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Scientific Tracks & Abstracts
Day 1

Euro Dementia 2018

Sessions:

Alzheimer's Imaging and Clinical trials | Dementia Care Practice & Awareness | Mixed Dementia Animal | Models & Translational Medicine | Therapeutic Targets & Mechanisms for Treatment | Others

Session Chair
Don Kulasiri
Lincoln University, New Zealand

Session Introduction

Title: **Pilot of an innovative and simple history based screening tool for dementia community clinics and acute medical hospitals**
V R Badrakalimuthu, Parklands Hospital, UK

Title: **The importance of platform in public-private partnerships and social care for Alzheimer's disease**
Manabu Tamura, Ministry of Economy, Trade and Industry, Japan

Title: **The future of Dementia & Alzheimer and the unexpected bioenergetic role of Neuromelanin**
Arturo Solís Herrera, Human Photosynthesis® Research Centre, Mexico

Title: **Neuropathophysiology cross-talk between Alzheimer's disease and Parkinson's disease**
Li Zeng, National Neuroscience Institute, Singapore

Title: **MRI in Dementia: Characteristics and Relationship of Atrophy and Cognitive Function**
Nedim Ongun, Burdur State Hospital, Turkey

Title: **The role of N-acetylglucosaminyltransferase III in Alzheimer's disease progression**
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Title: **Time to wake up to Sleep disorders in Dementia**
Santosh Bangar, MIND Clinic for Senior Citizens, India

Title: **Targeting hypoxic signals as a therapeutic approach to Alzheimer´s Disease**
Mario Durán-Prado, University of Castilla-La Mancha, Spain

Vellingiri Raja Badrakalimuthu, J Alzheimers Dis Parkinsonism 2018, Volume 8
DOI:10.4172/2161-0460-C3-042

FORGET: Pilot of an innovative and simple history based screening tool for dementia community clinics and acute medical hospitals

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FORGET, an innovative way of capturing clinical history whilst screening patients presenting with a symptom of cognitive impairment for a diagnosis of dementia. Thirty consecutive liaison assessments were carried out using history collection based on FORGET and MMSE. Sensitivity, specificity, positive predictive value, negative predictive value and odds ratio were calculated for FORGET (cut-off 3+), in diagnosing dementia. Out of 30 referrals to liaison, 20 were diagnosed with dementia. A score of more than 3 on FORGET had a sensitivity of 95% (95% CI, 75.13%–99.87%), specificity of 90% (95% CI, 55.50%–99.75%), PPV of 95% (95% CI, 75.13%–99.87%) and NPV of 90% (95% CI, 55.50%–99.75%) for diagnosis of dementia. The odds ratios for diagnosis of dementia with FORGET score of 3+ was 171 (95% CI 9.569–3055.681; p=0.0005; z=3.49) and for MMSE of 27 and lower, were odds ratio 19.133 (95% CI, 0.88–415.90; p=0.060; z=1.879). Combined cut –off of MMSE of 27 and lower and FORGET of 3+ provided an odd ratio of 273 (95% CI, 10.197–7309.229; p=0.0008; z=3.344) for a diagnosis of dementia. FORGET as a screening tool at a score of more than 3 has a good sensitivity and specificity and is associated with significant odds ratio for the diagnosis of dementia.

Recent Publications

1. P Prevezanos, V R Badrakalimuthu and T Eddy (2017) When mania is not mania: a case of frontotemporal dementia. Progress in Neurology & Psychiatry 21(3):8–9.
2. V R Badrakalimuthu (2017) FORGET: A screening tool for dementia. Geriatric Medicine Journal 47(7):20–23.
3. V R Badrakalimuthu and S Barclay (2013) Do people with dementia die at their preferred location of death? A systematic literature review and narrative synthesis. Journal of Age & Aging 1–7.
4. V R Badrakalimuthu (2013) Cholinesterase inhibitors for co-morbid Alzheimer's disease dementia and schizophrenia: Systematic review and meta-analysis. WJNS 3:57–60.
5. V R Badrakalimuthu and A Tarbuck (2012) Anxiety: the hidden element in dementia. Advances in Psychiatric Treatment 18:119–128.

