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 PKCβ 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 PKCβ (-/-) mutant mice to investigate the contribution of endogenous PKCβ 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 PKCβ activation promote the inhibition of the lipid droplet-selective autophagy process, opening the way to new therapeutic intervention approach.

Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.02.381

EW0768

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

P. Petrikis (Lecturer in psychiatry)[1,*], P. Voulgari[2], V. Boumba[3], D. Archimandriti[2], P. Skapinakis[4], V. Mavreas[4]
[1] *Department of Psychiatry, Ioannina, Greece*
[2] *Ioannina School of Medicine, Department of Internal Medicine, Ioannina, Greece*
[3] *Ioannina School of Medicine, Laboratory of Internal Medicine and Toxicology, Ioannina, Greece*
[4] *Ioannina School of Medicine, Department of Psychiatry, Ioannina, Greece*
[*] *Corresponding author.*

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-β2 (TGF-β2) 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-β2 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-β2 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-β2 levels is observed in all patients and with all antipsychotic medications used. TGF-β2 may be a marker of clinical efficacy.

Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.02.382

EW0769

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

B. Sadeghi
Shahid Beheshti University of Medical Sciences, Physiology, Neuroscience Research Center, Tehran, Iran

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), Aβ1-42 injected (5 μL/rat) and Aβ1-42 plus ghrelin-treated animals were designed. Ghrelin was administered after an ICV injection of Aβ1-42. 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 Aβ1-42 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 Aβ-induced AD. Results suggest that ghrelin may be considered as a promising therapeutic agent to alleviate cognitive deficits of AD.

Disclosure of interest The author has not supplied his/her declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.02.383

EW0770

Relationship between taste thresholds and antidepressant response: Preliminary findings

S. Srivastava[1,*], A. Kumar[1], V. Agarwal[2], P. Chubey[1], L. Donaldson[3], J. Potokar[4]
[1] *King George's Medical University, Geriatric Mental Health, Lucknow, India*
[2] *King George's Medical University, Postgraduate Department of Psychiatry, Lucknow, India*
[3] *University of Nottingham, Faculty of Medicine and Health Sciences, Nottingham, United Kingdom*
[4] *University of Bristol, Department of Community Medicine, Bristol, United Kingdom*
[*] *Corresponding author.*

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.