Supplemental Information

I. Supplemental figures

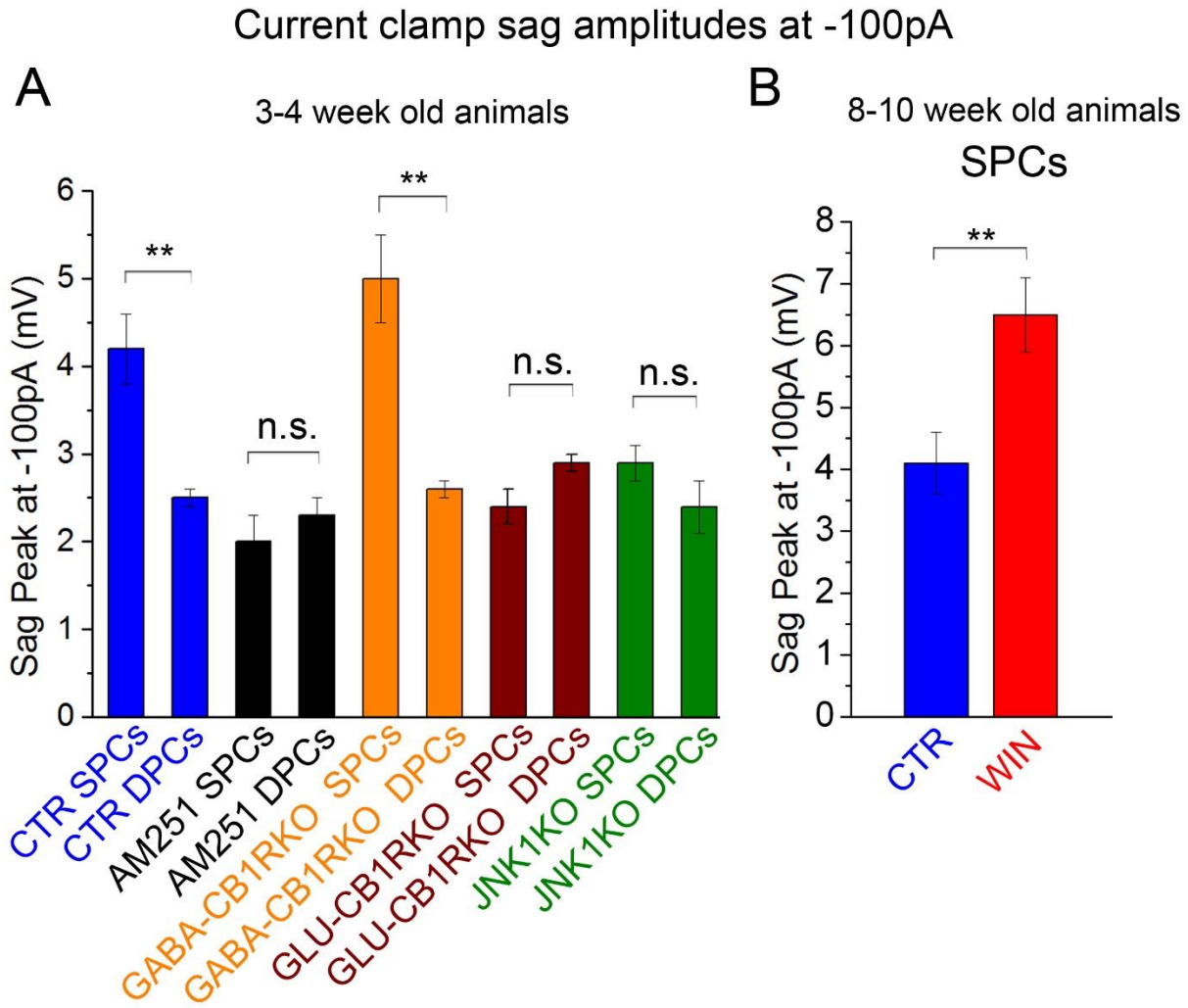

Figure S1 associated to Fig 1

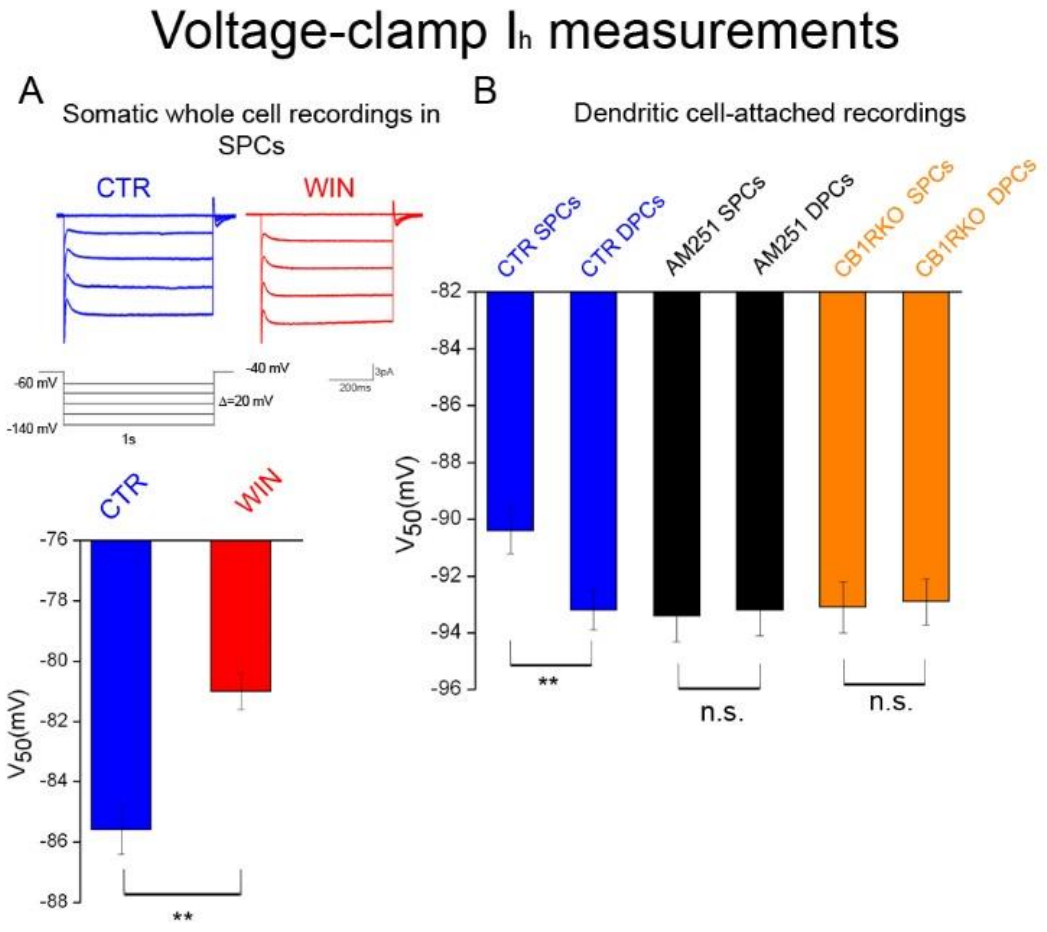

Figure S2 associated to Fig 2

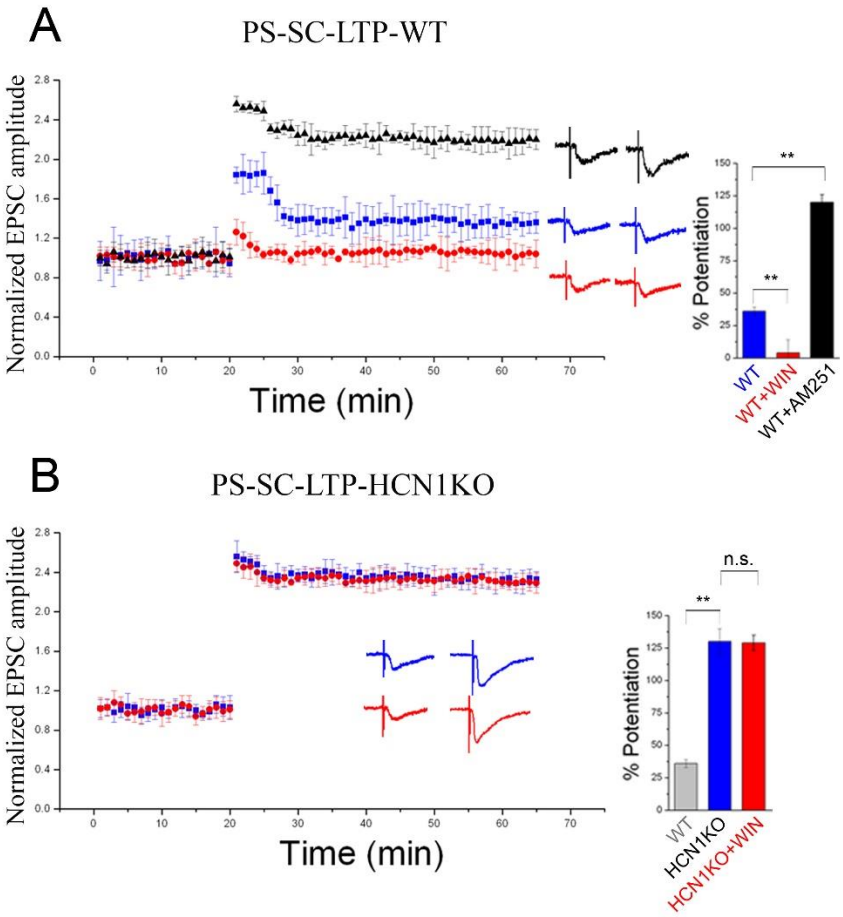

Figure S3 associated to Fig 6

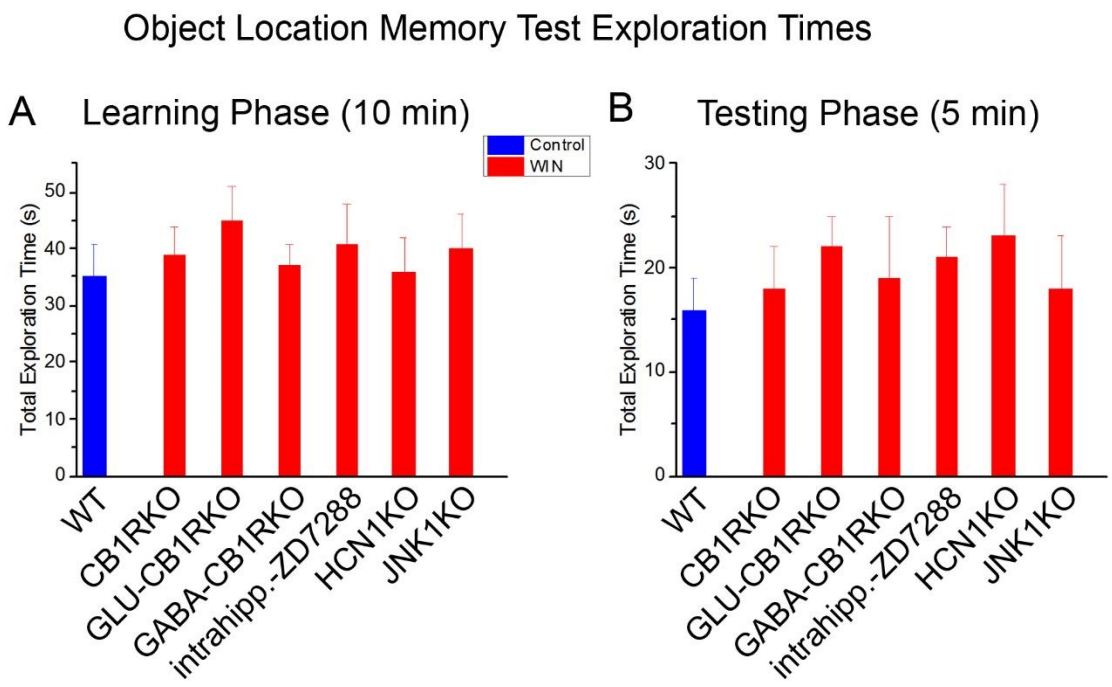

Figure S4 associated to Fig 7

II. Legend to Supplemental Figures

Figure S1: CB1R modulation affects the sag amplitude similarly in young and adult mice in SPCs and its inhibition abolishes the differences in sag amplitudes between SPCs and DPCs

A) Peak sag amplitude recorded at -100 pA in 3-4 week old animals in control condition (CTR) or after AM251 application in WT, GABA-CB1RKO, GLU-CB1RKO, and JNK1KO mice in SPCs and DPCs. The stimulation protocol was the same as in Fig 1A. The effect on sag amplitude was similar for all current injection between -400 and -100 pA (see Table S1 and S2 for values of all traces); n= 11-15 per experimental group: CTR SPCs: 11; CTR DPCs: 13; AM251 SPCs: 13; AM251 DPCs: 12; GABA-CB1RKO SPCs: 12; GABA-CB1RKO DPCs: 11; GLU-CB1RKO SPCs: 15; GLU-CB1RKO DPCs: 12; JNK1KO SPCs: 13; JNK1KO DPCs: 11.