Biograpy

Vellingiri Raja Badrakalimuthu is a Consultant Old Age Psychiatrist at Parklands Hospital, Basingstoke UK. His liaison team was shortlisted for one among four best OPMH teams in the UK in 2015. He has published 23 articles in reputed journals and books.

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Notes:

Manabu Tamura et al., J Alzheimers Dis Parkinsonism 2018, Volume 8
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The importance of platform in public-private partnerships and social care for Alzheimer's disease

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In patients with Alzheimer's disease (AD) who do not have surgical indications, anti-AD therapy is usually administered, while there are only four more or less effective drugs available. Of note, currently no medication exists, which can cure AD and all therapeutics may only slow the disease progression. Considering that, as expected, the number of AD patients will increase, gradually making it more difficult for doctors to manage all cases of dementia, which will require modification of the healthcare system in general. It is particularly important in Japan, with a now rapidly aging society; the government should clearly recognize possible increase in the incidence of dementia. Currently, there are a variety of neuroimaging initiatives for AD worldwide (ADNI, CATI, etc.), and several clinical trials have been initiated (EPAD consortium, A4, GAP foundation, Memento, etc.). Japan thus needs international collaboration with medical practitioners and scientists from other countries. On the other hand, it is important to create AD platform in a way of new and innovative public-private partnership (PPP). This AD platform should aim at dementia research as the main target (e.g., risk reduction, prevention, early diagnosis, treatment, and life support), and also to establish common paradigm directed at development of the registry, investigated patients cohort, and biobank. Collaboration of private enterprises, academia and public entities (e.g., basic science in MEXT, clinical application in MLHW, integration of different fields in METI) may have an immediate impact on the development of dementia research. In addition, Japan may further accelerate AD studies at a new system for medical research and development (Japan Agency for Medical Research and Development–AMED), which has extended its activities and currently grips project and budget management (e.g., new orange plan in MLHW). There are no doubts that fair evaluation of clinical results in AD considering COI (conflict of interests) should be done. In addition, it is important to make this information available to public in an easy-to-understand and accurate manner. Practical analysis of social environmental factors leading to AD, appropriate preventive measures, and treatment results are required in the future, since currently there is no therapeutic agent for AD with clearly confirmed clinical efficacy.

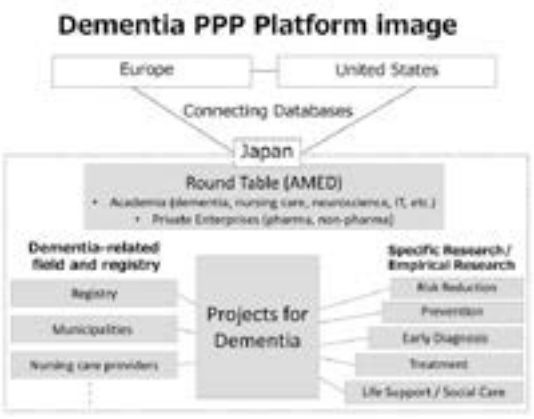

Figure 1. Scheme for PPP dementia platform.

Recent Publications

1. Saito T, Tamura M, Chernov M F, Ikuta S, Muragaki Y, et al. (2018) Neurophysiological monitoring and awake craniotomy for resection of intracranial gliomas. Progress in Neurological Surgery 30:117–158.
2. Aonuma S, Gomez-Tames J, Laakso I, Takakura T, Tamura M, et al. (2018) A high-resolution computational localization method for transcranial magnetic stimulation mapping. Neuroimage 172:85–93.

