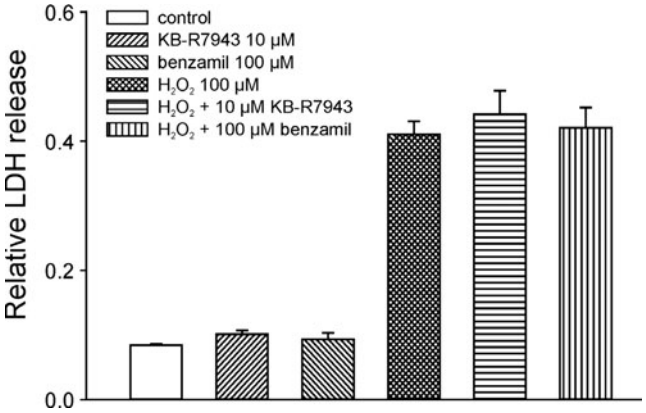
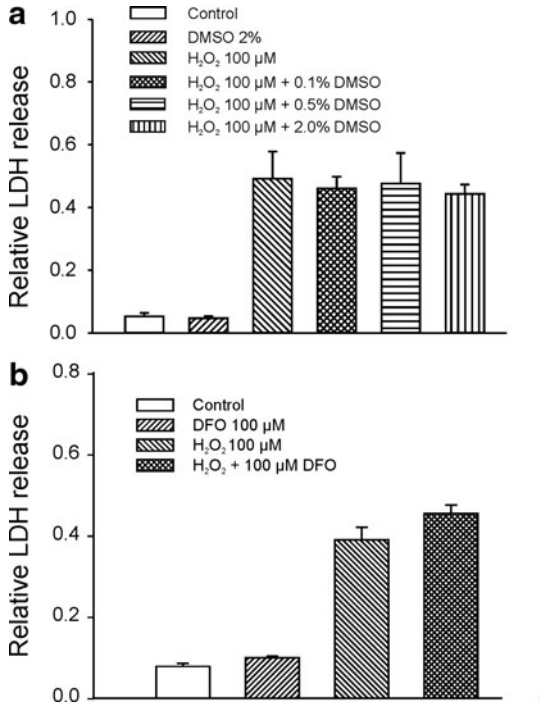
Supplementary Data



SUPPLEMENTARY FIG. S1. Evidence that activation of Na^+-Ca^{2+} exchange system is not responsible for hydrogen peroxide (H_2O_2)-induced glutamate receptor-independent neuronal injury. Summary data demonstrating the lack of protection against H_2O_2-induced glutamate receptor-independent neuronal injury by blockers of the Na^+-Ca^{2+} exchange system. Neurons were treated with 100 μM H_2O_2 in the absence or presence of 10 μM 2-(2-(4-(4-nitrobenzyloxy)phenyl)ethyl)isothiourea (KB-R7943) or 100 μM benzamil for 1 h in the presence of (5R,10S)-(+)-5-Methyl-10,11-dihydro-5H-dibenzo[a,d]cyclohepten-5,10-imine, 6-cyano-7-nitroquinoxaline-2,3-dione and nimodipine. Relative lactate dehydrogenase (LDH) release was measured 6 h after the H_2O_2 exposure. One hour treatment with 100 μM H_2O_2 in the absence of KB-R7943 or benzamil induced 0.41 ± 0.02 of relative LDH release ($n = 4$). In the presence of 10 μM KB-R7943 or 100 μM benzamil, relative LDH release by 100 μM H_2O_2 was 0.44 ± 0.04 and 0.42 ± 0.03, respectively. $n = 4$ wells in each group, $p > 0.05$ between H_2O_2 alone and H_2O_2 with KB-R7943 or benzamil.



SUPPLEMENTARY FIG. S2. Hydroxyl radicals are not responsible for H_2O_2-induced glutamate receptor-independent neuronal injury. (a) Summary bar graph showing the lack of protection on H_2O_2-induced neuronal injury by hydroxyl radical scavenger dimethyl sulfoxide (DMSO). Various concentrations of DMSO were added into culture wells 10 min before and during 1 h incubation with 100 μM H_2O_2. Relative LDH release was measured 6 h after exposure of neurons to H_2O_2 in the absence or in the presence of DMSO. In the absence of DMSO, 1 h exposure to 100 μM H_2O_2 induced a relative LDH release of 0.49 ± 0.09 ($n = 4$). In the presence of 0.1%, 0.5%, or 2% of DMSO, the relative LDH release was 0.46 ± 0.04, 0.47 ± 0.09, or 0.44 ± 0.03, respectively ($n = 4$ wells in each group, $p > 0.05$ between control and different DMSO groups). Addition of 2% DMSO in the control medium did not affect the background LDH release. **(b)** Summary data showing the lack of protection on H_2O_2-induced neuronal injury by membrane permeable iron chelator deferoxamine (DFO). Neurons were incubated with the medium containing 100 μM DFO 10 min before and during the exposure to 100 μM H_2O_2. LDH release was measured 6 h after H_2O_2 exposure. In the absence of DFO, 1 h exposure to H_2O_2 induced a relative LDH release of 0.39 ± 0.03 at 6 h. In the presence of 100 μM DFO, the relative LDH release was 0.45 ± 0.02 ($n = 8$ in each group, $p > 0.05$).