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Prefrontal excitation-inhibition balance and social cognition in bipolar disorder

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Background
Bipolar disorder (BD) is a mental disorder characterized by episodic mood dysregulation. Even in the euthymic state of BD, social cognitive deficits remain significant. Such impairment impacts patients with BD in their daily life and may cause unfavorable functional outcomes. The imbalance of excitatory glutamatergic and inhibitory GABAergic systems in the default network, which modulate corticolimbic systems, is a hypothetical neural underpinning of social cognitive deficits in BD.

Aims & Objectives
The current study aimed to investigate the balance of glutamate and GABA in cortical mid-line structures of BD and its association with social cognitive deficits.

Methods
Twenty-five euthymic BD patients and forty-three healthy controls (HCs) were recruited. The glutamatergic and GABAergic metabolites in the medial prefrontal cortex (mPFC) and post-medial cortex (PMC) were measured by proton magnetic spectroscopy (^{1}H-MRS), which is an imperative technique to study endogenous metabolites non-invasively in vivo. We also used several validated social cognitive tasks to assess different domains of social cognition , including Diagnostic Analyses of Nonverbal Accuracy, Taiwanese Version (DANVA-2-TW, measuring emotion recognition) the Reading Mind in the Eyes (RMET); the Chinese Theory of mind Task (cToM); and Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT).

Results
The negative association between the emotion recognition and glutamatergic-GABAergic balance in the mPFC was observed in the BD I patients ($r = -0.588$, $p = 0.017$) but not in HCs ($r = 0.005$, $p = 0.973$), which might be contributed from the positive association between mPFC GABAergic function and emotion recognition in the BD I patients ($r = 0.535$, $p = 0.033$).Similarly, we also found a negative association between emotional intelligence and glutamatergic-GABAergic balance in the mPFC in the bipolar patients ($r = -0.455$, $p= 0.017$) and a positive association between emotional intelligence and GABAergic function ($r = 0.504$, $p = 0.007$) in the mPFC in the BD patients.

Discussion & Conclusion
The BD group has worse social cognition, which was associated with their glutamatergic-GABAergic imbalanced status. The association observed in BD may suggest a possible modulatory role of excitation-inhibition balance in the default mode network (DMN), which may contribute to their social cognitive deficits. Further investigation into the association between social cognition, excitation-inhibition balance in the midline structure, and DMN activity may advance our neurobiological understanding of BD.