3. Suzuki A, Maruyama T, Nitta M, Komori T, Ikuta S, et al. (2018) Evaluation of DNA ploidy with intraoperative flow cytometry may predict long-term survival of patients with supratentorial low-grade gliomas: analysis of 102 cases. Clinical Neurology and Neurosurgery 168:46–53.
4. Takakura T, Muragaki Y, Tamura M, Maruyama T, Nitta M, et al. (2017) Navigated transcranial magnetic stimulation for glioma removal: prognostic value in motor function recovery from postsurgical neurological deficits. J Neurosurg 127(4):877–891.
5. Motogi J, Sugiyama Y, Laakso I, Hirata A, Inui K, Tamura M, Muragaki Y, et al. (2016) Why intra-epidermal electrical stimulation achieves stimulation of small fibres selectively: a simulation study. Phys Med Biol 61:4479–4490.

Biograpy

Manabu Tamura got his Physician's License in 1995 and completed his PhD in 2009 from Wakayama Medical University and moved to Tokyo Women's Medical University (Advanced Techno-Surgery and Neurosurgery) in 2017. He is the Deputy Director of Bio-Industry Division, Commerce and Service Industry Policy Group, Ministry of Economy, Trade and Industry. He has published more than 55 papers in reputed journals and has been serving at the Japanese Board of Neurological Surgery.

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The future of dementia and Alzheimer's and the unexpected bioenergetic role of neuromelanin

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Dementia means a decline in mental ability severe enough to interfere with daily life. Alzheimer´s disease (AD) is the most common type of dementia. Mental functions frequently impaired are: memory, language and communication, attention, concentration, reasoning and judgment, interpretation of visual perception. Usually symptoms start out slowly and gradually get worse. Memory loss is often one of the earliest symptoms of Alzheimer´s. Unfortunately, there is not a cure. So far, it is an erroneous belief that the brain gets energy burning (oxidizing) glucose. However, among the contradictions inside that theory, we have the fact that glucose and oxygen are not combined spontaneously within the blood or plasma, but until they are inside the cell, despite the combination of oxygen is abrupt and is not easily controlled. The foregoing is only a sample of the controversies that can be found in trying to explain the mechanisms by which glucose is the source of energy. In fact, glucose is the universal building block precursor, but cannot provide the energy that its own metabolism requires. Energy may be defined as everything that produces a change; our body and the brain take it from the light through the dissociation of the water molecule. The dissociation of the water molecule is performed by chlorophyll in the plants, and in the CNS the neuromelanin. Cognitive alterations in dementia and Alzheimer´s are extensive, which is congruent with the observed fact that in any system, when energy is the problem, the fault is widespread. This explains that the depigmentation of the substantia nigra and the locus coeruleus are a frequent finding in dementia and AD. Brain chemical reactions are surprisingly accurate, therefore the energy they require is surprisingly accurate, and it is precisely the way neuro melanin releases energy, in the form of H2 and high-energy electrons.

Recent Publications

1. Solis Herrera, A Arias Esparza, M C Solis Arias, P E Barreto, George Li, et al. (2016) Unsuspected intrinsic property of melanin to dissociate the water molecule can be used for the treatment of CNS diseases. CNS & Neurological Disorders–Drug Targets 15:2.
2. Aliev Gjumrakch, Solis Herrera, A Li, Yi Kaminsky, Yuri G Yakhno, et al. (2013) Human photosynthesis, the ultimate answer to the long term mystery of Kleiber´s law or E=M3/4: implications in the context of gerontology and neurodegenerative diseases. Open Journal of Psychiatry 3:408–421.
3. Solis Herrera A, Arias Esparza M C and Solis Arias M P (2013) Intracellular free chemical energy and neurodegenerative diseases. World J. Med. Med. Sci. Res. 1(2):012–025.

Biograpy

Arturo Solis Herrera has completed his MD at the School of Medicine in the Instituto Politécnico Nacional, México and Post-doctoral studies from Facultad de Medicina, de la Universidad Nacional Autónoma de, México. He is the Director and Founder of Human Photosynthesis® Research Centre. He has published more than 45 papers in several journals and has been serving as an Editorial Board Member.

