

## Supplementary Figure Legends

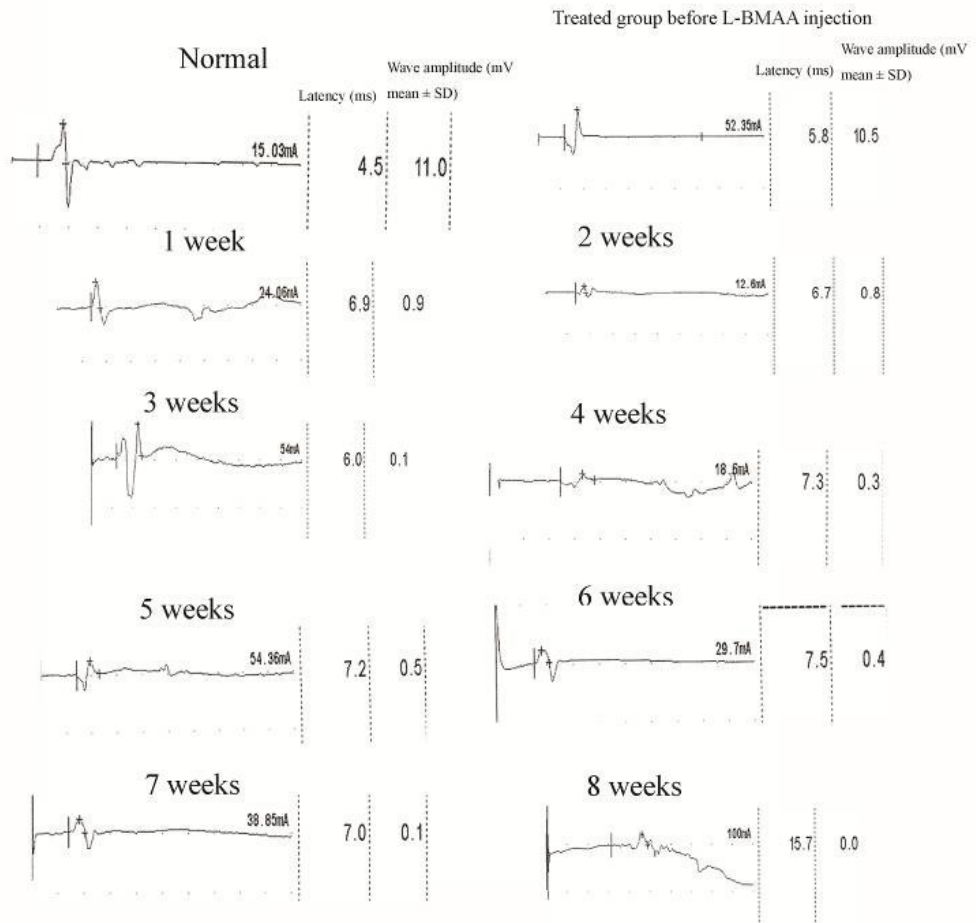
**Supplementary Figure 1.** L-BMAA injection induced clinical severity, as detected by motor-evoked potential (MEP) latencies and amplitudes. Figures shows MEP waveforms at each time point. MEP amplitude became significantly lower following L-BMAA injection compared to rats in the control group, and latency was also significantly prolonged, especially from four weeks to eight weeks post L-BMAA injection.

**Supplementary Figure 2.** Analysis of motor unit potentials in L-BMAA-treated rats. Mean amplitude and mean duration of MUPs, as well as the percentage of polyphasic potentials in L-BMAA-treated rats, were much higher than rats in the control group.

