

Leptin ameliorates ischemic necrosis of the femoral head in rats with obesity induced by a high-fat diet

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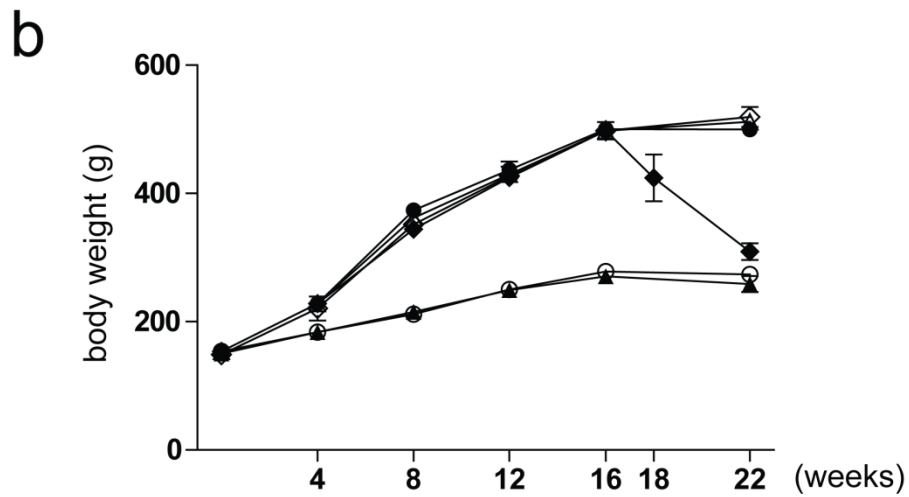
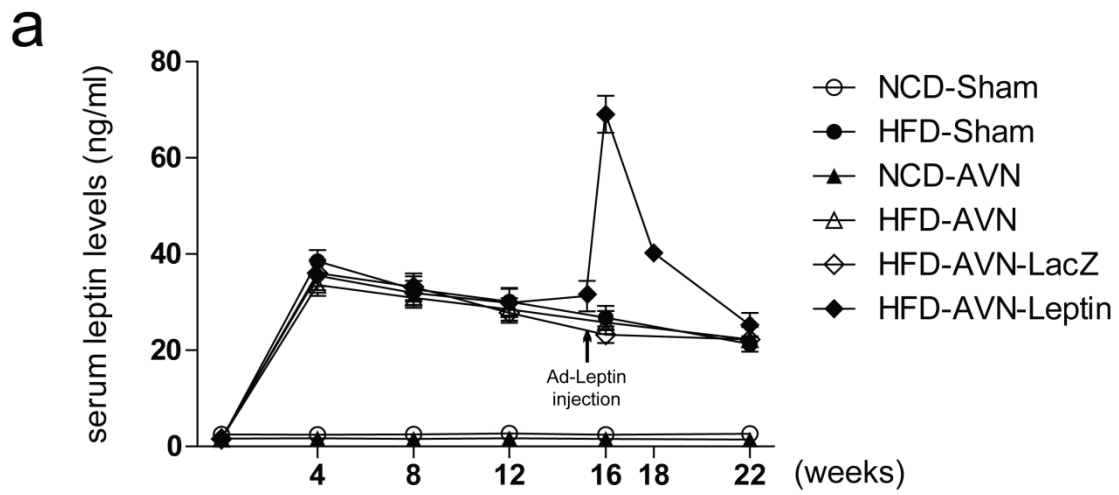
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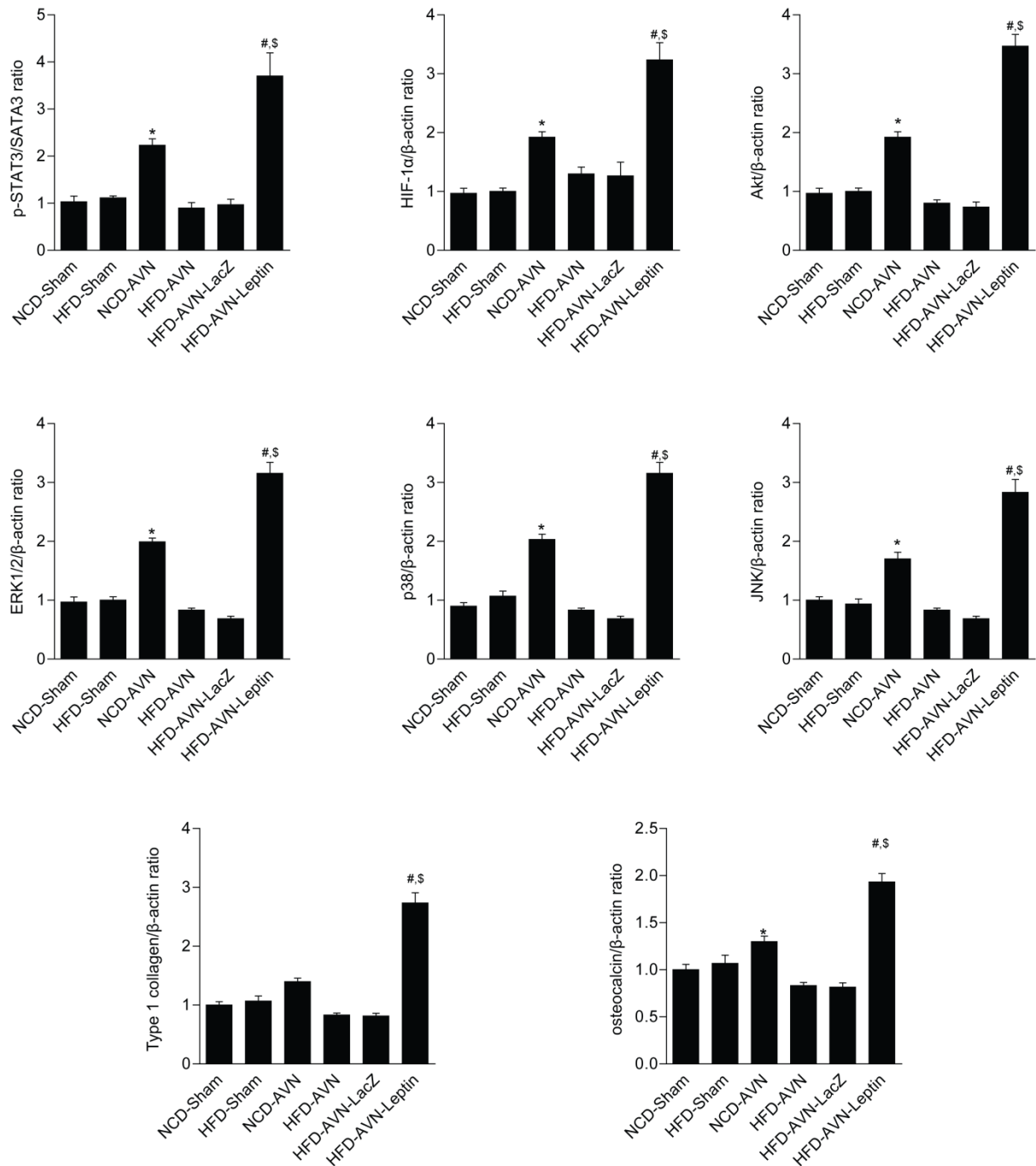
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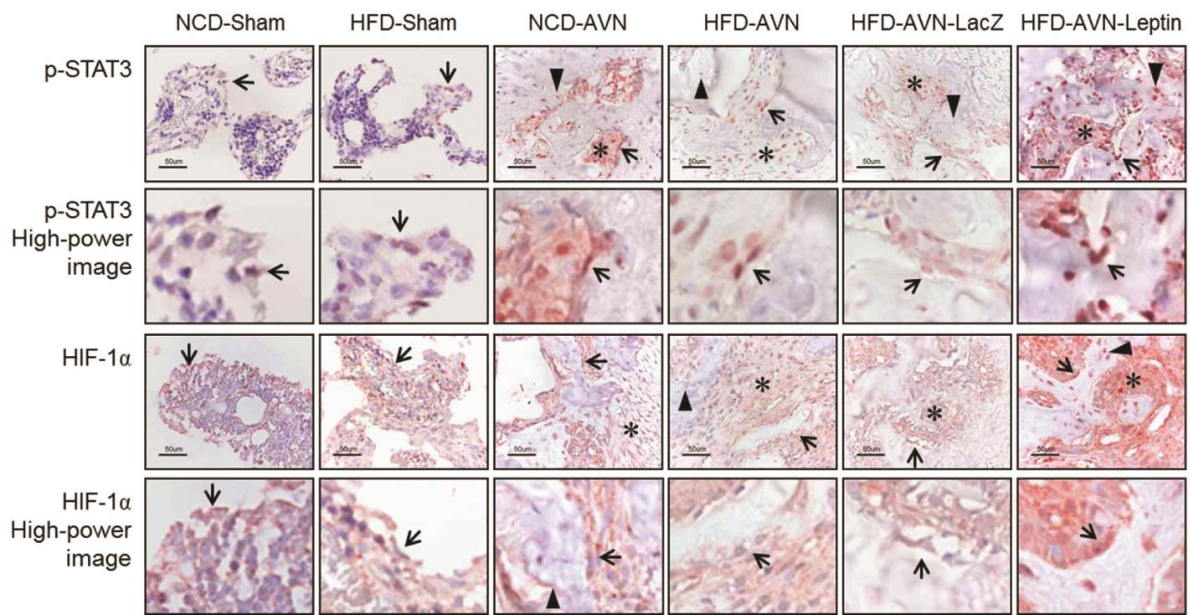
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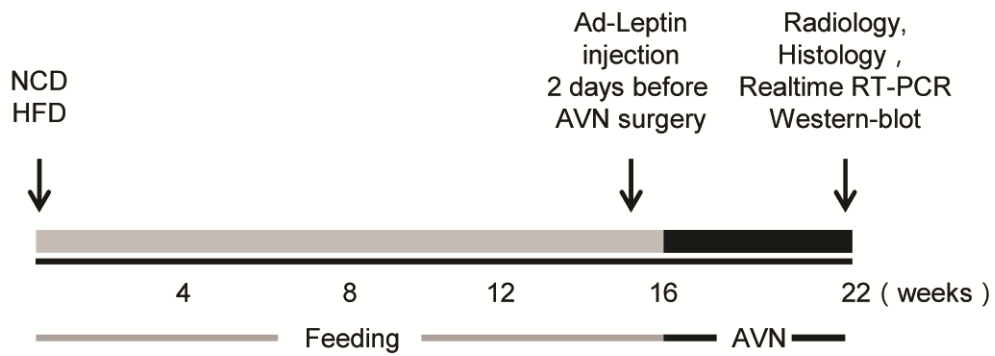