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Li Zeng et al., J Alzheimers Dis Parkinsonism 2018, Volume 8
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Neuropathophysiology cross-talk between Alzheimer's and Parkinson's disease

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Background: Leucine-rich repeat kinase 2 (LRRK2) mutations are the most common genetic cause of familial and sporadic Parkinson's disease (PD). Amyloid precursor protein (APP) is an important molecule in Alzheimer's disease (AD). Although recent research revealed that there might be common pathophysiological and genetic links between these two diseases, little is known about the potential biological interplay between LRRK2 and APP.

Materials & Methods: Here, we used LRRK2 G2019S mouse models and induced pluripotent stem cell (iPSC)–derived neurons from PD patients to investigate the potential pathophysiological interplay between LRRK2 and APP in PD.

Results: We demonstrate here that LRRK2 interacts with APP and LC/MS/MS analysis revealed LRRK2 phosphorylates APP at threonine-668 (T668). This in turn stimulates the production of the APP intracellular domain (AICD) and enhances AICD nuclear translocation, which is associated with the dopaminergic neuron loss in aging LRRK2 G2019S mutant mice. Importantly, we found that the expression of AICD exacerbates LRRK2 G2019S-induced neurotoxicity, whereas phospho-mutant AICD (T668A) has no effect on LRRK2 G2019S-induced neuronal loss in vitro and in LRRK2 G2019S mutant mice. Moreover, dopaminergic neurons generated from LRRK2 G2019S patient-derived induced pluripotent stem cells exhibit significantly elevated phospho-APP T668 levels relative to their counterparts derived from healthy individuals. The level of T668- phosphorylated APP is also significantly increased in LRRK2 G2019S post-mortem human brain tissues. Treatment with LRRK2 inhibitors reduces phosphorylation at T668 in LRRK2 G2019S patient's dopaminergic neurons and in LRRK2 G2019S mutant mice.

Discussion: APP and LRRK2 are physically and functionally linked, suggesting a shared pathway between the two most common neurodegenerative diseases that may help explain their overlapping pathology, especially in advanced stages of the respective disease.

Conclusions: APP is a substrate of LRRK2, and its phosphorylation promotes AICD function and neurotoxicity in PD.

Biograpy

Li Zeng is currently working as Senior Research Scientist and Principal Investigator at Neural Stem Cells Research Laboratory at National Neuroscience Institute, Singapore. He has published more than 25 papers in reputed journals and has been serving as an Editorial Board Member of repute.

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MRI in dementia: Characteristics and relationship of atrophy and cognitive function

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Introduction: The prevalence of dementia is rapidly increasing in developed countries because of significant increase in aging population. In order to be able to make a faster and more accurate decision, the importance of imaging in diagnosis and treatment is increasing steadily. Magnetic resonance imaging (MRI) is one method by which the extent, impact and possible etiology of regional brain pathology can be quantitatively assessed. The aim of this study is to reveal the imaging properties of dementia and investigate the relationship between MRI and cognitive score within the vascular dementia (VaD) and Alzheimer's disease (AD).

Methods: 1024 patients diagnosed with dementia were evaluated retrospectively and grouped according to clinical features. Demographic characteristics and risk factors were recorded. MRI scans were scored using Scheltens' scale and visual analogue scale. The Mini-Mental State Examination (MMSE) was used to assess the cognitive status. Patients were compared based on MRI measurements and MMSE scores.

Results: 242 patients without an MRI were excluded. 398 patients were probably suffering from AD with NINCDS-ADRDA criteria; 249 patients were probably suffering from VaD with NINDS-AIREN criteria and 135 patients were with other dementia subgroups. In MRI ratings, global atrophy (GA) and white matter hyperintensity (WMH) scores were significantly higher in VaD group ($p<0.001$) and medial temporal atrophy (MTA) scores were significantly higher in AD group ($p<0.001$). Age, WMH and MTA were significantly related to GA in VaD and AD groups. While WMH and MTA were associated with both groups, age was also associated with MTA in AD group. MMSE scores were associated with MTA in both VaD and AD. There was a significant association between MMSE and WMH in the VaD group but not in the AD group.

