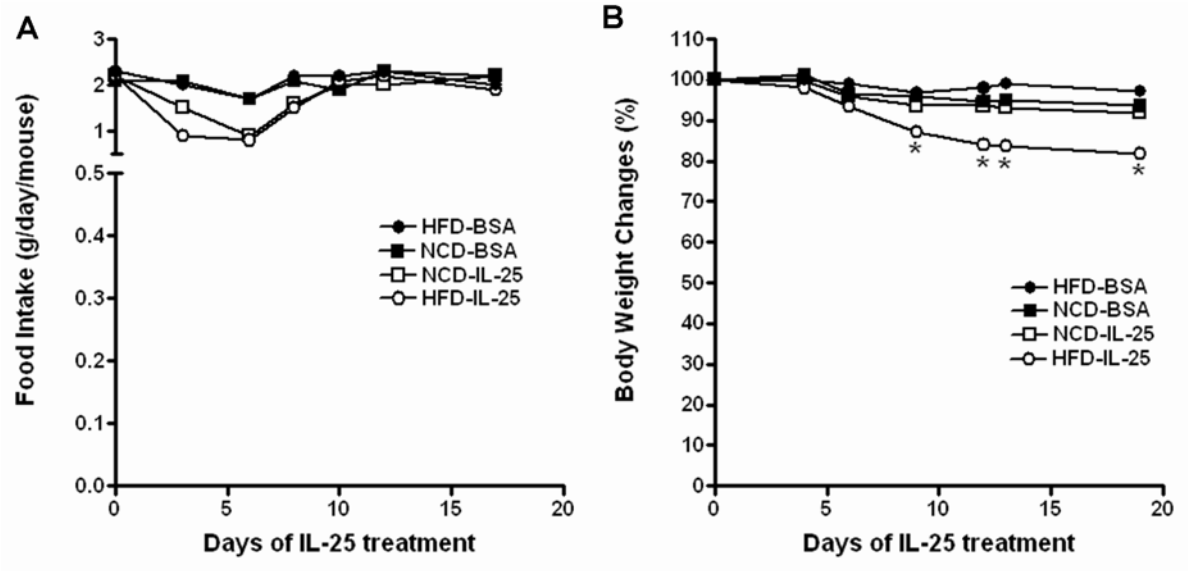

Figure S1. Administration of IL-25 affects the food intake and body weight. Mice were fed the high-fat diet (HFD) or normal control diet (NCD) for ~14 weeks, re-grouped, and then received injections of IL-25 or BSA for three weeks. Food intake (A) and body weight (B) were monitored during the course of IL-25/BSA treatment. Data shown are average food intake per mouse per day (A) or mean ± SEM for body weight (B). * $P<0.05$ versus HFD-BSA.

