, cell signaling and the biochemical parameters in diseases.

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Muhammad Asif Rasheed, J Alzheimers Dis Parkinsonism 2018, Volume 8
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In silico identification of novel ApoE4 inhibitor for Alzheimer's disease therapy

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ApoE4 is a major genetic risk factor due to its increased incidence of developing Alzheimer's disease. The study was designed to predict such compounds that may help in designing drugs 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). Six synthetic compounds (in clinical trials) were docked with target proteins to compare and analyze the docking results with natural compounds. Compounds S-allyl-L-cysteine, epicatechin gallate and fulvic acid show 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 showed high binding affinity of -7.2, same as that of natural compound S-allyl-L-cysteine which showed high binding affinity of -7.1 but did not show hydrogen bonding with any amino acid residue. Protein-protein interactions of ApoE4 showed physical and functional interaction with related proteins. Our study predicted 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.

Biography

Muhammad Asif Rasheed recently completed PhD studies from Huazhong Agricultural University, Wuhan, China. He has his expertise in Bioinformatics and passion in improving health and wellbeing. He applied different bioinformatics approaches to predict the virulence factors in *Mycoplasma bovis* bacteria. He simultaneously published review articles by applying different bioinformatics tools on proteins related to liver cirrhosis. Currently, he is working on therapeutic aspects of Alzheimer's disease.

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Notes:

Etrat Hooshmandi et al., J Alzheimers Dis Parkinsonism 2018, Volume 8
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Carbamylated erythropoietin-FC protects hippocampus against Aβ induced memory deterioration in a rat model of Aβ toxicity: Considering Akt/GSK-3β, MAPKs and MMP-2

Etrat Hooshmandi[1], Fereshteh Motamedi[1], Maryam Moosavi[2], Rasoul Ghasemi[1] and **Nader Maghsoudi[1]**
[1]Shahid Beheshti University of Medical Sciences, Iran
[2]Shiraz University of Medical Sciences, Iran

Alzheimer's disease (AD) is a debilitating neurodegenerative disease, characterized by extracellular deposition of senile plaques, mostly amyloid β-protein (Aβ) and neuronal loss. It has been reported that erythropoietin (EPO) has neuroprotective effects in some models of neurodegenerative disease but because of its hematopoietic side effects, its derivatives lacking hematopoietic bioactivity is recommended. This study evaluated the neuroprotective effects of carbamylated erythropoietin-FC (CEPO-FC) against beta amyloid induced memory deficit. Adult male Wistar rats weighing 250–300 g were cannulated bilaterally into CA1. Aβ25–35 was administered intra hippocampally for four consecutive days (5 μg/2.5 μL/each side/day). CEPO-FC (500 or 5000 IU) was injected intraperitoneally during the days 4–9. Learning and memory performance of rats was assessed on days 10–13 using Morris water maze and then the hippocampi were isolated and the amount of activated forms of hippocampal MAPKs subfamily, Akt/GSK-3β and MMP-2 were analyzed by western blot. The behavioral results revealed that CEPO-FC treatment in both 500 and 5000 IU significantly reversed Aβ-induced learning and memory deterioration. Molecular analysis showed an increment of MAPKs and MMP-2 activity and an imbalance in Akt/GSK-3β signaling after Aβ25–35 administration. CEPO-FC treatment prevented the elevation hippocampal of P38, ERK, MMP-2 activity and also Akt/GSK-3β signaling impairment induced by Aβ25–35; however, it had no effect on c-Jun N-terminal kinase (JNK). It seems that CEPO-FC prevents Aβ-induced learning and memory deterioration and modulates hippocampal MAPKs, Akt/GSK-3β and MMP-2 activity. This study suggests CEPO-FC can be considered as a potential therapeutic strategy for memory deficits like AD.

Biography

Etrat Hooshmandi is a PhD student of Neuroscience at Shahid Beheshti University of Medical Sciences, Tehran, Iran. Her thesis and projects are about therapeutic targets and molecular signaling in rat models of Alzheimer's diseases.

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Notes:

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Scientific Tracks & Abstracts
Day 2

Dementia 2018

Day- 2

SESSIONS

Alzheimer's Disease: Diagnosis and Symptoms | Managing Dementia | Alzheimer's disease Imaging | Causes and Prevention of Alzheimer's | Vascular Dementia | Alzheimer's Clinical Trials and Studies

Chair: Peace Tsawodzi, Change for Zongo Youth, Ghana

SESSION INTRODUCTION

Title: Ionizing radiation for awakening LPS treated BV-2 microglia cells
Weonkuu Chung, Kyung Hee University Hospital, South Korea

Title: Pharmacological & non-pharmacological approaches to live well with Dementia
Arooge Shafi, Gosford Private Hospital, Australia

Title: Cortical thickness and surface area networks in Alzheimer's disease and behavioral variant frontotemporal dementia.
Vesna Vuksanovic, University of Aberdeen, UK