Conclusion: This study is important for the evaluation of dementia related imaging properties. We conclude that clinical dementia is accompanied by brain tissue loss, regardless of etiology. These results may affect future investigations aimed at primary or secondary prevention of VaD and AD. Although selected treatments may vary, clinical outcomes are likely to be tied to slowing or preventing brain tissue loss.

Biograpy

Nedim Ongun has completed his Neurology Residency and is working as a Neurologist. He is also pursuing his PhD in Physiology at the university. He has published more than 25 papers in reputed journals and has been serving as a Referee and Editorial Board Member.

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The role of N-acetylglucosaminyltransferase III in Alzheimer's disease progression

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The pathogenic mechanism of Alzheimer's disease (AD) has not been clearly defined, and many factors have been discovered to explain this multifactorial disorder. The alteration of glycoprotein glycans in AD has been highlighted recently. It has been reported that the bisecting N-acetylglucosamine (GlcNAc) levels were higher in the cerebrospinal fluid of most AD patients, which indicates that N-acetylglucosaminyltransferase III (GnT-III), a glycosyltransferase responsible for synthesizing a bisecting GlcNAc residue, may play an important role in the development of AD. In our previous studies, we demonstrated that the levels of GnT-III and bisecting GlcNAc were increased in AD models, and glucagon-like peptide-1 (GLP-1) receptor agonists could downregulate aberrant expression of GnT-III through the Akt/GSK-3β/β-catenin signaling pathway in neurons. Here, we further explored the role of GnT-III in AD progression. We overexpressed GnT-III in PC12 cells and found that the intracellular reactive oxygen species (ROS) was increased significantly in GnT-III overexpressing cells. The mitochondrial structure was damaged and the mitochondrial membrane potential ($\Delta\psi m$) tested by JC-1 probe was lower in GnT-III overexpressing cells, which indicated that the mitochondrial function might be damaged by aberrant GnT-III expression. Besides, we also tested the GLP-1 receptor signaling mediated by GLP-1 and found that overexpression of GnT-III disrupted the normal GLP-1 receptor signaling in neurons. In conclusion, our findings reveal that GnT-III could be a potential therapeutic target for AD.

Recent Publications

1. Wang Y, Chen S, Xu Z, Chen S T, Yao W B, et al. (2018) GLP-1 receptor agonists downregulate aberrant GnT-III expression in Alzheimer's disease models through the Akt/GSK-3β/β-catenin signaling. Neuropharmacology 131:190–199.
2. Chen S, Yin L, Xu Z, An F M, Liu A R, et al. (2016) Inhibiting receptor for advanced glycation end product (AGE) and oxidative stress involved in the protective effect mediated by glucagon-like peptide-1 receptor on AGE induced neuronal apoptosis. Neurosci Lett. 612:193–198.
3. An F M, Chen S, Xu Z, Yin L, Wang Y, et al. (2015) Glucagon-like peptide-1 regulates mitochondrial biogenesis and tau phosphorylation against advanced glycation end product-induced neuronal insult: Studies in vivo and in vitro. Neuroscience 300:75–84.

Biograpy

Ying Wang is a PhD student of Microbiology and Biochemical Pharmacy at the China Pharmaceutical University. She is investigating the molecular pathogenesis of Alzheimer's disease, especially about the relationship between protein glycosylation and Alzheimer's disease.

