Supplementary table 1 – Primary and secondary antibodies used in the study.

Antibodies	Host*	Diluition	Source
Albumin	G	1:4000 (IF)	Novus Biologicals, NB600-41532
ERK1/2	R	1:1000 (WB)	Cell Signaling Technology, 9102
GFP	R	1:300 (IF)	Cell Signaling Technology, 2956
Leptin	R	1:1000 (WB)	Peprotech, 500-P68
Nestin	М	1:400 (IF)	Merck, MAB353
pERK1/2 (Thr202/Tyr204)	R	1:2000 (WB), 1:3000 (IHC, IF)	Cell Signaling Technology, 4370
pSTAT3 (Tyr705)	R	1:1000 (WB), 1: 900 (IHC, IF)	Cell Signaling Technology, 9145
pSTAT3 (Tyr705)	G	1:600 (IF)	Santa Cruz Biotechnology, sc-7993
STAT3	М	1:1000 (WB)	Cell Signaling Technology, 9132
Vimentin	G	1:400 (IF)	Santa Cruz Biotechnology, sc-7557

TABLE 1A	Primary	antibodies.
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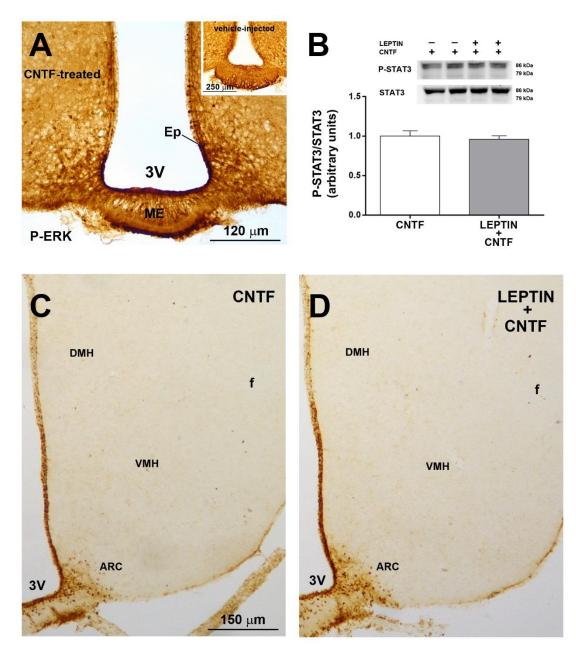
* R, rabbit; M, mouse; G, goat; WB, western blotting; IHC, immunohistochemistry; IF, immunofluorescence.

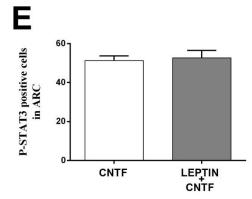
Conjugated to	React*	Dilution	Source
Fluorophore	R	1:400 (IF)	Invitrogen, A21206
Fluorophore	R	1:400 (IF)	Invitrogen, A31572
Fluorophore	G	1:400 (IF)	Invitrogen, A11055
Fluorophore	G	1:400 (IF)	Invitrogen, A21432
Fluorophore	G	1:400 (IF)	Invitrogen, A21082
Fluorophore	Μ	1:400 (IF)	Invitrogen, A21202
Peroxidase	R	1:200 (IHC)	Vector Laboratories, BA-1000
Peroxidase	М	1:5000 (WB)	Jackson ImmunoResearch, 715-036-150
Peroxidase	R	1:5000 (WB)	Jackson ImmunoResearch, 711-036-152

TABLE 1B | Secondary antibodies.

* R, rabbit; G, goat; M, mouse.

