# Connectivity within the Default Mode Network after Traumatic Axonal Injury

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Traumatic axonal injury (TAI) is a common consequence of TBI in which the brain's white matter is mechanically torn by deceleration and rotational forces. Injury to axons after this type of injury causes significant impairments in cognitive functioning, but the association between disruption of structural connections (i.e., axons) and the brain's functional connectedness is not well understood. Studies examining integrity of white matter after TAI have found significant compromise to structures likely involved in the connectivity of the default mode network (DMN), a reliably elicited functional neural network with clinical implications. The discriminant and prognostic utilities of the DMN following traumatic axonal injury (TAI) have not been previously investigated.

This broad investigation was comprised of two related studies examining the utility of neuroimaging modalities as biomarkers of TAI. Resting-state magnetic resonance imaging (RS-MRI) and diffusion tensor imaging (DTI) sequences were acquired 6-11 months post-injury using a 3T scanner from 25 patients with TAI and 17 controls. Functional and neurocognitive outcomes were assessed the same day. The first study examined the utility of three approaches analyzing DMN integrity using RS-fMRI. The purpose was to identify the utility of each approach to distinguish between healthy and brain-injured individuals, and determine whether observed differences have clinical significance. The second study integrated functional and structural connectivity measures of the DMN to determine whether compromise to functional connectivity within this network can be explained by the degree of white matter compromise commonly observed after TAI.

The first study concluded that connectivity within the DMN is compromised after TAI, as all three methods demonstrated good ability to discriminate between healthy and injured brains. The second study suggests the functional disconnectedness within the DMN is in part due to compromise in structural connections observed after TAI. Neither the degree of functional or structural compromise to the DMN has clinical implications in TAI. In general, the two investigations suggest the DMN undergoes compromise after TAI, and connectivity between nodes of the network are valid markers of axonal injury.