

and suicidal behavior in psychosis. Although clozapine is associated with a low likelihood of extrapyramidal symptoms and other neurological side effects, weight gain and metabolic side effects are well known in clinical practice exposing the patient to a greater risk of cardiovascular disorders, premature death, as well as psychosocial issues leading to non-adherence. The mechanisms underlying this pharmacologically activated disorders are still controversial. Based on our *in vitro* results, we have characterized *in vivo* the effects of the selective PKC β inhibitor, Ruboxistaurin (LY-333531) on a preclinical model of long-term clozapine-induced weight gain. Cell biology, biochemistry and psychomotor tests have been performed on wild type and PKC β (-/-) mutant mice to investigate the contribution of endogenous PKC β and its pharmacological inhibitor on the neuroleptic effect of clozapine. Lastly, we also shed light on a novel aspect of the mechanism underlying of clozapine-induced weight gain, demonstrating that the clozapine-dependent PKC β activation promote the inhibition of the lipid droplet-selective autophagy process, opening the way to new therapeutic intervention approach.

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Changes in the cytokine profile in first episode, drug-naïve patients with psychosis after short-term antipsychotic treatment

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Introduction An increasing body of evidence suggests that antipsychotic medication can cause immunological changes that could be attributed to the amelioration of psychotic symptoms or the metabolic side effects of the drugs. So far, the results of the studies remain controversial.

Objective Our aim was to compare the levels of interleukin (IL) IL-2, IL-6 and transforming growth factor- β 2 (TGF- β 2) in drug-naïve, first-episode patients with psychosis before and after six weeks of antipsychotic medication.

Methods Thirty-nine first episode patients with psychosis were enrolled in the study. Serum levels of IL-2, IL-6 and TGF- β 2 were measured by enzyme linked immunosorbent assay (ELISA) before and six weeks after the initiation of antipsychotic medication. In addition, clinical psychopathology was assessed using Positive and Negative Syndrome Scale (PANSS) before and after treatment.

Results Serum levels of IL-2 were significantly higher in the study group six weeks after the initiation of antipsychotic treatment ($P < 0.001$) while TGF- β 2 levels were decreased ($P < 0.001$) and IL-6 levels were slightly reduced ($P < 0.004$).

Conclusion The changes in cytokine levels may be attributed to the action of antipsychotic medication and the remission of psychopathology. The reduction in TGF- β 2 levels is observed in all patients and with all antipsychotic medications used. TGF- β 2 may be a marker of clinical efficacy.

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Amelioration of impaired hippocampal cognitive performance in Alzheimer's disease via long-term intervention with ghrelin

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Introduction Alzheimers disease (AD) is a neurodegenerative disorder characterized by loss of memory and cognitive deficits. Ghrelin is a peptide hormone which has been linked to neuroprotection, memory and learning processes.

Objectives This study investigated the effects of ghrelin-induced memory retention on amelioration of cognitive deficits via restoration of long-term potentiation (LTP) and induction of synaptic plasticity in hippocampal CA3, using a rat model of AD induced by amyloid- β (1-42) injection.

Methods Five groups of male rats (230–270 g) including ghrelin-treated (200 ng/rat, [ICV], daily for two weeks), A β 1-42 injected (5 μ L/rat) and A β 1-42 plus ghrelin-treated animals were designed. Ghrelin was administered after an ICV injection of A β 1-42. To assess cognitive performance and the motor dysfunction, passive avoidance tests and open-field were performed, respectively. Step-through latency (STL) was evaluated as learning and memory index. Intrahippocampal field potential recordings were done.

Results Results showed that following A β 1-42 injection, STL and induction of LTP were significantly decreased whereas ICV injection of ghrelin significantly enhanced memory retention by improvement of STL and restitution of LTP in the CA3 with increased EPSP slope and PS amplitude, suggesting the involvement of ghrelin in postsynaptic mechanisms of hippocampal LTP.

Conclusions It was revealed that neuroprotective effects of chronic ghrelin not only can enhance but also can restore LTP in the CA3 area in A β -induced AD. Results suggest that ghrelin may be considered as a promising therapeutic agent to alleviate cognitive deficits of AD.

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Relationship between taste thresholds and antidepressant response: Preliminary findings

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Introduction In healthy volunteers, light acting through serotonin pathways, decreases the threshold for sweet, but not salt taste; similar to SSRI paroxetine. In depressive disorders, there is deficiency of serotonin throughput, which is remedied by SSRI medications, and results in improvement in symptoms of depression. Thus, we report on taste thresholds before and after SSRI treatment.

Objectives To study the variation in thresholds for sweet with SSRI treatment in depressed patients in short- and long-term.