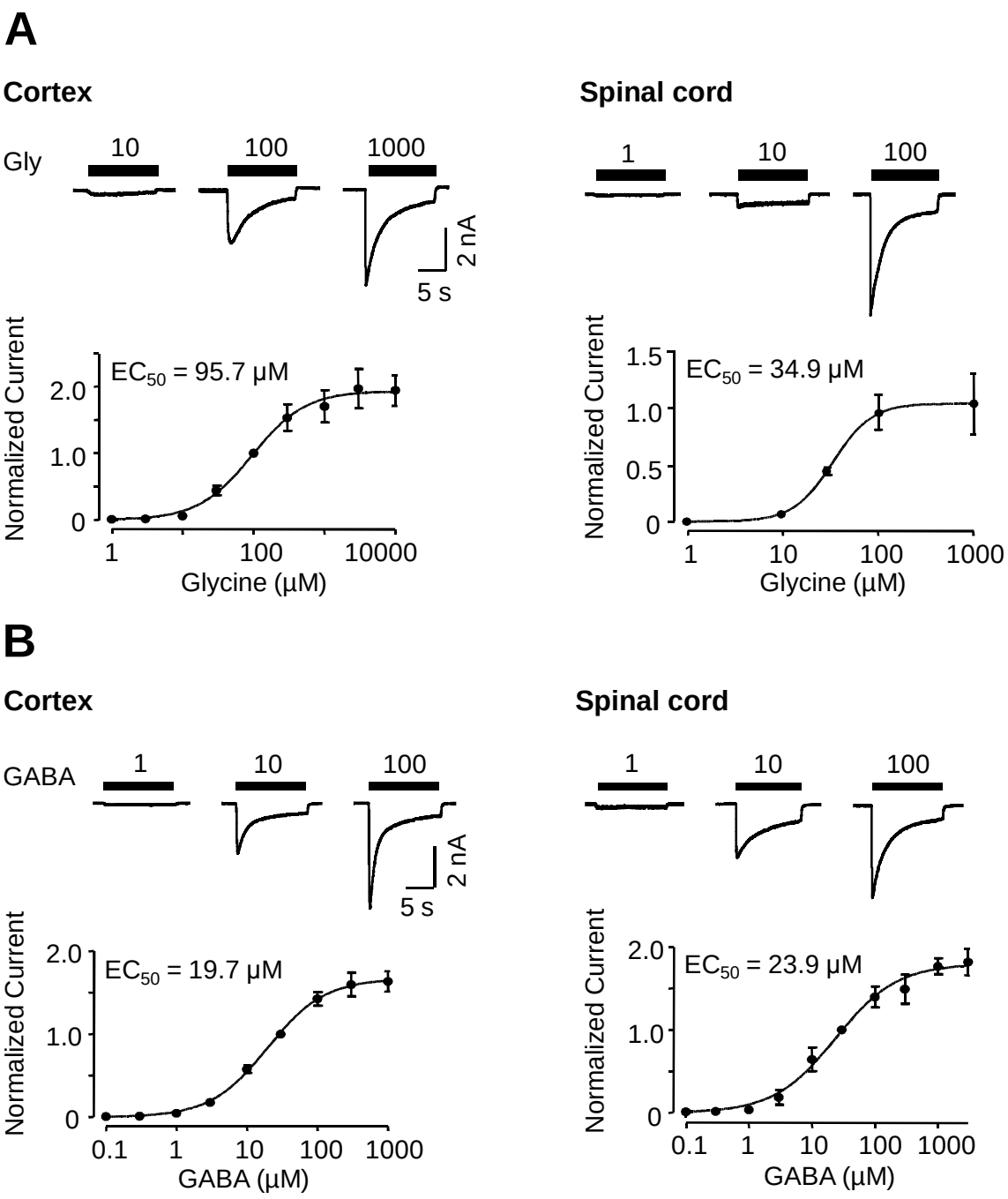
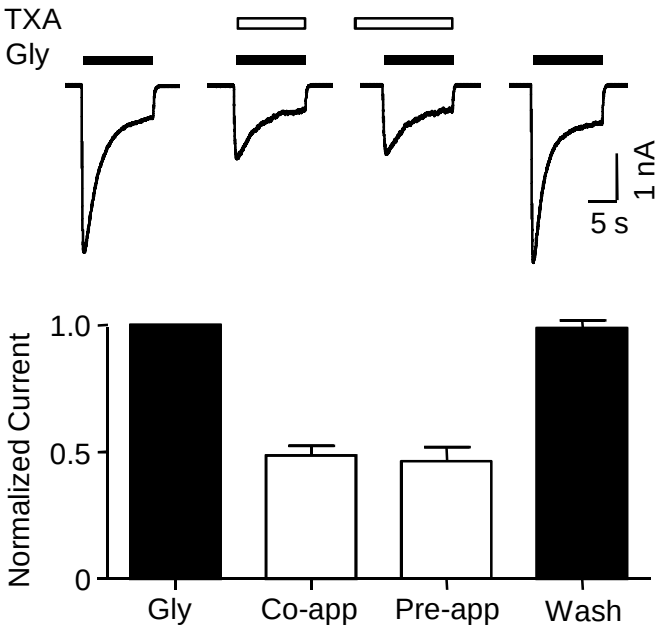
