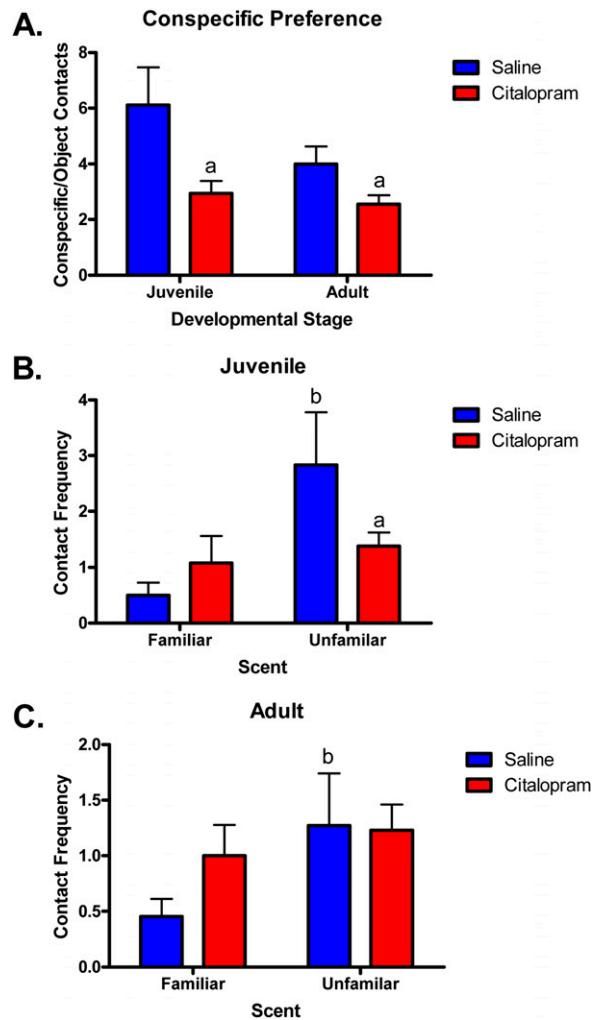
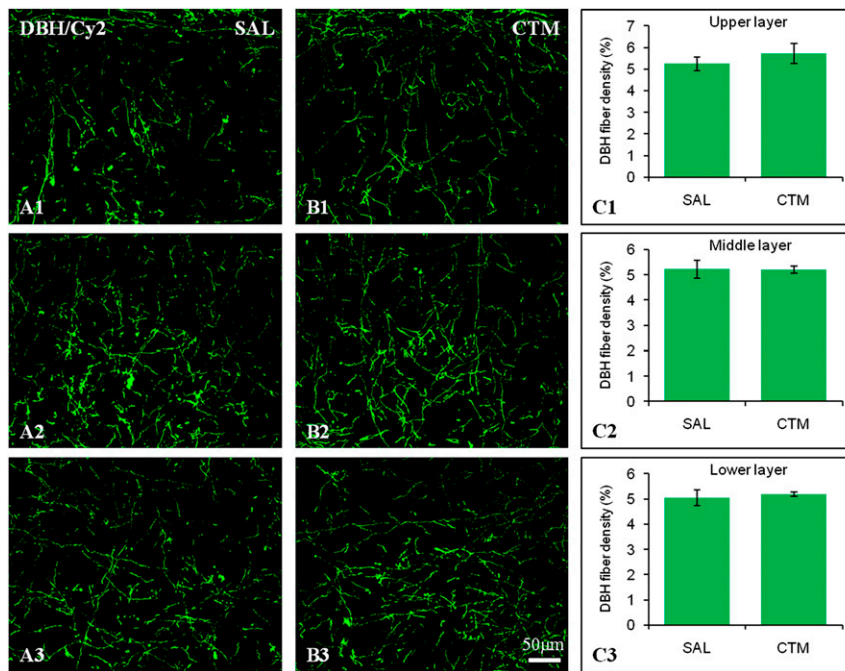


