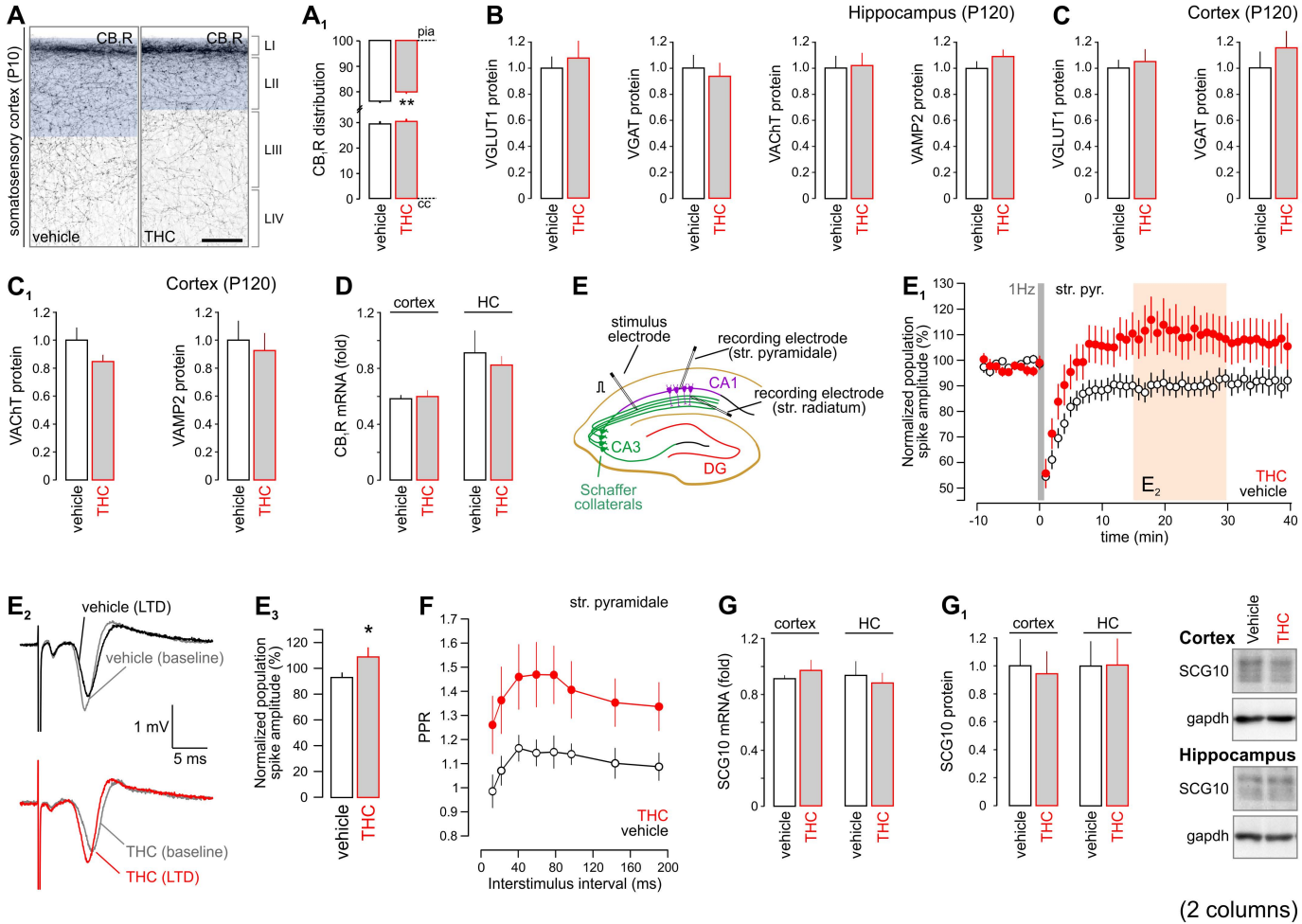
A
somatosensory cortex (P10)
CB1R
CB1R
LI
LII
LIII
LIV
vehicle
THC
A1
CB1R distribution
pia
cc
**
vehicle
THC
B
Hippocampus (P120)
VGLUT1 protein
VGAT protein
VAChT protein
VAMP2 protein
C
Cortex (P120)
C1
D
CB1R mRNA (fold)
cortex
HC
E
stimulus electrode
recording electrode (str. pyramidale)
recording electrode (str. radiatum)
CA1
CA3
DG
Schaffer collaterals
E1
1Hz
str. pyr.
Normalized population spike amplitude (%)
E2
THC
vehicle
time (min)
E2
vehicle (LTD)
vehicle (baseline)
1 mV
5 ms
THC (baseline)
THC (LTD)
E3
*
F
str. pyramidale
PPR
Interstimulus interval (ms)
G
SCG10 mRNA (fold)
G1
SCG10 protein
Cortex
Hippocampus
SCG10
gapdh
Vehicle
THC

