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Day 1

Gregory Yeh et al., J Alzheimers Dis Parkinsonism 2019, Volume 9
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Stress-induced alzheimer's disease

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Alzheimer's Disease (AD) is a chronic but deadly neurodegenerative disorder. A significant burden on global public health, a cure for AD is still elusive. Recent clinical trials based on pathogenic theories of extra/intracellular protein aggregation resulting from oxidative stress or other environmental insults have encountered setbacks. Here, we report a significant and serendipitous case of an AD patient. It is the first time to follow a single patient over 32 plus years, where AD symptoms have presented and remised repeatedly. The effects of stress resulting in numerous ailments, e.g. memory loss, brain atrophy, high blood pressure, inflammations, decrease of immunity, etc. were observed in the five episodes of severe stress, indicating that the disease is stress-induced. An anti-stress lifestyle involving seven daily anti-stress methods were implemented, which remarkably led to the recovery of memory and retardation of disease progression. We discovered a relationship between stress/stress hormones and strain/effects of stress hormones and the pathways leading to a stress-induced molecular mechanism that accounts for the toxic free radicals (oxidants) and Aβ and Tau (anti-oxidants). Our mechanism may also be applied to other neurodegenerative diseases related to stress effects on proteins, e.g. alpha-synuclein in Parkinson's disease, superoxide dismutase in amyotrophic lateral sclerosis, IAPP amyloid in type-2 diabetes, etc. As chronic traumatic encephalopathy and post-traumatic stress disorder both lead to AD, the anti-stress program may very well be of help there, all indicating that the stress and the molecular mechanism deduced from can be a significant finding in recent years.

Biography

Gregory Yeh earned his B.S. degree in physics from Holy Cross College in 1957, his M.S. degree in engineering physics from Cornell University in 1960, and his Ph.D. degree in polymer physics from Case Institute of Technology in 1966. From 1960-64, he worked as a research physicist at Goodyear Tire and Rubber Company and then as a senior research physicist at General Tire and Rubber Company. After completing postdoctoral studies at Case Institute of Technology in 1966, Professor Yeh joined the faculty of the University of Michigan College of Engineering as an assistant professor in 1967. He was promoted to associate professor in 1969 and professor in 1972.

His work, documented in 80 scientific publications and numerous invited presentations at scientific meetings all over the world, spanned a wide range of timely topics, with emphasis on the morphology and kinetics of single and multiple polymeric systems and on solid-state polymer processing and deformation. He also made seminal contributions to the morphology and kinetics of strain-induced crystallization of polymers and to the elucidation of chain conformation in amorphous polymers.

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Ramel A Carlos et al., J Alzheimers Dis Parkinsonism 2019, Volume 9
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Obstructive sleep apnea in various cognitive disorders

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Introduction: Various research studies suggested an association between Obstructive Sleep Apnea (OSA) and various cognitive disorders, including Alzheimer's disease. The degree of OSA has been directly correlated with the severity of cognitive impairment. Stroke and vascular diseases are significant comorbidities in these patients. We report the occurrence of OSA in patients with various cognitive disorders on the Island of Guam and correlate the severity of OSA with the results of the neuropsychological testing and neuroimaging studies.

Method: A retrospective review of medical records of patients evaluated in The Neurology Clinic with the diagnosis of OSA in patients with various cognitive impairments from July 2016 to July 2018 was conducted. These include patients with Alzheimer's disease, vascular dementia, unspecified dementia and mild cognitive impairment.

Result: There were 375 patients with various cognitive impairments and 16% have been diagnosed with OSA. Among patients with OSA, 46% have severe OSA, 38% have moderate OSA and 16% have mild OSA. Severe impairment on Global Cognitive Scores (GCS) was seen in 60% of patients with severe OSA, 44% of moderate OSA and 20% of mild OSA. Moderate GCS were seen in 29% of patients with severe OSA, 39% of patients with moderate OSA and 30% of patients with mild OSA. Evidences of silent stroke were seen in 25% of patients and another 31% have leukoaraiosis on their neuroimaging studies. The occurrences of vascular diseases including hypertension, diabetes mellitus, hyperlipidemia and cardiac disorders were higher in those with severe and moderate OSA compared to those with mild OSA and without OSA.

Conclusion: Obstructive sleep apnea is a common comorbidity of patients with various forms of cognitive impairment. The severity of OSA correlates with the degree of impairment on neuropsychiatric testing. Neuroimaging studies demonstrated evidences of silent stroke and leukoaraiosis among these patients.

Biography

Ramel A Carlos is currently working as Neurologist at The Neurology Clinic at Guam, USA. He has published numerous research papers and articles in reputed journals and has various other achievements in the related studies. He has extended his valuable service towards the scientific community with his extensive research work.

