Supplemental figure 1. Characterization of the LPS-induced Loss of Mouse Nigral Neurons.

To investigate the selectivity of the neurodegeneration, double-label immunofluorescence was performed by incubating brain sections with antibodies against a neuron-specific nuclear protein (Neu-N, 1:2000, Chemicon, Temecula, CA) and tyrosine hydroxylase (TH), followed by incubation with Alexa-488 (green) and Alexa-594 (red) conjugated secondary antibodies (1:1000). Neurons in general were labeled with anti-Neu-N antibody (red). Dopamine neurons were labeled with both anti-TH antibody in cytoplasm (green) and anti-Neu-N antibody in nuclei. In the pars compact region of the LPS-injected substantial nigra, the number of dopamine neurons was significantly fewer than that in the NS-injected side in nTg, M7KO and M83KO mice. The number of non-dopaminergic neurons (nuclear Neu-N immunoreactivity only) was not significantly decreased. In SYNKO mice, the number and the intactness of the intricate network of the dopaminergic fibers in the LPS-injected side did not show significant change, compared with the NS-injected side. NS: normal saline.

Supplemental figure 2. LPS-induced Activation of Astroglia in the Mouse Substantial Nigra

One week after stereotaxic injection of LPS and saline (NS), the brain sections were immunostained with an antibody against glial fibrillary acidic protein (GFAP, 1:1000, Dako, Carpinteria, CA) to determine the activation of astroglia. In the NS-injected substantial nigra, astroglia exhibited the resting morphology. In the LPS-injected substantial nigra, astroglia exhibited activated morphology with increased expression of GFAP. Note the activation status of astroglia did not show significant difference among different genotypes.