A

B

C

Figure 1. The integration strategy of network pharmacology in GA alleviating SONFH. (A) 2D structure of GA. (B) Venn diagram of GA and ONFH. (C) Core target network diagram of GA and ONFH.
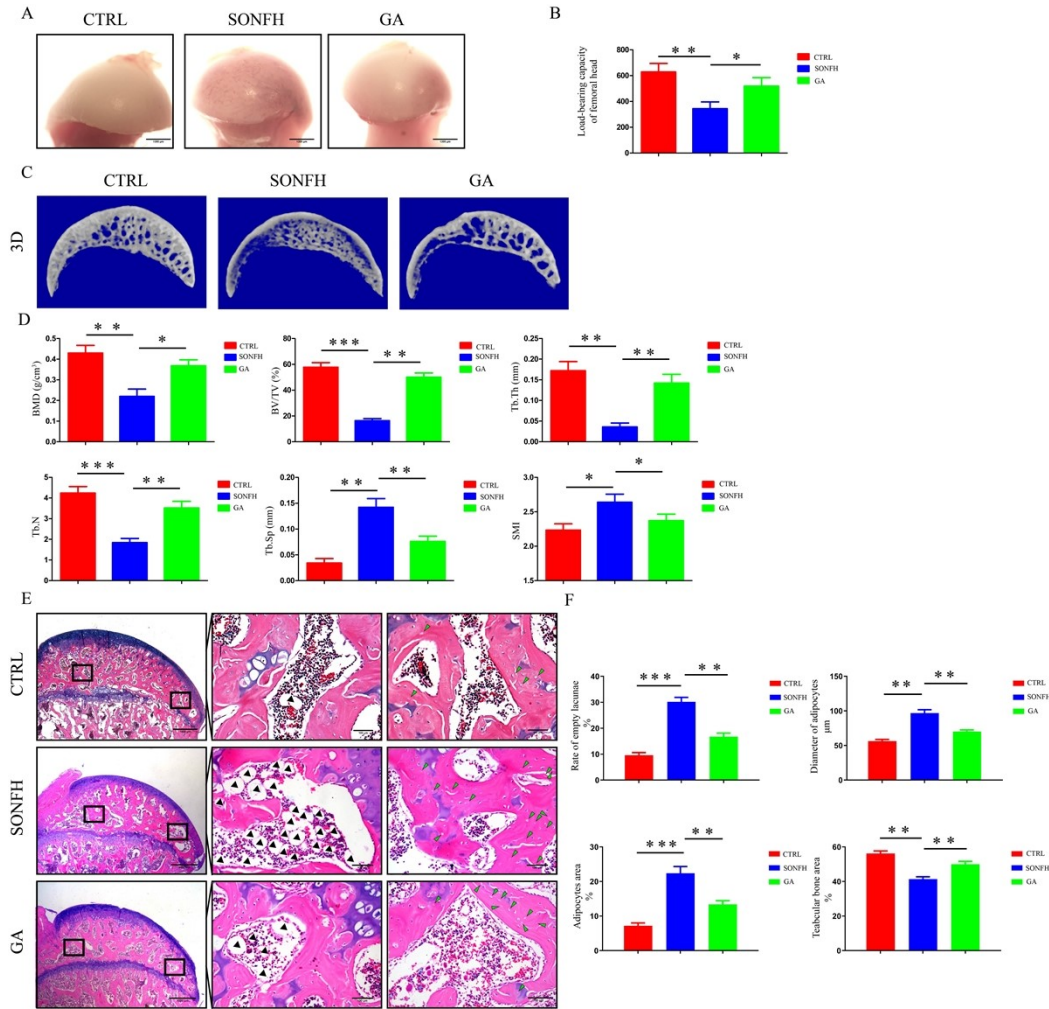
Figure 2. GA improved the microstructure of bone trabeculae and histopathological changes of SONFH rats. (A) Gross morphology of the femoral heads. (B) The load-bearing capacity of femoral heads. (C) μCT scanning images of the femoral heads. (D) Quantitative analysis of BMD, BV/TV, Tb.Th, Tb.N, Tb.Sp and SMI. (E) Histological images (ABH/OG) of the femoral heads. (F) Histomorphometric analysis of ABH/OG staining. The adipocytes are indicated by black arrows and the empty lacunae are indicated by green arrows. GA group (treated with GA at a dosage of 