**INTRODUCTION**

65 million people are currently being treated for epilepsy, a disease characterized by recurring seizures. Seizures are caused by abnormal hyperactivity of glutamatergic neurons ultimately leading to a neurotoxic environment, neuronal death, and an increase in proinflammatory cytokines, such as interleukin-1β (IL-1β). We discovered IL-1R1, the cognate receptor for IL-1, is expressed on glutamatergic neurons in the dentate gyrus, which may indicate neuronal IL-1R1 potentially plays an unknown role during epileptogenesis. Therefore, it is important to examine the role of neuronal IL-1R1 in a model of epilepsy.

**OBJECTIVES**

Our objectives were 1) to identify a relationship between IL-1R1 and seizures; and 2) to examine the effect of chronic IL-1 signaling on glutamatergic neurons in epilepsy.

**MATERIALS AND METHODS**

Intracerebroventricular (ICV) injections: Animals are anesthetized, and with the to the chart below, 4 days after injections, animals are euthanized and perfusion fixed with 4% paraformaldehyde or 24 h after KA injection the animals were used for Kainic acid (KA) injections. Intracerebroventricular (ICV) injections: Animals are anesthetized, and with the to the chart below, 4 days after injections, animals are euthanized and perfusion fixed with 4% paraformaldehyde or 24 h after KA injection the animals were used for Kainic acid (KA) injections.

**EXPERIMENTAL DESIGN**

**RESULTS**

IL-1R1 is expressed on glutamatergic neurons in the dentate gyrus of the hippocampus

Contra

**CONCLUSIONS**

- IL-1R1 is located on glutamatergic neurons in the dentate gyrus.
- Neuronal IL-1R1 expression is upregulated during KA induced seizures.
- IL-1R1 does not initially change average seizure severity.
- Chronic neuronal IL-1R1 signaling may alleviate seizure severity via an unknown mechanism.
- Further research will focus on the effects of cell type specific IL-1R1 signaling on seizure severity.

**REFERENCES**

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