The neural substrate of reversal learning in obsessive-compulsive disorder: an event-related fMRI study

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Obsessive-compulsive disorder (OCD) is a chronic invalidating neuropsychiatric disorder with a lifetime prevalence of about 2-3%. The essential features of OCD are recurrent inappropriate and anxiety-provoking thoughts (obessions) that lead to repetitive behaviours (compulsions). These features characterize this disease phenomenologically as a disorder in cognitive flexibility within an emotional context.
Reversal learning can be defined as the ability to alter behaviour when reinforcement contingencies change (Rolls, 1999) and can therefore be regarded as a measure of cognitive flexibility within an emotional/motivational context. Moreover, the processing of reversal learning is believed to be subserved by the orbitofrontal cortex in humans (e.g. Fellows et al., 2003). Previous neuropsychological studies in OCD that used tasks presumed to activate this brain region demonstrated impairments to various degrees (Abbruzzese et al., 1997; Nielen et al., 2002). However, neuroimaging studies showed the orbitofrontal cortex to be dysfunctional in resting-state paradigms consistently (e.g. Baxter et al., 1987). Neurocognitive tasks aimed at activating the orbitofrontal cortex within a neuroimaging environment have not been performed so far in OCD patients.
In the present study, the objective was to assess regional brain activity in OCD patients compared to normal controls while they performed a reversal learning task during functional magnetic resonance imaging (fMRI). In order to maximally avoid susceptibility artefacts for OFC signal, we used a sequence sensitive to signal in this area (Deichmann et al., 2003).
In the current presentation, preliminary results are discussed for 14 OCD subjects.

Abbruzzese et al. (1997) Neuropsychologia 35: 907-12
Baxter et al. (1987) Arch Gen Psychiatr 44: 211-18
Deichmann et al. (2003) Neuroimage 19: 430-41
Fellows et al. (2003) Brain 126: 1830-37
Nielen et al. (2002) J Affect Dis 69: 257-60
Rolls (1999). Neurocase 5: 301-12

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