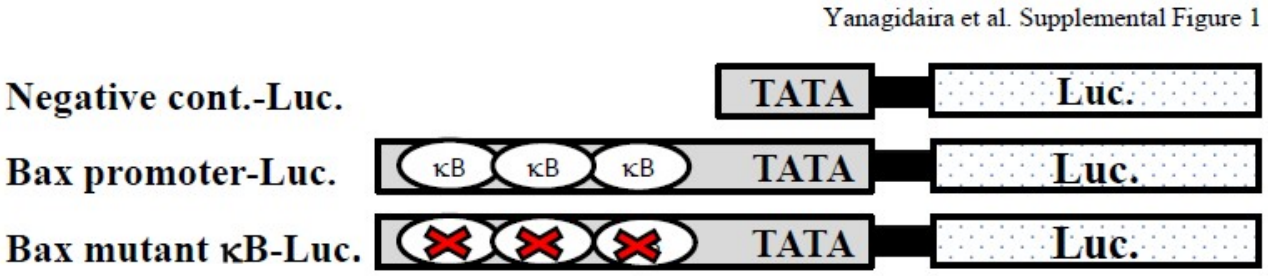
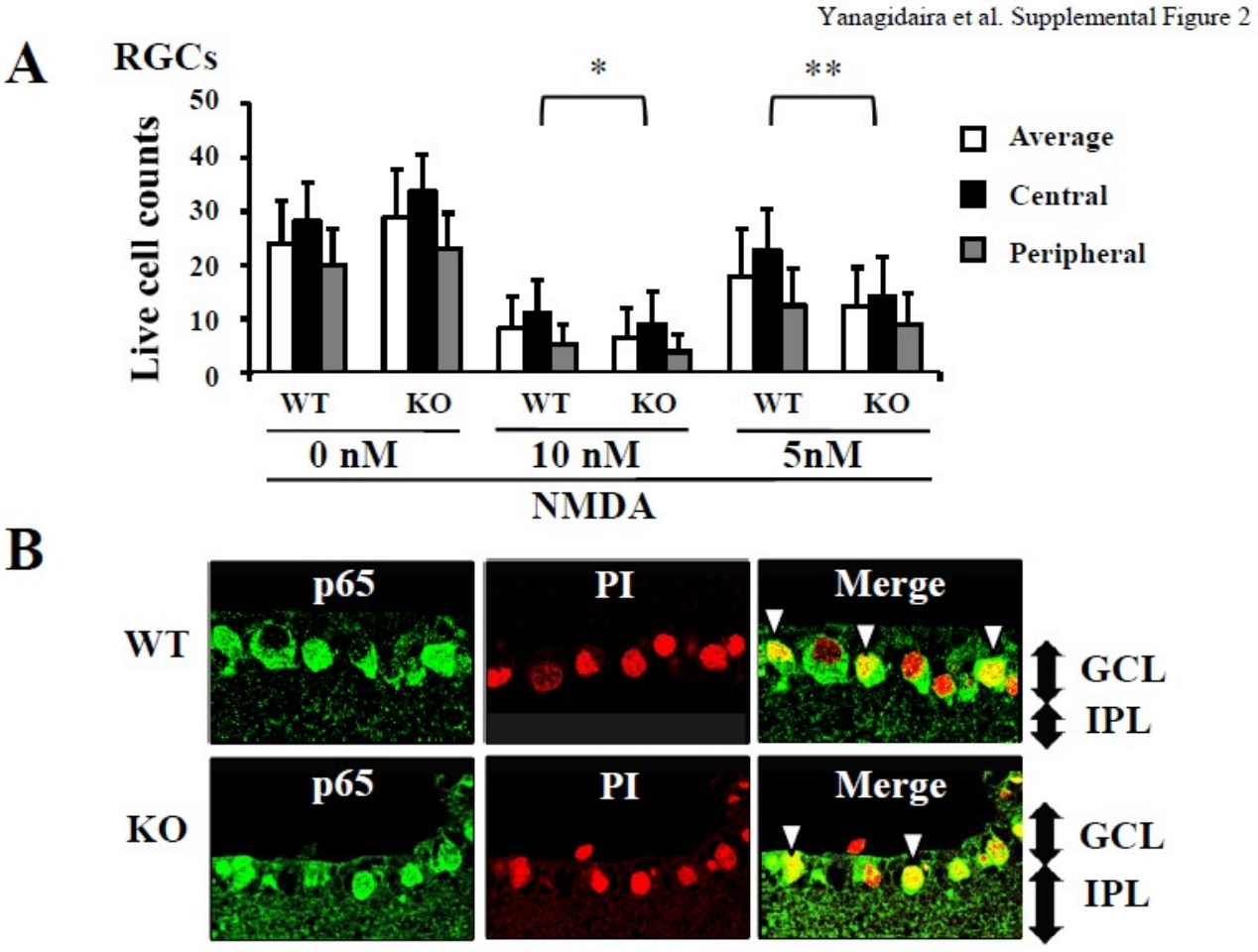
Supplemental Information