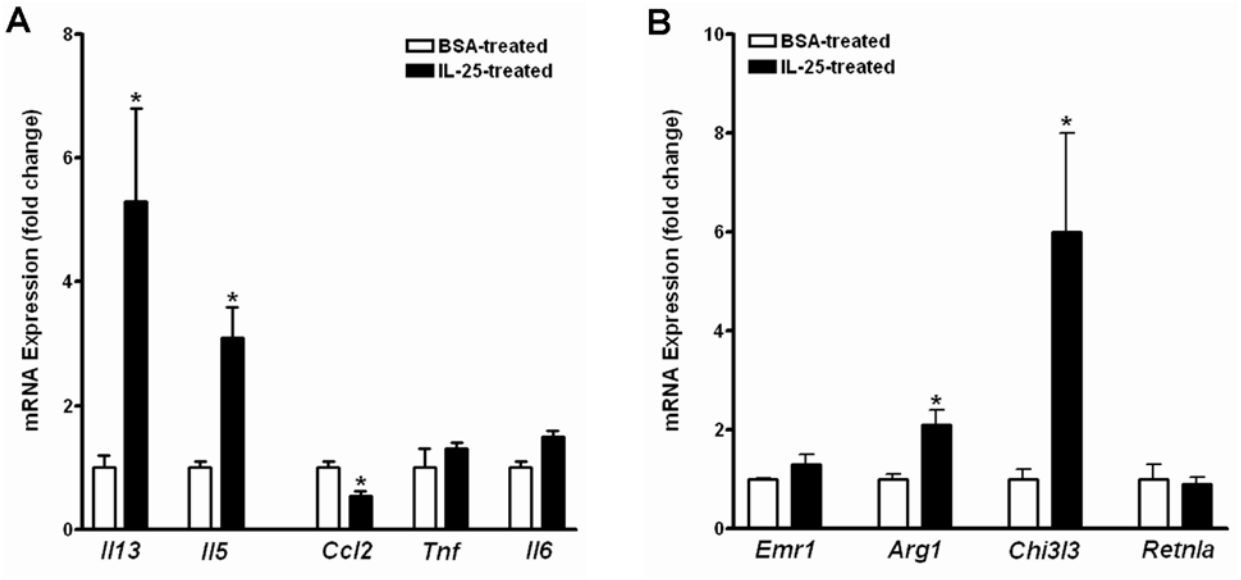

Figure S2. Administration of IL-25 to mice fed a normal control diet induces a type 2 cytokine response and alternative activation of Kupffer cell/macrophage in the liver. Mice were fed a normal control diet for 14 weeks, re-grouped, and then received injections of IL-25 or BSA for three weeks. (A) Hepatic expression of cytokines; (B) Hepatic expression of markers for Kupffer cell/macrophage activation. The fold changes are relative to BSA-treated after normalization to 18s rRNA. Data shown are mean ± SEM. **P*<0.05 versus respective BSA-treated.

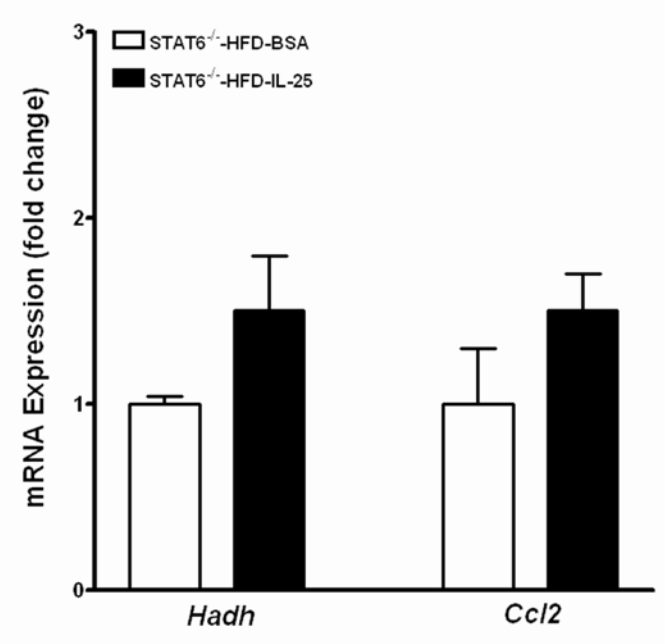

Figure S3. Administration of IL-25 does not alter the expression of Hadh and Ccl2 in mice deficient in STAT6. STAT6$^{-/-}$ mice were fed a high-fat diet (HFD) for ~14 weeks, re-grouped, treated with IL-25 or BSA for three weeks. Hepatic gene expression of *Hadh* and *Ccl2* was examined by qPCR. The fold changes are relative to STAT6$^{-/-}$-HFD-BSA mice after normalization to 18s rRNA. Data shown are mean ± SEM.

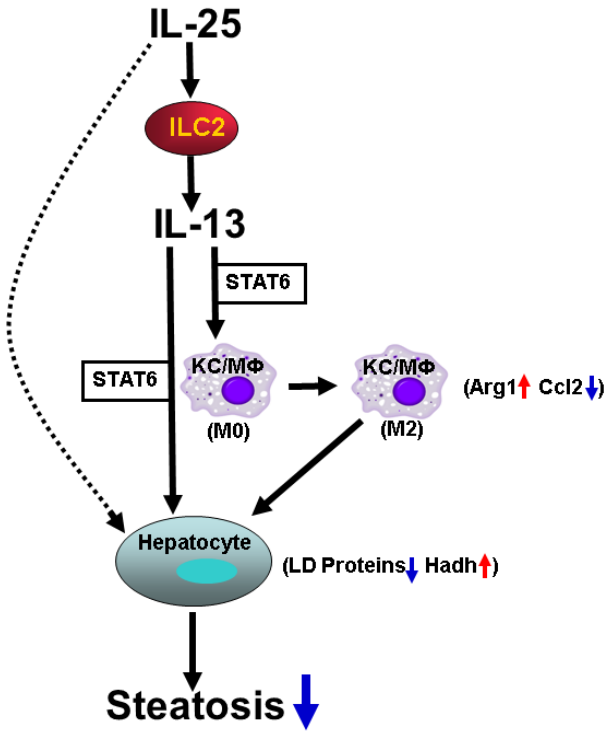

Figure S4. Hypothetical model of IL-25 on hepatic steatosis. IL-25 in the liver stimulates ILC2 to release IL-13 that in turn acts on hepatocytes as well as Kupffer cells via STAT6-depedent pathways, which leads to alterations in the expression of LD-associated proteins and factors that control lipid metabolic pathways thereby decreasing hepatic steatosis.