ELECTRONIC SUPPLEMENTAL MATERIAL (ESM)

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CREBH mediates metabolic inflammation to hepatic VLDL overproduction and hyperlipoproteinemia

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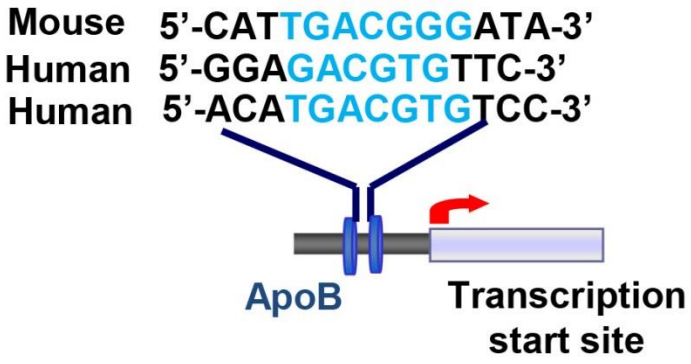
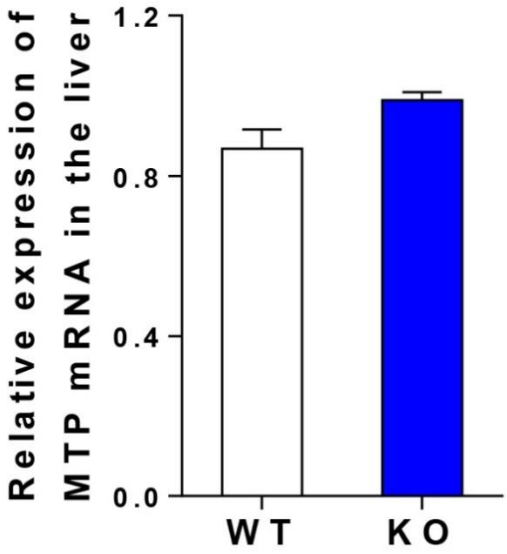
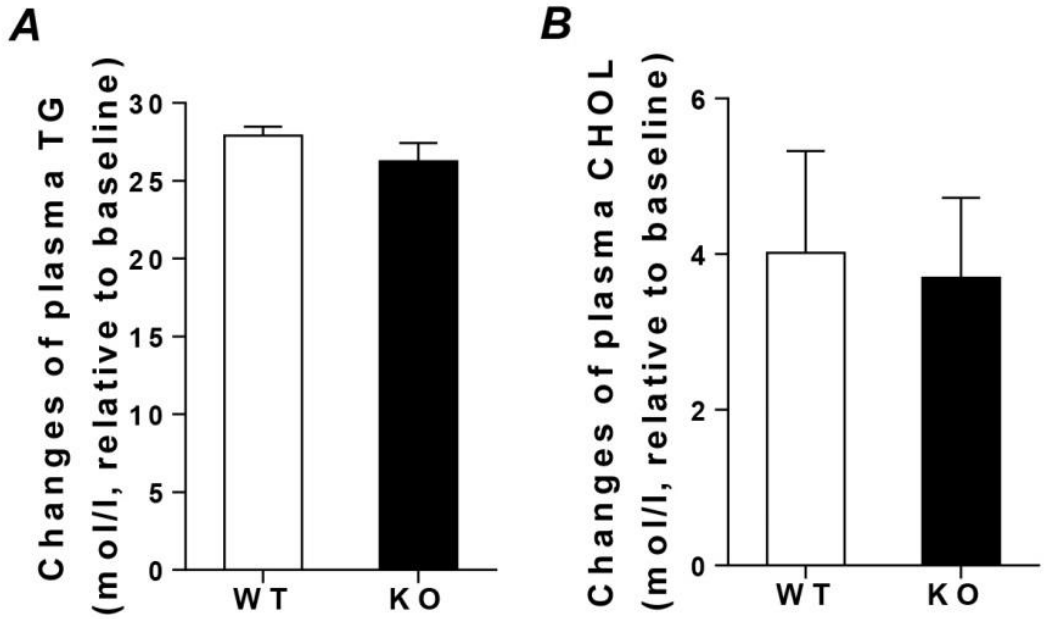