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Time to wake up to sleep disorders in dementia

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In the last few years, there has been an increase in the studies of sleep disorders and their role in the pathogenesis of dementia. Most notably, obstructive sleep apnoea (OSA) and rapid eye movement (REM) sleep behaviour disorder have been closely studied. We carried out a narrative review of OSA to study its association with Alzheimer's disease and mild cognitive impairment (MCI). OSA is a very common, yet undiagnosed sleep disorder, with high prevalence in older people. OSA is characterized by repetitive cessation or reduction of airflow due to upper airway obstruction. It is a well-known risk factor for vascular illnesses and has been implicated in the pathogenesis of stroke, hypertension and cardiac arrhythmias. The resultant chronic intermittent hypoxia and hypercapnia in undiagnosed OSA can result in cognitive impairment. Furthermore, OSA with cognitive impairment shares some features with Alzheimer's disease, such as involving genetic predisposition ApoE4, hippocampus and synaptic plasticity abnormalities. On balance, OSA has negative effects on cognition, most likely in the domain of attention, verbal and visual delayed long-term memory and executive functions. A still unanswered question is whether these deficits are primarily a consequence of sleep fragmentation and/or hypoxemia, or whether they co-exist independently from OSA. A thorough clinical examination with an emphasis on a comprehensive sleep history is cornerstone in the assessment of OSA. However, sleep study is required to make a definitive diagnosis. First line of treatment includes modification of lifestyle, for e.g., reduction of body weight, quitting smoking and alcohol use. The gold standard treatment is continuous positive airway pressure (CPAP); however, tolerability can be an issue, especially in older people diagnosed with dementia. Another important sleep problem important in neurodegenerative illnesses is REM sleep behaviour disorder (RBD), classed as a parasomnia. It has well established links with synucleinopathies, such as Parkinson's disease, Lewy body dementia (LBD) or multiple system atrophy. However, RBD can occur in patients with Alzheimer's disease, a non-synucleinopathy.

Recent Publications

1. Santosh Bangar, Abhishek Shastri, Hany El-Sayeh, and Andrea E Cavanna (2016) Women with epilepsy: clinically relevant issues. Functional Neurology 31(3):127–134.
2. Mehboob Yaqub, Yasir Akbar and Santosh Bangar (2016) Tourette's syndrome: Is there a causal link to violence? International Journal of Endorsing Health Science Research 4(1):7–14.
3. Abhishek Shastri, Santosh Bangar and John Holmes (2016) Obstructive sleep apnoea and dementia–is there a link? International Journal of Geriatric Psychiatry 31(4):400–5.

Biograpy

Santosh Bangar was trained in the UK to achieve a CCT in Geriatric Psychiatry with an Endorsement in Liaison Psychiatry of Older People. To pursue his interest in Neuropsychiatry, he studied at the University of Birmingham and achieved a merit. He practises as a Consultant Geriatric Neuropsychiatrist with special interest in Sleep Disorders. He has published articles in Obstructive Sleep Apnoea, Epilepsy, Tourette's syndrome and Delirium. He has been invited to international and national conferences and has served as a Peer Reviewer for prominent scientific journals.

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Targeting hypoxic signals as a therapeutic approach to Alzheimer's disease

