Supplementary Materials: Compound K Attenuates the Development of Atherosclerosis in ApoE$^{-/-}$ Mice via LXRα Activation

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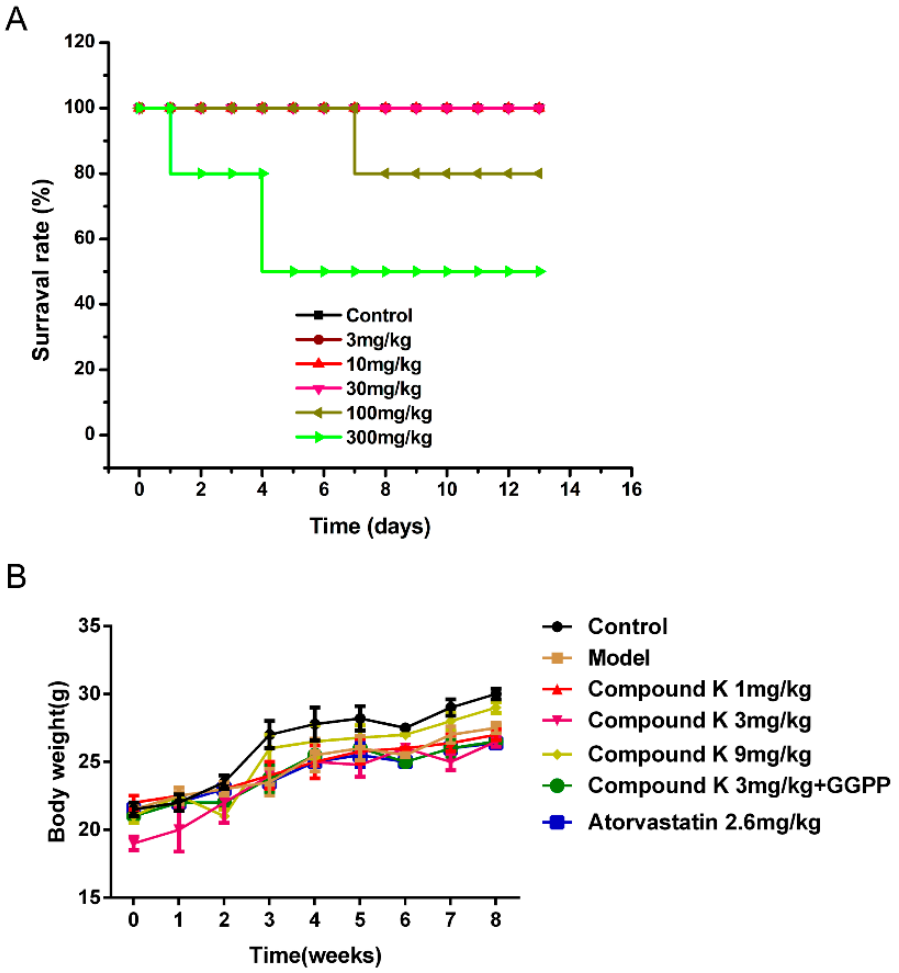

Figure S1. Potential acute toxicity of Compound K in vivo. Compound K of different concentration was administered to mice i.p. once. Survival ratio was recorded after 14 days. There was no mouse got dead until the compound K dose reached 100 mg/kg (**A**); During the in vivo experiments, animals were weighed every week. There was no significant alteration in the body weight (**B**). Data of weight are presented as mean ± SEM (n = 6), and analysed by ANOVA with Dunnett's post-hoc analysis.

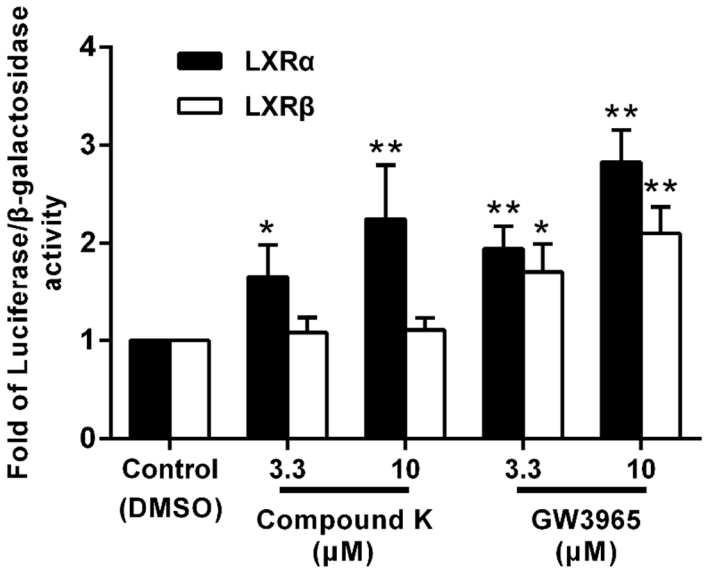

Figure S2. Compound K selectively actives LXR-α in HEK293T cell line. The HEK293T cells were transfected with hLXREx3TK-Luc as a reporter plasmid, pCMX-hLXR-α or pCMV-hLXR-β as expression vectors, and pSV-β-galactosidase was used to normalise the transfection efficiencies. Cells were treated with increasing concentrations of compound K (3.3, 10 μM). GW3965 (3.3, 10 μM) was used as positive control. After incubation, the cells were lysed, and assayed for luciferase and β-galactosidase activities. The results are expressed as relative luciferase activity (fold difference compared to negative control). Data are presented as mean ± SEM (n = 3, each in duplicate). * $p < 0.05$ vs. control, ** $p < 0.01$ vs. control.