Conclusions. Our preliminary findings suggest that ACCN1 (ASIC2) gene could be involved in modulating the susceptibility of BD patients to develop renal dysfunctions induced by chronic Li treatment.

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

http://dx.doi.org/10.1016/j.eurpsy.2017.01.317

O096

Association between two single-nucleotide polymorphism of TAAR1 gene and suicide attempts

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Introduction. TAAR1 is a G protein-coupled receptor expressed broadly throughout the brain. Recently, TAAR1 has been demonstrated to be an important modulator of the dopaminergic, serotonergic and glutamatergic activity.

Aims. Assessment of the relation between two single-nucleotide polymorphisms of TAAR1 gene, suicide attempts and alcohol abuse.

Methods. A total of 150 Polish patients were included, 59 subjects after suicide attempt vs. 91 controls. The chosen SNPs (rs759733834 and rs9402439) were studied using RFLP-PCR methods. The Hardy-Weinberg equilibrium was tested in control group.

Statistical tests. Chi² or Yeates Chi² Test were used.

Results. The mean age of study subjects and controls was: 38 ± 12.3 and 42 ± 12.8 respectively; 40% study males vs. 54% male controls. We did not observe the association between the carriage of the genotypes GG, GA and AA of rs759733834 polymorphisms in either of the groups. The distribution of genotypes in respect to rs9402439 polymorphism (CC, CG, GG) was also insignificant. Among patients with alcohol dependence, the frequency G allele of rs9402439 polymorphism was lower compared to non-addicted ones (27 vs. 47%) P < 0.01.

Conclusions. TAAR1 polymorphisms rs759733834 and rs9402439 are not related to suicide attempts. The carriage of allele G of rs9402439 polymorphism is related to lower risk of alcohol addiction OR 0.40 95%CI 0.20–0.81. To our knowledge, this is the first study on the TAAR1 receptor and the risk of suicide and it might offer a new insight into genetic etiology of TAAR1 receptor.

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

http://dx.doi.org/10.1016/j.eurpsy.2017.01.318

O098

The effects of deep-brain magnetic stimulation (DMS) on white matter deficits: New mechanism in major depressive disorder (MDD) treatment

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Deep-brain magnetic stimulation (DMS) is an effective therapy for various neuropsychiatric disorders including major depression disorder. The molecular and cellular mechanisms underlying the impacts of DMS on the brain remain unclear. Studies have reported abnormalities in the white matter of depressive brains, suggesting the involvement of myelin and oligodendrocyte pathologies in the development of major depressive disorder. In this study, we use a cuprizone induced demyelination animal model to generate depressive-like behaviours and white matter and oligodendrocyte damages. Meanwhile, we treated the animal with DMS 20 minutes daily during the cuprizone challenge or recovery period. Behavioural tests, including nesting, new object recognition, working memory and depression-like behaviours were tested periodically. Histological staining and western blotting were used to examine the underlying mechanism of DMS. We found that DMS reverse cuprizone induced behavioural deficits in acute demyelination but not during the recovery period. DMS alleviated demyelination and inflammation induced by cuprizone. During the recovery period, DMS had no impacts on overall neural progenitor cell proliferation, but enhanced the maturation of oligodendrocyte. This data suggest that DMS may be a promising treatment option for improving white matter function in psychiatric disorders and neurological diseases in future.