B) Peak sag amplitude recorded at -100 pA in 8-10 week old WT animals in control condition (CTR) or after WIN application in SPCs. The stimulation protocol was the same as in Fig 1A. The effect on sag amplitude was similar for all current injection between -400 and -100 pA (see Table S1 for values of all traces); n= 11-12 per experimental group: CTR 12; WIN 11

Data are represented as mean ± standard error (SE); **p<0.01; n.s.: not significant by unpaired Student's t-test.

Figure S2: CB1R modulates I_h activation recorded at the soma in SPCs and its inhibition abolishes the differences in I_h activation between SPCs and DPCs

A) Top: Example of step hyperpolarizations (1 s duration) evoking slowly activating and non-inactivating inward currents from whole cell somatic patches of SPCs. Middle: Hyperpolarizing step protocol in voltage clamp configuration. Bottom: Summary data of V_{50} values measured in SPCs in control condition (CTR, blue) and after WIN application (red).

B) Summary data of V_{50} values measured in control condition (CTR) or after AM251 application in WT and in CB1RKO mice in SPCs and DPCs; n=11-14 per experimental group: CTR SPCs 12; CTR DPCs 13; AM251 SPCs: 11; AM251 DPCs 13; CB1RKO SPCs 13; CB1RKO DPCs 14.

Data are represented as mean ± standard error (SE); **p<0.01; n.s.: not significant by unpaired Student's t-test.

Figure S3: Effects of CB1R-mediated I_h modulation on pairing protocol-induced LTP of Schaffer collateral in SPCs.

A,B) Left: Summary plots and example traces of the normalized EPSC amplitudes from whole cell recordings in SPCs before and after pairing protocol stimulation (PS) at the Schaffer collateral (SC) inputs (PS-SC-LTP); the examples traces were acquired 10 min before and 60 min after PS. Right: Summary bar graphs of the percent of potentiation of the EPSC amplitude 60 minutes after TBS compared to the EPSC amplitude 10 minutes before TBS in the various experimental conditions. The percent of potentiation is calculated as follows: $((Amplitude_{after} - Amplitude_{before}) / (Amplitude_{before})) \times 100$. **A)** Responses from WT mice recorded either in control perfusate (CTR, blue), or in the presence of WIN (red) or in the presence of AM251 (black); n=11-13 per experimental group: CTR 13; WIN 11; AM251 13. **B)** Responses from HCN1KO mice recorded in control perfusate (blue) or in WIN (red); n=13 per experimental group (note that, for clarity and easy comparison, in the bar graph we reported in grey the % of potentiation in WT mice in control perfusate shown in blue in panel A)

Data in the bar graphs are represented as mean ± standard error (SE); **p<0.01; n.s.: not significant by one-way ANOVA followed by Tukey's post-hoc test.