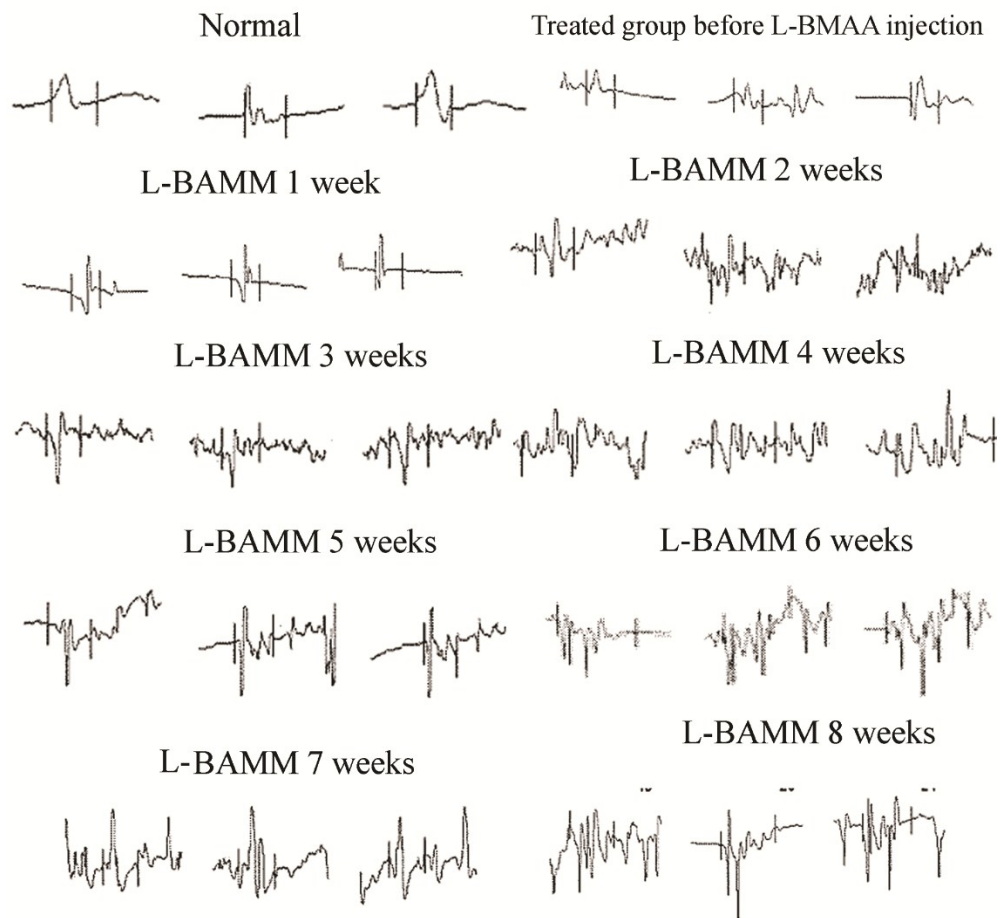
**Supplementary Figure 3.** (A-D): L-BMAA injections increased GSK3 $\beta$  expressions in CNS of treated rats. GSK3 $\beta$  expression was upregulated, especially at 6-8 weeks post-injections in L-BMAA-treated rats, as shown by immunostaining of GSK3 $\beta$ , and counterstained with hematoxylin. (E-H): L-BMAA injections increased tau-5 expressions in CNS of treated rats. Expression of tau-5 increased with time in L-BMAA-treated rats as shown by immunostaining, and counterstained with hematoxylin. SC: spinal cord; Scale bar = 100  $\mu$ m.

**Supplementary Figure 4.** Gradual loss of neurons in the CNS of L-BMAA-treated rats are shown by nissl staining (A-O). SC: spinal cord; BC: brain cortex; H: hippocampus; Scale bar = 100  $\mu$ m.

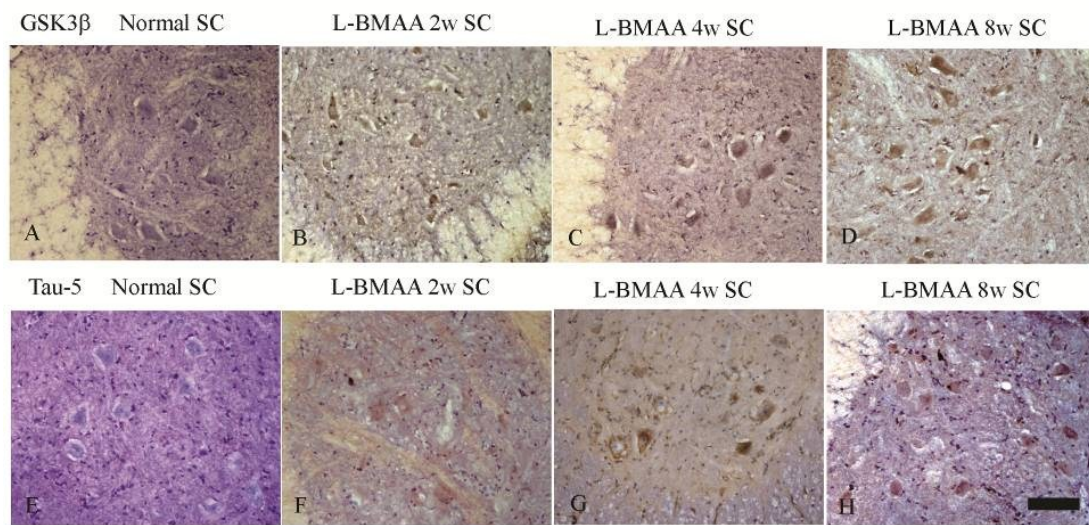
# Supplementary Figure 1



## Supplementary Figure 2



### Supplementary Figure 3



**Supplementary Figure 4**

