

Background: Early life adversity has been identified as a potentially causal factor in the development of mental disorders. Little is known, however, about the association between various types of early life adversities and social cognitive function in adults with major psychiatric disorders, such as schizophrenia, borderline personality disorder, bipolar disorder and major depressive disorder. We conducted a systematic review aimed at elucidating possible underlying cognitive mechanisms that may form the pathway between early life adversities and social cognitive dysfunction.

Methods: Relevant studies were identified via electronic and manual searches of the literature, and included peer reviewed English language articles published up to May 2017. Quality of individual articles was assessed using the quality evaluation scale.

Results: A total of 15 studies were included in the systematic review with the quality assessment scores ranging from 2 to 5 (out of 6). The majority of the studies demonstrated that various types of early life adversities, specifically physical neglect, emotional and sexual abuse and insecure attachment, are significantly associated with social cognitive function.

Discussion: Presented in the context of an attachment model, we conclude that childhood adversity results in poor internal working models, selective attention towards emotional stimuli and greater difficulties with emotional self-regulation. The importance of these findings for development of interventions which diminish the adverse effects of childhood maltreatment on social cognition is discussed.

S7. NEOSENSITIZATION TO MULTIPLE DRUGS FOLLOWING DRUG REACTION WITH EOSINOPHILIA AND SYSTEMIC SYMPTOMS SYNDROME (DRESS)

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Background: Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) syndrome is associated with severe skin eruptions, fever, hematological abnormalities, and multi-organ involvement. Although aromatic anticonvulsant drugs have been frequently associated with the manifestation of DRESS syndrome, its induction following treatment with non-aromatic anticonvulsants, such as valproate, has rarely been reported. Moreover, there are limited data regarding the development of neosensitization related to chemically unrelated drugs following an episode of DRESS syndrome.

Methods: Here, a case of neosensitization to multiple drugs is described. The present case report describes a female patient who experienced neosensitization to amoxicillin, olanzapine, and quetiapine following the manifestation of DRESS syndrome induced by valproate.

Results: A 50-year-old woman with a 15-year history of schizophrenia was being treated with lithium (1200 mg) and quetiapine (600 mg) about 1 month, but due to high lithium serum concentrations, the lithium was changed to valproate (600 mg). Seven days later, the patient developed a whole-body skin rash, facial edema, and hyperthermia. Laboratory tests revealed an abnormal white cell count ($25.2 \times 10^3/\mu\text{L}$ with 6% eosinophils) and aspartate transaminase (AST) and alanine transaminase (ALT) concentrations of 2729 IU/L and 2749 IU/L, respectively. At that time, the patient had no any other medical history including drug allergy. A diagnosis of DRESS syndrome due to valproate treatment was established by a consulting dermatologist. As a result, all medicines were discontinued because of severe hepatitis, and intravenous methylprednisolone (60 mg per day) was administered for 1 week. The skin rash, fever, and liver dysfunction progressively disappeared. After

discharge, the patient was treated with quetiapine (200 mg). However, she became lost to follow up after 6 months. Approximately 3 years later, the patient was admitted to a local hospital for psychotic symptoms aggravation because she was not taken antipsychotics for 3 years. She treated with lithium (900 mg), sulpiride (600 mg), risperidone (2 mg), and quetiapine (100 mg) for 2 weeks. Additionally, the patient initiated treatment with amoxicillin for acute tonsillitis. On the first day of amoxicillin intake, she developed fever, diffuse erythematous macules on her trunk, and facial edema, and she was transferred to a general hospital via the emergency department. To control her psychotic symptoms she is prescribed olanzapine, haloperidol and quetiapine step by step but all these medications develop fever, skin rash and abnormal AST/ALT. Finally she was given amisulpiride which had not been previously prescribed. Within 2 months, the patient's psychotic symptoms had gradually decreased and ultimately remitted.

Discussion: To our knowledge, this is the first case report of neosensitization to multiple drugs after valproate-induced DRESS syndrome. A thorough search of Pubmed was performed to identify similar cases, which confirmed that no cases of hypersensitivity to amoxicillin or neosensitization to multiple drugs after a valproate-related DRESS episode have been reported. Furthermore, only two studies have reported possible neosensitization to amoxicillin following DRESS episodes induced by carbamazepine, and only one case reported neosensitization to amoxicillin following a DRESS episode induced by allopurinol.

S8. RELATIONSHIP BETWEEN ALLOSTATIC LOAD AND POOR FUNCTIONAL CAPACITY IN YOUTH AT ULTRA-HIGH RISK FOR PSYCHOSIS

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Background: Current pathophysiological models of psychotic disorders suggest that stress contributes to the aetiology and trajectory of the disorder. Allostatic load (AL), a multisystem index of immune, neuroendocrine and metabolic dysregulation, is thought to represent the cumulative biological impact of stress. Two recent studies suggest that AL is elevated in patients with first-episode psychosis and related to psychotic symptoms and poor social and occupational functioning. Here, we investigate the relationship between AL and clinical outcomes in individuals at ultra-high risk for psychosis.

Methods: AL was measured in a sub-group of participants of the NEURAPRO study, a multicentre randomized-controlled trial of omega-3 polyunsaturated fatty acids versus placebo in people aged 13 - 40 at UHR for psychosis. A total of 106 participants who underwent additional biomarker analysis were included in the present study. Biomarkers for the AL index were selected based on (1) representation of several physiological systems including the cardiovascular, neuroendocrine, immune, and metabolic systems, (2) use in previous AL research, and (3) associations with disease risk. We adopted a scaled AL algorithm whereby each marker proportionally contributes to the overall AL index. Clinical outcomes were assessed 3 and 12 months after study intake using the Social and Occupational Functioning Assessment Scale (SOFAS), the Brief Psychiatric Rating Scale (BPRS), the Scale for the Assessment of Negative Symptoms (SANS), the Montgomery-Asberg Depression Rating Scale (MADRS) and the Young Mania Rating Scale (YMRS). We hypothesised that AL would be (1) associated with higher symptoms scores and reduced functioning at baseline and (2) related to more severe symptoms and reduced functioning at the 3 and 12 month assessments. These hypotheses were tested by calculating Pearson correlation coefficients and by using linear regression modelling, respectively.