



**SUPPLEMENTARY FIG. S4.** Effect of agonists and inverse agonists of GABA<sub>A</sub> receptors under ischemic conditions. **(A)** Response of the regular spiking (RS) neuron model to GABA<sub>A</sub> receptor agonists under ischemic conditions. The values of extracellular potassium and intracellular chloride are shown in the upper panel. Under these conditions, the model reaches depolarization block and GABA<sub>A</sub>R agonists are unable to rescue the neuron from depolarization block. Ischemic conditions were modeled as an increase in extracellular potassium concentration and continuous activation of GABA<sub>A</sub>Rs by the increased extracellular GABA levels seen in ischemia, but not post-traumatic brain injury (TBI). **(B)** Response of the RS neuron model to GABA<sub>A</sub> receptor inverse agonists under ischemic conditions. GABA<sub>A</sub> receptor inverse agonists partially rescue the neuron from depolarization block under ischemic conditions.