EPP0765

Clinical predictors of kynurenine pathway aberrations in schizophrenia and bipolar disorder

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Introduction: Schizophrenia and bipolar disorder are severe mental illnesses that are known to have a considerable overlap in underlying pathophysiological mechanisms. More specifically, disturbances in the kynurenine pathway have been hypothesized as processes bridging altered immune responses and clinical manifestations of these illnesses.
Objectives: The aim of this study was to investigate the abnormalities in serum kynurenine metabolites in schizophrenic and bipolar patients and the impact of clinical factors.
Methods: Four patient groups were included in the current study: 1) Acute bipolar inpatients (n=205); 2) stable bipolar outpatients (n=116); 3) acute schizophrenia inpatients (n=111) and 4) stable schizophrenia outpatients (n=75); and one healthy control group (n=185). Clinical symptoms were established using symptom severity scales. The quantitative determination of serum kynurenine metabolites was performed using LC-MS/MS. General linear model and multivariate linear regression analyses were used to perform the statistical analysis with JMP Pro 15.
Results: In line with previous research, the results indicate that serum kynurenine metabolites are disturbed in schizophrenic and bipolar patients compared to healthy controls. Whereas no differences were observed between schizophrenia and bipolar disorder, illness state and duration of illness clearly impacted kynurenine metabolite levels. Acutely ill patients had significantly lower levels compared to stable patients, which seemed to be driven by psychotic symptoms.
Conclusions: To conclude, the results confirm the involvement of the kynurenine pathway in the pathophysiology of schizophrenia and bipolar disorder by lowered peripheral kynurenine metabolite level. In addition, an important role of acute psychotic symptoms and longer illness duration on these metabolite aberrances is demonstrated.

Disclosure: No significant relationships.
Keywords: kynurenine; schizophrénia; BIPOLAR; inflammation

EPP0764

The role of inflammation and cortisol in the relationship between social cognition abilities and later emotional or behavioural problems: evidence from a UK birth cohort

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Introduction: Deficits in social cognition have been associated with the onset of emotional and behavioural problems, but the biological mechanisms underlying this relationship remain unclear.
Objectives: This study examined whether diurnal cortisol patterns, systemic inflammation, or both, explained the association between social cognition difficulties and subsequent emotional and behavioural symptoms.
Methods: The sample consisted of 714 individuals from the Avon Longitudinal Study of Parents and Children (ALSPAC) with valid data on cortisol measures (age 15 years) and emotional or behavioural problems (age 17 years). Social cognition abilities were measured at 8, 11, and 14 years old. Inflammation was measured using serum levels of interleukin 6 (IL-6, age 9 years) and C-reactive protein (CRP, age 9 and 16 years). Bayesian structural equation modelling was used to investigate the mediating effect of cortisol or inflammation on the association between social cognitive difficulties and emotional or behavioural problems.
Results: Children with social cognition difficulties were associated with later emotional and behavioural problems. Flattened diurnal cortisol slope was associated with the hyperactivity/inattention problem two years later. Mediation analyses revealed that lower morning cortisol significantly mediated the associations between social communication difficulties at 8 years with hyperactivity/inattention and conduct problems in adolescence, with the adjustment of inflammation and all covariates. Systemic inflammation was not related to social cognitive difficulties or future emotional and behavioural problems.
Conclusions: The finding suggests that social cognition is related to cortisol activities longitudinally. It also expands the evidence that adolescents with behavioural problems are characterised by hypoactivity of the hypothalamic-pituitary-adrenal (HPA) axis.

Disclosure: No significant relationships.
Keywords: social cognition; general population; Cortisol; prospective cohort study

EPP0765

Investigation of anti-NMDA receptor antibodies in first episode psychosis patients

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Introduction: Anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis is an autoimmune limbic encephalitis, where psychiatric symptoms are often the initial presentation dominant initially. These patients are mainly admitted to psychiatric wards, due to first episode psychosis (FEP).
Objectives: Multiple studies analysed whether anti-NMDAR antibodies were present in the sera of schizophrenic patients, but results have not verified this hypothesis. It is possible, however, that unknown autoimmune antibodies play a role in FEP, similarly to anti-NMDAR antibodies.