Supplementary information

A non-ionotropic activity of NMDA receptors contributes to glycine-induced neuroprotection in cerebral ischemia-reperfusion injury

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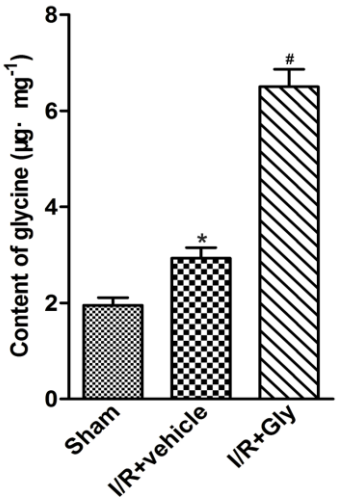

Figure S1. Intracerebroventricular injection of glycine leads to the elevation of glycine in the ischemic brain tissue. Intravenous injection of (100 μg/100 g) at 3.0 h after ischemia-reperfusion increases the levels of glycine in the ischemic brain tissue at 30 min following glycine injection (n=6, *P<0.05 vs. Sham; #P<0.05 vs. I/R+Vehicle; ANOVA test). Gly: glycine.

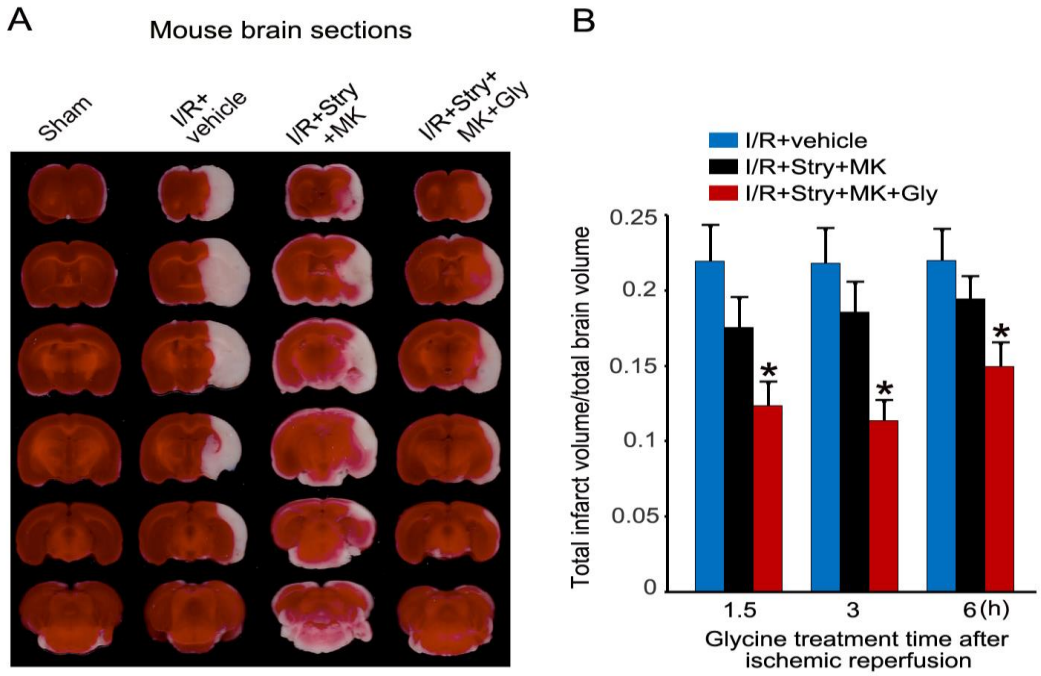

Figure S2. Glycine treatment reduces the infarct volume of ischemic mouse brain independent of glycine receptor activation and the channel activity of NMDARs. (**A**) Sample images of TTC strained-mouse brain sections collected at 24 h after ischemia onset in a mouse MCAo model. Glycine (100 μg/100 g, icv) was administered at 3 h following ischemia-reperfusion (I/R). At 30 min prior to glycine injection, MK-801 (8.0 μg/100 g, icv) and

strychnine (1.2 µg/100 g, icv) were injected. (**B**) Summarized quantification data of (A) indicate that glycine treatment at 1.5, 3, or 6 h following I/R reduces infarct volume after glycine receptors and NMDARs are inhibited (n = 6; ANOVA test, *P < 0.05 vs. I/R+Stry+MK). Stry: Strychnine; Gly: glycine; MK: MK-801