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

X. Zhang[1,*], H. Yan[1], S. Shah[2], G. Yang[2], X. Zhao[1], J. Zhu[2], X. Zhang[1], J. Li[1], Y. Zhang[1], Q. Chen[2], A. Mattay[2], W. Yue[1], D.R. Weinberger[2], D. Zhang[1], H. Tan[2]
[1] *Peking University, Institute of mental health, Beijing, China*
[2] *Lieber Institute for Brain Development, Neuroimaging Core, Baltimore, USA*
* *Corresponding author.*

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

A. Abkhoo
Students' Scientific Research Center SSRC, Tehran University of Medical Sciences, Tehran, Iran, Medicine, Tehran, Iran

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

A. Barcelos*, A. Lopes, M. Bernardo, C. Adriana
Hospital Garcia de Orta, EPE, Psiquiatria e Saúde Mental, Almada, Portugal
* *Corresponding author.*

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.