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Mourad Tayebi, J Alzheimers Dis Parkinsonism 2019, Volume 9
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Single domain antibody fragments reverse cognitive function deficits and amyloid plaques in alzheimer's disease animal models

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An active and promising area of research for Alzheimer's disease (AD) is immunotherapy using antigens (active) or antibodies (passive) that target AD neuropathology. Senile plaques contain the beta-amyloid (Aβ) peptide that is derived from a longer precursor protein, amyloid precursor protein. Amyloid beta is produced as either a 40 or 42 amino acid peptide, the latter being more fibrillogenic and toxic than the shorter isoform. Initially produced as a soluble peptide, Aβ subsequently can form oligomers, a molecular complex of monomer units. Aβ oligomers are highly toxic to neurons and particularly damaging to synapses. There is strong evidence that oligomer accumulation may seed plaque aggregation and serves as an early molecular target for preventing AD. Interestingly, oligomers can be detected by antibodies based upon structure with less of a need to target the amino acid sequence of an individual protein making antibody development for oligomers a fascinating area to pursue. Antibodies developed against oligomers may be able to bind several mis-folded proteins implicated in neurodegenerative diseases. Immunotherapy studies have typically relied on the use of anti-Aβ antibodies targeting plaques in transgenic mouse models of AD, and subsequently translated to human clinical trials. However, the success rate of these translational studies has been limited. We have previously developed and characterized unique anti-Aβ single domain antibodies derived from camelids. These antibodies, we called PRIOAD, were able to (i) cross the in vitro and in vivo blood brain brain (BBB) in mice rats and in vitro human BBB model; (ii) bind with high affinity to soluble oligomers derived from synthetic and native human Aβ but not their monomeric and fibrils counterparts; and (iii) not induce neurotoxic effects and host immune responses in mice. PRIOADs were evaluated for their prophylactic and therapeutic efficacy in several AD animal models. Following a 2-weekly intraperitoneal administration of PRIOAD for 3 months, there was a significant reduction of Aβ plaque burden in these animals. More importantly, PRIOADs led to complete reversal of the cognitive deficits in these animals. The study was very encouraging and will be expanded to include larger number of animal cohorts prior to translation into human clinical trials.

Biography

Mourad Tayebi is a Professor of Biomedical Sciences and Director of Higher Degree Research at the School of Medicine, Western Sydney University. Mourad's research focuses on developing effective therapies and early diagnosis for Alzheimer's disease. Mourad's team developed novel therapeutics with the ability to transmigrate across the blood brain barrier and reverse cognitive deficits in animal models of Alzheimer's disease.

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Masoomeh Mohamadpour, J Alzheimers Dis Parkinsonism 2019, Volume 9
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Manufacture of herbal capsule for alzheimer's patients by incensole acetate, melittin and curcumin

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Alzheimer's disease is one of the major causes of dementia and the major degenerative brain disease in the world. Treatment of this disease is one of the main concerns of the World Health Organization in the next century. Alzheimer's patients are known to have progressive and chronic memory impairment, speech worsening and functional impairment. There are currently no definitive treatments for Alzheimer's disease. But herbal medicine can reduce the symptoms and make life easier for the patient, in other words, the process of further treatment means preventing the progression of the disease and controlling the symptoms, mental and behavioral disorders caused by it. Nowadays, due to the complications of many chemical drugs and patients prefer for the use of herbal medicines, there is a good platform for the development of this approach, to make the herbal capsule from the incensole acetate, miltinin and curcumin combination to help develop and alleviate the symptoms of Alzheimer's disease. The product should be completely natural without any chemical elements. In sterile conditions, the ingredients are mixed with 250 mg of incensole acetate, 150 mg of curcumin, 100 mg of maltitol by spatula, in the form of a homogeneous mixture of powdered toothpaste. The powder mixture was weighed to 500 mg with weights accurately in mg and then poured 'into 500 mg capsules. The mixture shows improvement in the Alzheimer's disease treatment.

Biography

Masoomeh Mohamadpour is currently working as an Assistant Professor at the Neuroscience Research Center, Iran University of Medical Sciences. Her research interest is in herbal medicine in Alzheimer's disease.

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Investigating the autophagy mechanisms of orientin in lipopolysaccharide-stimulated BV2 microglia cells

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Background & Aim: Neuro inflammation is a primary risk factor of Neurodegenerative Diseases (ND), with microglia cells under pathological conditions directly contributing to neuro inflammation. Induced autophagy has been known to therapeutically reduce neuro inflammation without exacerbating the pathological condition of the disease. Existing treatments for inducing autophagy in neuronal setting are few but effective, with some noted to have reached clinical trials phase II and much scientific support for new compounds to modulate autophagy in a neuronal setting. Hence, this study aims to focus on the autophagic inducing potential of orientin on lipopolysaccharides-stimulated BV2 microglial cells.

Method: BV2 microglia cells pre-treated with orientin at maximum non-toxic dose or MNTD (15 μM) and ½ MNTD (7.5 μM), for a 3-hour period, followed by induction of neuro inflammation via 0.1 μg/mL of Lipopolysaccharide (LPS) stimulation. Autophagolysosome production was qualitatively determined with Acridine Orange (AO) staining and expression of autophagy pathway proteins was analyzed via Western Blot analysis.

Result: The induction of intracellular autophagolysosomes, under MNTD and ½ MNTD treatment of orientin qualitatively determined by AO staining confirmed the near completion of autophagy, with particular noteworthy observation of low complete neuronal death. Western Blot results showed up regulation of autophagy proteins Beclin-1, ATG5 and LC3-II, highlighting upregulation of key autophagy pathways in autophagy vacuole formation.

Conclusion: Orientin possesses significant likelihood of contributing to field of autophagy inducing therapeutic agents for targeting neuro inflammation in neurodegenerative diseases. Its autophagy inducing properties most likely stem from its ability to directly affect the mTOR signaling pathways by down regulating PI3K-I/Akt and MAPK/Erk 1/2 signaling pathway, encouraging future studies are required to refine this hypothesis.

Biography

Joshua Kuruvilla has completed his graduation in Medical Biotechnology in Biological Sciences.

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