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Abnormalities in metabolite concentrations in tourette's disorder and obsessive-compulsive disorder—A proton magnetic resonance spectroscopy study

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ABSTRACT

Introduction: Abnormal glutamatergic transmission in cortico-striato-thalamo-cortical (CSTC) circuits is thought to be involved in the pathophysiology of Tourette's disorder (TD) and obsessive-compulsive disorder (OCD). Using proton magnetic resonance spectroscopy, the current study aimed to investigate regional concentrations of glutamatergic compounds in TD and OCD patients in comparison to healthy control subjects (HC).
Material and methods: Twenty-three TD patients, 20 OCD patients and 22 HC were included. Short echo-time single-voxel 3T MRS was obtained from dorsal anterior cingulate cortex (dACC) and midline bilateral thalamus.
Results: The 3-group comparison showed a significant difference in choline concentration in the thalamus. Thalamic choline was highest in OCD patients, showing a significant difference with TD, and a trend compared to HC (post-hoc analyses). Glutamine in dACC correlated negatively with tic severity scores in TD patients, while glutamate in thalamus correlated positively with anxiety severity scores in OCD patients.
Conclusions: These findings suggest subtle differences in metabolites in CSTC areas between TD and OCD. Alterations of choline concentrations seem to be both regional (only in thalamus, not in dACC) and disease specific in OCD pathology. The findings need replication in larger groups, but encourage further research into glutamatergic metabolites in TD and OCD.

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1. Introduction

Tourette's disorder (TD) and obsessive-compulsive disorder (OCD) are neurodevelopmental disorders with repetitive behaviours as their core symptoms. TD is characterized by motor and phonic tics while OCD is characterized by obsessions (recurrent intrusive thoughts) and compulsions (repetitive ritual behaviour). Up to 66% of adult TD patients have comorbid obsessive-compulsive (OC) symptoms (Freeman et al., 2000). TD and OCD share a strong yet complex genetic relationship as evidenced by family and genetic studies (Davis et al., 2013; Leckman et al., 1995). At the neuronal level, both disorders are associated with dysfunctions in cortico-striato-thalamo-cortical (CSTC) circuits (Menzies et al., 2008). The parallel CSTC circuits originate from the medial prefrontal cortex (mPFC)/anterior cingulate cortex (ACC), the orbito-frontal cortex (OFC) or the dorsolateral prefrontal cortex (DLPFC), and connect to the thalamus via their respective targets within the striatum (Alexander et al., 1986). The parallel CSTC circuits have been related to sensori-motor, cognitive and emotional processing (Alexander et al., 1986). The ACC, as an important node in the CSTC circuits, has a functional role in initiation and modula-

