Supporting Information

Shi *et al.* 10.1073/pnas.0808701106

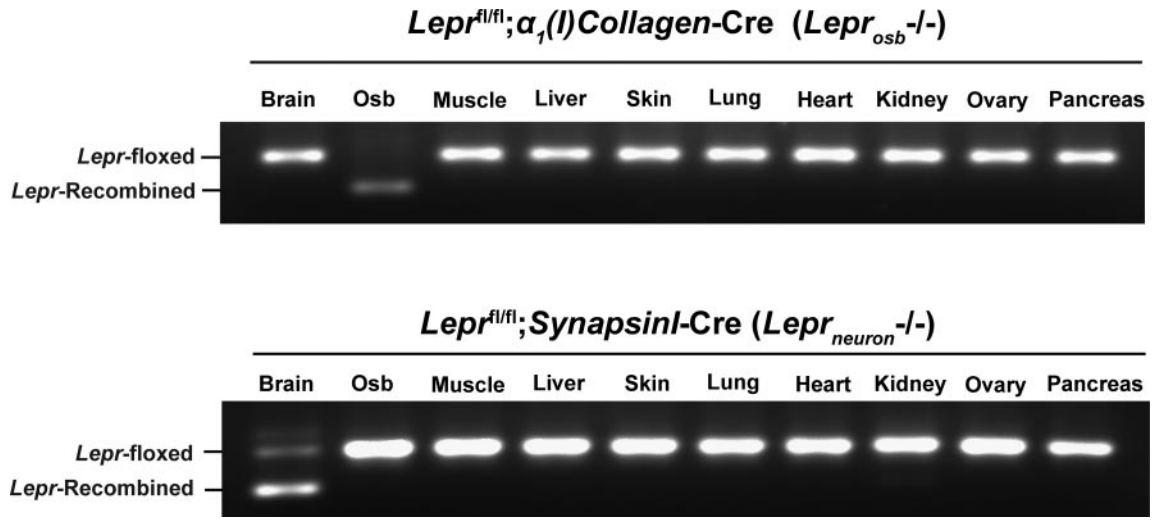

Fig. S1. Recombination analysis of the *Lepr* locus in osteoblasts and brain. Representative results of PCR-based genotyping of *Lepr* in various tissues/organs of α_1(*I*)*Collagen*-Cre, *Lepr*$^{fl/fl}$, and *SynapsinI*-Cre;*Lepr*$^{fl/fl}$ mice. The same primers and PCR program have been used as described [McMinn JE, *et al.* (2004) An allelic series for the leptin receptor gene generated by CRE and FLP recombinase. *Mamm Genome* 15:677–685].

Table S1. Comparison of the anthropomorphic indices between the *l/l* mice and their WT littermates

Indices	WT	*l/l*
Body weight, g	20.7 ± 0.4	20.1 ± 0.2
Fat pad weight, g	0.218 ± 0.027	0.122 ± 0.015*
Urinary epinephrine, ng/mL per mmol creatinine	19.0 ± 5.0	41.1 ± 16.6
Urinary norepinephrine, ng/mL per mmol creatinine	26.1 ± 3.3	49.1 ± 8.1*
Serum leptin, pg/mL	4.2	2.3*
Body temperature, °C	38.3 ± 0.1	38.4 ± 0.1

Twelve-week-old female mice were used in these analyses, $n = 8–10$ per group for the analysis of body weight and gonadal fat mass; $n = 5$ for urinary epinephrine and norepinephrine content; and $n = 3$ for body temperature measurement.

*$P < 0.05$, mean ± SEM. Serum leptin level data have been derived from Bjørnholm M, *et al.* (2007) Mice lacking inhibitory leptin receptor signals are lean with normal endocrine function. *J Clin Invest* 117:1354–1360.