Title: Amyloidogenesis under experimental neurodegeneration in rats
Zagrebin V L, Volgograd State Medical University, Russia

Title: Ranolazina decrease inflammation and oxidative stress in neural cells in primary culture
Soraya L Valles, University of Valencia, Spain

Weonkuu Chung et al., J Alzheimers Dis Parkinsonism 2018, Volume 8
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Ionizing radiation for awakening LPS treated BV-2 microglia cells

Weonkuu Chung, Mi Ju Chung, Dong Wook Kim, Chan Woo Kim, Jong Kil Lee, Hak Young Lee, Geon-Ho Jahng and **Se Young Choi**
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The aim of this study was to investigate the mobility of microglia when irradiated with low dose radiation (5.4 Gy/3 fr and 9 Gy/5 fr) after inhibiting by high dose lipopolysaccharide (LPS) in microglia cells. BV-2 microglia cells were treated with various concentrations of LPS to determine the optimal concentration to inhibit their mobility and the change of microglia's mobility was defined with wound healing assay. The same experiment was conducted to confirm the effect of low dose irradiation on microglia's mobility after treating with LPS. As a result, we saw that 2 μg/mL of LPS suppressed the mobility of movement distance greatly for microglia cells to 2.96 μm. So, then we selected a 2 μg/ml of LPS for BV-2 cells as optimal concentrations. We found that LPS (2 μg/mL) inhibited microglia cells mobility significantly to 1.62 μm ($p<0.001$). Compared with the control group and the irradiated group, the control group and the RT5 group (9 Gy/5 fr) showed similar median movement distances (12.5 μm vs., 15.6 μm $p=0.98$). That means 9 Gy radiation dose didn't inhibit mobility of microglia cells. But we couldn't see that 5.4 Gy/3 fr group made enhanced effect for mobility of microglia cells. When the control group and the LPS+RT5 (9 Gy/5 fr) group were compared, we could define ionizing radiation make enhance median mobility from inhibited status to as like normal condition. (control G 13.79 μm vs., LPS + RT5 12.14 μm, $p=0.84$). In Summary, the activity of BV-2 cells was reduced by high dose LPS (2 μg/mL). It was also confirmed that the migration of suppressed BV-2 cells was similar to that of the control group when treated with low doses (9 Gy/5 fr) of radiation. We have found that low dose irradiation is one of the ways to increase the activity of microglia cells and we will continue to enhance mobility of microglia cells. If we know that ionizing irradiation can control microglia activity, we can use a radiation treatment for neurodegenerative disease such as Alzheimer's disease.

Biography

Weonkuu Chung did his Research Fellowship in Radiation Oncology Biology Lab, Duke Medical School. Durham, USA during March 1999- February 2000. During April 2009-February 2010 he worked as Professor in Radiation Oncology, Konyang University Hospital. From March 2010, he is working as Professor in Radiation Oncology, Kyung Hee University Hospital at Gangdong. He is a regular member of The Korean Society for Radiation Oncology, ESTRO and ASTRO.

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Arooge Shafi, J Alzheimers Dis Parkinsonism 2018, Volume 8
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Pharmacological & non-pharmacological approaches to live well with Dementia

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Dementia is a neurodegenerative syndrome of progressive decline in memory, intellect, social skills and overall function. Alzheimer´s disease accounts for approximately 75% of all dementia cases. Three out of 10, aged above 85 have dementia. In 2018, 425,416 people were living with Dementia in Australia, 55% amongst who were females. Number will rise to 1 million by 2056. Dementia is the second leading cause of death and also the single greatest cause of disability among older Australians. Initial approach is to find a balance between autonomy and safety. Anti- dementia meds, and rehabilitation & psychosocial interventions only delay cognitive & functional decline, however; don't prevent or modify dementia. Severity of behavioral & psychological symptoms of dementia (BPSD) increases the burden on their family. It is also one of the main reasons for nursing home placement. Worsening of BPSD should be first managed by excluding any underlying cause. Limited use of antipsychotics is recommended in BPSD. Average life expectancy after being diagnosed is around 10 years, depends on the type of Dementia as well. Main focus is to live well with Dementia through support, services, activities and exercise. Important to raise the issues of advanced health care directives, driving and future care arrangements early on. In 2018, dementia is estimated to cost Australia more than $15 billion.

Biography

Arooge Shafi practices at Gosford Private Hospital (North Gosford) & Brisbane Waters Private Hospital (Woy Woy) NSW, Australia. Area of his work are Stroke, major neurological disorders, Cognitive Impairment/Dementia, any brain or spinal cord injury. Mechanical fall, fractures, joint replacements, amputation, and chronic pain. Pulmonary, Cancer and Cardiac Rehab. Also reconditioning post prolong medical and surgical admission.