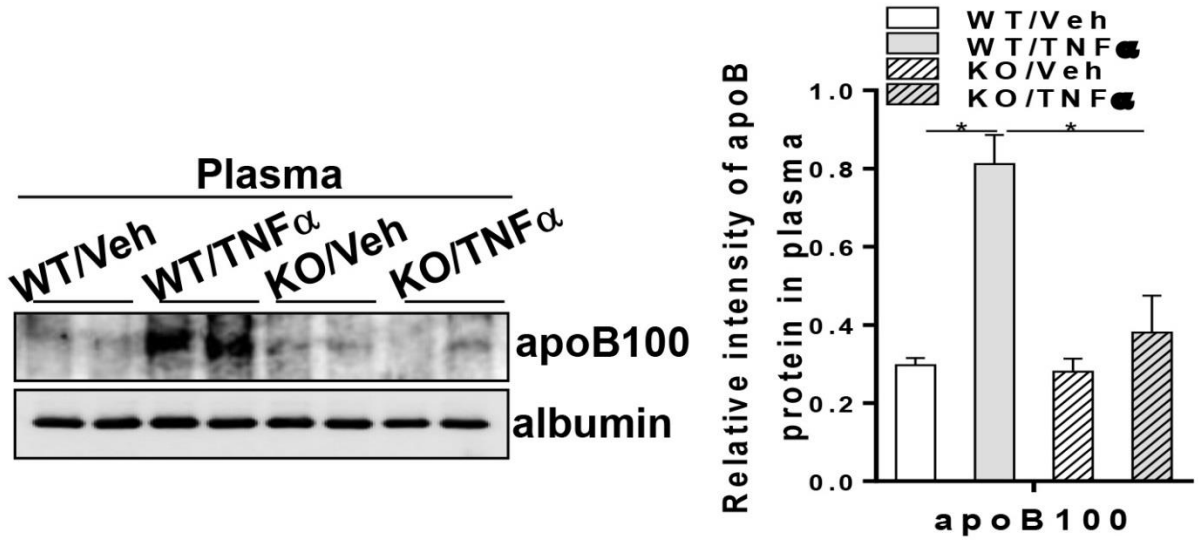
Supplementary Figure 1. The proposed CREBH binding elements present in mouse and human apoB gene promoters.



