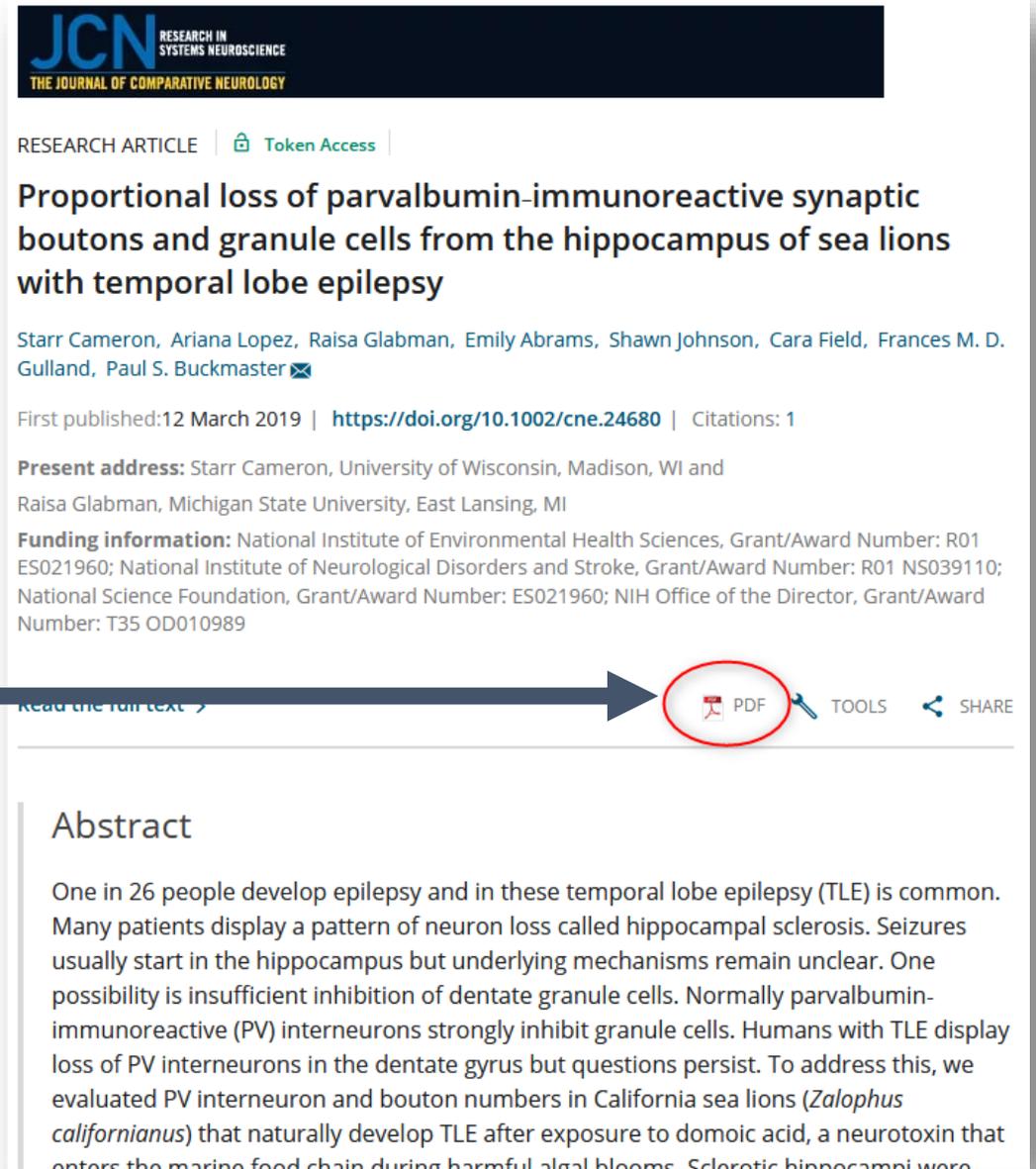


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Proportional loss of parvalbumin-immunoreactive synaptic boutons and granule cells from the hippocampus of sea lions with temporal lobe epilepsy

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Abstract

One in 26 people develop epilepsy and in these temporal lobe epilepsy (TLE) is common. Many patients display a pattern of neuron loss called hippocampal sclerosis. Seizures usually start in the hippocampus but underlying mechanisms remain unclear. One possibility is insufficient inhibition of dentate granule cells. Normally parvalbumin-immunoreactive (PV) interneurons strongly inhibit granule cells. Humans with TLE display loss of PV interneurons in the dentate gyrus but questions persist. To address this, we evaluated PV interneuron and bouton numbers in California sea lions (*Zalophus californianus*) that naturally develop TLE after exposure to domoic acid, a neurotoxin that enters the marine food chain during harmful algal blooms. Sclerotic hippocampi were

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Immunoreactive synaptic boutons in the hippocampus of sea lions

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Abstract
One in 26 people develop epilepsy and in these temporal lobe epilepsy (TLE) is common. Many patients display a pattern of neuron loss called hippocampal sclerosis. Seizures usually start in the hippocampus but underlying mechanisms remain unclear. One possibility is insufficient inhibition of dentate granule cells. Normally parvalbumin-immunoreactive (PV) interneurons strongly inhibit granule cells. Humans with TLE display loss of PV interneurons in the dentate gyrus but question persists. To address this, we evaluated PV interneuron and bouton numbers in California sea lion (*Zalophus californianus*) that naturally develop TLE after exposure to domoic acid, a neurotoxin that enters the marine food chain during harmful algal blooms. Sclerotic hippocampi were identified by the loss of Nissl-stained hilar neurons. Stereological methods were used to estimate the number of granule cells and PV interneurons per dentate gyrus. Sclerotic hippocampi contained fewer granule cells, fewer PV interneurons, and fewer PV synaptic boutons, and the ratio of granule cells to PV interneurons was higher than in controls. To test whether fewer boutons was attributable to loss versus reduced immunoreactivity, expression of synaptotagmin-2 (sy2) was evaluated. Sy2 is expressed in boutons of PV interneurons. Sclerotic hippocampi displayed proportional losses of sy2-immunoreactive boutons, PV boutons, and granule cells. There was no significant difference in the average numbers of PV- or sy2-positive boutons per granule cell between control and sclerotic hippocampi. These findings do not address functionality of surviving synapses but suggest reduced granule cell inhibition in TLE is not attributable to anatomical loss of PV boutons.

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