SUPPLEMENTARY MATERIAL FOR:

Novel crosstalk between ERK MAPK and p38 MAPK leads to homocysteine-NMDA receptor mediated neuronal cell death

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Pharmacological inhibitors	Physiological action	Source	Concentration used
SB203580	selective ATP-competitive inhibitor of p38 MAPK	EMD Millipore, Billerica, USA	5 μM (Davies et al. 2000; Poddar et. al. 2010)
SB202190	selective ATP-competitive inhibitor of α and β isoforms of p38 MAPK	EMD Millipore, Billerica, USA	7.5 μM (Davies et al. 2000)
SB239063	selective ATP-competitive inhibitor of α and β isoforms of p38 MAPK	EMD Millipore Billerica, USA	10 μM (Iegos et al. 2002)
PD98059	selective inhibitor of the activation of MEK1/2 (upstream kinase of ERK1/2)	EMD Millipore Billerica, USA	15 μM (Alessi et. al. 1995; Poddar and Paul. 2009)
U0126	selective non-competitive inhibitor of MEK1/2 (upstream kinase of ERK1/2)	EMD Millipore Billerica, USA	20 μM (Favata et.al. 1998; Davies et al. 2000 Stanciu et.al. 2002)
FR180204	selective ATP-competitive inhibitor of ERK1 and ERK2	EMD Millipore Billerica, USA	4 μM (Ohori et. al. 2005)
MK801	selective non-competitive NMDA receptor antagonist	Sigma Aldrich, St Louis, USA	5 μM (Yun et. al. 1998; Poddar and Paul. 2009
APV	selective competitive NMDA receptor antagonist	Sigma Aldrich, St Louis, USA	200 μM (Paul et. al. 2003; Poddar and Paul. 2009)

Table S1. Summary of all pharmacological inhibitors used in the study, their physiological action and sources.

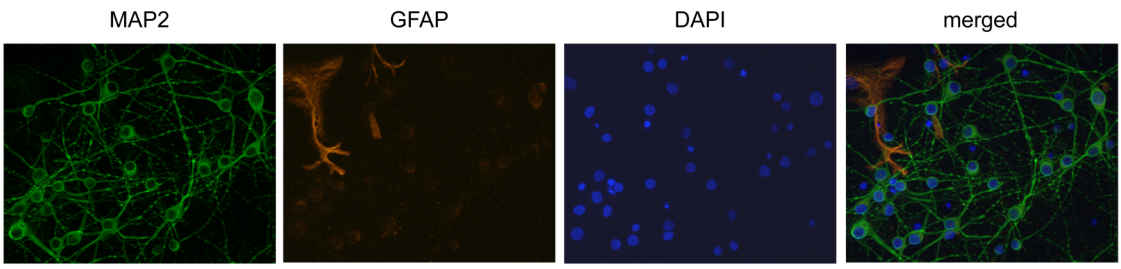

Figure S1. Purity of primary neuronal cultures. Neuronal cultures were processed for immunocytochemical staining with anti-MAP-2 (neuronal marker, green) and anti-GFAP (astrocyte marker, red) antibodies and counterstained with DAPI (blue). Quantitative analysis (1500 cells from 3 experiments) showed that 95.63 ± 0.67% ($p < 0.0001$) of the cells stained for anti-MAP-2 antibody indicating that ~ 95% of the cells are neurons.