Figure S4: Total exploration time for the different strains during OLM task

A, B) Bar graphs showing the total exploration time during **(A)** learning phase (10 min) or **(B)** testing phase (5 min) in vehicle-treated control animals (blue) and in WIN-treated WT, CB1RKO, GLU-CB1RKO, GABA-CB1RKO, WT injected intrahippocampally with ZD7288, HCN1KO and JNK1KO mice. The n values are the same as indicated in Fig 7.

Note that all groups showed statistically similar exploration times in both the learning and testing phases. Statistical analysis: one-way ANOVA followed by Tukey's posthoc test.

III. Supplemental tables

Table S1 associated to Fig 1

Strain	Treatment	-400pA	-350pA	-300pA	-250pA	-200pA	-150pA	-100pA	-50pA
3-4 weeks WT	---	**9.3±0.8**	**8.5±0.6**	**8.0±0.8**	**7.2±0.5**	**6.4±0.5**	**5.8±0.5**	**4.2±0.4**	**2.7±0.6**
	WIN	12.2±0.6	10.9±0.2	10.0±0.6	9.8±0.3	9.4±0.3	8.0±0.3	7.3±0.4	4.9±0.3
	WIN + AM251	8.9±0.5	8.8±0.1	8.2±0.2	7.5±0.4	6.3±0.5	6.0±0.4	4.5±0.4	3.1±0.4
	AM251	6.9±0.3	6.5±0.3	6.2±0.3	5.0±0.3	4.0±0.1	3.0±0.2	2.6±0.3	2.3±0.6
	ZD7288	0.4±0.4	0.3±0.2	0.1±0.1	0.4±0.5	0.3±0.1	0.2±0.1	0.1±0.1	0.4±0.1
	WIN + ZD7288	0.6±0.8	0.2±0.4	0.3±0.5	0.1±0.2	0.4±0.2	0.5±0.2	0.2±0.1	0.3±0.2
	AM251 + ZD7288	0.2±0.3	0.2±0.2	0.3±0.3	0.1±0.4	0.5±0.1	0.1±0.1	0.2±0.1	0.1±0.1
	JZL	11.9±0.4	11.1±0.3	10.8±0.5	10.0±0.3	9.6±0.5	8.2±0.8	7.0±0.4	3.2±0.5
	URB	8.6±0.6	8.2±0.4	7.8±0.3	7.5±0.4	6.9±0.2	6.0±0.4	4.8±0.3	2.9±0.2
	P38-inh	9.8±0.2	9.2±0.2	8.5±0.2	8.0±0.2	6.8±0.3	6.1±0.3	4.5±0.4	3.1±0.4
	JNK-inh-intra	7.2±0.3	6.3±0.2	5.9±0.3	5.3±0.3	3.7±0.2	3.0±0.2	2.9±0.2	2.7±0.5
	WIN + P38-inh	11.6±0.2	10.8±0.3	9.8±0.5	9.5±0.1	8.9±0.6	8.2±0.6	6.9±0.3	3.2±0.4
	WIN + JNK-inh-intra	7.5±0.7	6.9±0.4	6.2±0.4	5.6±0.2	4.2±0.2	3.3±0.2	2.9±0.2	2.3±0.3
	aniso-intra	12.3±0.8	11.9±0.3	11.0±0.3	10.2±0.5	9.6±0.7	8.6±0.2	7.5±0.4	3.0±0.2
	WIN + aniso-intra	11.9±1.0	11.5±0.3	10.8±0.4	10.6±0.3	9.8±0.4	9.0±0.4	8.0±0.4	3.5±0.4
	AM251 + aniso-intra	12.1±0.5	11.1±0.2	10.5±0.6	10.1±0.1	9.5±0.5	8.4±0.2	7.8±0.3	3.1±0.3
	JNK-inh-extra + aniso-intra	7.1±0.6	6.7±0.1	5.9±0.4	5.2±0.2	4.5±0.3	3.9±0.3	3.2±0.2	2.5±0.1
	P38inh + aniso-intra	11.8±0.4	11.6±0.3	10.8±0.5	9.8±0.3	8.9±0.6	8.3±0.4	7.8±0.1	3.2±0.2
	ODQ	7.1±0.3	6.4±0.4	6.1±0.7	5.5±0.6	5.0±0.3	3.8±0.2	2.9±0.3	2.1±0.3
	WIN + ODQ	6.8±0.4	6.5±0.2	6.2±0.3	5.7±0.4	4.5±0.5	3.5±0.2	2.8±0.2	2.0±0.4
	AM251 + ODQ	6.9±0.5	6.3±0.5	5.9±0.4	5.5±0.5	4.6±0.6	3.1±0.4	2.4±0.6	1.9±0.4
	aniso-intra + ODQ	7.0±0.4	6.7±0.6	6.4±0.5	5.2±0.4	4.2±0.4	3.5±0.3	2.6±0.4	2.1±0.3
	Gallein	7.2±0.3	6.5±0.2	5.9±0.4	5.0±0.4	4.1±0.4	3.4±0.1	2.5±0.2	1.9±0.2
	Gallein + WIN	7.0±0.3	6.1±0.3	5.6±0.2	4.9±0.3	3.9±0.5	2.8±0.3	2.0±0.3	1.6±0.2
	CPTIO	6.5±0.4	6.2±0.5	5.5±0.3	4.9±0.2	4.0±0.3	2.4±0.3	2.2±0.3	2.0±0.2