# Supporting Information

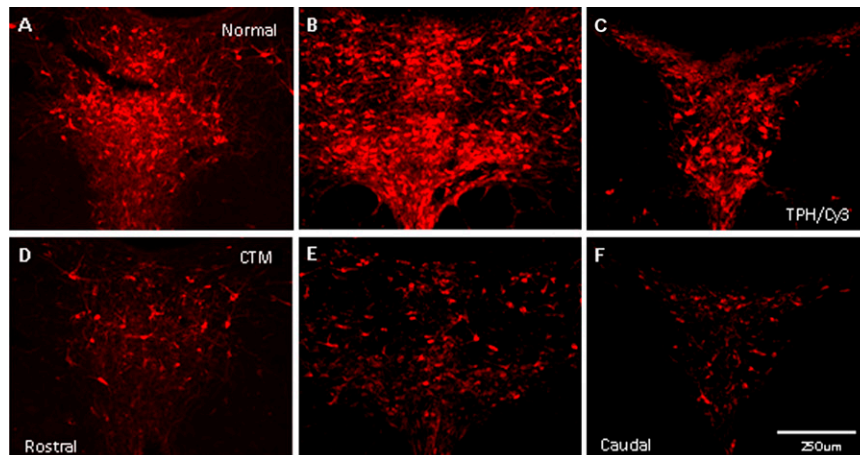
Simpson et al. 10.1073/pnas.1109353108



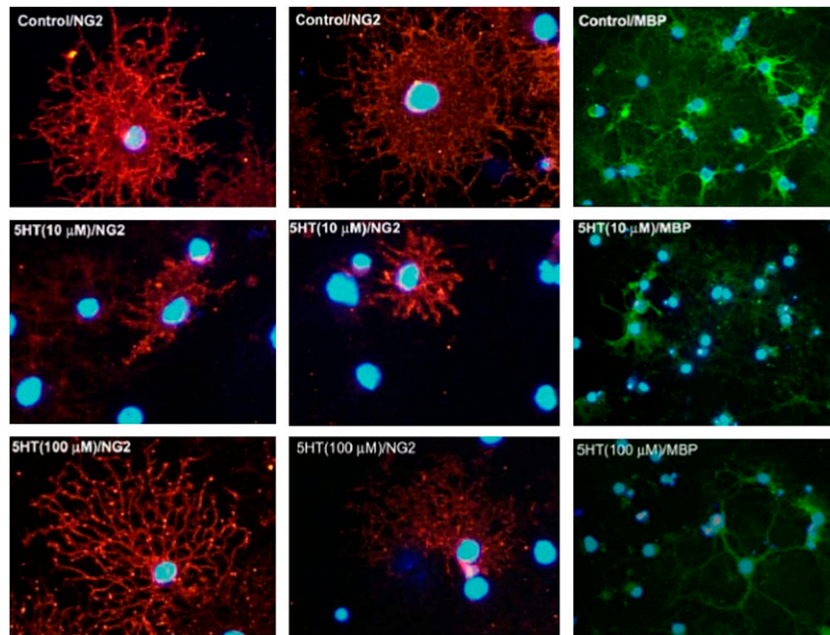
**Fig. S1.** Persistent effects of perinatal citalopram (CTM) exposure on response to novelty and social preference. Data represent the mean  $\pm$  SEM of 6–12 subjects per group for juvenile [postnatal days 30–40 (P30–P40)] and adult (P60+) response to a novel conspecific rat vs. a novel object (A) and response to a novel scent vs. a familiar scent (B and C). ANOVA revealed significant effects of neonatal drug exposure (A, B, and C) and scent familiarity (B and C). <sup>a</sup> $P < 0.05$  vs. saline-exposed rats; <sup>b</sup> $P < 0.05$  vs. familiar scent.