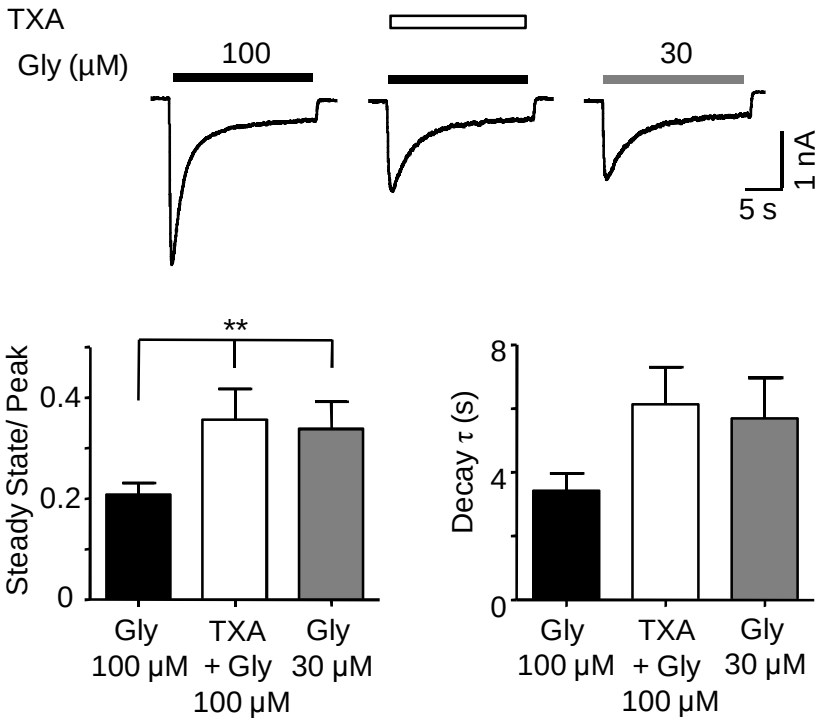