50mg/kg/d via oral administration for 6 weeks). Data are expressed as mean ± SD, *$P <$ 0.05, **$P <$ 0.01, ***$P <$ 0.001 (vs SONFH).
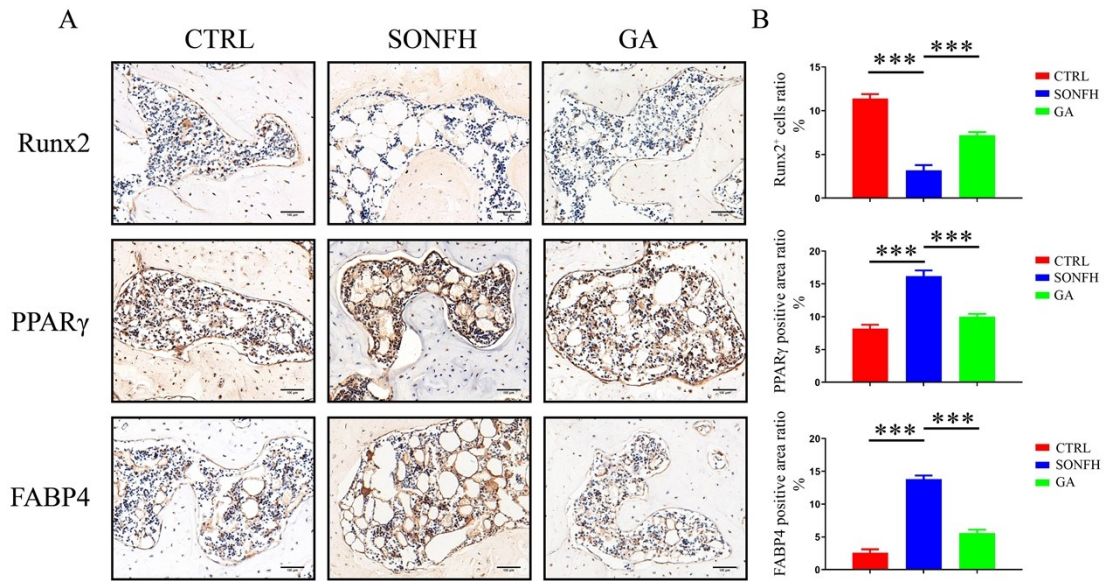
Figure 3. GA promoted osteogenic differentiation and suppressed adipogenic differentiation in SONFH rats. (A) IHC staining of osteogenic and lipogenic-associated proteins of the femoral heads. (B-D) Quantitative analysis of Runx2, PPARγ and FABP4. Data are expressed as mean ± SD, ***$P < 0.001$ (vs SONFH).
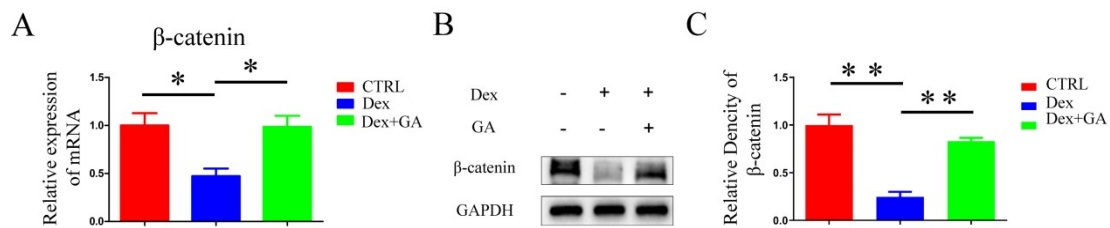

Figure 4. GA reversed the decrease in β-catenin levels caused by Dex. (A) The mRNA level of β-catenin. (B,C) The protein level of β-catenin. The Dex concentration was 1 μM and the GA concentration was 80 μM. Data are expressed as mean ± SD, *$P < 0.05$, **$P < 0.01$ (vs Dex).