Ruprecht-Karls-Universität Heidelberg
Medizinische Fakultät Mannheim
Dissertations-Kurzfassung

Neuroimaging biomarkers in Alcohol Use Disorder: clinical relevance and relapse prediction

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Alcohol Use Disorder (AUD), as a chronically relapsing disorder, is a worldwide public health problem. Neuroimaging studies reported alterations related to alcohol use from various aspects, which could be potential biomarkers in AUD. However, AUD's neuroimaging features need further description, and the underlying neural mechanisms are still not fully understood. This study aimed to identify neuroimaging biomarkers in AUD from the aspects of brain iron accumulation and neural patterns decoding alcohol cues, and their clinical relevance and relapse prediction using these novel biomarkers.

This study was based on secondary analyses of previous datasets. To examine brain iron accumulation, 186 individuals with AUD and 274 healthy participants were included. Quantitative Susceptibility Mapping (QSM), an emerging MRI technique developed for quantifying tissue magnetic susceptibility, was performed to measure the whole-brain iron level. Using an alcohol cue-reactivity task, functional magnetic resonance imaging (fMRI) data from 238 AUD individuals and 229 healthy participants were used to identify neural patterns during processing visual cues of alcohol. The processes of visual object recognition and reward appraisal of alcohol cues were separately modeled using Representational Similarity Analysis. To develop biomarkers in AUD, whole-brain iron levels and decoding involvements during cue-reactivity task were compared between AUD individuals and healthy participants. The relationship between drinking patterns and biomarkers was explored, and the decoding involvements during a cue-reactivity task were used for predicting relapse within six months. Moreover, connectivity analyses of fMRI were conducted to investigate communications between neural patterns and the rest of the brain.

Whole-brain analyses of QSM showed that the susceptibility in dorsal striatum (putamen and caudate) among AUD individuals was higher than in healthy participants, and was positively related to the Obsessive Compulsive Drinking Scale scores and the amount of drinking in the past three months. During decoding alcohol cues, AUD individuals, compared to healthy individuals, showed higher involvement of motor-related brain regions in the process of visual object recognition, and their reward, habit and executive networks were more engaged in appraising reward values. Connectivity analyses showed that the involved neural systems were widely connected with higher cognitive networks during alcohol cue processing in AUD individuals, and decoding involvements of frontal eye fields and dorsolateral prefrontal cortex could contribute to relapse prediction.

Higher iron accumulation in the dorsal striatum was observed in AUD. This surrogate for the brain iron level was linearly associated with compulsive drinking patterns and the recent amount of drinking, which provides a new biomarker in relation to brain iron accumulation for clinical practice. The decoding neural patterns during alcohol cue-reactivity provide insight into how AUD individuals differently decode alcohol-cues compared to healthy participants, from the componential processes of visual object recognition and reward appraisal. These identified patterns are suggested as biomarkers and potential therapeutic targets in AUD.