

ORAL SESSION: Neurodegeneration: from high risk groups to transdiagnostic signatures

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Oral Sessions

Presentations

Changes in functional connectivity associated with vascular burden in person at-risk of AD

Cross-sectional studies suggest that cardiovascular risk factors are associated with resting-state functional connectivity (FC) in aging and Alzheimer's disease (AD) (Liu 2013, Haight et al. 2015, Rashid et al. 2019, Zonneveld et al. 2019). However, evidence is missing regarding vascular-related longitudinal changes in FC. We investigated whether plasma cholesterol and blood pressure levels are associated with changes in brain FC in asymptomatic individuals at risk for AD.

Presenter

Theresa Köbe, McGill University Montréal, Quebec
Canada

Transcriptomic Analysis of Alzheimer's Disease Associated Brain Hypometabolism

Alzheimer's disease (AD) starts decades before clinical symptoms appear. Low glucose utilization in regions of the cerebral cortex mark early AD and is informative for clinical diagnosis. Fluorodeoxyglucose positron emission tomography (FDG-PET) is a primary tool for the diagnosis of dementia and its subtypes. FDG-PET measures glucose metabolism within the brain [Buckner et al., 2005; Friedland et al., 1983].

Presenter

Sejal Patel, PhD, Centre for Addiction and Mental Health
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Canada

Network Diffusion Model Enhances Predictions of Future Tau-PET Burden in Alzheimer's Patients

The aggregation of tau is being increasingly recognized as a trustworthy biomarker preceding the appearance of Alzheimer's disease (AD) symptoms. Predicting a patient's future tau burden is important for clinical trial design as this prediction can assist resource planning and inform patient inclusion decisions. Here we use a group of subjects clinically diagnosed with either AD dementia (N=28) or mild cognitive impairment (MCI) likely due to AD (N=60) to investigate whether a network-based pathological spread model could predict future tau accumulation based on the regional standardized uptake value ratios for tau (tau-SUV_R) and amyloid (amyloid-SUV_R) determined at baseline.

Presenter

Pablo Damasceno, UCSF
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Generalizable, reproducible, and interpretable imaging biomarkers for Alzheimer's disease

Brain structural alterations are promising biomarkers for tracking disease progression and diagnosing Alzheimer's disease (AD). Deep learning methods have increasingly been used in the computer-aided diagnosis of AD due to their ability to learn to optimize feature representation and robustness. Also, a comprehensive evaluation of the neurobiological basis and generalizability of the end-to-end machine learning system should be given the highest priority[1-2].

Presenter

Dan Jin, Institute of Automation Chinese Academy of Sciences Beijing, Beijing
China

Brain disorders taxonomy from a transcriptomics point of view

Traditionally, human diseases have been identified and categorized based on their symptoms and phenotypes. While helpful in certain aspects, these categories still have many limitations. There is a high degree of variability among patients labeled within a disease category as well as accompanying comorbidities. Many disorders are considered 'spectrum' disorders with a wide range of clinical presentation, such as autism, schizophrenia or dementia [1]. We examined gene-disease associations (GDA), as well as disease-disease associations (DDA), of brain disorders from a transcriptomic perspective, using microarray gene expression data derived from human brain.

Presenter

Yashar Zeighami, Dr., McGill University Montreal, Quebec
Canada

Discovering Propagation Pattern of Neurodegeneration across Brain Networks

Alzheimer's disease (AD) is a complex neurodegenerative disease characterized with disconnection syndrome, where the brain network is progressively disrupted by neuropathological process. The neuropathological disruption may be caused for decades before an individual reached the AD stage. However, the pattern of the pathological burden spreading across brain regions along time is still unclear. Recent advances in non-invasive and in-vivo neuroimaging technology allow us to measure the pathophysiology burden in the whole brain. Thus, it is timely to learn the mechanism of cognitive decline from the unprecedented amount of longitudinal neuroimaging data. In this paper, we propose a novel linear mixed-effect model to explore three data science hypotheses one after another: (1) Does AD differentially affect hub nodes in the network? (2) Does the AD-related neurodegeneration start from hub node and propagate across network in a prion-like manner? (3) Does the propagation pattern follow the connectome pathway or spatial vicinity?

Presenter

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