P31.02

Blunted prolactin response to fenfluramine following add-on fluvoxamine

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The study examined the hypothesis that fluvoxamine, an agent effective in ameliorating negative symptoms of schizophrenia when added to antipsychotic treatment, acts by modifying the serotonergic system. Prolactin and cortisol response to fenfluramine challenge was examined before and after add-on fluvoxamine treatment in 12 medicated chronic schizophrenia patients. Prolactin response to fenfluramine was significantly blunted after fluvoxamine treatment. The data supports the hypothesis that add-on fluvoxamine acts by modifying the serotonergic system in schizophrenic patients. Reduction in serotonergic responsiveness following fluvoxamine treatment is similar to that following clozapine and is consistent with clinical evidence showing effectiveness of both in treatment of negative symptoms.

P31.03

Release of prolactin but not of adenocorticotropic hormone increases significantly in lactate-induced panic attacks

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In contrast to pronounced psychopathological effects, the stress hormones ACTH and cortisol are not increased during sodium lactate- induced panic attacks. To further investigate this phenomenon, we measured prolactin, another potentially stress-sensitive hormone.

We studied the plasma prolactin and ACTH responses to 20 minutes infusions with 0.5 molar sodium lactate (10 ml/kg body weight) and placebo (isovolemic normal saline) in eight patients with panic disorder and eight sex- and age-matched normal controls.

In comparison to placebo, lactate infusion led to enhanced prolactin secretion in all subjects studied; this increase of plasma prolactin was significantly elevated ($p<0.05$) in panickers compared to non-panickers. In contrast, ACTH did not increase significantly after lactate vs. placebo, both in panickers and non-panickers.

The mechanisms for this differential endocrine stress response of prolactin and ACTH in lactate-induced panic is still unclear. The potential influence of modulatory peptides and neurotransmitters will be further investigated.

P32. Neuroimmunology

P32.01

Marker of bipolar mania: soluble IL-2 & transferrin receptors in serum

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We studied plasma soluble interleukin-2 receptor (sIL-2R), sIL-6R, Clara cell protein (CC16), and transferrin receptor (TfR) levels in 57 bipolar manic (DSM-IV) patients aged 16 to 45 years during both acute mania and subsequent remission (YMRS < 12). The results were compared with age-and sex- matched healthy controls. The mean plasma TfR levels in both acute mania and subsequent remission were elevated and independent of any clinical or immune-inflammatory variables. There is none alternation of circulating CC16 and SIL-6R. Dosage of valproate, total number of prior affective episodes, YMRS scores, and a negative first-degree family history of mood disorder may play a contributory role in the elevation of plasma sIL-2R levels during acute mania. (adjusted $R2 = 0.229$, $P< 0.0025$). Our findings demonstrate that increasing plasma sIL-2R and TfR levels in bipolar disorder are associated with illness itself rather than pharmacological effects and individual variations (e.g. BMI). Increasing plasma TfR levels without alternation of plasma sIL-6R might be considered as a trait of bipolar disorder and plasma sIL-2R as a state marker of illness severity during acute mania.

P32.02

Neuroplasticity in major depression may be indicated by S100B

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Objectives: Neurodegenerative mechanisms contribute to the pathophysiology of major depression. S-100B is a astroglial peptide which exerts multiple neurotrophic effects on sero-toninergic neurons, axonal growth and synaptogenesis. S-100B has been reported to be in-creased in diseases with neuronal cell damage or degeneration and is therefore a candidate to indicate neuronal restructuring in major depression.

Method: S-100B plasma levels were determined in 25 patients with major depression and 25 matched healthy controls using an immunofluorometric sandwich assay.

Results: Patients with major depression showed significantly increased S-100B levels compared to healthy controls. S-100B plasma levels were significantly positively correlated with the relative reduction of depressive symptoms after four weeks of treatment. This effect was pronounced in patients of the melancholic subtype. In a linear regression model, only S-100B and severity of depressive symptoms upon admission revealed a significant predictive effect on therapeutic response.

Conclusions: The results indicate that the neuroprotective functions of S-100B might counterbalance neurodegenerative mechanisms that are involved in the pathophysiology of major depression and in the response to antidepressant treatment.

P33. Neurophysiology

P33.01

Word recognition memory in healthy subjects at risk for depression

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Event related brain potential (ERP) studies of memory in depression have shown impaired working memory and recollection processes indicated by a reduction of the "old/new effect". In word recognition experiments, this repetition effect is modulated by changes of different ERP components, e.g. the N400 or LPC. Furthermore, changes of these components were also observed in non-depressive patients with a history of an affective disorder. Therefore, the question has to be raised, whether these findings may also be found in healthy subjects at risk for depression.

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