Background

- Focal cortical dysplasia (FCD) is the underlying cause of pharmacoresistant epilepsy in many children.
- Surgical resection is the treatment of choice.
  - Not always curative.
- Mammalian target of rapamycin (mTOR) pathway has recently been implicated in FCD pathogenesis.
- Current mouse model has led to increased neuronal size, lamination defects, and spontaneous seizures.
- However, no developmental timeline has been created so far.

Methods

- Continuous video-EEG recordings.
- For both FCD and control mice age 1 month or 6 months.
- Fluorescent immunostaining of tissue from control and FCD mice.
- Cell counts performed using Neurolucida software.

Results

- Video-EEG for 5 control and 6 FCD mice showed:
  - No seizure activity in control mice.
  - 0.1 - 3.8 seizures/hour for FCD mice.
- Dyslamination and abnormal migration start p7 and worsen until p21.
- Ongoing efforts:
  - How to adapt video-EEG to neonatal and juvenile mice.

Conclusions

- FCD mice seize spontaneously with the surprising early finding of a trend toward decreased seizures with age.
- Neuronal migration is highly affected, as expected, in these animals, and worsens after p7, perhaps due to increased glial cell proliferation.

Implications

- Characterize time course with which EEG and developmental migration patterns change in FCD mice.
- Determine the best time course for treatment of children with this devastating type of epilepsy.