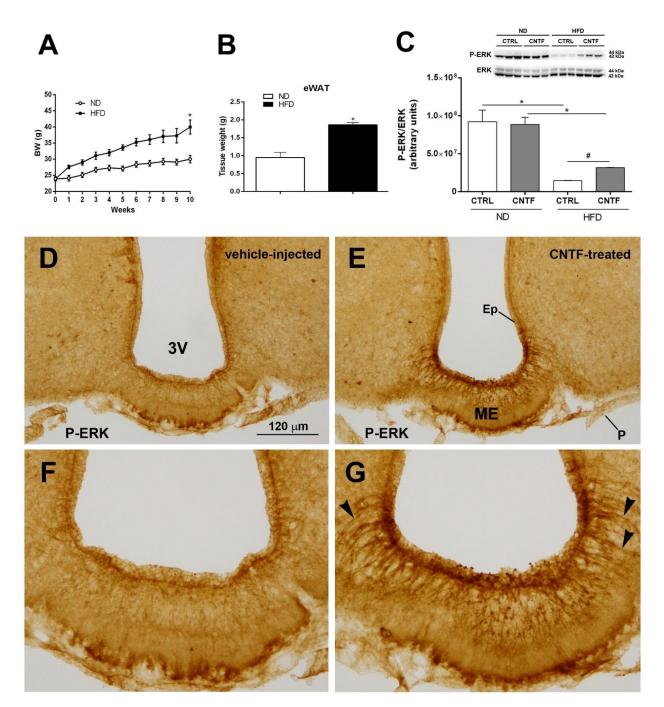
Supplementary figure 1





Supplementary figure 1 - CNTF administration to obese db/db mice. A: Representative immunoperoxidase image showing P-ERK immunoreactivity in the ependyma (Ep) lining the infundibular recess of the third ventricle (3V) from a db/db mouse treated with CNTF. The inset shows the corresponding region from a vehicle-injected mouse. B: Representative immunoblot and quantification of P-STAT3 content in hypothalamic protein extracts from db/db mice treated with CNTF or CNTF/leptin. Data (3 mice) are mean \pm SEM (one-way ANOVA). C and D: Representative P-STAT3 immunoperoxidase staining from db/db mice treated with CNTF (C) or CNTF/leptin (D). ARC, arcuate nucleus; VMH, ventromedial hypothalamus; DMH, dorsomedial hypothalamic nucleus; f, fornix; All pics were taken from coronal hypothalamic sections at bregma – 1.70 mm. E: Number of P-STAT3-positive neurons in the ARC of CNTF-and CNTF/leptin-treated mice from three coronal sections. Data (3 mice) are mean \pm SEM (unpaired Student's t-test).

Supplementary figure 2



Supplementary figure 2 – Activation of ERK signaling by CNTF in HFD obese mice. A: Body weight of mice fed a normal diet (ND) and a high fat diet (HFD) for 10 weeks. Data (3 mice) are mean \pm SEM, (unpaired Student's t-test), *p<0.05 comparison between ND and HFD mice. B: Tissue weight of the epididymal fat depots from ND and HFD mice. Data (3 mice) are mean \pm SEM, (unpaired Student's t-test), *p<0.05 comparison between ND and HFD mice. C: Representative immunoblot and P-ERK quantification in hypothalamic protein extracts from vehicle-injected (CTRL-ND) and CNTF-treated (CNTF-ND) mice fed a normal diet (ND) and vehicle-injected (CTRL-HFD) and CNTF-treated (CNTF-HFD) mice fed a HFD. Data (3 mice) are mean \pm SEM, *p<0.05 comparison

between ND and HFD mice, #p<0.05 comparison between CNTF-treated and vehicle-injected HFD mice (one-way ANOVA). D-G: Representative immunoperoxidase images showing P-ERK immunoreactivity in a vehicle-injected HFD mouse (D and F) and in a CNTF-treated HFD mouse (E and G). In this latter, arrowheads indicate tanycyte-like projections that are strongly positive for P-ERK. F and G are enlargements of the median eminence (ME) of D and E, respectively. 3V, third ventricle; Ep, ependymal layer, P, pial surface. All pics were taken from coronal hypothalamic sections at bregma – 1.70 mm.