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Disrupted Structural Connectivity in Prefrontal Areas in Borderline Personality Disorder

Disturbances are seen in white-matter structures responsible for the appropriate inhibition and control of emotions.

Many of the core features of borderline personality disorder (BPD; irritability, impulsivity, emotional instability) suggest disruption in frontolimbic brain circuits. Imaging studies have shown disruptions in the uncinate fasciculus, a white-matter tract connecting prefrontal regions to paralimbic temporal regions. The present researchers used diffusion tensor imaging to examine white-matter integrity in prefrontal fiber tracts in 18 women with BPD and 38 psychiatrically healthy controls.

Significant increases in radial diffusivity were seen in the right prefrontal white matter of people with BPD in a region correlated with the uncinate fasciculus, inferior anterior thalamic radiation, and fronto-occipital fasciculus. Similar increases in apparent diffusion coefficient (ADC) values for these right prefrontal regions were seen. Symptom severity was correlated with imaging findings: dysphoria negatively correlated with ADC values in the anterior thalamic radiation, and self-perception positively correlated with fractional anisotropy in the right corona radiata.

COMMENT

The white-matter structures identified here (uncinate fasciculus, anterior thalamic radiation) are components of frontolimbic and frontothalamic loops, which are responsible for the appropriate inhibition and control of emotions. These particular white-matter tracts continue to develop well into adulthood. This raises the interesting possibility that the disruption seen in this study, increased radial diffusivity (which suggests myelin disruption), is related to delayed maturation or underdevelopment of this part of the brain in individuals with BPD. Added to the growing evidence that stimulating circuit activity (at least in some parts of the cortex) can influence myelination (*Front Cell Neurosci* 2018 Nov 19), these findings have important implications for neuromodulatory treatments of BPD.

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Nenadifá I et al. Diffusion tensor imaging in borderline personality disorder showing prefrontal white matter alterations. **Compr Psychiatry** *2020 May 1; [e-pub]. (https://doi.org/10.1016/j.comppsych.2020.152172)*

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