Supplemental Figure 1

Concentration-dependent currents for glycine and GABA in cortical and spinal cord neurons. **(A)** Currents evoked by glycine (1 – 1000 µM) in cortical and spinal cord neurons. The corresponding concentration–response plots show that EC_{50} = 95.7 ± 16.2 µM (*n* = 5) in the cortex and the EC_{50} = 30.9 ± 14.6 µM (*n* = 5) in the spinal cord. **(B)** Currents evoked by GABA (1 – 1000 µM) in cortical and spinal cord neurons. The corresponding concentration–response plots show that the EC_{50} = 19.7 ± 2.3 µM (*n* = 4 – 6) in the cortex and the EC_{50} = 23.9 ± 2.4 µM (*n* = 5 - 7) in the spinal cord. Data are mean ± SEM.

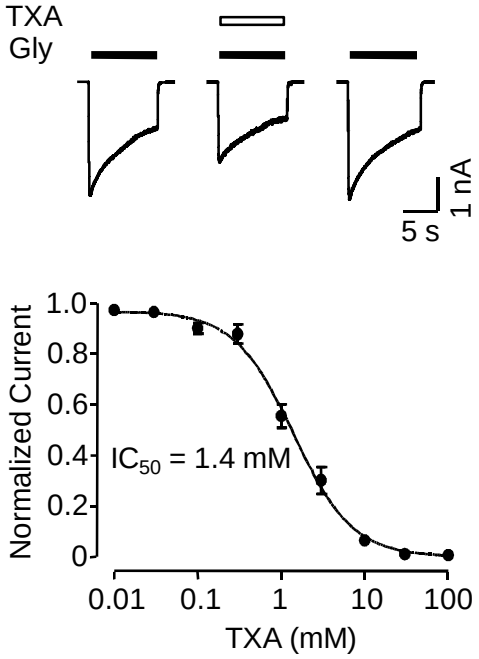


Supplemental Figure 2
TXA inhibition is similar whether TXA is pre-applied or co-applied with glycine. TXA (1 mM) was co-applied with glycine (100 µM) or pre-applied 30 s prior to glycine. Pre-application did not affect the extent of TXA inhibition (*n* = 5). Data are Mean ± SEM.