	CPTIO + WIN	6.3±0.6	6.0±0.5	5.4±0.4	5.0±0.4	3.8±0.4	2.6±0.2	2.2±0.1	1.9±0.2
	L-NAME	6.8±0.5	5.8±0.5	5.0±0.3	4.7±0.4	4.0±0.3	3.0±0.2	2.4±0.3	2.2±0.1
	L-NAME + WIN	6.7±0.4	6.1±0.6	5.7±0.3	5.1±0.3	3.7±0.3	2.8±0.2	2.0±0.4	1.8±0.2
	DDOA	7.2±0.6	6.5±0.4	6.0±0.4	5.6±0.2	5.0±0.2	3.9±0.1	3.0±0.2	2.7±0.2
	DDOA + WIN	9.5±0.6	8.6±0.5	7.8±0.3	7.1±0.3	6.4±0.2	5.7±0.2	4.8±0.3	4.0±0.1
8-10 weeks WT	---	8.9±0.9	8.0±0.5	7.6±0.7	7.3±0.5	6.8±0.3	6.0±0.4	4.5±0.5	3.1±0.4
	WIN	11.8±0.8	11.1±0.4	10.4±0.5	9.5±0.3	9.0±0.5	8.4±0.4	7.4±0.3	4.1±0.2
CB1RKO	---	6.5±0.7	6.1±0.5	5.5±0.1	4.9±0.3	4.2±0.2	3.5±0.4	3.1±0.4	2.0±0.2
	WIN	6.1±0.3	5.8±0.1	5.1±0.5	4.5±0.3	3.7±0.1	3.0±0.3	2.5±0.3	2.3±0.3
GLU-CB1RKO	---	6.6±0.2	5.4±0.6	5.3±0.6	4.2±0.5	3.2±0.3	2.3±0.2	2.0±0.2	2.2±0.4
	WIN	6.3±0.4	5.6±0.4	4.6±0.4	4.0±0.2	3.8±0.3	3.8±0.1	2.6±0.2	2.0±0.3
GABA-CB1RKO	---	8.6±0.6	8.4±0.3	7.8±0.5	7.0±0.4	6.6±0.4	5.9±0.3	5.0±0.5	3.1±0.4
	WIN	10.9±0.4	10.6±0.5	10.0±0.3	9.6±0.1	8.7±0.5	8.6±0.4	8.1±0.1	3.2±0.3
Selective $CB1R^+$ SPCs	---	9.1±0.5	8.3±0.5	7.9±0.4	7.1±0.6	6.4±0.4	5.7±0.5	4.8±0.3	3.0±0.2
	WIN	11.4±0.6	10.7±0.7	9.9±0.3	9.7±0.4	8.9±0.7	8.5±0.6	7.9±0.5	3.4±0.4
Selective $CB1R^-$ SPCs	---	6.5±0.4	5.7±0.5	5.2±0.6	4.4±0.4	3.5±0.5	2.6±0.4	2.3±0.3	1.9±0.2
	WIN	6.7±0.5	5.9±0.6	5.3±0.6	4.6±0.7	4.0±0.4	3.1±0.5	2.5±0.2	2.1±0.3
HCN1KO	---	0.2±0.3	0.1±0.4	0.1±0.1	0.3±0.3	0.1±0.6	0.2±0.1	0.1±0.1	0.1±0.1
	WIN	0.2±0.5	0.2±0.5	0.1±0.5	0.1±0.5	0.2±0.3	0.1±0.2	0.1±0.1	0.1±0.1
JNK1KO	---	7.1±0.2	6.5±0.3	5.8±0.3	5.5±0.2	4.9±0.2	3.6±0.3	2.9±0.1	2.5±0.4
	WIN	6.8±0.3	6.2±0.2	6.1±0.4	5.8±0.2	4.0±0.4	3.2±0.3	2.6±0.2	2.2±0.3
JNK2KO	---	8.4±0.4	7.8±0.3	7.0±0.5	6.3±0.4	5.5±0.3	4.2±0.4	3.2±0.2	2.3±0.4
	WIN	9.6±0.2	9.0±0.4	8.5±0.3	7.8±0.1	6.9±0.5	6.0±0.2	4.9±0.3	3.0±0.4

