The effects of CLCN2 knockout on epileptic absence seizures

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Epilepsy is a neurological disorder affecting about 1 percent of Americans. Epileptic seizures are caused by nerve cell activity in the brain and can vary from being nearly undetectable to long periods of shaking. Idiopathic generalized epilepsy includes a group of epileptic disorders that are believed to have a strong genetic basis. The seizures of interest here are petit mal seizures, or absence seizures. Absence epilepsy is one of the most common forms of childhood epilepsy. It is characterized by brief, frequent seizures and affects children from the ages of 4 to early adolescence. Absence seizures are also characterized by generalized spike-and-slow wave electrical discharges (SWD), which can be detected using electroencephalography (EEG). Due to the prevalence and comorbidities associated with absence epilepsy, it is important to study it further. Based on research published in a 2003 paper by Haug et al, we decided to examine the effects of a chloride channel gene CLCN2 in a mouse model. Data was collected via EEG recordings of mice over a 5-day period. Two mouse models were used: wild-type mice and CLCN2 knockout mice. EEG recordings were scored for spike-and-wave discharges to determine seizure occurrence. Due to time constraints, only limited data was obtained. This data confirmed the hypothesis that CLCN2 KO mice would have increased seizures. However, the insufficient data prevented us from creating a significant conclusion. Nevertheless, important procedural information was found to aid in future experiments.