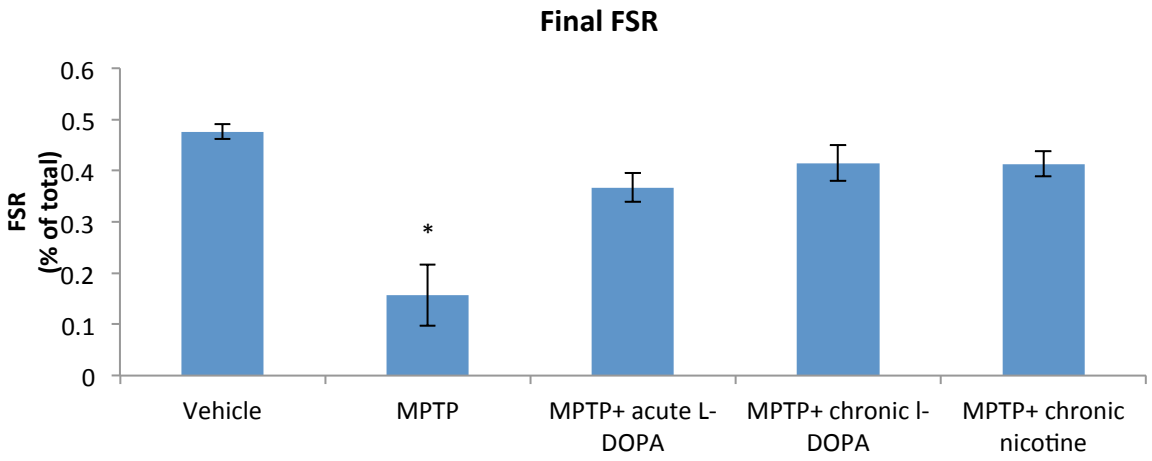
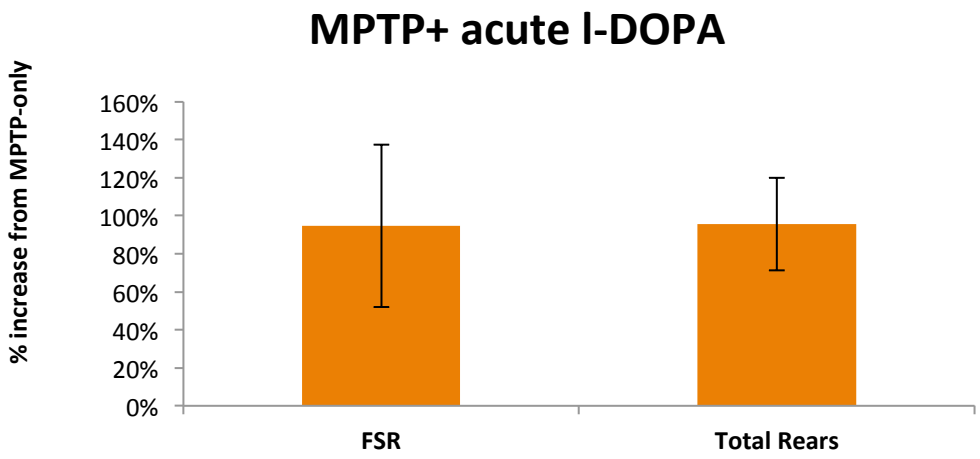
Effect of Nicotine and *l*-dopa on MPTP-induced Rearing





1.) mice injected with MPTP for 2 wks (4 mg/kg and then 8 mg/kg), then administered either *l*-dopa (15 mg/kg) or nicotine (0.4 mg/kg) first, followed by MPTP (16 mg/kg and then 32 mg/kg).