Table S2 associated to Fig 1

Strain	Treatment	-400pA	-350pA	-300pA	-250pA	-200pA	-150pA	-100pA	-50pA
WT	---	**6.9±0.5**	**6.0±0.6**	**5.7±0.4**	**4.8±0.4**	**4.0±0.3**	**3.5±0.1**	**2.5±0.1**	**1.3±0.2**
	WIN	7.1±0.6	5.9±0.4	6.0±0.2	5.1±0.4	3.8±0.1	3.3±0.3	2.4±0.2	1.2±0.1
	AM251	6.9±0.5	6.1±0.8	5.8±0.1	5.0±0.5	4.1±0.2	3.2±0.2	2.4±0.3	1.4±0.2
	ZD7288	0.3±0.1	0.2±0.6	0.3±0.1	0.1±0.6	0.2±0.1	0.1±0.4	0.3±0.2	0.1±0.1
	WIN + ZD7288	0.2±0.1	0.1±0.5	0.1±0.1	0.3±0.3	0.2±0.1	0.1±0.1	0.2±0.3	0.1±0.2
	gallein	6.9±0.4	5.9±0.6	5.5±0.4	4.9±0.5	4.2±0.3	3.8±0.6	2.9±0.4	1.8±0.4
	Aniso-intra	9.2±0.5	8.3±0.3	7.9±0.4	6.4±0.2	5.9±0.1	4.8±0.3	4.0±0.3	2.9±0.3
	Jnk-inh-extra	6.7±0.4	6.2±0.5	5.9±0.4	4.9±0.3	4.3±0.2	3.8±0.1	2.9±0.1	1.5±0.2
	Jnk-inh-intra	7.0±0.5	6.3±0.6	5.7±0.5	5.0±0.3	4.2±0.4	3.4±0.2	2.4±0.3	1.1±0.3
CB1KO	---	7.1±0.6	6.0±0.4	5.6±0.3	4.7±0.2	3.9±0.2	3.1±0.3	2.3±0.2	1.2±0.1
	WIN	6.9±0.5	5.8±0.6	5.8±0.4	5.0±0.4	4.0±0.3	3.3±0.2	2.4±0.1	1.3±0.3
GLU-CB1RKO	---	7.1±0.5	5.9±0.4	6.0±0.3	4.8±0.3	3.8±0.2	3.2±0.1	2.3±0.3	1.2±0.2
	WIN	7.0±0.6	6.1±0.5	5.9±0.4	4.9±0.1	3.7±0.4	3.1±0.4	2.4±0.2	1.1±0.2
GABA-CB1RKO	---	6.9±0.4	5.9±0.5	5.7±0.2	5.0±0.2	4.1±0.2	3.0±0.2	2.6±0.1	0.9±0.1
	WIN	6.8±0.8	5.8±0.4	5.8±0.3	4.7±0.4	4.0±0.3	3.2±0.2	2.5±0.1	1.4±0.3
Selective $CB1R^+$ DPCs	---	7.0±0.6	6.3±0.5	5.6±0.4	4.9±0.3	3.9±0.5	3.4±0.2	2.9±0.4	1.5±0.6
	WIN	6.8±0.7	6.3±0.3	5.8±0.5	4.8±0.3	4.0±0.4	3.2±0.3	2.7±0.4	2.0±0.2
Selective $CB1R^-$ DPCs	---	7.1±0.6	6.2±0.5	5.4±0.6	4.9±0.5	4.0±0.5	3.2±0.2	2.5±0.3	1.3±0.4
	WIN	6.8±0.5	6.2±0.5	5.5±0.6	4.7±0.6	3.9±0.4	3.3±0.5	2.4±0.3	1.8±0.5
HCN1KO	---	0.2±0.1	0.2±0.1	0.2±0.2	0.1±0.1	0.3±0.2	0.2±0.1	0.2±0.2	0.2±0.1
	WIN	0.1±0.1	0.1±0.1	0.1±0.1	0.2±0.2	0.1±0.2	0.2±0.2	0.1±0.1	0.2±0.2
JNK1KO	---	6.9±0.5	6.2±0.4	6.0±0.2	4.8±0.2	3.8±0.3	3.3±0.2	2.4±0.2	1.2±0.3
	WIN	7.0±0.6	5.9±0.5	6.1±0.3	5.1±0.3	4.0±0.1	3.2±0.1	2.3±0.1	1.2±0.1
JNK2KO	---	6.8±0.8	6.1±0.6	5.8±0.2	4.9±0.2	4.1±0.2	3.4±0.2	2.4±0.4	1.3±0.2
	WIN	6.9±0.8	5.8±0.4	5.9±0.1	5.0±0.3	3.9±0.1	3.1±0.1	2.5±0.3	0.9±0.1

Table S3 associated to Fig 2

	Treatment	V_{50} SPCs (mV)	V_{50} DPCs (mV)
Dendritic WT	---	-90.4 ± 0.8	-93.2 ± 0.7**
	WIN	-86.8 ± 0.6	-92.6 ± 0.9**
	WIN + AM251	-91.2 ± 0.7	-91.4 ±0.8
	AM251	-93.4 ± 0.9	-93.2 ± 0.9
Dendritic CB1RKO	---	-93.1 ± 0.9	-92.9 ± 0.8
	WIN	-92.9 ± 0.9	-93.0 ± 0.6
Somatic WT	---	-84.6 ± 0.5	-88.2 ± 0.8
	WIN	-81.1 ± 1.1	-89.1 ± 0.7

Table S4 associated to Fig 2

	Maximal tail current (pA)
CTR	128.2 ± 6.5
WIN	125.4 ± 8.4
AM251	121.6 ± 7.3
WIN+AM251	128.4 ± 5.8
CB1RKO	138.4 ± 5.6
CB1RKO+WIN	132.2 ± 7.9

Table S5 associated to Fig 1

	-400pA	-350pA	-300pA	-250pA	-200pA	-150pA	-100pA	-50pA
CTR	-42.8±0.6	-38.7±0.7	-34.8±0.6	-30.3±0.8	-25.0±0.6	-19.7±0.3	-13.7±0.3	-7.1±0.2
WIN	-43.8±0.8	-39.0±0.6	-34.3±0.9	-29.2±1.1	-23.9±0.6	-18.3±0.5	-12.4±0.3	-6.3±0.3
AM251	-42.2±0.6	-37.5±0.4	-32.5±0.5	-27.6±0.9	-22.5±0.8	-17.2±0.7	-11.9±0.4	-5.9±0.2

