OPINION

The structural covariance network's rich-club connectivity is related to memory processes in mild cognitive impairment and alzheimer's disease

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ABSTRACT

Despite the fact that Medio temporal lobe volume changes are well-known features of Alzheimer's Disease (AD), grey matter volume changes may occur throughout the brain. Because of the underlying network structure, these distributed changes are not independent and can be described using a Structural Covariance Network (SCN).

Key Words: *Dementia*

INTRODUCTION

The most common cause of dementia is Alzheimer's disease (AD). The neuropathological hallmarks of alzheimer's disease are amyoid amyloid plaque accumulation, tau tangle formation, and neurodegeneration. Furthermore, hippocampus volume changes and memory decline are well-known features of Alzheimer's disease. Cortical volume and thickness changes may also occur outside of the Medio temporal lobe. These distributed changes are not independent, but may manifest as coherent changes as a result of the underlying network structure. Specific patterns of cortical thinning are common in alzheimer's disease and are thought to be indicative of disease progression. As a result, studying cortical organization could provide new insights into the progression of Alzheimer's disease.

Measures of brain morphology (for example, cortical thickness) have previously been shown to correlate across different brain regions. These correlations are thought to be related to functional and axonal connectivity, which underpins the Structural Covariance Network (SCN). The SCN has previously been shown to be less efficiently organized in AD compared to healthy controls. These studies relied on group-level SCNs, which were created by estimating the strength of connections between brain regions based on correlations of morphological measurements across a group of subjects. As a result, individual network characteristics were lacking, limiting future statistical analyses that can link SCNs to individual (e.g., demographic and clinical) factors and provide a better understanding of pathophysiology.

To address this limitation, several individual SCN methods for estimating subject specific structural covariance connectivity have been developed. For example, the author proposed a distance-based approach in which a subject's contribution is estimated in relation to a reference group. Furthermore, the author proposed an alternative method based on a cube-based approach, in which small 3D cubes represent nodes and connections are estimated based on grey matter morphology similarities. This individual SCN method builds on previous research by demonstrating that less efficiently organized SCNs are associated with poor cognitive performance and faster atrophy rates. Furthermore, changes in SCNs have been linked to amyloid and tau pathology, highlighting their potential in AD research. The goal of this study was to characterize cortical organization in cognitively impaired older people using the rich-club subnetwork of the Structural Covariance Network (SCN) and its relationship to cognitive function. The study's main finding is that a loss of nodal degree in the SCN's rich-club subnetwork correlates with lower memory performance and a smaller hippocampal volume in a memory clinic sample. The presence of hub nodes is one of the primary characteristics that distinguish random networks from efficiently organized networks such as the brain. The brain network's hubs tend to form a densely connected rich-club subnetwork, with a higher rich-club configuration corresponding to a greater number of connections between the network's hubs. In the current study, we found that a loss of rich-club configuration, i.e., a loss of connections between the hubs, is associated with impaired cognitive performance in the memory domain, as well as hippocampal volume loss.

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Previously, a lower nodal degree in the SCN's fronto-temporal regions was linked to memory loss. We show that the effect of nodal degree on memory performance is more widespread throughout the brain, specifically involving connections between hub nodes (i.e., the rich-club).

The more densely connected hub regions require more blood flow and metabolic activity due to higher rates of neural processing and information flow. It has previously been debated whether increased metabolic activity interacts with tau pathology in alzheimer's disease. Furthermore, hub regions in alzheimer's disease patients were found to be more prone to lesions than peripheral regions. This shows that hub region, and thus the rich-club subnetwork, are especially vulnerable in AD. Our findings support this theory and suggest that the vulnerability of hubs may provide an underlying mechanism for memory retrieval issues in AD. Potential age and cognitive performance differences were assessed using a one-way ANOVA test, while differences in the categorical variables sex and education were assessed using a chi-squared test. To assess potential group differences in the RCC, a one-way ANOVA test was used. Following that, the relationship between RCC and memory performance was examined using multivariable linear regression, with RCC as the dependent variable and raw VLT-dr scores as the independent variable, while controlling for age, gender, education level, and diagnostic group using two dummy variables representing MCI and AD. Similarly, the relationship between RCC and hippocampal volume was investigated using multivariable linear regression, which controlled for age, gender, total intracranial volume, and diagnostic group with two dummy variables representing MCI and AD. DTI analysis was previously used to study the rich-club phenomenon in relation to AD, revealing that connections in the rich-club were affected in early-onset AD. Other DTI studies, however, have found that the extent of rich-club configuration remains largely intact in AD. This, together with the current study's findings, suggests that the SCN has the potential to provide unique knowledge on interregional cortical associations in subjects with cognitive impairments, as well as valuable information to supplement DTI. The distance-based SCN is proposed as a quantitative MRI processing method that provides information on neurodegeneration and the underlying network of distributed dependent cortical regions in relation to cognitive function and is suitable for use in large population scale studies. We found that decreased rich-club connectivity in the SCN is associated with poor memory performance and hippocampal atrophy, implying that a loss of degree in the rich-club subnetwork may be associated with underlying memory loss in the context of MCI and dementia. This study adds to the growing body of evidence linking hub region interconnectivity to cognitive impairments and atrophy associated with alzheimer's disease. Future studies, preferably longitudinal, prospective or retrospective, should look into whether rich-club connectivity can be used to predict disease progression.