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Vesna Vuksanovic et al., J Alzheimers Dis Parkinsonism 2018, Volume 8
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Cortical thickness and surface area networks in Alzheimer's disease and behavioral variant frontotemporal dementia

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Motivated by prior data of cortical regional volume differences, we investigated changes in cortical structural networks in Alzheimer's disease (AD) and behavioral variant frontotemporal dementia (bvFTD). We estimated structural correlation from magnetic resonance image (MRI) measures of cortical thickness and surface area at 68 regions, in a total of 628 participants (202 healthy elderly (HE), 213 bvFTD and 213 AD). We used network modules (i.e., groups of regions that have a high density of connections within them, with a lower density of connections between groups) to estimate changes in cortical networks that attribute globally, locally and at the lobe level. We found that the strength of structural correlation differs in bvFTD and in AD group compared to HE. Global correlation of regional thinning is a marker of bvFTD condition and the surface area correlation is a marker of AD. Cortical thickness and surface area correlational networks show a quite distinctive hub like organization, which also differs both from normal and between the two forms of dementia. We conclude that bvFTD and AD are associated with structural imaging markers of brain network organization differently.

Biography

Vesna Vuksanovic is working as a Research Fellow at the University of Aberdeen, Biomedical Imaging Centre. She has her specialization in Neuroimaging in Health and Neurodegenerative Diseases. Her research interests include developing models of the brain as a network of complex interacting components; application of these models in the context of brain disorders in dementia and understanding the progression of neurodegenerative processes using computational modeling of neuroimaging data.

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Amyloidogenesis under experimental neurodegeneration in rats

Zagrebin V L[1], **Yermilov V V**[1], **Zub A V**[1], **Antoshkin O N**[2], **Sagrsyan S A**[1] and **Lipov D S**[1]
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Introduction: One of the mechanisms in the pathogenesis of proteinopathy is the formation of two types of histopathological inclusions: senile plaques with the main role of β-amyloid peptides formed during specific cleavage of amyloid precursor protein (APP), and neurofibrils formed by pathological forms of protein. Both of these components are currently considered as potential molecular targets, the impact of which will allow to slow down or stop the development of the neurodegenerative diseases.

Aim: The aim of the work was to identify amyloid proteinopathy in the cerebral cortex during experimental neurodegeneration.

Materials & Methods: The study was performed on 20 white male rats, 24 months of age. The physical and process stress effect was reproduced for seven days.

Discussions & Results: Neurons of experimental group had a predominantly large bright, almost transparent nucleus, occupying more than half of the cytoplasm. The nuclei were eccentrically located with segregated nucleoli of irregular elongated shape. The basophilic substance was practically not detected in the cytoplasm when stained with thionin. Neurons with clear signs of apoptosis were noted. Congo red staining for amyloid revealed intracellular inclusions. Extracellular fibrillar structures were also found. In the group of intact animals, chromatin in the nucleus was clearly pronounced and a sufficient amount of basophilic substance was observed in the perikaryon.

Conclusions: Neurodegenerative disturbance in neurons of the cerebral cortex are characterized by apoptosis and proteinopathy with a pronounced predominance of intracellular and extracellular amyloid.

Biography

Zagrebian V L completed his PhD from the Volgograd State Medical University. Later he become the Vice Dean of Medical Faculty and was elected as Head of the Department of Histology, Embryology and Cytology of Volgograd State Medical University. He was elected as President of the Federation of Youth Scientific Societies of Medical Universities of Russian Federation. He is also an Associate Professor, who has published more than 150 papers in reputed journals and has been serving as an Issuing Editor of a scientific journal of Volgograd State Medical University.

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Soraya L Valles et al., J Alzheimers Dis Parkinsonism 2018, Volume 8
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Ranolazina decrease inflammation and oxidative stress in neural cells in primary culture

Soraya L Valles, Adrian Jorda, Martin Aldasoro, Patricia Marchio, Constanza Aldasoro, Sol Guerra-Ojeda, M Dolores Mauricio and **Jose M Vila**
University of Valencia, Spain

Ranolazine is a piperizene drug used in heart attack. This drug deserves not only to be clinically studied but also registered as medicine in particular against serious diseases including cardiovascular disease and this drug has been approved by European Medicament Agency. Our group has demonstrated a decrease of inflammation and oxidative stress in neural cells of primary culture by adding Aβ1-42 compared with control cells. Also, in this investigation we detect neural decrease in cell death induced by addition of the toxic peptide. Future studies looking for ranolazine action in other brain cells such as oligodendroglia or microglia will be assayed. Also, a clinical study of patients with Alzheimer' disease will be necessary.

Biography

Soraya L Valles are working as Assistant Professor in Department of Physiology, School of Medicine and University of Valencia, Spain. She is specialized in biochemical and molecular biology. Her areas of research interests are discovery of novel pharmaceutical products that can be developed to novel drugs in particular anticancer and Alzheimer's disease and discover the pathways produced by the drugs in illness as well as elucidating and understanding the mechanisms of action.

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