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Recent evidences have consistently reported lower glutamate (Glu) levels in various brain regions, including the medial prefrontal cortex (mPFC), in chronic schizophrenia but findings in the early (EP) or in the prodromal phase of the disorder are equivocal. Although regular cannabis use has been associated with an increased risk of subsequent psychosis and with a perturbed Glu signalling, to date, the critical question of whether or not Glu abnormalities exist in EP and are related to cannabis use remains unanswered. Magnetic resonance spectroscopy was used to measure [Glu_{mPFC}] of 35 EP subjects (18 of whom were regular cannabis users) and 33 healthy controls (HC). For correlative analysis, neuropsychological performances were scored by a comprehensive cognitive battery. [Glu_{mPFC}] was lower in EP users comparing to both HC and EP non-users ($P=0.001$ and $P=0.01$, respectively), while no differences were observed between HC and EP non-users. In EP users Glu declined with age ($r=-0.46$; $P=0.04$) but this relationship was not observed in non-users. Among neuropsychological profiles, working memory was the only domain that differentiates patients depending on their cannabis use, with users having poorer performances. In summary, our research revealed that cannabis use in EP is associated with Glu decreased levels, which are normally not seen in the early phase of the disorder. This finding is in line with previous ¹H-MRS studies in cannabis users without a psychotic disorder and sheds light for the role of cannabis use in the progression of the disease.

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

<http://dx.doi.org/10.1016/j.eurpsy.2017.02.324>

EW0711

Molecular targets of the ethanol and original anticonvulsant in the treatment of alcohol dependence

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Objective Chronic exposure to alcohol causes neuroadaptive changes in the brain, which leads to the recurrence of the disease. Promising in this area is to find new safe and effective pharmacological agents acting on molecular targets of influence of alcohol in the CNS.

Methods Experiments were performed on male rats Wistar and male mice (CBAx57Bl/6)F1.U. Experimental animals were formed alcohol dependence, based on long-term use of alcohol solution. Animals in a state of alcohol dependence were injected original anticonvulsant meta-chloro-benzhydryl-urea. We evaluated parameters orienting-exploratory behavior and emotional reactivity of the animals in the test “open field”, the cellular and humoral immune response. Properties of benzodiazepine receptors of

the brain examined radioreceptor method using selective ligands [³H]flunitrazepam and [³H]Ro5-4864.

Results Chronic exposure to ethanol resulted in a significant change in the parameters of the experimental animal behavior and emotional reactivity in the test “open field”, observed suppression of immune response (~40%), and increase in the number of receptors on 54.8–59.4% associated with reduced receptor affinity. Administration of meta-chloro-benzhydryl-urea led to the abandonment of the use of ethanol, recorded a correction of the above immunological and behavioral disorders due to alcohol intoxication. Properties of benzodiazepine receptors in the brain of experimental animals receiving the drug at a dose of 100 mg/kg for 14 days, indicators affinity and receptor density were close to the values in the control group.

Conclusions Anticonvulsant has a modulating effect on the functional activity of the nervous and immune systems, reduces compulsive craving for alcohol.

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

<http://dx.doi.org/10.1016/j.eurpsy.2017.02.325>

EW0712

Serotonergic modulation of cognition: An acute challenge

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Abstract Serotonin is well known to affect the multifaceted construct of impulsivity. Lowering brain serotonin levels is shown to increase impulsive choice in delay-discounting tasks (1) but improves response inhibition in stop-signal paradigms. (2) Administration of the antidepressant citalopram in healthy people increases tendency to perform go choices in a Go/No-Go task independent of outcome valence (3). It is rather unclear thought how serotonergic neurotransmission affects several aspects of cognition. We administered a single dose of 20mg escitalopram, a selective serotonin reuptake inhibitor, to 66 healthy participants, aged 18–45 years old, in a double-blind, randomized, placebo-controlled, parallel-groups study. Acute escitalopram administration had a beneficial effect on inhibitory control with reduced stop-signal reaction time observed in the treatment group. Participants made significantly more errors in a probabilistic learning task and had lower accuracy during the discrimination stage in an instrumental learning task thus indicating a learning impairment. More errors in the CANTAB intra-extra dimensional set shift task were also observed in the escitalopram-treated group. Our findings following acute administration of a clinically relevant dose of escitalopram show a dissociate role for serotonin in modulating cognition mediated by a potentially differential modulation of fronto-striatal loops.

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

<http://dx.doi.org/10.1016/j.eurpsy.2017.02.326>

EW0713

Microstructural and metabolic disorders in CC of juvenile schizophrenia patients

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