Data from large representative epidemiological samples, such as the National Co-morbidity Survey Replication, indicate high co-occurrence of major depressive disorder and alcohol misuse and dependence. Possible mechanisms include common risk factors, affective disorder inducing alcohol use, and alcohol use inducing affective disorder. Overall the findings for a common predisposition are not very strong, but common genetics contributing to the induction of both disorders seem to include the cholinergic muscarinic 2 receptor gene, clock genes and possibly MTHFR. Attempts to group depression and alcoholism into alcohol-induced depression and depression as an independent disorder, or alternatively into externalizing and internalizing alcoholics (characterized by high levels of anxiety and depression), have not gained common acceptance. Data indicate more than one pathway, with differences in subgroups specifically for males and females. A possible mechanism underlying the co-occurrence may be stress vulnerability and alteration of stress vulnerability within the context of major depressive disorder and chronic alcohol use. The interaction seems to be specifically pertinent for an increased risk of relapse. Our understanding of alcohol dependence and major depressive disorder has been based to a considerable degree on animal models. Preclinical co-morbidity studies so far have been rare; one reason being that results vary substantially according to the applied model. Currently the gene/environment interaction and the role of epigenetic processes are increasingly getting into the focus of research, which promises to further our understanding of the mechanism of co-morbidity for alcoholism and major depressive disorder.

P0058

Kinetic of a new drug in patients with alcoholism

T.V. Shushpanova [1], V.Y. Semke [2], T.P. Novozheyeva [1]. [1] *Neurobiology Laboratory, Mental Health Research Institute, Tomsk, Russia* [2] *Mental Health Research Institute, Tomsk, Russia*

Objective: We investigated effect of Galodif® on activity of liver cytochrome P-450 system of alcoholics from Russians and Tatars.

Methods: 68 patients were examined. The concentration of antipirine in saliva was determined by spectrophotometry assay (B.B. Brodie 1949, in Semenjuk A.V. modification, 1982). Pharmacokinetic parameters were counted as follows: period of half elimination (T1/2, h), total clearance (Cl t, ml/min), middle time of residual (MRT, h) middle time of elimination (MET, h), area under pharmacokinetic curve (AUC, mkgh/ml).

Results: T1/2 (h) was 8,81±5,23 before treatment and 4,37±2,31* after treatment with Galodif; Clt (ml/min) 113,42±38,67 and 137,37±54,00;MRT (h) 11,44±5,43 and 3,69±0,60*; MET (h) 6,03±2,10 and 4,64±1,83*; AUC (mkgh / ml) 7,05±5,74 and 6,39±2,18 respectively (*-differences between values of pharmacokinetic parameters are reliable according to l-criterion by Kolmogorov-Smirnov $p<0,05$). Galodif reduces period of half-elimination, significant decrease of middle time of residual drug in organism and middle elimination time. Total clearance increased. Under influence of Galodif elimination of antipirine increased that suggested induction of liver microsomal monooxigenases cytochrome P-450 system in Russian alcoholic patients. Influence of Galodif on antipirine pharmacokinetics parameters in Tatar alcoholic patients: T1/2 (h) 11,19±2,95 and 2,57±0,69*; Clt (ml/min) 71,108±11,58 and 116,23± 9,40*; MRT (h) 8,66±1,13 and 2,60±0,46*; MET (h) 5,71±0,57 and 3,68±0,49*; AUC (mkg h/ ml) 11,58±1,71 and 7,30±1,04*. Galodif ability for induction of liver monooxigenases of patients from different ethnic groups is to be taken into account during clinical application. Individual sensitivity of organism to drug is caused by biochemical and anthropo-morpho-physiological polymorphism.

P0059

The usefulness of atypical antipsychotics in psychiatric comorbidities with addictions (case report)

M.I. Simu [1], M. Codreanu [1], C. Hudita [1], R. Ionescu [2]. [1] *Saint Stelian Hospital for Toxicodependency and Psychiatry, Bucharest, Romania* [2] *Obregia Hospital, Psychiatry Department, Bucharest, Romania*

Introduction: The long-term outcome of patients with addictive disorders is closely linked to the bio-psycho-social variables, the interactions between factors and, to psychiatric comorbidities.

Our case report is about a 28 years old male with an 8-years history of heroin dependence, and occasionally cocaine, ecstasy abuse.

4 years ago, he started a long-term Methadone maintenance treatment, and during this time he had several relapses and hospitalizations, most of them generated by recurrent major depressive episodes.

At his last admission in the detox clinic, transferred from the Emergency Hospital with a chronic amphetamine intoxication, minor ECG and EEG abnormalities, he had paranoid thoughts, delusional-hallucination behavior, ambivalence toward his mother, ideas of culpability, dysphoria, occasional suicidal thoughts, insomnia and fatigue, also opiates and amphetamines urine positive tests. At that moment he was under treatment with Methadone (75 mg/day ongoing) and Venlafaxine (stopped). He received an atypical antipsychotic as co-therapy (Quetiapine titrated up to 400 mg/day). After 3 weeks of hospitalization the symptomatology improved, with delusional thoughts and behavior remission.

In ambulatory, the patient was maintained on: Methadone 75 mg/day, Quetiapine 300 mg/day and received CBT, with a high compliance to pharmaco- and psychotherapy.

Conclusions: The patient outcome based on screening tests, CGI, MADRS and Quality of Life Scales proved the usefulness, efficiency and high tolerability of an atypical antipsychotic in the acute phase (psychotic-affective episode secondary to amphetamine intake) and also in long-term therapy for the prevention of drug and potentially addictive substances subsequent to depressive episodes along with Methadone.

P0060

Office based opiate treatment

M.G. Singh. *Department of Psychiatry, VAMC, Hampton, VA, USA*

Opiod dependence is a significant problem in the United States Of America and is undertreated .Only 20 % of patients get help inspite of effective treatments being available .The United Nations report that worldwide approximately 16 million people abuse opiates but only 7.8% receive treatment.

The new office based opiate agonists improve access to patients that are otherwise reluctant to use the federally supervised Methadone or Opiate treatment programs. I will review the assessment of opiate dependence and treatment options available and present my experience to date with patients on Buprenorphine.

In 2002 the US Food and Drug Administration approved 2 sublingual formulations of Buprenorphine for treatment of opiate addiction

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