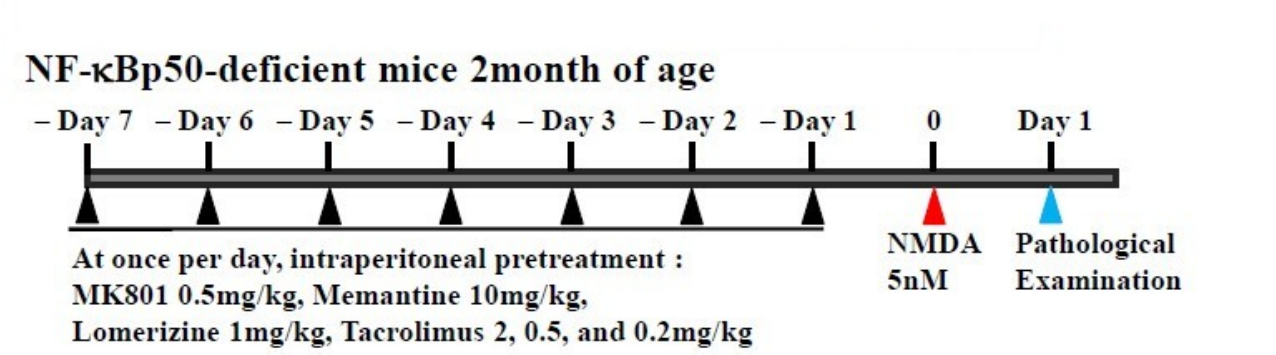
Supplemental Figure 1. Diagram of the Bax-promoter-Luc. and its respective mutant NF-κB binding sites (κB) promoter construct and negative control are illustrated.



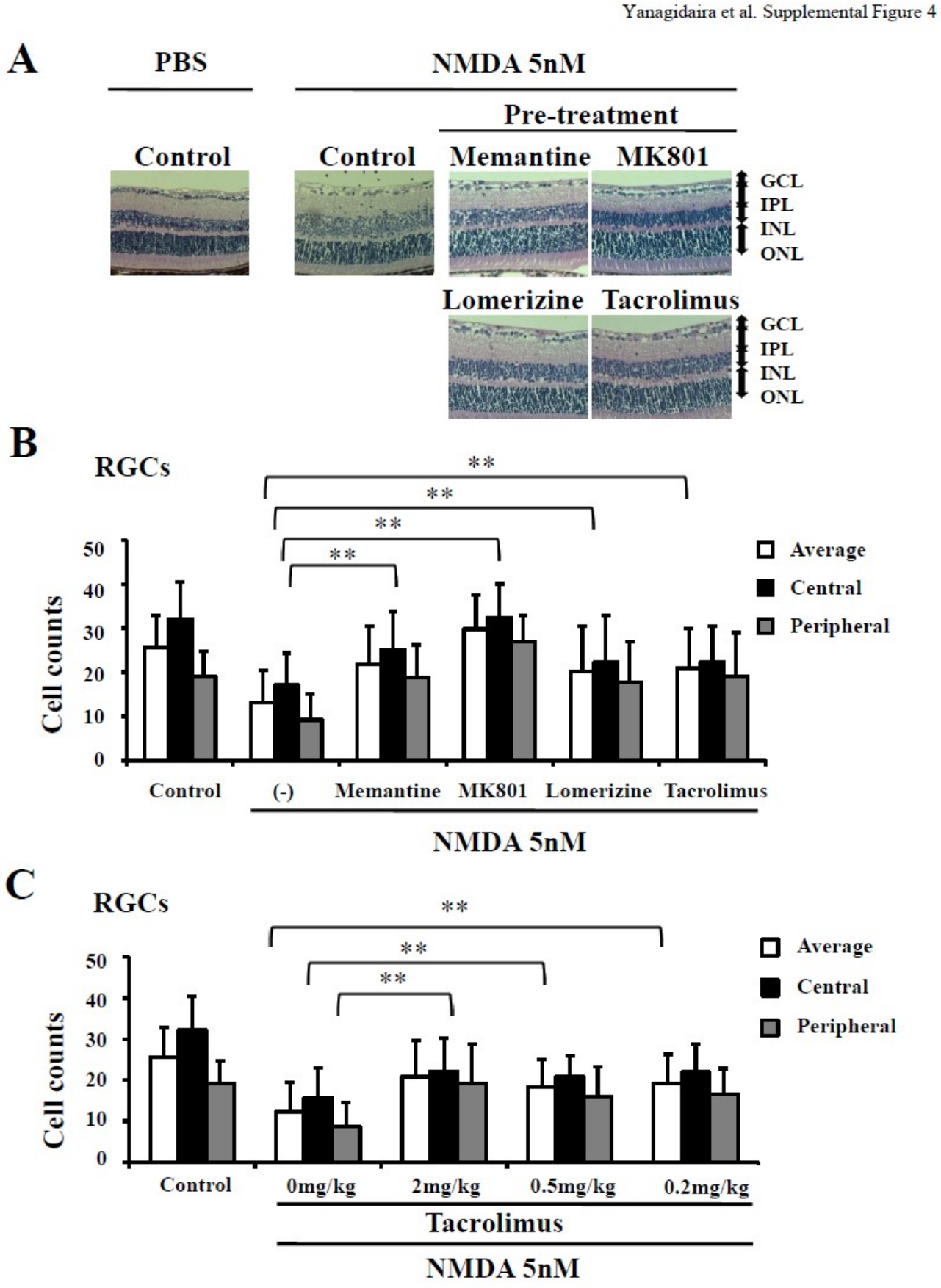
Supplemental Figure 2. The sensitivity of retina against N-methyl-D aspartate (NMDA) toxicity. **A:** The decreasing live cell number in GCL of p50-deficient mice after treatment with NMDA indicated concentration. The live cells were counted in length of 0.35 mm at 0.3 mm each from the edge of optic disc. Bar graphs show the number of cells in GCL at time before and 24 h after NMDA treatment. The average number of cells per field was analysed by the two-tailed *t*-test. Data are mean _ SEM (n = 6–7 mice). * $P < 0.01$ *vs.* wild-type mice. **$P < 0.01$ *vs.* WT mice. **B:** Immunohistochemistry for detection of p65 localizations in the retina after NMDA treatment. After

