Kommunikation und Medien

Philipp Kreßirer

Pettenkoferstr. 8a
80336 München

Tel: +49 (0)89 4400-58070
Fax: +49 (0)89 4400-58072
E-Mail: philipp.kressirer@med.uni-muenchen.de
Web: www.klinikum.uni-muenchen.de

EXPERTEN-STATEMENTS

Prof. Yaakov Stern, Chief of Neurocognitive Science Division, Department of Neurology, Columbia University, New York, USA

Opportunities and challenges of reserve research: I will give a brief history of the concepts underlying reserve, including brain reserve, cognitive reserve and brain maintenance. The research approaches to understanding the neural basis of these concepts will be discussed. I will then describe the progress of a program designed to come to consensus on operational definitions for terms related to reserve and resilience among human and animal researchers.
I will also share some new data exploring the neural implementation of cognitive reserve.

Prof. Arfan Ikram, Head Department of Epidemiology, Erasmus University Medical Centre Rotterdam, Niederlande

Dementia has a considerable component of genetic heritability with recent genome-wide association studies (GWAS) identifying several genetic variants related to risk of dementia. Additionally, several GWAS have also identified risk variants linked to endophenotypes of reserve. Individually, these genetic variants have a very modest effect, but collectively the genetic burden can be considerable and can even be used for risk stratification purposes.
The question remains though to what extent this genetic risk is amenable by lifestyle interventions. This information is important not only for individual management of people worried about their dementia risk, but it can also provide leads to develop preventive strategies that effectively target high-risk persons.
This talk will discuss important work in this area and provide directions for future research. Recent findings from the UK Biobank and the Rotterdam Study will be highlighted.

Prof. Norbert Müller, Ärztlicher Direktor, Marion von Tessin Memory Zentrum, München

The pathogenetic mechanisms of Alzheimer's disease (AD) are up to now only partly understood. There is no doubt that 'immunosenescence', the aging of the (healthy) immune system, leads to impaired immune function and that aging is the main risk factor for AD. Also beyond doubt is that neuroinflammation plays a key role in the pathophysiology of the disease. However, whether inflammation is an underlying cause or a resulting condition in AD remains unresolved. At higher ages, communication in the peripheral and CNS immune systems, including both the initiation of the immune process and the down-regulation of inflammation, are impaired; this impaired communication might be one of the main factors contributing to the immune pathology of AD. The innate (monocytes, macrophages) and adaptive immune systems (T- and B-cells) have been shown to be upregulated in aging and AD. Mounting evidence indicates that microglia activation contributes to neuronal damage in neurodegenerative diseases, but beneficial aspects of microglia activation have also been identified. The possible role of C-reactive protein as inflammation marker is discussed. In this regard, we discuss the limitations and advantages of the protective effects of non-steroidal anti-inflammatory drugs (NSAIDs) and anti-inflammatory treatment options and identify possible future implications for AD therapy that might result from this underlying neuroinflammation.

Prof. Robert Perneczky, Leiter Abteilung für Gerontopsychiatrie und Alzheimer Therapie- und Forschungszentrum, Klinik und Poliklinik für Psychiatrie und Psychotherapie, LMU-Klinikum

Neuroinflammation in Alzheimer's disease; the ActiGliA study: The accumulation of pathologic proteins such as amyloid-β (Aβ) and tau in the brain characterise Alzheimer's disease (AD), but other changes including the degeneration of brain networks and a response of the immune system also play key roles. Brain structural and functional connectivity become impaired increasingly as AD progresses. Alterations of the intrinsic connectivity of brain resting state networks and fibre tracts correlate with disease severity and can be measured in vivo using BOLD functional magnetic resonance imaging (fMRI) and diffusion tensor imaging (DTI) respectively. The role of glial activation in the neuropathology of AD has also been widely recognised both as a consequence and as a contributing factor for the formation of Aβ and tau aggregation. Immune surveillance in the

Kommunikation und Medien

Tel: +49 (0)89 4400-58070
Fax: +49 (0)89 4400-58072
E-Mail: philipp.kressirer@med.uni-muenchen.de
Web: www.klinikum.uni-muenchen.de

brain is mainly due to astroglial and microglial cells and microglial activation can be measured reliably by TSPO positron emission tomography (PET). To understand better important associations between network degeneration and immune response in AD, we initiated the ActiGliA prospective cohort study in 2018. Here we present initial results of the first ≈100 patients, including baseline clinical and biomarker characteristics of the cohort and topographical associations between disease-stage dependent microglial activation and resting state connectivity changes. In line with previous findings, we confirm that brain functional and structural connectivity are disrupted and microglial activity is elevated in AD; however, we also show that significant inter-subject heterogeneity exists, warranting more detailed correlation with individual characteristics including symptom severity and Aβ and tau status.

Prof. Michael Ewers, Professor für Neuroimaging, Institut für Schlaganfall- und Demenzforschung, LMU-Klinikum

Michael Ewers' research focuses on neuroimaging-assessed brain changes in Alzheimer's disease, based at the Institute for Stroke and Dementia Research (ISD). The major research interest is to understand structural and functional network changes that accelerate or delay the onset of dementia symptoms. For reserve, a major approach is to use graph theoretical analysis to both resting state and task-based MRI in order to identify functional brain changes that underlie higher resilience of cognitive abilities during the course of Alzheimer's disease. The identification of such protective brain mechanism are sought to be targeted by therapeutic intervention in secondary prevention of Alzheimer's disease.

Kommunikation und Medien

Tel: +49 (0)89 4400-58070
Fax: +49 (0)89 4400-58072
E-Mail: philipp.kressirer@med.uni-muenchen.de
Web: www.klinikum.uni-muenchen.de