ABSTRACT

INTRODUCTION

RESULTS

CONSTITUTIVE LOSS OF SHANK3 IN MICE PRESERVES ASSOCIATIVE LEARNING AND MEMORY AFTER TRAUMATIC BRAIN INJURY

Trauma is the leading cause of death and disability in patients between the ages of 1-44 with TBIs contributing to a nearly a third of deaths. Presently, approximately 2% of the U.S. population are afflicted with disabilities and behavioral deficits as a result of a TBI. Despite promising preclinical data, clinical trials have failed to produce effective therapies for this highly morbid injury process. The lack of effective therapies has led to an increased interest in understanding the genetic differences involved in cognition and neurodegeneration. This has led to the study of Shank3, which plays a key role in the synaptic loss associated with Alzheimer’s disease (AD).

METHODS

HYPOTHESIS

CONCLUSION

We hypothesized that mice with the constitutive loss of Shank3 would attenuate deficits in learning and memory after traumatic brain injury.

CONSTITUTIVE LOSS OF SHANK3 IN MICE PRESERVES ASSOCIATIVE LEARNING AND MEMORY AFTER TRAUMATIC BRAIN INJURY

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INTRODUCTION

The CDC estimates that nearly 3 million Americans sustain a traumatic brain injury each year with a high degree of long-term neurocognitive morbidity. TBI results in a variety of neuropathological hallmarks including the generation of amyloid beta oligomers (AβOs). AβOs at the synapses incite dysfunction and degradation of synaptic receptors. Shank3's newly discovered role as an AβO receptor may be one of the putative mechanisms behind the neurocognitive protection seen after the constitutive loss of Shank3.