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2274th Conference



12th International Conference on

# **Alzheimer's Disease & Dementia**

October 29-31, 2018 | Valencia, Spain

# Keynote Forum Day 1

Dementia 2018

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Petr Zach
Charles University, Czech Republic

### Immunohistological analysis of neuronal length changes in patients with Alzheimer's disease

We measured the postmortem length of the pyramidal neurons in the cortical layer III in five cortical regions (anterior cingulate gyrus, posterior cingulate gyrus, entorhinal-, perirhinal-, and parahippocampal cortices) in control and Alzheimer's disease patient groups. Our hypothesis was that length of the pyramidal neurons would be smaller in the Alzheimer's disease group and also there would be shift in right left asymmetry. We found pyramidal neurons length asymmetry in controls in anterior and posterior cingulate gyri and in Alzheimer's disease patients only in entorhinal cortex. However, control-Alzheimer's disease group pyramidal neuron length comparison revealed no significance in perirhinal cortex, left side shorter in Alzheimer's disease patients compared to control group in parahippocampal gyrus, both left and right side shorter in Alzheimer's disease patients compared to control group in entorhinal and posterior cingulate cortex and right side shorter in Alzheimer's disease patients compared to control group in anterior cingulate gyrus. Also we measured numbers of DAPI and Fluoro-Jade B stained cells in previously menitoned cortical areas. Our hypothesis was that in Alzheimer's disease group there would be more cells stained with Fluoro-Jade B compared to controls. We found higher numbers of Fluro-Jade stained cells in all cortical regions in patients with Alzheimer's disease compared to controls. However, only anterior cingulate cortex showed significant increase in their numbers in patients with Alzheimer's disease. Our conclusion is that degeneration in Alzheimer's disease is caused more by shrinkage of the neurons rather then by reduction of their numbers.

### **Biography**

Petr Zach completed his PhD in Neurosciences in 2001. He is studying Classical Neuroanatomy of the brain in Alzheimer's disease by using MRI techniques. He is Head of the Department of Anatomy and also an Author of more then 25 publications in journals with impact factor. He teaches Anatomy/Psychiatry And Pharmacology to medical students at the Charles University.

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**Notes:** 



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Magdalena Turek
Care Homes Zapiecek, Poland

### Occupational burnout in caregivers working in care homes in Poland

We approach the problem of occupational burnout and the specificity of the profession of caregiver in our presentation with emphasis on the predictors of psychophysical exhaustion and work disappointment. The study part involved seventy seven employees of nursing homes and social welfare homes, sixty four women and thirteen men with different levels of education from different towns with a diverse population and the average age of participants were 38.76 years. Caregivers are understood as professionals, not dependent families. General hypothesis was verified, according to which among the carers of dependents there is a varied level of burnout syndrome dependent from the tested correlates. We verified three detailed hypotheses regarding the relationship between occupational burnout and its selected correlates like sociodemographic factors, a sense of loneliness and stress. The results obtained confirmed general hypothesis about the occurrence of a differentiated burnout syndrome among the caregivers of dependents and also hypotheses regarding the association of occupational burnout with the feeling of loneliness and stress. It was also possible to confirm partially the validity of the hypothesis regarding the relationship of occupational burnout (in relation to some predictors) with sociodemographic factors such as age, place of residence and marital status. In contrast to most studies on occupational burnout conducted in our country, our work showed that caregivers living alone complain less about the lack a sense of professional effectiveness and psychophysical exhaustion than people in relationships.

### **Biography**

Magdalena Turek experienced manager with a demonstrated history of working in care homes for the elderly who suffer Alzheimer's disease and dementia. A specialist in the field of Alzheimer care with relentless curiosity, intellectual and creative personality. Strong professional with a Master's degree in Psychology from SWPS University of Social Sciences and Humanities, BA (Hons) degree in Tourism Management from Bournemouth University and course in Psychology of Addiction from Oxford University. Member of British Psychological Society BPS.

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# Keynote Forum Day 2

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# **Alzheimer's Disease & Dementia**

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# Mourad Tayebi

Western Sydney University, Australia

### Investigating age-related dementia in natural higher mammalian models

Proteinopathies such as Alzheimer's disease (AD) and Parkinson's disease (PD) are a group of disorders thought to be caused by abnormal folding or misfolding of beta apploid (Ab) and a group of the caused by a protein prot abnormal folding or misfolding of beta amyloid (Ab) and α-synuclein, respectively. Their pathogenesis is not well understood due to unresolved molecular mechanisms. This is further complicated by the lack of proper natural disease models that might be effective in aiding the investigation of the molecular mechanisms underlying these disorders. Dogs spontaneously deposit human-type Ab as they age and thus are a natural higher mammalian model of aging. The canine Aβ precursor protein (APP) is virtually identical to human APP. Previous studies demonstrated that aging dogs spontaneously accumulate human-type AB and parallel declines in cognition. Further, the outcomes of immunotherapy studies in aged dogs has predicted human clinical trial outcomes; clearance of  $A\beta$  plaques with little cognitive benefits. In more recent work, we show that canine derived  $A\beta$ was toxic to human neuronal cell lines and led to aggregation of human Aβ. Eastern grey kangaroos (EGK) display a typical movement disorder presentation associated with grass phalaris poisoning. We show that this disorder, known as phalaris staggers displays a parkinsonian type syndrome with associated Parkinson's-like signs and neuropathology, including synucleinopathy and neuromyopathy. Studies of proteinopathies have typically used transgenic mouse models and subsequently translated to human clinical trials. However, the success rate of these translational studies has been limited and unfortunately resulted in negative outcomes and some with adverse events. It is critical to identify and validate natural higher mammalian models of proteinopathies to investigate the molecular mechanisms underlying these disorders and test therapeutic outcomes prior to translation to human clinical trials.

### **Biography**

Mourad Tayebi is an Associate Professor in Biomedical Sciences at the School of Medicine at Western Sydney University, Australia. He is an International Expert in the field of protein misfolding diseases, with specific focus on investigating the molecular mechanisms underlying pathogenic protein misfolding and characterizing the misfolding associated with these disorders. His team is very active in the development of early blood diagnostic test screen for Alzheimer's and effective therapies for neurodegenerative diseases.

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