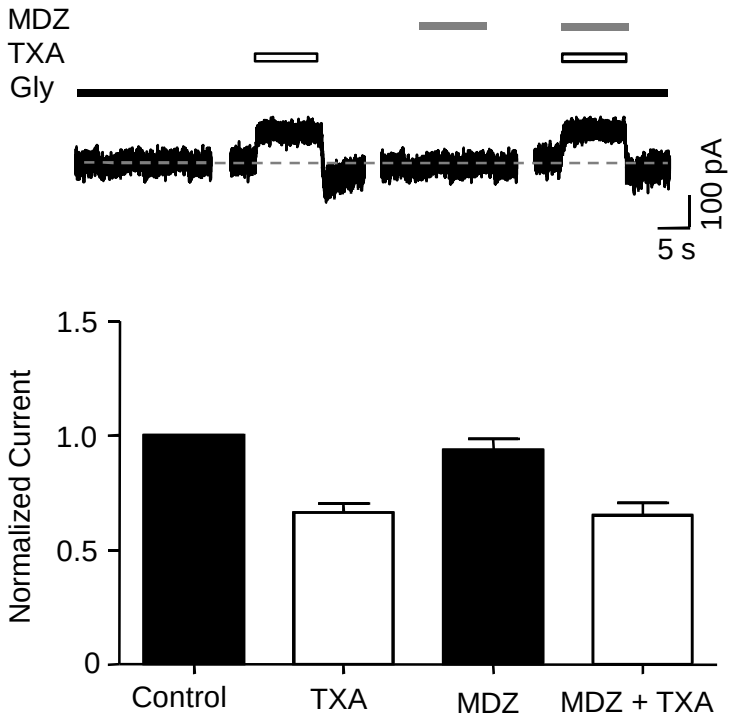


Supplemental Figure 3

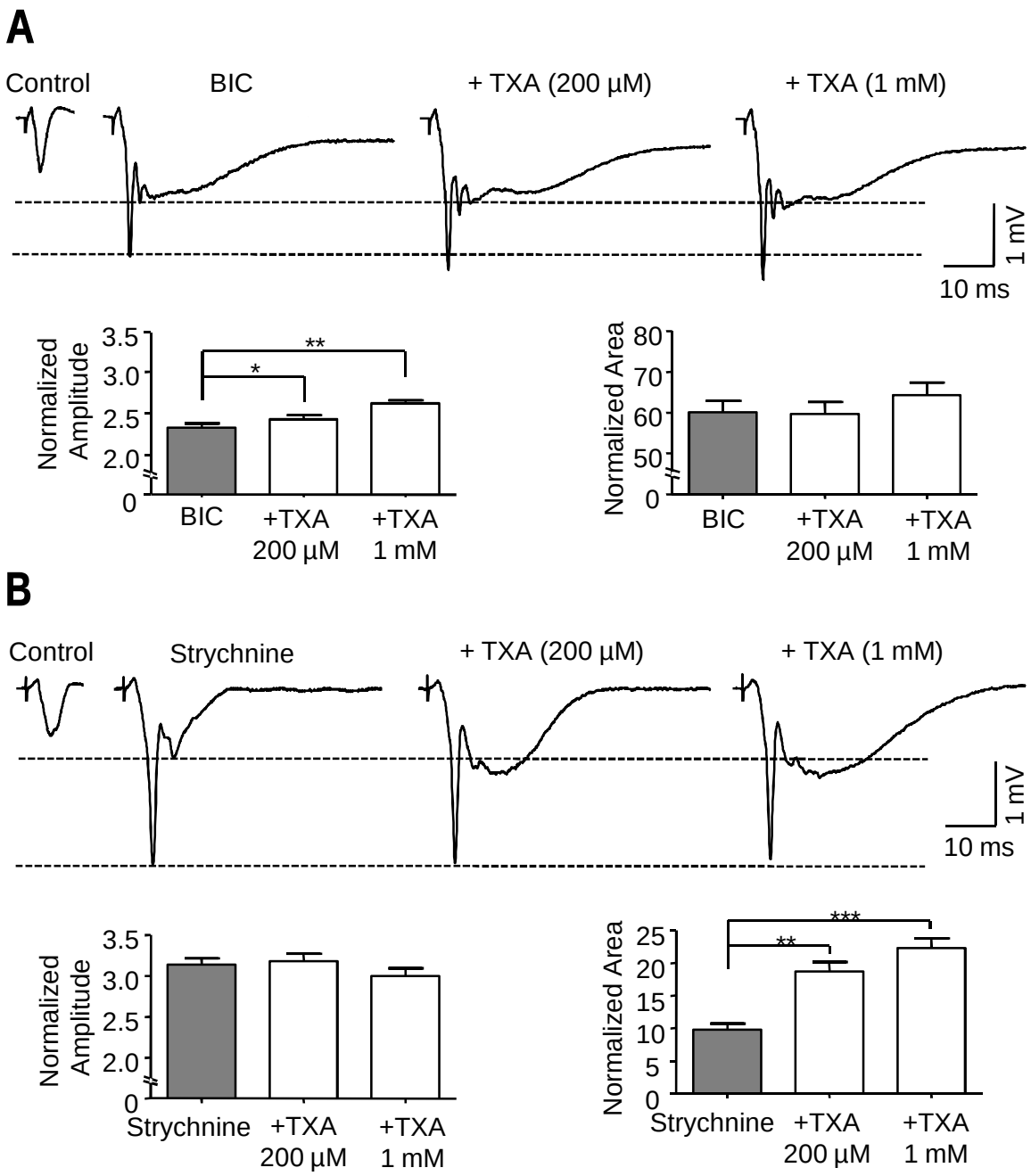
TXA inhibition of glycine receptors decreases receptor desensitization. Prolonged applications of glycine (100 and 30 μM) in the absence and presence of TXA were recorded to determine the steady state of the current. The chart on the left shows the ratio between the steady state and the peak of the current while the chart on the right shows the decay τ as a measure of receptor desensitization. In the presence of TXA the steady state/ peak ratio and the decay τ increase from 0.21 ± 0.02 and 3.4 s ± 0.5 s to 0.36 ± 0.06 and 6.1 s ± 1.1 s, respectively. A similar steady state/peak ratio and decay τ is observed with currents activated by glycine (30 μM; 0.34 ± 0.05 and 5.7 s ± 1.3 s). Data are mean ± SEM.



Supplemental Figure 4
TXA inhibits glycine currents in spinal cord neurons. Inhibition of glycine (30 µM)-activated currents by TXA (1 mM) in spinal cord neurons and the corresponding concentration–response plot (IC_{50} = 1.4 ± 0.1 mM, *n* = 5 – 7). Data are mean ± SEM.

