Supplemental Figure 2: The CB1 antagonist rimonabant impairs within-session extinction. Animals were extinguished in a manner similar to those shown in figure 2D and the data between the two groups was normalized similarly. Animals received either vehicle (100% DMSO) or rimonabant (5 mg/kg, i.p.), an antagonist at the CB1 receptor, 30 minutes prior to extinction training/testing. Note that in these experiments, CB1 antagonist-treated animals demonstrate a deficit in within-session extinction. Additionally, CB1 antagonist-treated animals have been previously shown to demonstrate robust deficits in extinction retention\textsuperscript{20,21}. Further, it has been shown that post-extinction training administration of a CB1 antagonist does not block extinction\textsuperscript{20}, indicating that the endocannabinoid system is likely not involved in the consolidation of extinction. This differs markedly from the pattern of extinction seen in TrkBt1-infected animals (present study), where within-session extinction is normal but extinction retention is selectively impaired. This suggests that within-session extinction may be cannabinoid dependent, whereas consolidation of extinction (but not within session extinction) may be dependent upon BDNF-mediated activation of the TrkB receptor. Notably, blocking either within-session extinction (e.g., with CB1 antagonist treatment) or the consolidation of extinction (e.g., with TrkBt1 infection of the amygdala) lead to deficits in extinction retention, suggesting that within-session extinction may be a necessary-but-not sufficient condition for extinction retention.