PLATFORM ABSTRACTS

Network preservation analysis reveals dysregulated gene modules shared between Alzheimer's disease and temporal lobe epilepsy

Anna Harutyunyan[1], Alison Anderson[2], Nigel Jones[2, 1], Patrick Kwan[2, 1]

1. *Department of Medicine, Royal Melbourne Hospital, The University of Melbourne, Parkville, VIC, Australia*
2. *Department of Neuroscience, Central Clinical School, Monash University, Clayton, VIC, Australia*

Patients with Alzheimer's disease (AD) have increased risk of epilepsy. Shared pathophysiological features between AD and temporal lobe epilepsy (TLE) have been reported.

Objectives

To identify commonly dysregulated groups of genes and biological pathways in AD and TLE using a bioinformatic approach.

Methods

We included data from publicly available large microarray datasets of gene expression in brain tissues obtained from 128 individuals with TLE, 91 individuals with late onset AD and 81 non-demented controls (NDC). Using Network Analysis we constructed three consensus coexpression networks: AD vs. NDC, TLE vs. NDC, and AD vs. TLE. We then employed network preservation statistics to assess the preservation of network properties between the three consensus networks.

Results

NDC groups. Within these modules, sub-modules that annotate to immune system and axon myelination processes were the most differentially preserved between AD vs. TLE and TLE vs. NDC networks, suggesting shared processes between TLE and AD. Additionally, we identified a small module in AD and TLE networks that was not detected in the NDC group. Genes in this module annotate to inflammatory cytokines and interleukins. Across all data sets modules that associated with general homeostatic systems, metabolism and cell organization were similarly preserved.

Conclusion

This analysis identified gene modules associated with inflammatory processes to be significantly preserved in TLE and AD brain, implicating neuroinflammation as a potential common mechanism between these two diseases. Targeting neuroinflammation might represent a viable strategy to prevent epileptogenesis in AD patients.