(after 7 and 14 days) the memory function was improved. The administration of HAL in a single dose and in chronic treatment produced a worsening of spatial memory, statistical significant after 14 days of treatment. CBZ at doses of 30 mg/kg did not induce catalepsy after single and chronic treatment, but HAL (0.15 mg/kg) induced a very strong catalepsy already after a single administration as well as after chronic treatment.

Summarizing, it can be suggested that carbamazepine has a memory enhancing effect after chronic treatment, which is probably caused by the influence of this drug on systems of brain neurotransmitters. CBZ can be regarded as a subsidiary therapy agent in memory deficits, e.g. in bipolar affective illness.

P.3.089 Antioxidant defence system in patients with a first psychotic episode. Effect of antipsychotic treatment

P. Lopez[1]*, J.A. Micó[2], A. González-Pinto[1], A. Aldama[1], C. Gonzalez[1], F. Mosquera[1], O. Rojas[2]. [1]*Santiago Apostol Hospital, Psychiatry, Vitoria, Spain;* [2]*University of Cadiz, Neurosciences, Spain*

Objective: It has been proposed that oxidative stress-mediated impairment of neuronal processes is involved in the pathophysiology of neuropsychiatric diseases such as schizophrenia. The primary antioxidant defence is enzymatic involving catalase (CAT) and glutathione peroxidase (GPx). Glutathione (GSH) is the main non-protein antioxidant and plays a critical role in protecting cells from damage by reactive oxygen species (ROS) generated by dopamine (DA) metabolism, which it is known to be altered in schizophrenia. The aim of the study was to determine the basal activity of the antioxidant defence system in patients with a first psychotic episode, compared with matched healthy control subjects, and the effect of treatments on these antioxidant defences.

Method: This is a prospective and longitudinal study in consecutively admitted patients with a first psychotic episode. First psychotic episode is defined as the first time patient displayed positive psychotic symptoms of delusions or hallucinations. This study aims to follow them up for one year. Patients are treated with antipsychotics (no clozapine). The data presented here are obtained form blood samples taken upon arrival into the emergency room and after four weeks ($n=10$) which were processed following standard procedures. In addition, samples were taken from healthy volunteers that were matched for sex and age ($n=18$). Enzyme activities and GSH levels were determined by spectrophotometric assays in haemolisates of erythrocytes. Total antioxidant status was also determined by a spectrophotometric assay in serum. The data were analyzed by T-test for un-paired samples (patients versus controls) and paired samples (four weeks versus admission).

Results: These preliminary results reported a decrease ($p<0.05$) in total antioxidant status of patients at baseline (0.850 ± 0.043 mM) and after 4 weeks (0.886 ± 0.054 mM) versus control (1.164 ± 0.082 μM). GSH levels were lower ($p<0.05$) in blood of patients at admission (284.4 ± 19.195 vs 338.844 ± 15.468). After 4 weeks, GSH levels (337.2 ± 31.526) were not significantly different from control subjects. GPx and CAT activities of patients were not different from control subjects neither at admission nor after 4 weeks.

Discussion: These preliminary results show that a decrease in antioxidant defence may be involved in the pathophysiology of psychosis. GSH deficit may be involved in membrane peroxidation and microlesions related to dopamine. Antipsychotic treatment seems to normalize these levels. This finding may have a significant impact on improving strategies directed to neuroprotection in the treatment of psychosis. The ongoing study would provide more extensive data from a greater n and a 1 year follow-up of these patients, providing data of the effect of long term antipsychotic administration. Supported by the Stanley Foundation (RC-003)

P.3.090 Pseudophaeochromocytoma associated with clozapine- amisulpride combination therapy

C. Kalaitzi*, K. Gazi, K. Papoutsakis, E. Neroutsos. *Elefsina's General Hospital, Psychiatry, Neo Heraklio – Athens, Greece*

The augmentation of clozapine with amisulpride has been documented to be efficient and well tolerated. For this, we have adopted it in our everyday practice for almost a year now. We report on the case of a 33 years old male with treatment- resistant schizophrenia and suboptimal response to clozapine. The patient was receiving 600 mg of clozapine when started on amisulpide. Two weeks later and while on 200 mg of amisulpride, the patient presented hypertension (mean BP 185/105 mmHg) and tachycardia (mean 121 bpm).

24 hour urinary catecholamine concentration (μmol) was noradrenaline 1.23 (<0.59) and vanillylmandelic acid 59 (<35). CT scan revealed no evidence of phaeochromocytoma. Amisulpride was withdrawn and symptoms resolved within a week. Clozapine has been reported to cause increases in plasma noradrenaline concentrations up to 471%, a postulated mechanism being the inhibition of presynaptic reuptake mediated by alpha2 adrenergic receptors. About a dozen of cases of clozapine induced pseudophaeochromocytoma have been described in the literature. Amisulpiride, which has a high affinity for presynaptic alpha2 adrenoreceptors, may have added the last drop to make this increase a clinically significant one. Pathophysiological mechanisms as well as implication for clinical practice are being discussed.

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P.3.091 A comparison of the effects of quetiapine and olanzapine on glucose metabolism in patients with schizophrenia

D. Henderson[1]*, P.M. Copeland[2], C.P. Borba[1], D.D. Nguyen[1], A.E. Evins[1], H. Zhang[3], D. Hayden[3], O. Freudenreich[1], C. Cather[1], D. Schoenfeld[3], D.C. Goff[1]. [1]*Massachusetts General Hospital/Harvard Medical School, Psychiatry, Boston, MA, USA;* [2]*Massachusetts General Hospital/Harvard Medical School, Medicine and Endocrinology, USA;* [3]*Massachusetts General Hospital/Harvard Medical School, MGH Biostasticis, USA*

Purpose: The rate of type II diabetes mellitus (DM II) may be higher among patients with schizophrenia than in the general population. Furthermore, recent case reports have linked treatment-emergent DM II in non-obese patients with schizophrenia with the use of some atypical antipsychotics (clozapine and olanzapine in particular). However, few studies have investigated the effects of newer atypical antipsychotics such as quetiapine. We compare