"converted" into physical symptoms. Contemporary neuroscience has shown that the neural correlates for "repression" or memory suppression include dorsolateral prefrontal (DLPFC) cortex activation and hippocampal deactivation. Our objectives were: 1. Test this mechanism in CD and 2. Explore the neural correlates of the associated sensorimotor symptom.

Method Stressful events were elicited from the Life Events and Difficulties Schedule interview in 12 motor CD patients and 13 healthy controls and rated by a blinded panel for their likelihood to cause CD: severely threatening events were categorised as "escape" if their consequences might plausibly be mitigated by illness. In a block-design functional magnetic resonance imaging (fMRI) task, recall of those events (Escape condition) were compared to the recall of equally threatening non-escape control events (Severe condition).

Results Relative to controls, patients showed significant increased left DLPFC and decreased left hippocampus activity during the Escape versus Severe condition (compatible with memory suppression) and increased right supplementary motor area (SMA) and temporo-parietal junction (TPJ) activity. Patients failed to activate the right inferior frontal cortex (rIFC) during both conditions. Connectivity between amygdala and motor areas (SMA and cerebellum) was enhanced in patients relative to controls.

Conclusion These data offer support for the notion that the way adverse events are processed cognitively can lead to physical symptoms. A plausible mechanism for the onset of these symptoms may stem from abnormal emotional control (DLPFC, rIRC) leading to memory suppression (hippocampal) with symptomatic alterations in motor planning and body schema (SMA, TPJ).

009 THE NEURAL CORRELATES OF FREUDIAN "REPRESSION" IN CONVERSION DISORDER

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Objective Freud proposed that in Conversion disorder (CD), the affect attached to stressful memories is "repressed" and