Figure S1. Sal B improves the oxidative stress state and inhibits liver fibrosis. Under normal circumstances, the generation and elimination of ROS are in a state of dynamic equilibrium. When the body is stimulated by free radicals, the dynamic balance of ROS production and clearance is destroyed, resulting in liver damage. The accumulation of ROS in long-term liver injury can activate the TGF-β/Smad signaling pathway to promote liver fibrosis and TGF-β_1 can promote the production of ROS, thus forming a vicious cycle. In the present study, Sal B inhibited liver fibrosis by attenuating the oxidative stress state via the activation of the Nrf2/HO-1 signaling pathway and inhibition of pSmad2L/C. ROS, reactive oxygen species; Sal B, salvianolic acid B; p-, phosphorylated; Smad2C, Smad2 at COOH-terminal; Smad2L, Smad2 at linker region; HO-1, heme oxygenase-1; Nrf2, nuclear factor erythroid-2-related factor 2; TGF-β_1, transforming growth factor β 1; ALT, alanine transaminase; AST, aspartate aminotransferase; SOD, superoxide dismutase; MDA, malondialdehyde; GSH, glutathione.