NMDA treatment, p65 translocations to the nuclei are clearly observed in cells of GCL of both WT and p50-deficient mice. Scale bar = 25 mm. KO, p50-deficient mice; WT, wild-type mice.

Yanagidaira et al. Supplemental Figure 3

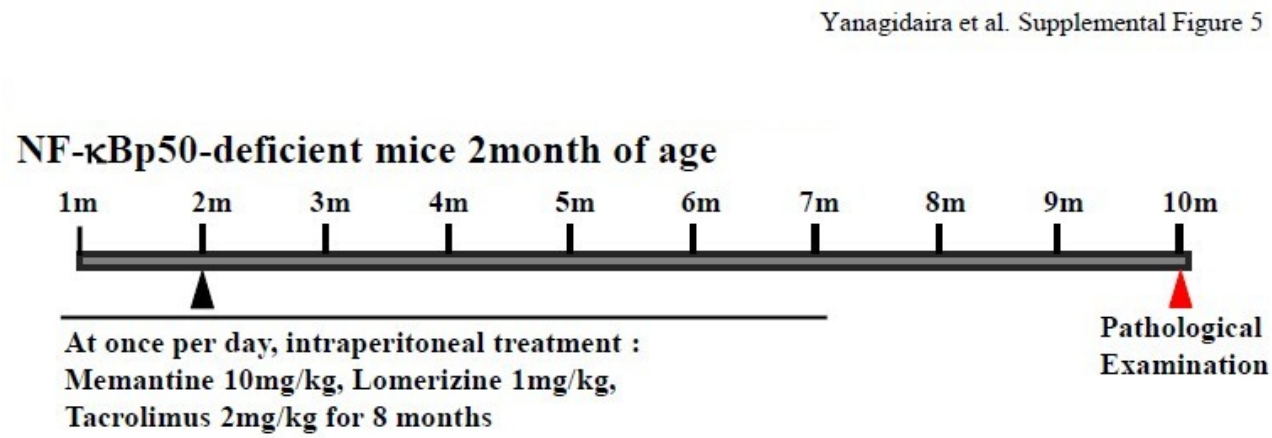


Supplemental Figure 3. Diagram of the transient experiment for studying the neuroprotective effects of chemical reagents on NMDA-induced neurotoxicity.

