Childhood physical neglect and mitochondrial copy number in patients with bipolar disorders

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Background Childhood trauma is consistently associated with psychiatric disorders and common chronic diseases well into adulthood. Compelling evidence has shown that mitochondrial dysfunction and increased oxidative stress are common features in bipolar disorder (BD). However, the association between childhood trauma and mitochondrial DNA copy number (MCN) or oxidative stress remains elusive in patients with BD.

Aims & Objectives The aim of this study is to explore the association between childhood trauma and MCN or oxidative stress in clinically stable patients with BD.

Methods 26 healthy controls and 70 euthymic patients diagnosed with BD according to the DSM 5 criteria were recruited. The patients' psychopathology was evaluated using the 17-item Hamilton Depression Rating Scale (HDRS) and the 11-item Young Mania Rating Scale (YMRS). Childhood Trauma Questionnaire (CTQ) was applied to evaluate 5 subtypes of childhood trauma, including emotional abuse, physical abuse, sexual abuse, emotional neglect and physical neglect. Leukocyte MCN and oxidative stress were assessed using a LightCycler Instrument (Roche, Mannheim, Germany) and ln-transformed to be normal distribution. SPSS Statistics 20.0 (SPSS Inc., Chicago, IL) was used for all of these analyses. The threshold for statistical significance was set at p less than 0.05 (two-tailed).

Results Half of the healthy controls and 46% of the patients were men. The mean ages of the healthy controls and patients were 33.2±12.64 and 37.90 ± 13.72 years, respectively. The HADS and YMRS were 1.83± 2.09 and 0.76± 1.40, respectively. Emotional neglect (74.3%), physical neglect (55.7%), and emotional abuse (47.1%) were the most common reported childhood trauma subtypes, followed by physical abuse (34.3%) and sexual abuse (37.1%). After adjusting for age, sex and BMI, physical neglect scores were inversely correlated to ln-MCN (r=-0.30, p=0.013). Oxidative stress (delta CT) was not correlated with childhood trauma after adjustment.

Discussion & Conclusion Childhood trauma may induce neuro-oxidative and immune-inflammatory pathways coupled with lipid and protein oxidation, which may play a role in severity of illness. Limited data in BD patients indicates that childhood trauma is also associated with increased inflammatory biomarkers, which lead the hypothesis that MCN may be involved. Our results support the association between childhood physical neglect and MCN in patients with BD. Other subtypes of childhood trauma were not associated with MCN after adjustment. Further study is needed to explore the mechanism underlying MCN, oxidative change in BD patients with childhood trauma.