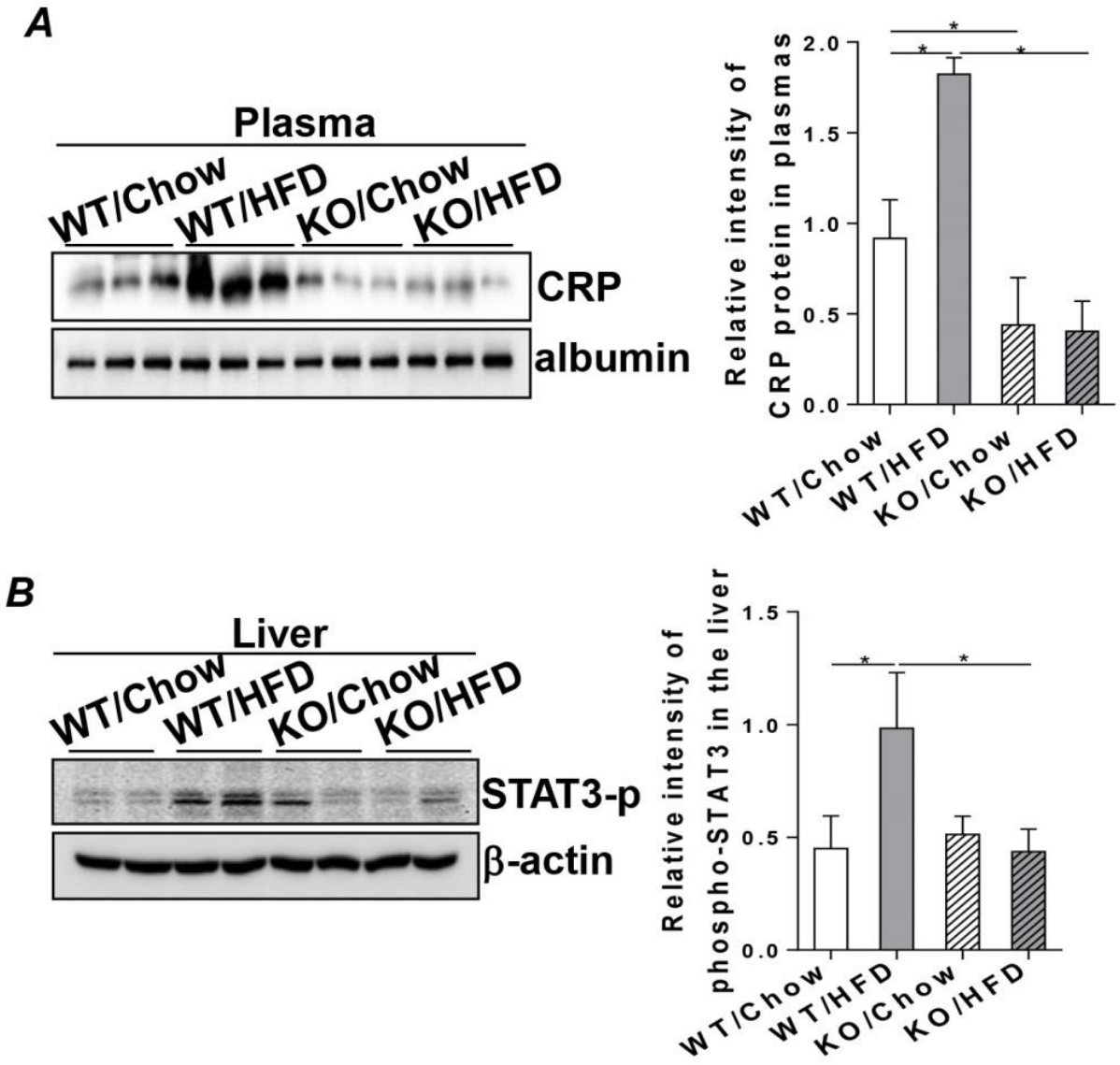
Supplementary Figure 2. MTP mRNA from livers of WT and KO mice after a 12h fast.



Supplementary Figure 3. Blood was collected from WT and KO mice following a 5h fast for baseline control (0h). Mice were then treated with poloxamer (500mg/kg, ip. injection). Mice were sacrificed at 3h after poloxamer treatment. Blood samples were collected for determining the changes of triglyceride (A) and cholesterol (B) contents between baseline (0h) and 3h.



Supplementary Figure 4. WT and KO mice were fasted for 6h. Mice were then given either recombinant mouse TNFα (12μg/200g body weight) or vehicle (Veh), saline, as control via ip. injection. Plasmas were collected at 5h post-treatment for immunoblotting the secreted VLDL-apoB100 and albumin. Right panel shows quantification of apoB100 signal intensity to loading control albumin. Results are shown as mean ± SEM, n=5-6/group. *P < 0.05 versus controls.



Supplementary Figure 5. WT and KO mice were fed with either a chow or a HFD (60% caloric from fat, Dyets #103938) for 7 weeks. Blood samples and liver tissues were collected at the endpoint for analysis. (A) Immunoblotting of liver phosphor-Stat3 and total Stat3. (E) Immunoblotting of plasma C-reactive protein (CRP). Quantifications of Stat3 and apoB protein signal intensity were shown in the right panels. Results are shown as mean ± SEM, n=5-6/group. *P < 0.05 versus controls.