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EV0602

Effect of chronic exposure of Losartan in mouse prenatal alcohol exposure (PAE) model

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Background and aims Foetal alcohol syndrome (FAS) is a condition that currently affects 1% of babies born in Europe and North America. It is characterised by memory impairment, developmental delay and distinctive facial features. This research uses a mouse prenatal alcohol exposure (PAE) model to explore the effects of PAE on learning, memory and to explore the potentially beneficial effects of common drugs previously shown to have cognitive enhancing effects in both humans and animals.
Methods Sixty mice ($M = 30$ $F = 30$) C57 mice were exposed to 5% ethanol throughout pregnancy. After weaning the offspring received Losartan (10 mg/kg) via their drinking water for 8 weeks. At 3 months, learning and memory was assessed using the novel object recognition paradigm.
Results PAE caused a significant decrease in offspring body weight. Treatment with Losartan caused no growth impairment or renal damage. Novel object recognition indicated that PAE caused male offspring to spend significantly less time exploring the novel object than controls and that treatment with Losartan had the effect of improving awareness of the novel object both in the control and alcohol group and decreasing anxiety ($P \leq 0.05$). A significant opposite effect was noticed in the female alcohol progeny when compared to the male alcohol progeny ($P \leq 0.05$). Losartan in female alcohol progeny had no effect on anxiety. Male control Losartan spent more time exploring the novel object than male alcohol Losartan ($P \leq 0.05$).
Conclusions Losartan had no deleterious effects on the development of the animals, and was able to improve learning and memory in control animals without effect in PAE mice.
Disclosure of interest The author has not supplied his declaration of competing interest.

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EV0603

Kleefstra syndrome: Considerations about treatment strategy in 2 patients with a causative Ehmt1 mutation and apathy

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Introduction Kleefstra syndrome [OMIM: 610253] is caused by a 9q34.3 micro-deletion or an intragenic mutation in the EHMT1 gene. Its core phenotype comprises intellectual disability, childhood hypotonia and distinct dysmorphisms. The syndrome can be associated with congenital anomalies, epilepsy, cardiac arrhythmias and a typical sleep pattern. Starting from adult age, a regressive phenotype may develop.
Objectives Further delineation of the neuropsychiatric phenotype.
Aims Formulating a comprehensive treatment approach.
Methods Detailed examination of two patients with EHMT1 mutation.
Results Patient 1, male aged 34 years, showed recurrent behavioral problems with aggression and self-injuries as well as obstipation. Elsewhere, a diagnosis of autism was established. Aged 24, he suffered from some epileptic seizures. Recently, paroxysmal atrial fibrillation was diagnosed. Neither treatment with pipamperone and risperidone nor with valproate was effective for behavioral control. Array analysis and metabolic screening did not reveal abnormalities. Whole exome sequencing revealed an intragenic EHMT1 mutation. Patient 2, female aged 53 years, was known with childhood epilepsy and developed gradual decline of general functioning with motor slowing from her third decade. In her thirties, a mood/anxiety disorder was suspected for which several antidepressants were given without any effect. Array analysis was normal. A pathogenic nucleotide deletion was identified resulting in a frame-shift in exon 21 of the EHMT1 gene. In both patients marked apathy was observed (AES = 62 and 64, respectively).
Conclusions Apathy syndrome in Kleefstra syndrome should be differentiated from depression and autism. Apart from treatment with selected psychotropics, individually targeted contextual measures should always be implemented.
Disclosure of interest The authors have not supplied their declaration of competing interest.

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e-Poster viewing: Mental health care

EV0604

Innovative home based assertive outreach service for treatment of schizophrenia in Larkano, Pakistan (SOUL): Programme implementation and outcomes at the end of five years

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Introduction There is a significant service gap in provision of essential treatment to patients with severe mental disorders in low-income countries, which leads to increased mental health disability and bigger disease burden on the families and society. The SOUL programme is a first of its kind in the country, which utilizes assertively engaging patients at their homes.
Objectives The key objectives are early recognition, treatment and psychosocial support to patients with the diagnosis of schizophrenia. Additional objectives include social recovery of the patients, psycho education to family members and generating clinical and functional outcomes.
Methods Programme design developed by host psychiatry department through stakeholder consultation. Training was undertaken for programme team and included training on use of outcome measures namely Brief Psychiatric Rating Scale (BPRS), Clinical Global Impression (CGI) and Global Assessment of Functioning (GAF). Hosting carers and families meetings on regular intervals serve the purpose of family psycho-education and receiving informal feedback about the service.
Results Preliminary findings on clinical and functional outcomes of cohort of 125 patients recruited over continual basis over 5 years are presented. Complex community intervention shows significant change in all outcome scales (with good effect size) with before and after analysis at one year. The programme demonstrated excellent engagement with patients and very low dropout rate.
Conclusions Low cost community intervention involving trained doctor and psychiatric nurses working under close supervision of a