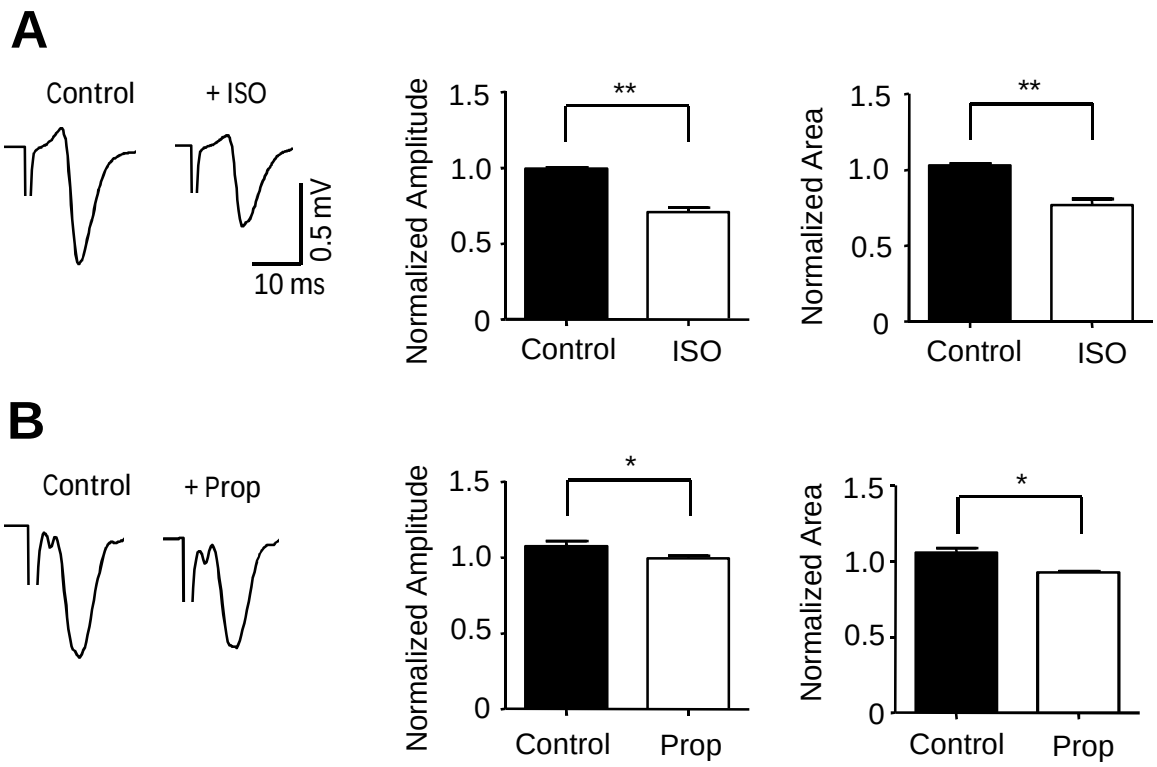


Supplemental Figure 5
Midazolam failed to reverse TXA inhibition of current evoked by low concentrations of glycine. The chart shows that coapplication of midazolam (MDZ 1 μM) with TXA (0.1 mM) had no effect (65.1% ± 5.5% of control) on TXA-mediated inhibition of glycine (10 μM) current (*n* = 5). Data are mean ± SEM.



Supplemental Figure 6

TXA increases the amplitude of the evoked field responses in the presence of either bicuculline or strychnine. **(A)** Bicuculline (10 µM) increased the amplitude (234.2% ± 5.3%) and the area (6074.0% ± 214.4%) of the evoked field responses as compared to control (*n* = 5). Subsequent coapplication of TXA (200 µM and 1 mM) further enhanced the amplitude of the evoked field potential without affecting the area. **(B)** Strychnine (10 µM) increased the amplitude (313.8% ± 7.9%) and area (1082.2% ± 89.2%) of the evoked field responses as compared to control (*n* = 5). Subsequent coapplication of TXA (200 µM and 1 mM) further enhanced the area of the evoked field potential without affecting the amplitude. * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$ vs. bicuculline or strychnine alone. Data are mean ± SEM.



Supplemental Figure 7

Isoflurane and propofol attenuate evoked field potentials in cortical slices. **(A)** Evoked field potentials recorded in regular ACSF in the absence and presence of isoflurane (ISO, 250 µM). The charts show that isoflurane reduces the amplitude (71.1% ± 2.8% of control) and the area (76.9% ± 4.1% of control) of the evoked field response (n = 5). **(B)** Evoked field potentials recorded in the absence and presence of propofol (Prop, 1 µM). The charts show that propofol also reduces both the amplitude (92.6% ± 1.6% of control) and the area (87.6% ± 3.2% of control) of the evoked field response (n = 5) but this effect is not as pronounced as that of isoflurane. * $P < 0.05$, ** $P < 0.01$ vs. control. Data are mean ± SEM.