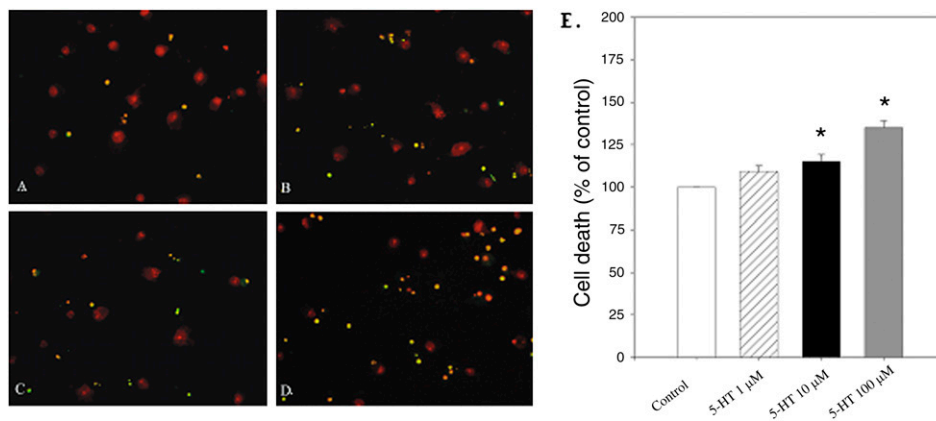
**Fig. S2.** Representative dopamine  $\beta$ -hydroxylase (DBH) immunoreactive (-ir) fiber density in the male rat primary somatosensory cortex after CTM (P8–P21) exposure and then assessed at adult. Note the rather similar DBH-ir fiber labeling density between saline (SAL) and CTM-exposed tissues. *A1* and *B1* are from layers 1 and 2/3; *A2* and *B2* are from layer 4 (middle layer); and *A3* and *B3* are from layer 5/6. (Scale bar: 50  $\mu$ m.) The semiquantitative data (*C1*, *C2*, and *C3*;  $n = 4$  from each group) revealed an almost identical DBH fiber density between saline and CTM-exposed animals.



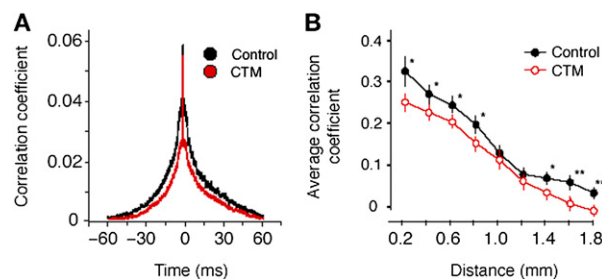
**Fig. S3.** Representative photomicrographs through the dorsal raphe nucleus show the rostral to caudal distribution of Cy3-labeled, tryptophan hydroxylase (TPH)-ir cells in untreated (*A–C*) and CTM-treated (20 mg/kg; P8–P21) (*D–F*) male rats. Note that in the drug-treated subject, TPH expression was reduced along the entire extent of the dorsal raphe nucleus neuraxis. However, this decrement was particularly evident within midline subregions. (Scale bar: 250  $\mu$ m.)



**Fig. 54.** Representative examples of morphological changes in oligodendrocyte (OL) cell cultures treated with different concentrations of serotonin (5-HT). Note the disruption (shortening, thickening, puncta, and polarization) of OL processes after treatment with 10  $\mu\text{M}$  (Middle) and 100  $\mu\text{M}$  (Bottom) 5-HT vs. controls (Top). NG2 (red; a marker for OL progenitor cells), MBP (green; a marker for mature OLs), and DAPI (blue; a marker for nuclei).



**Fig. 55.** Treatment with 5-HT induces cell death in cultures of immature OLs. Immature OLs were treated with various concentrations of 5-HT [normal/control (A), 1  $\mu\text{M}$  (B), 10  $\mu\text{M}$  (C), and 100  $\mu\text{M}$  (D)] and then processed for TUNEL staining. (E) The relative proportion of cell death was semiquantitatively calculated from a baseline measure (100%), according to the number of OLs that expressed positive TUNEL staining [TUNEL+/propidium iodide+ (total)]. TUNEL-labeled profiles (green) and nuclei (propidium iodide counterstaining; red) were recorded, and averages were obtained over six randomly selected fields (100 $\times$ ). Data were derived from three independent experiments. \* $P > 0.05$  vs. control.



**Fig. 56.** Cortical desynchronization in rats (P22–P27) after 2 wk of 10 mg/kg CTM exposure. (A) Mean normalized cross-correlation functions and SEM for primary auditory cortex neurons in controls (black) and CTM-exposed (red) rats separated by 1 mm or less. (B) Average correlation coefficient and SEM (Materials and Methods) as a function of distance for neuron pairs in controls (black) vs. CTM-exposed (red) rats.