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Hypoxia, "hypoxic signals" as increased mitochondrial radical oxygen species levels and hypoperfusion occurs early in Alzheimer`s disease, inducing white matter lesions and correlating with dementia. Indeed, these abnormalities turn towards a cerebral microvascular pathology which accompanies age-related cognitive dysfunction and neurodegeneration. Therefore, alleviating cerebral microvascular pathology through the blockade of early hypoxic signals becomes a promising strategy to slow down Alzheimer`s progression. We have described that hypoxic signals (as oxidative stress) in early stages preceding vascular damage, beta amyloid deposition and appearance of brain parenchymal hypoxia, are premorbid and prodromal indicators of Alzheimer`s in the 3xTg-AD murine model of the disease. Moreover, circulating beta amyloid peptide damages the cerebral microvasculature through mechanisms that involve an increase in hypoxic signals, specifically in the mitochondrial compartment of endothelial cells, as it is an increase in superoxide ion, driving to endothelial cell death and therefore, to a compromise of cerebral microvessels function. Our results obtained with endothelial cells cultures, exposed to beta amyloid peptide, in vitro, clearly indicate that hypoxic cell responses can be blocked using a mitochondrial protector as coenzyme Q10, a lipophilic antioxidant involved in electrons transport from the mitochondrial complex I to complexes II and III, which results in a protective effect against beta amyloid cell toxicity through raising the whole cell metabolic status. We have recently assayed the effect of targeting mitochondrial hypoxic signals, in vivo, in 3xTg-AD mice. Animals were fed from prodromal stages of the disease with ubiquinol (the reduced form of coenzyme Q10) diet, compared to vehicle diet and wild type mice. Firstly, hippocampal chronic inflammation and peripheral leukocytes-oxidative stress found in 3xTg-AD mice were reversed by ubiquinol, which was mirrored by a reversion of the mice neuropathological status. Brain parenchyma hypoxia, exacerbated in vehicle-fed 3xTg-AD mice also in colocalization with large and abundant beta amyloid plaques, disappeared upon intervention with ubiquinol as well as the amount and size of beta amyloid plaques. This is correlated with a reduction in collagen deposition in the basal lamina of brain microvessels, a clear indicator of an improved brain microvessels function. Altogether, our results indicate that combating hypoxic signals from early prodromal Alzheimer´s stages could be a successful strategy to improve microvessels function, reducing parenchymal hypoxia, accumulation of beta amyloid plaques and, maybe, improving the delivery of memantine.

Recent Publications

1. Frontiñán-Rubio, Javier, Sancho-Bielsa, Francisco Javier, Peinado, Juan Ramón, LaFerla, Frank, Giménez-Llort, Lidia, Durán-Prado, Mario and Alcain, Francisco Javier () Sex-dependent colocalization of hipoxia and beta-amyloid plaques in hippocampus and enthorhinal cortex in 3xTg-AD mice is reversed by long-term treatment with ubiquinol and vitamin C. In review (second) in Mol. Cell. Neurosci.
2. Durán-Prado Mario, Javier Frontiñán, Raquel Santiago-Mora, Juan Ramón Peinado, Cristina Parrado-Fernández, et al. (2014) Coenzyme q10 protects human endothelial cells from β-amyloid uptake and oxidative stress-induced injury. PLoS One 9(10): e109223.
3. Torres-Lista Virginia, Cristina Parrado-Fernández, Ismael Alvarez-Montón, Javier Frontiñán-Rubio, Mario Durán-Prado, et al. (2014) Neophobia, NQO1 and SIRT1 as premorbid and prodromal indicators of AD in 3xTg-AD mice. Behav. Brain Res. 271:140-6.

Biograpy

Mario Durán-Prado has a Degree in Biochemistry (with Honors, 2000, University of Córdoba), Master's Degree in Biochemistry and Molecular Biology (Magna Cum Laude, 2002, University of Córdoba) and PhD in Sciences (Magna Cum Laude, 2007, University of Córdoba). After a Post-doctoral training at the Group of Neuroimmunology of Inflammatory and Autoimmune Diseases, IPBLN-CSIC (2008-2010), he joined the newly-created Faculty of Medicine of Ciudad Real (University of Castilla-La Mancha, Spain) as Associate Professor in Cell Biology, being member of the Group of Oxidative Stress and Neurodegeneration (GEON). He works on neuropeptides/GPCRs and their involvement in neuroendocrine, oncological and cardiovascular pathologies. Since 2010, his research focuses towards the role of oxidative stress in the initiation and progression of neuropathologies as brain tumors and Alzheimer`s disease, focusing in the development of novel therapeutic approaches using diverse translational and multidisciplinary research models. He holds four national and international projects as Principal Investigator, also as a collaborator in two active projects funded by the H2020. He has more than 40 publications (h-index=20), some of them in high impact factor journals (>7).

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