Table S6 associated to Figs 1 and 2

	Treatment	R_{in} (MΩ)[a]	V_m (mV)
Soma	**CTR**	**71 ± 7**	**-66.4 ± 1.2**
	WIN	67 ± 6*	-64.2 ± 2.1*
	WIN + AM251	81 ± 4*	-69.2 ± 1.3*
	AM251	84 ± 6*	-69.9 ± 1.4*
Dendrites	**CTR**	**25 ± 3**	**-69.9 ± 0.9**
	WIN	15 ± 2**	-67.2 ± 1.1*
	WIN + AM251	40 ± 4**	-71.0 ± 1.3*
	AM251	44 ± 4**	-70.9 ± 1.2*

IV. Legend to Supplemental Tables

Table S1: Raw data of the effects of modulation of the CB1R-I_h pathway on sag amplitudes in SPCs
Sag amplitude raw values (in mV) in SPCs for all the current injections between -400 and -50pA in WT and in genetically modified animals in control condition (---) and after different treatments (as indicated in the table). Data are presented as mean ± SE. The n values are reported in the legend of Fig 1.

Table S2: Raw data of the effects of modulation of the CB1R-I_h pathway on sag amplitudes in DPCs
Sag amplitude raw values (in mV) in DPCs for all the current injections between -400 and -50pA in WT and in genetically modified animals in control condition (---) and after different treatments (as indicated in the table). Data are presented as mean ± SE. The n values are reported in the legend of Fig 1.

Table S3: Raw data of the effects of WIN and AM251 on dendritic and somatic V_{50} in SPCs and DPCs
Raw values of half maximal I_h activation (V_{50}) in SPCs and DPCs obtained in dendritic cell attached or somatic whole cell recordings in control condition (---) and after treatments (as indicated in the table) in WT and CB1RKO

mice. Data are presented as mean ± SE. **p<0.01 DPCs vs. SPCs by Student's T-test. The n values are reported in legend of Fig 2.

Table S4: Raw data of the maximal tail currents
Raw values of maximal tail current recorded at -140mV in dendritic cell-attached voltage clamp recordings in SPCs and DPCs in control condition (CTR) and after treatments (as indicated in the table) in WT and CB1RKO mice. Data are presented as mean ± SE. The n values are reported in the legend of Fig 2

Table S5: Peak hyperpolarization amplitudes induced by current steps in SPCs
Raw data of the peak hyperpolarization in control condition and in presence of WIN or AM251 in WT animals for all the current step injections. Peak hyperpolarization raw values are reported as difference between the hyperpolarized peak and the resting membrane potential (-70mv) in mV. Data are presented as mean ± SE. The n values are reported in legend of Fig 1.

Table S6: Effect of WIN and AM251 on input resistance (R_{in}) and resting membrane potential (V_m) in SPCs.
Raw values of R_{in} and V_m recorded in somatic and dendritic whole cell configuration in control condition (CTR) and after WIN and/or AM251 application. R_{in} was measured from current injection ranging between -50 and +50 mV. Dendrite recordings were performed 250-300 µm from the soma. *p<0.05; **p<0.01 Experimental vs. CTR by Student's t-test. The n values are reported in legend of Fig 1 and 2.

V. Supplemental Experimental Procedures

Mice: CB1R mutants: In order to delete CB1Rs only in specific cell types, we used a mouse line in which the CB1R coding region is flanked by two *loxP* sites (CB1R-floxed, $CB1R^{f/f}$) and can therefore be deleted by Cre recombinase. GLU-CB1RKO mice were obtained by crossing $CB1R^{f/f}$ mice (Marsicano et al., 2003) with NEX-Cre mice (Kleppisch et al., 2003), and GABA-CB1RKO mice were generated by crossing $CB1R^{f/f}$ (Marsicano et al., 2003) with Dlx5/6-Cre mice (Monory et al., 2006). Ai9/$CB1^{f/f}$ mice were generated by crossing $CB1^{f/f}$ mice with B6;129S6-Gt(ROSA)26Sor$^{tm9(CAG-tdTomato)Hze}$/J (also known as Ai9). $CB1R^{f/f}$ mice were in a predominantly C57BL/6J background (backcrossed for seven generations; stock 000664; Jackson Laboratory).
Forebrain specific HCN1KOs: Based on the fact that the forebrain-restricted HCN1 deletion depends on expression of Cre under the CamKII promoter, wich turns on late in postnatal development (3-4 weeks), we used 8-10 weeks old animals for all the studies involving these mice in order to be sure that HCN1s were completely depleted.

