quality of life in some patients with cognitive impairment due to NPH.

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

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1024

EV0695

Rural and urban childhood environment effects on episodic memory

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Introduction Childhoods in urban or rural environments may differentially affect risk for neuropsychiatric disorders. Here, we leveraged on dramatic urbanization and rural-urban migration since the 1980s in China to explore the hypothesis that rural or urban childhoods may differentially influence memory processing and neural responses to neutral and aversive stimuli.

Objectives Explore the underlying mechanisms of childhood environment effect on brain function and neuropsychiatric risk.

Methods We examined 420 adult subjects with similar current socioeconomic status and living in Beijing, China, but with differing rural ($n = 227$) or urban ($n = 193$) childhoods. In an episodic memory paradigm scanned in a 3 T GE MRI, subjects viewed blocks of neutral or aversive pictures in the encoding and retrieval sessions.

Results Episodic memory accuracy for neutral stimuli was less than for aversive stimuli ($P < 0.001$). However, subjects with rural childhoods apparently performed less accurately for memory of aversive but not neutral stimuli ($P < 0.01$). In subjects with rural childhoods, there was relatively increased engagement of bilateral striatum at encoding, increased engagement of bilateral hippocampus at retrieval of neutral and aversive stimuli, and increased engagement of amygdala at aversive retrieval ($P < 0.05$ FDR corrected, cluster size > 50).

Conclusions Rural or urban childhoods appear associated with physiological and behavioural differences, particularly in the neural processing of aversive episodic memory at medial temporal and striatal brain regions. It remains to be explored the extent to which these effects relate to individual risk for neuropsychiatric or stress-related disorders.

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

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1025

e-Poster Viewing: Neuroscience in Psychiatry

EV0696

Possible Involvement of Endogenous Opioids and Nitric Oxide in the Anticonvulsant Effect of Acute Chloroquine Treatment in Mice

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Introduction Chloroquine, a 4-aminoquinoline derivative, has long been used for the treatment of malaria and rheumatological disorders, including rheumatoid arthritis and systemic lupus erythematosus. Accumulating evidence now suggests potential use of chloroquine as a neuroprotectant. Studies have shown that nitric oxide (NO) pathway is involved in the chloroquine actions. Considering the fact that nitrergic neurotransmission plays a crucial role in the central nervous system functioning, in the present study we evaluated whether nitrergic system is involved in the anticonvulsant effects of chloroquine in a model of clonicseizure in mice.

Methods Clonic seizure threshold was determined by infusion of pentylenetetrazole (PTZ, 0.5%) at a constant rate of 1 mL/min into the tail vein of male Swiss mice (23–29 g). Minimal dose of PTZ (mg/kg of mice weight) needed to induce clonicseizure was considered as an index of seizure threshold.

Results Chloroquine (5 mg/kg, acutely 30 min before test, intraperitoneally), i.p significantly increased the seizure threshold. Acute co-administration of a non-effective dose of the non-selective NO synthase (NOS) inhibitor, L-NAME (L-NG-Nitro-L-arginine methyl ester hydrochloride,5 mg/kg, i.p.) or the selective inhibitor of neuronal NOS, 7-NI (7-nitroindazole, 40 mg/kg, i.p.) with an effective dose of chloroquine (5 mg/kg) inhibited its anticonvulsant effects. Co-administration of a non-effective dose the selective inducible NOS inhibitor, aminoguanidine (100 mg/kg, i.p.) with chloroquine 5 mg/kg did not alter its anticonvulsant effects.

Conclusion Chloroquine increases the PTZ-induced clonic seizure threshold in mice. We demonstrated for the first time that nitric oxide signaling probably through neuronal NOS could be involved in the anticonvulsant effects of chloroquine in this model of seizure in mice.

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

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1026

EV0697

Cannabis and confabulation: An intrusive relationship

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Introduction The association between the neurocognitive impact of cannabis use and deficits in working and declarative memory is well documented. Studies with cannabis users suggest that recognition memory is particularly susceptible to cannabinoid acute intoxication. Studies carried out in the 1970s using free memory tests, showed that cannabis users not only named fewer words having also a tendency to evoke intrusive memories. Interestingly, a recent study has exposed an association between cannabis consumption and increased likelihood of creating fake memories.

Objectives The main objective of this work is to do literature revision, framing old data with recent works, exposing the relationship between cannabis consumption and memory confabulation/intrusion.

Methodology Literature review, comparison and description of empirical data [1].

Results Recent studies show that both cannabis users and abstinents are more susceptible to create false memories, not being able to identify trap stimuli as events that never occurred.