(2 columns)

Fig. S1 **Unchanged expression levels for select synaptic proteins and CB_1Rs in adult offspring born from THC-exposed pregnancies.** (**A**) *In utero* THC exposure altered the distribution of CB_1R^+ axons, which accumulated in superficial cortical laminae receiving particularly rich CB_1R^+ innervation. (**A_1**) Quantitative analysis of the transverse depth of layers I-II (*blue* in "A") harboring a dense CB_1R^+ axonal meshwork and VI (*not shown*) on postnatal day (P)10. "Pia" and "cc" refer to the *pia mater* and corpus callosum, respectively, to denote the location of particular laminae in "A". Data were expressed as percentage of the total thickness of the cortical mantle. Serial sections spanning the primary somatosensory cortex in n = 3-5 *male* mice/group were analyzed. Western blot analysis of hippocampal (**B**) and cortical (**C,C_1**) tissues revealed that prenatal THC exposure does not impose long-lasting modifications to any of the proteins indispensable for presynaptic vesicle release (Blakely *et al*, 2012). n = 4-6 *male* mice/group were analyzed. (**D**) Similarly, CB_1R mRNA expression was unchanged in offspring prenatally exposed to THC ($n \geq 4$ *male* mice/group). (**E**) Positioning of the stimulus and recording electrodes in hippocampal slices used for acute *in vitro* recording of field excitatory postsynaptic potentials (fEPSPs, str. radiatum) and population spikes (str. pyramidale) in the CA1 subfield upon orthodromic stimulation of Schaffer collateral inputs (Takahashi *et al*, 2006). (**E_1**) Low-frequency stimulation of Schaffer collateral inputs in vehicle-treated mice suppressed neuronal population activity in the str. pyramidale. In THC-exposed offspring, however, this suppression was only transient and rapidly recovered with a trend towards moderate potentiation instead. Orange rectangle denotes the subset of data points that were statistically evaluated in E_3. (**E_2**) Example traces of baseline and of follow-up after LTD induction in offspring exposed *in utero* to vehicle or THC. (**E_3**) Quantitative assessment of population activity after LTD induction in vehicle (n = 6) *vs.* THC-exposed (n = 6) offspring. (**F**) Summary of the paired-pulse ratio (PPR) at different interstimulus intervals (ms). The PPR significantly increased in the CA1 str. pyramidale of THC-exposed (n = 5) offspring *vs.* vehicle controls (n = 5). PPR value of THC-exposed mice at each interstimulus interval was statistically compared to the corresponding value in vehicle-treated controls, and was found significantly different between the experimental groups ($p < 0.05$). (**G,G_1**) SCG10 mRNA (**G**) and protein (**G_1**) expression were unchanged in adult mice whose mothers were exposed to THC (Mato *et al*, 2004). $n \geq 4$ *male* mice/group from independent pregnancies were used in each experiment. Gapdh was used to normalize both mRNA and protein levels. Data were expressed as means ± s.e.m. *Scale bar* = 200 µm (A).

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