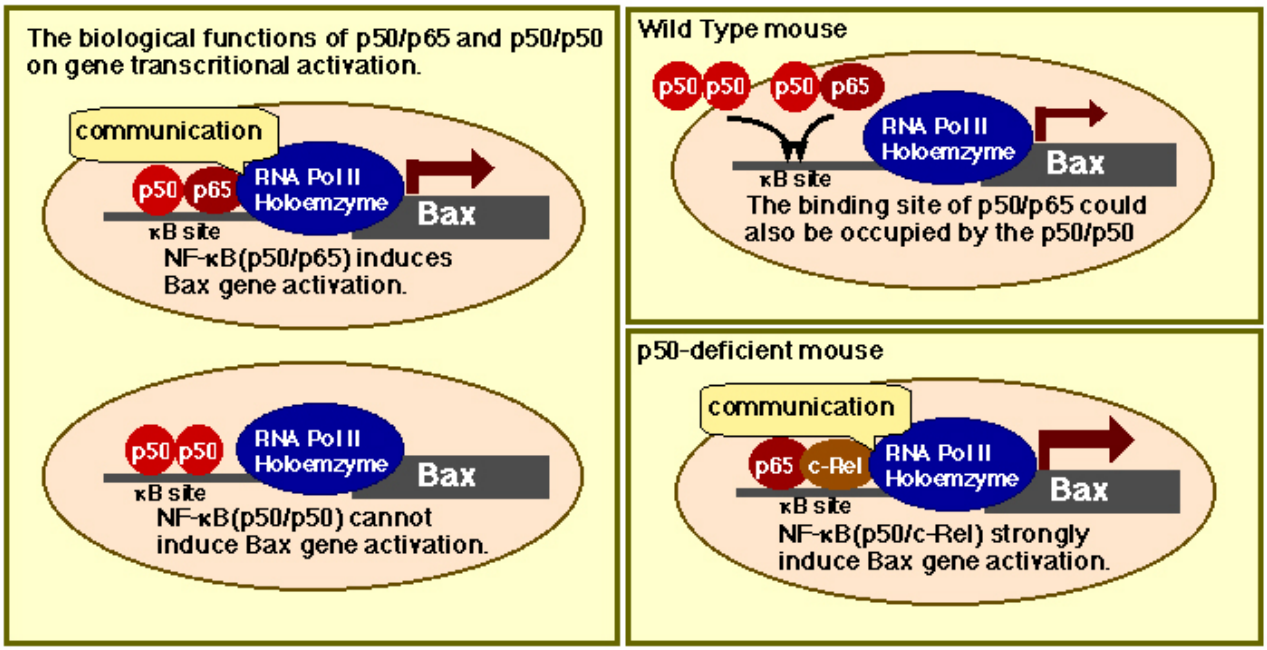


Supplemental Figure 4. Protective effects of chemical reagents against NMDA-induced neurotoxicity in p50-deficient mice. The protective effects of reagents against NMDA-induced

RGC death in the p50-deficient mice (KO). **A:** Histological examination of the retinas at 24 hours after the intravitreal injection of 5 nM NMDA. Original magnification; x10. In KO mice, 5 nM NMDA treatment for 24 hours markedly induced cell death in GCL, however, more cell survival was observed in mice pre-treated with chemical reagents. **B:** Surviving RGCs of KO mice pre-treated with a NMDA antagonist (MK801, memantine), a calcium blocker (lomerizine), a calcineurin inhibitor (tacrolimus), or vehicle. Intrapenetrial injection of memantine, MK801, lomerizine, and tacrolimus dramatically protects RGCs from the NMDA-induced neurotoxicity. Central (C); the number of cells 0.35mm in length 0.3 mm from the edge of the optic disc in the ganglion cell layer (GCL). Peripheral (P); the numbers of cells 0.35mm in length 0.3mm from the orra serata. Average; the average cell numbers of (C) plus (P). Data are the mean ±SD (n=each 7~13). Data was analyzed by the Mann-Whitney U-test (**P<0.0001). **C:** Dose-dependency of the neuroprotection by tacrolimus against NMDA-induced RGC death in KO mice. Values represent the mean ± SD. Bar graph shows surviving RGC numbers are significantly higher in the pre-treated mice (**P<0.0001). The NMDA-induced cell death was significantly prevented by the intraperitoneal injection of tacrolimus (0.2 mg/kg) one week (n=each 8~16). Data was analyzed by the Mann-Whitney U-test.



Supplemental Figure 5. Diagram of the experiment for studying the neuroprotective effects of chronic administration of chemical reagents on spontaneous optic neuropathy.



Supplemental Figure 6. Biological function of p50-p50 homodimer on NF-κB transcriptional activation as repressor.