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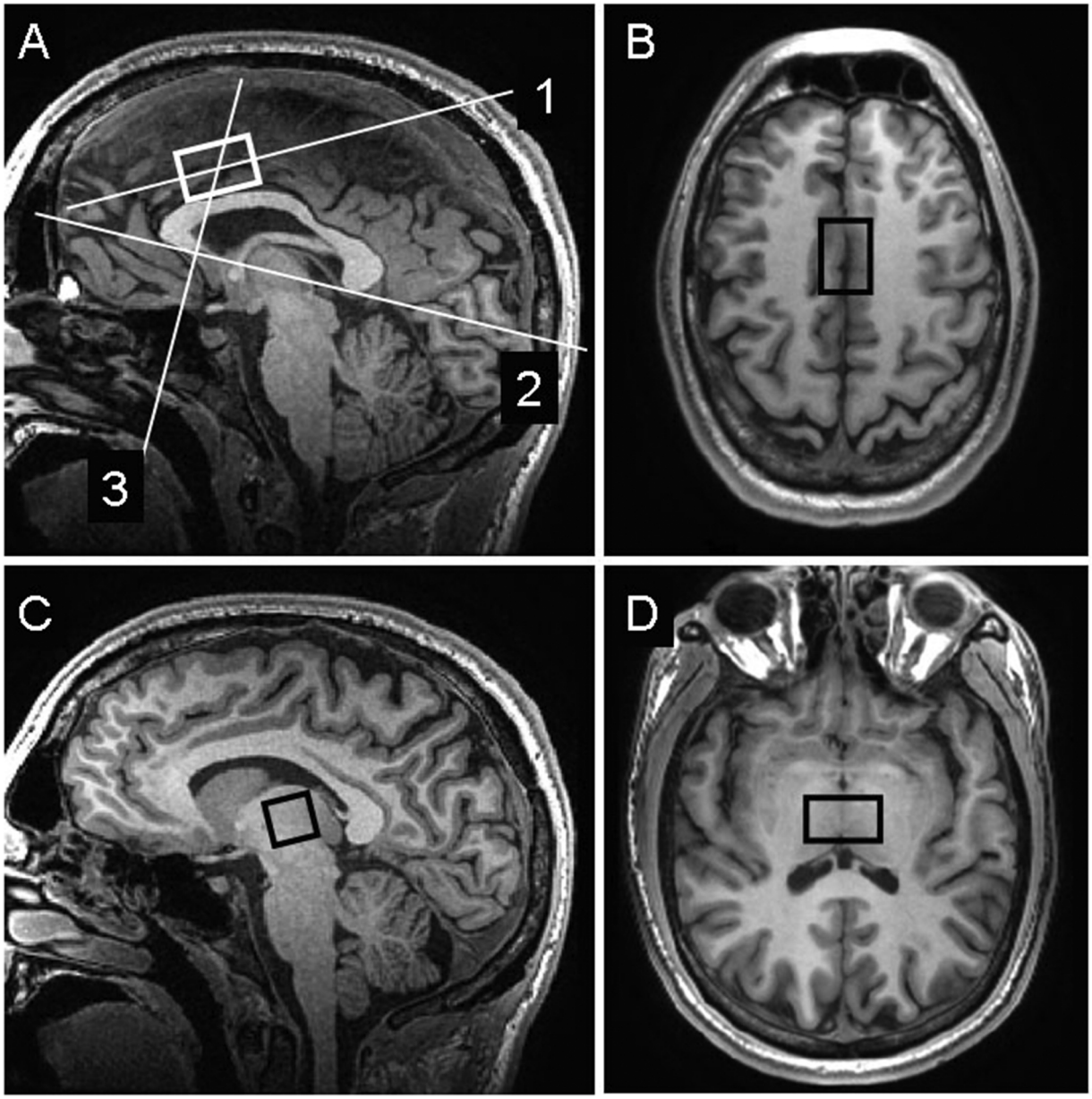

Fig. 1. The VOI of dACC was positioned parallel to the hippocampal axis (Fig. 1A, Line 1) touching the top of the corpus callosum, and over the two hemispheres symmetrically (Fig. 1B). The centre of the VOI in anterior-posterior direction was placed using a line (Fig. 1A, Line 3) through the anterior commissure and perpendicular to the anterior commissure – posterior commissure (AC-PC) line (Fig. 1A, Line 2) (Dou et al., 2013). The VOI of midline bilateral thalamus was positioned along the same oblique-transverse plane (Fig. 1C&D).

tion of goal-directed behaviour (Devinsky et al., 1995), and can be functionally divided into dorsal (cognitive) and ventral (emotional) regions (Bush et al., 2000). The dorsal ACC (dACC) strongly connects with premotor and supplementary motor areas (SMA) (Devinsky et al., 1995) whereas the ventral ACC connects to the limbic system, which primarily processes emotional and motivational information (Vogt et al., 1992). Another hub region in the CSTC circuits is the thalamus, which serves as a relay station between striatum and cortex, and is critically involved in motor function, perception and thought integration (Alexander et al., 1986). Abnormalities in ACC and thalamus have been demonstrated by multiple structural and functional imaging studies in both TD and OCD (Ganos et al., 2013; Menzies et al., 2008; Worbe et al., 2015).

Imbalance between excitatory and inhibitory neuronal projections in the various CSTC regions is suggested to alter development of these brain networks and is associated with neurodevelopmental disorders such as TD and OCD (Pauls et al., 2014; Ramamoorthi and Lin, 2011). Glutamate is the most important excitatory neurotransmitter in the brain, while gamma-aminobutyric acid (GABA) is the principal inhibitory neurotransmitter. Glutamate serves as the precursor of GABA through decarboxylation by glutamic acid decarboxylase. Glutamine and glutamate form an important cycle: glutamate that is released from neurons is metabolized into glutamine in the astrocytes. Glutamine is subsequently released by the astrocytes, and used mainly for glutamate synthesis in neurons (von Bohlen un Halbach and Dermietzel, 2001). Previous evidence from genetic, pharmacological, animal and post-mortem studies have shown that glutamatergic dysfunctions in CSTC circuits are related to TD and OCD pathology (Adamczyk et al., 2011; Delorme et al., 2004; Kalanithi et al., 2005; McGrath et al., 2000; Ramamoorthi and Lin, 2011).

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