***In vitro* electrophysiology**
Composition of sucrose-containing ACSF: mM: 85 NaCl, 75 sucrose, 2.5 KCl, 25 glucose, 1.25 NaH_2PO_4, 4 $MgCl_2$, 0.5 $CaCl_2$, and 24 $NaHCO_3$;
Composition of recording ACSF: 126 NaCl, 2.5 KCl, 26 $NaHCO_3$, 2 $CaCl_2$, 2 $MgCl_2$, 1.25 NaH_2PO_4, and 10 glucose**.**
Recording setup: Slices were visualized with an upright microscope (Olympus; BX61WI) with infrared–differential interference contrast (IR-DIC) optics. MultiClamp700B amplifier (Molecular Devices) was used for the recordings. Signals were filtered at 3 kHz using a Bessel filter and digitized at 10 kHz with a Digidata 1440A analog–digital interface (Molecular Devices). Series resistances were carefully monitored, and recordings were discarded if the series resistance changed >20% or reached 20 MΩ. The recorded traces were analyzed using Clampfit 10.2 (Molecular Devices).
Sag measurement: Pipette internal solution (in mM): 126 K-gluconate, 4 KCl, 10 HEPES, 4 ATP-Mg, 0.3 GTP-Na, 10 phosphocreatine and 0.2% biocytin, pH 7.2, 285-290 mOsm; Recording ACSF for these experiments also contained 0.5 mM 4-aminopyridine (4-AP) (to block voltage-gated K^+ channels), 1 µM tetrodotoxin (TTX; to block voltage-gated Na^+ channels), 0.1 mM $CdCl_2$ (to block Ca^{2+} channels), 1 mM $BaCl_2$ (to block inwardly rectifying K^+ channels), 5 µM NBQX (to inhibit AMPA receptors), 10 µM APV (to inhibit NMDA receptors), 10 µM bicuculline (to inhibit $GABA_A$ receptors), 22 µM CGP35348 (to inhibit $GABA_B$ receptors), 10 µM DHPG (to inhibit mGluRI), 3 µM LY341495 (to block mGluRII), 20nM MSOP (to block mGluRIII).
Dendritic I_h measurements: Internal solution (in mM): 120 KCl, 10 HEPES, 20 TEA-Cl, 5 4-amynopyridine, 2 $CaCl_2$, 1 $MgCl_2$ and 1 $BaCl_2$. The ACSF for these experiments contained 10 µM APV, 5 µM CNQX, 1 µM TTX, 10 mM TEA, 0.5 mM 4-AP, 0.1 mM $CdCl_2$, 1 mM $BaCl_2$, and 10 µM bicuculline. Leak subtraction was performed online in Clampex 9.2 (Molecular Devices, Union City, CA, USA) using eight sub-sweeps of polarity opposite to command voltages (note that active currents around resting membrane potential other than I_h were blocked by components of the internal solution, see above; Magee, 1998). After I_h recordings, gentle suction was applied in order to break the cell membrane and obtain whole cell configuration in order to measure RMP, and to allow biocytin to penetrate and diffuse into the cell for subsequent soma location identification. This procedure was necessary to distinguish SPCs from DPCs after the dendritic recordings.
Temporal summation of excitatory inputs and LTP: Glutamatergic EPSPs (temporal summation experiments) and EPSCs (LTP experiments) were investigated in the presence of picrotoxin and CGP55845 to block $GABA_A$ and $GABA_B$ receptors, respectively. The CA3 region was removed from the slice to prevent the emergence of epileptiform discharges after blocking GABA receptors. A concentric bipolar stimulating electrode was placed in stratum radiatum to evoke Schaffer collateral inputs. A patch electrode containing (in mM) 126 K-gluconate, 4 KCl, 10 HEPES, 4 ATP-Mg, 0.3 GTP-Na, 10 phosphocreatine and 0.2% biocytin was used to perform whole cell recordings in SPCs or DPCs to record the evoked EPSPs and EPSCs. Synaptic responses to various intensities of stimulation (3–10 mA, 0.1 ms) were recorded in the whole-cell configuration from the soma of individual CA1 PCs. In the summation experiments in GLU-CB1RKO mice, the train of EPSPs occasionally gave rise to action potential spikes at the fifth EPSP; in these cases, the amplitude of the 5th EPSP was calculated using the maximal amplitude before the spike generation.
Soma location idenfitication: Fixative solution contained 4% paraformaldehyde and 0.2% picric acid in 0.1mM phosphate buffer. The definition of the superficial and deep sublayers in the dorsal hippocampus used in this paper

was described previously (Lee et al., 2014): distance from the pyramidale-radiatum border: superficial sublayer: 0-20 µm; deep sublayer: 20-40 µm (for rationale and discussion, see Lee et al., 2014; Slomianka et al., 2011).
Viral Injections: After a recovery period following the injections, animals were returned to their home cage in the vivarium for at least 4 weeks to allow for expression of the protein, trans-synaptic retrograde transport of WGA-Cre to the soma, deletion of the *CB1R* gene, expression of red protein Tdtomato, and finally, degradation of previously expressed CB1Rs. Note that the majority of CB1Rs (70%) reside on the plasma membrane and have a half-life of 5 hours, whereas the remaining 30% of CB1Rs are assumed to be intracellular and have a half-life of 24-48 hours (Howlett et al., 2010). Therefore, at 4 weeks post- injection, we expect that there are no CB1Rs expressed in the red Tdtomato-expressing cells.
Behavioral experiments: Prior to training, mice were handled for 5 min per day for 5 d and habituated to the experimental apparatus for 5 min per day for 6 d in the absence of objects. The experimental apparatus was a white rectangular open field (30x23x21.5 cm). Learning and testing trials were video-recorded and analyzed by individuals who were blinded to the treatment condition of subjects.

VI. Supplemental References

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