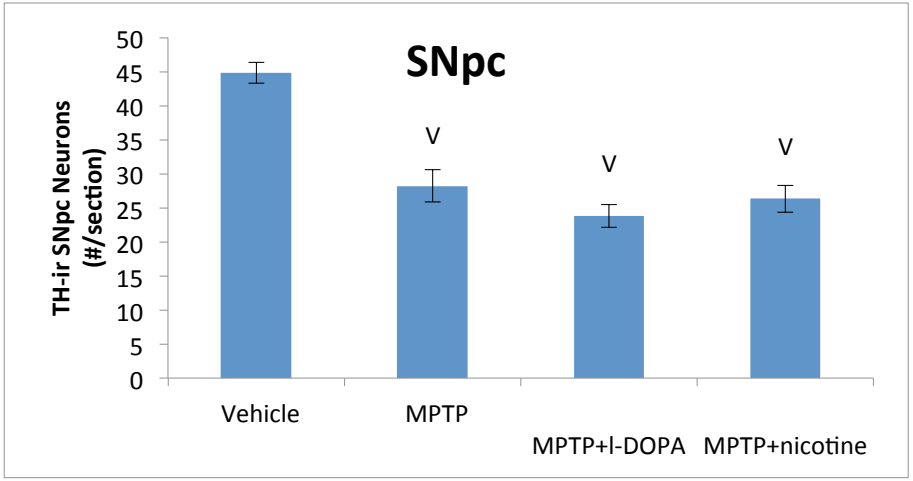
2.) MPTP only mice first behaviorally tested, then injected with acute *l*-dopa and tested again.

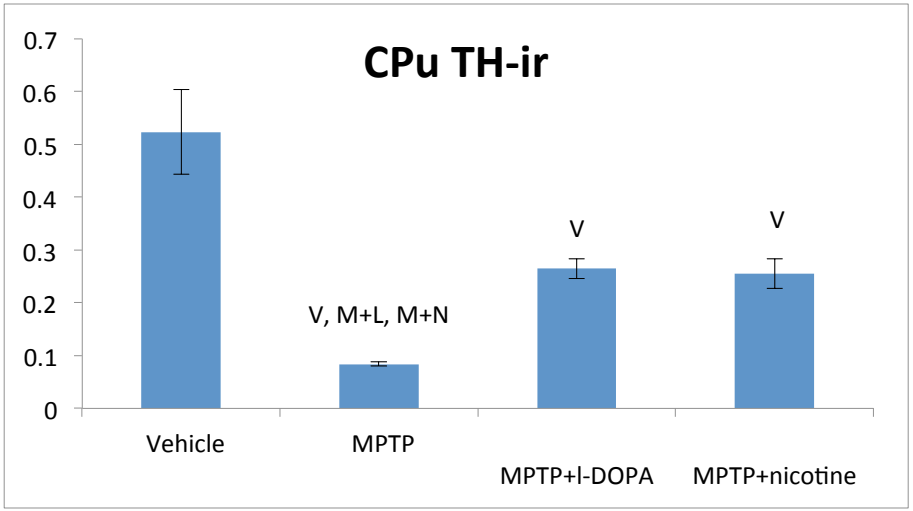
3.) Subchronic *l*-dopa or nicotine reverses the decrease in free standing rears compared to MPTP alone group.

4.) Acute *l*-dopa, given to MPTP only treated mice, results in an increase in both free standing and total rears compared to the baseline rearing behavior.

Does *l*-dopa reverse the effects of MPTP?

Effect of nicotine and *l*-dopa on tyrosine hydroxylase (**TH**) immunolabeling in the substantia nigra pars compacta (**SNpc**) and on dopamine nerve terminals in the caudate-putamen (**CPu**)





1.) subchronic administration of nicotine or *l*-dopa, starting 2 weeks after MPTP treatment, results in no change in the mean number of dopamine neurons in the substantia nigra compared to the MPTP only group.

2.) However, within the striatum (CPu), nicotine or *l*-dopa results in a 2x increase in the optical density of tyrosine hydroxylase (ie dopamine nerve terminals) compared to the MPTP only group.

3.) The data suggests that nicotine or *l*-dopa results in sprouting of new dopamine nerve terminals in the striatum, with no change in the number of dopamine cells in the substantia nigra.