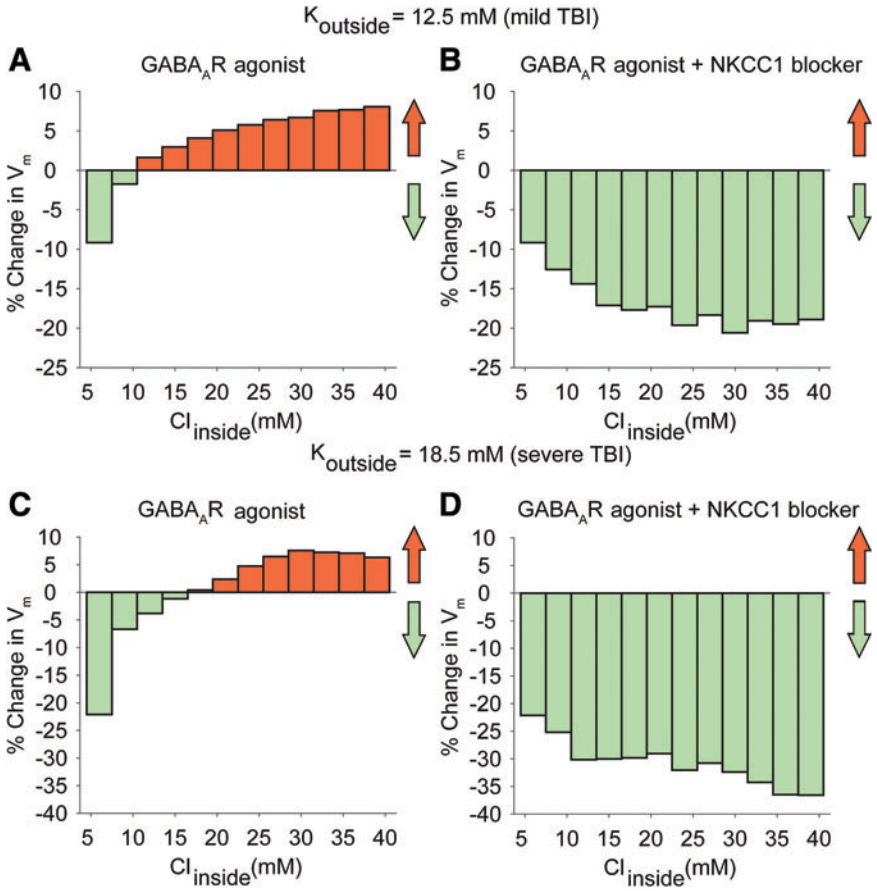

SUPPLEMENTARY FIG. S2. $GABA_A$ receptor agonists, in combination with Na^+-K^+-$2Cl^-$ (NKCC1) co-transporter blockers, can reduce the median membrane potential across a wide range of pathological ion concentations. **(A)** Effect of $GABA_A$ receptor agonists on the percent change in median membrane potential of the regular spiking (RS) neuron model for mild traumatic brain injury (TBI). Note that values >0 represent a pathological worsening reflecting depolarization of the median membrane potential, whereas values <0 represent therapeutic benefits caused by hyperpolarization of the median membrane potential. Under these conditions, $GABA_A$ receptor agonists lose their therapeutic efficacy as the intracellular chloride concentration exceeds the physiological range. **(B)** Same as in **A**, but for the combination of $GABA_A$ receptor agonist and NKCC1 co-transporter blocker. By blocking NKCC1 co-transporter and hence reducing intracellular chloride concentration, this combination of drugs can hyperpolarize the model neuron (and thus help to rescue it from depolariziation block) across a wide range of intracellular chloride concentrations. This suggests that this combination of drugs can have a therapeutic benefit in post-TBI brain states. (**C, D**) Same as **A, B,** but for $K_{outside} = 18.5$ mM, reflecting more severe TBI.