Supplementary Figure 1. Plasma leptin and body weight. Plasma leptin levels (A) and body weight (B) were measured in each group. Values represent mean \pm SEM for four animals in each group.



Supplementary Figure 2. Western blot analysis indicates both canonic and noncanonical leptin signaling pathways in the groups, respectively. The protein content was calculated by gray scale. Data are expressed as the mean SEM (n = 5 per group). *, p < 0.05 vs. NCD-Sham; #, p < 0.05 vs. NCD-AVN; \$, p < 0.01 vs. NCD-AVN; &, p < 0.01 vs. HFD-AVN-LacZ



Supplementary Figure 3. High power images (H &E, x400) stromal cells on bone surfaces. Expression of phosphorylated STAT3 and HIF-1 α were stronger in osteoblasts (arrows), osteoblasts (arrow heads), and stromal fibroblasts (asterisks) in HFD-AVN-Leptin compared with HFD-AVN or HFD-AVN-LacZ. Phosphorylated STAT3 and HIF-1 α expression in NCD-AVN was intermediate between HFD-AVN-Leptin and HFD-AVN.



Supplementary Figure 4. Experimental design. After 16 weeks of NCD or HFD feeding, AVN was surgically induced. Ad-Leptin was by intravenous injection 2 days before surgery-induced AVN. Animals were euthanized at 6 weeks after inducing AVN.