of adenosine in the CNS. In PTZ-kindling group gene expression of ENT-1 increased by 1.7 fold. Levetiracetam decreased significantly the mRNA expression of ENT1 by 0.5 fold change in hippocampus that supposed to prevent the influx of extracellular adenosine into the cells.
Conclusions: The results suggest that the neuroprotective effect of levetiracetam observed during the investigation could have a straight connection to its action on A_1 adenosine receptors.

559 | Proteomic analysis of a rat model of genetic generalised epilepsy with absence seizures

Debbie Chong[1]; Anna Harutyunyan[2]; Anup Shah[3,4]; Piero Perucca[1,2,3,4,5,6]; Nigel Jones[1,2,5]; Ralf Schittenhelm[3,4]; Alison Anderson[1,2,5]; Pablo Casillas-Espinosa[1,2,5]
[1]Central Clinical School, Monash University, Neuroscience, Melbourne, Australia; [2]University of Melbourne, Medicine (The Royal Melbourne Hospital), Parkville, Australia; [3]Monash University, Monash Proteomics & Metabolomics Facility, Clayton, Australia; [4]Monash University, Monash Biomedicine Discovery Institute, Clayton, Australia; [5]Alfred Health, Neurology, Melbourne, Australia; [6]Austin Hospital, Neurology, Heidelberg, Australia

Purpose: Absence epilepsy is the most common form of genetic generalised epilepsy (GGE). Its seizures are characterized by behavioural arrests, automatisms, and a 3 Hz spike-wave discharge on the EEG (Danober L et al. Prog Neurobiol 1998;55(1):27–57. Scheffer I et al. Epilepsia 2017;58(4):512–21.). While the symptoms of absence epilepsy are well-known, associated molecular changes are less understood. Proteomic analysis is increasingly utilised to elucidate molecular changes involved in epilepsy development. This has improved our understanding of disease progression and allows for the identification of potential therapeutic targets and biomarkers. The Genetic Absence Epilepsy Rats from Strasbourg (GAERS) are a well-validated GGE model. Like in humans, the proteomic changes resulting in GGE in GAERS are unclear. Here, we assess proteomic differences between the GAERS and Non-Epileptic Control (NEC) strains to explore the molecular mechanisms and to identify novel potential biomarkers and treatment targets in GGE.
Method: Liquid chromatography high-resolution tandem mass-spectrometry (LC-MS/MS) was performed using the somatosensory cortex (SCx) and thalamus of the GAERS (n = 6) and NEC rats (n = 6). Differentially expressed proteins between the groups were determined using the limma package in R. Enriched pathways were identified using g:Profiler.
Result: A total of 123 and 102 proteins were found to be significantly differentially expressed between GAERS and NEC in the thalamus and the SCx respectively. Among these proteins, aspartoacylase, glial fibrillary acidic protein, and glutamate metabotropic receptor 2 have been previously associated with absence epilepsy, and an additional 11 proteins were found to be previously associated with epilepsy in general. Pathway analysis identified terms that were mostly related to metabolism.
Conclusions: This study has identified both novel and previously identified absence epilepsy-associated proteins and candidate biological pathways. These results could be utilised to inform future biomarker and therapeutic target research.

567 | Identification of microRNAs as biomarker candidates for epilepsy-associated psychiatric comorbidities in animal models of epilepsy

Eva-Lotta von Rüden[1]; Heike Janssen-Peters[2]; R. Maarten van Dijk[1]; Isabel Seiffert[1]; Ines Koska[1]; Christina Möller[1]; Thomas Thum[2]; Heidrun Potschka[1]
[1]Ludwig-Maximilians-University Munich, Inst. of Pharmacology, Munich, Germany; [2]Hannover Medical School (MHH), Inst. of Molecular and Translational Therapeutic Strategies (IMTTS), Hannover, Germany

Purpose: Psychiatric comorbidities like anxiety and depression have been identified as prevalent and serious comorbidities, which have a major impact on quality of life in patients suffering from epilepsy. Therefore, there is a need to detect and manage these comorbidities.
We aimed to identify microRNAs as potential circulatory biomarkers for identification of patients with epilepsy-associated psychiatric comorbidities.
Method: The microRNA expression profile (750 microRNAs) was analyzed in blood samples of rats from the electrical post-status-epilepticus (SE) model (naïve $n = 5$, sham $n = 5$, epileptic $n = 6$). Based on missing values, p value ($P < 0.05$), fold change (<-1 and >1), correlation with seizure frequency and duration (coefficient <0.5) to exclude microRNAs directly related to epilepsy, and CT value (<30) microRNAs were preselected ($n = 11$). MicroRNA levels were correlated with selected behavioral and biochemical parameters (e.g. nest building, brain-derived neurotrophic growth factor (BDNF); Spearman). The regulation of preselected microRNAs was further assessed by quantitative real-time PCR in three well-described animal models of epilepsy (amygdala kindling with focal/generalized seizures, chemical induced SE).