Supplemental Figure Legends

Fig S1. Cycloheximide does not block the stimulatory effect of TNF-α on sLEPR release. Ad-LEPRb-infected HEK293 cells were pre-treated with or without 50 ug/ml cycloheximide (CHX and control, respectively) for 30 min followed by 20 hr treatment with vehicle or 20 ng/ml TNF−α in the continuous presence or absence of CHX. sLEPR levels in conditioned media were determined by ELISA (A), and LEPRb in cell lysates were determined by Western blotting (B).

Fig S2. Chloroquine does not block the stimulatory effect of TNF−α on LEPRb protein levels. LEPRb-transfected N2a cells were pre-treated with chloroquine (50 uM) for 30 min followed by 6 hr treatment with vehicle or TNF−α (50 ng/ml). LEPRb and α-tubulin levels in cell lysates were determined by Western blotting.

Fig S3. TNF−α activates multiple MAP kinase pathways. Ad-LEPRb-infected HEK293 cells were treated with TNF−α at the indicated concentrations for 6 hours. Levels of JNK/SAPK, p38 MAP kinase and p44/p42 (ERK1/2) and their phosphorylated counterparts were determined by Western blotting using antibodies specified to the phospho-protein or the total protein.

Fig S4. PMA stimulates LEPRb protein expression. Ad-LEPRb-infected HEK293 cells were treated with PMA (1 ug/ml) for 15 hrs. Levels of LEPRb in cell lysates and sLEPR in conditioned media were determined by Western blotting. A duplicate set of samples are shown.

Supplemental Figures

Fig S1

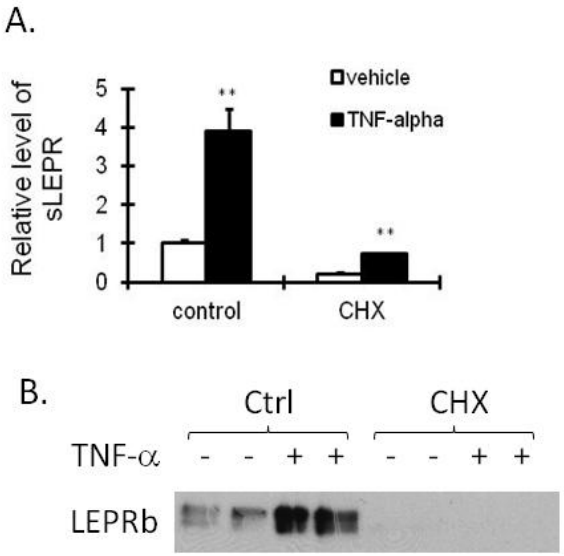

Fig S2

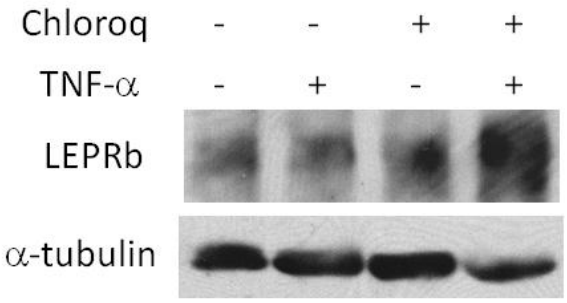

Fig S3

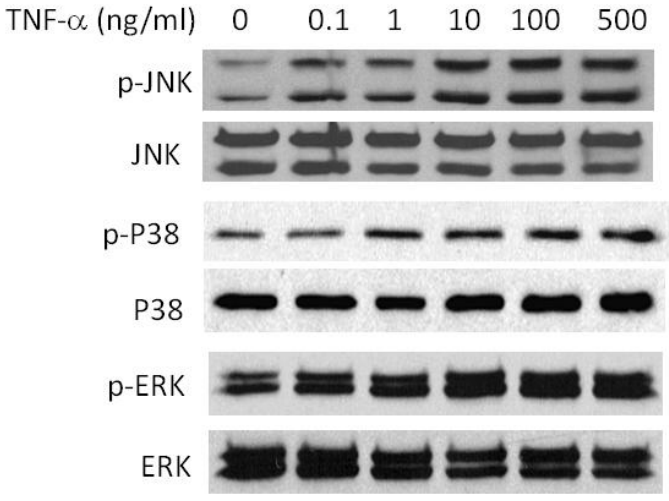

Fig S4