Discussion/conclusions Changes in perception and memory deficits are two common consequences of acute marijuana intoxication. The fact that these deficits remain during drug abstinence demonstrates the relevance of better understanding the mechanisms by which cannabinoids alter such cognitive functions. Reductions in the activation of brain areas comprised in the lateral and temporal lobe and in frontal cortex zones involved in the processes of attention and performance monitoring may be a possible explanation.

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

Reference

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http://dx.doi.org/10.1016/j.eurpsy.2017.01.1027

EV0698

Cannabidiol's role as a potential target in the treatment for schizophrenia

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Objectives Schizophrenia is a debilitating psychiatric disorder which places a significant emotional and economic strain on the individual and society-at-large. Unfortunately, currently available therapeutic strategies do not provide adequate relief and some patients are treatment-resistant. Therefore there is urgent need for the development of mechanistically different and less side effect prone antipsychotic compounds. Recently, the endocannabinoid system has emerged as a potential therapeutic target for pharmacotherapy that is involved in a wide range of disorders, including schizophrenia. Modulation of this system by the main psychoactive component in cannabis, Δ9tetrahydrocannabinol (THC), induces acute psychotic effects and cognitive impairment. However, the non-psychotropic, plant-derived cannabinoid agent cannabidiol shows great promise for the treatment of psychosis, and is associated with fewer extrapyramidal side effects than conventional antipsychotic drugs.
Methods The aim of this review is to analyse the involvement of the endocannabinoid system in schizophrenia and the potential role of cannabidiol in its treatment.
Results and conclusions There is still considerable uncertainty about the mechanism of action of cannabidiol as well as the brain regions which are thought to mediate its putative antipsychotic effect. Further data is warrant before this novel therapy can be introduced into clinical practice.
Disclosure of interest The authors have not supplied their declaration of competing interest

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1028

EV0699

Psychotic symptoms in patients with nmda antibodies

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Introduction This paper is a review of literature about the relation in some cases between psychotic symptoms and NMDA antibodies. Most of these cases are early observed and treated by psychiatry, observing torpid evolution and symptoms that are rarely observed in Psychiatry patients, like visual hallucinations or rapid fluctuations of symptoms.
Objectives Make a review of psychotic symptoms and NMDA antibodies, to think about other options when we are in front of some unusual cases in psychiatry, and it seems that "nothing is working"
Methods Systematic review of pub med literature, applying the keywords: "psychotic" and "NMDA antibodies" of last 5 years.
Results We found that in most of cases the patients presents Opisthotonus, catatonia, and rhythmic and non-rhythmic involuntary movements of the mouth and jaw, and most of them had a psychiatric evaluation for those symptoms. There was no response to antipsicotic treatment. The treatment with corticoids and rituximab was effective.
Conclusions In psychiatry we have to think in some cases that maybe "the patient could have something else than a psychiatric disease", most when we found that the symptoms has a rare presentation and the treatment is not effective.
We encourage our colleagues to "think outside the box" when something like this occurs, and hesitate about our own valuations of the patients, when the case is atypical strange.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1029

EV0700

Charles-bonnet syndrome: Hallucinations are in the eye of the beholder

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Introduction Charles-Bonnet Syndrome (CBS) is a clinical entity characterized for visual hallucinations in patients with severe vision impairment and preserved cognitive state. Its pathogeny is still unknown, limiting management options. For diagnosis neurological and psychiatric disorders must be discarded. Treatment is based in three pillars: explaining to the patient the origin and nature of the symptoms, treating the visual deficit when possible, and pharmacotherapy with anti-psychotics.
Objectives and aims To outline the main characteristics and etiopathogenic theories of the CBS, so as to improve diagnosis and treatment.
Methods Basing on a case followed in mental health consults, we made a systematic review of the articles published in Medline (PubMed) in the last 5 years, with the following keywords, Charles-Bonnet Syndrome, hallucinations, deafferentation, visual impairment.
Results We found that all our case and the reported ones had in common the nature and characteristics of the hallucinations, the presence of a trigger, usually a new medicament, and the functional MRI patrons of activity; those patrons located the loss of input prior to the association cortex, which appeared hyper-excitable in functional MRI.
Conclusions Although the aetiology and pathogeny of CBS is still unclear, present data suggests that the key mechanism may be a dysregulation in the homeostatic adaptation of the neural pathway when it is left without external input, traducing a hyper-function of a physiological process, probably mediated by acetylcholine, as opposed with other neuropsychiatric pathologies, in which the cortex is the primary affected area.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2017.01.1030

EV0701

Is borderline personality disorder a neuroendocrine disease?

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Introduction Borderline personality disorder (BPD) is a disabling heterogeneous psychiatric disorder characterized by poor affect