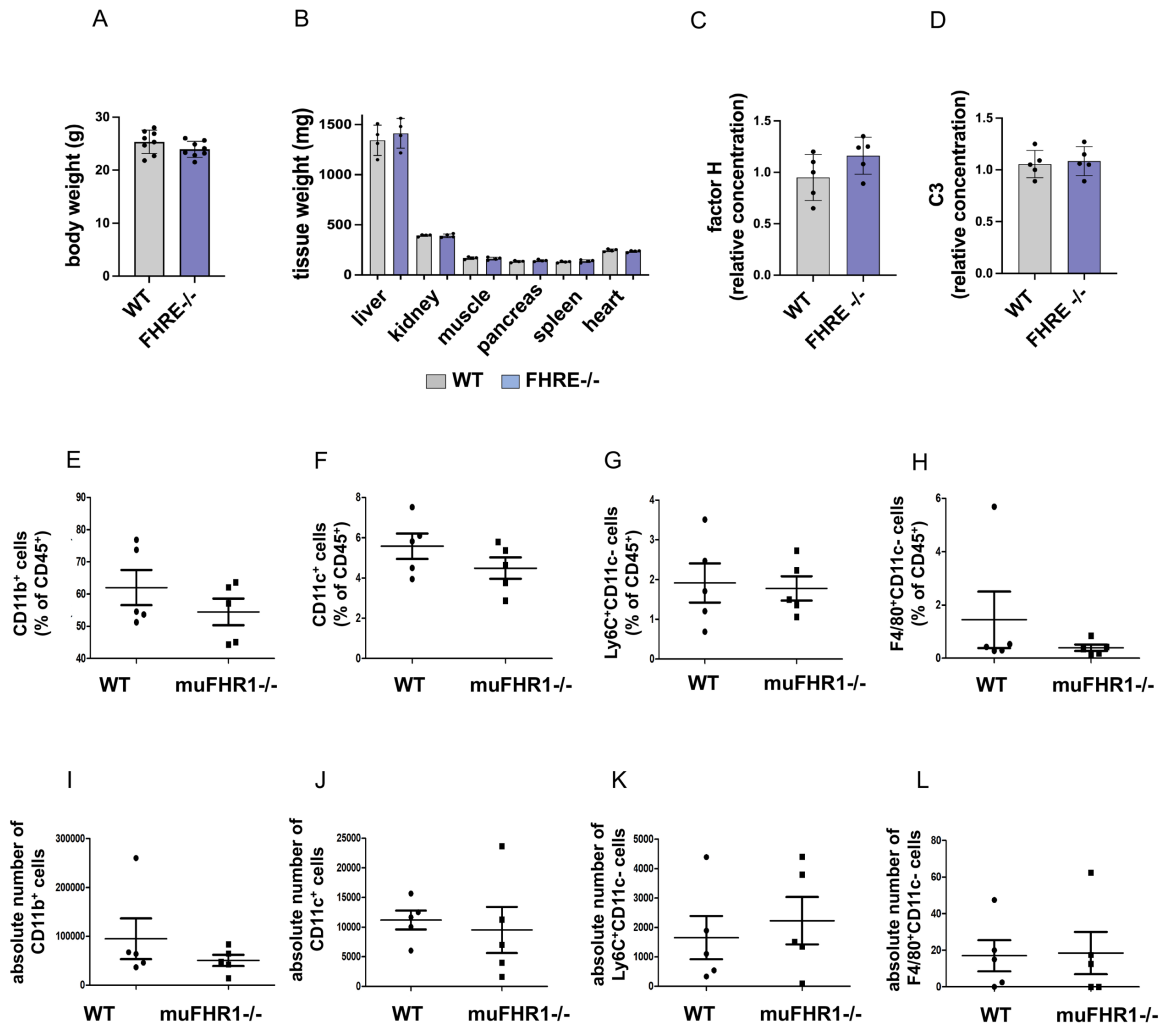
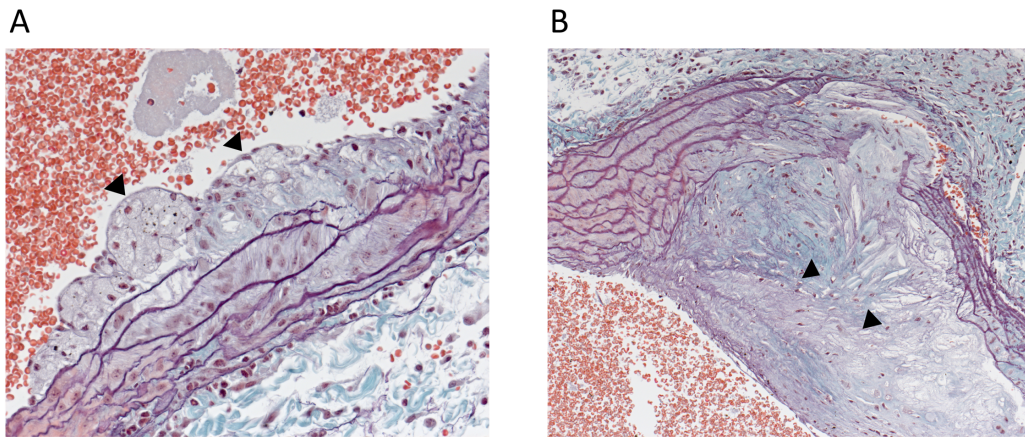


Deletion of the mouse homolog of human FHR1 (muFHR1) alleviates atherosclerosis in ApoE^{-/-} mice

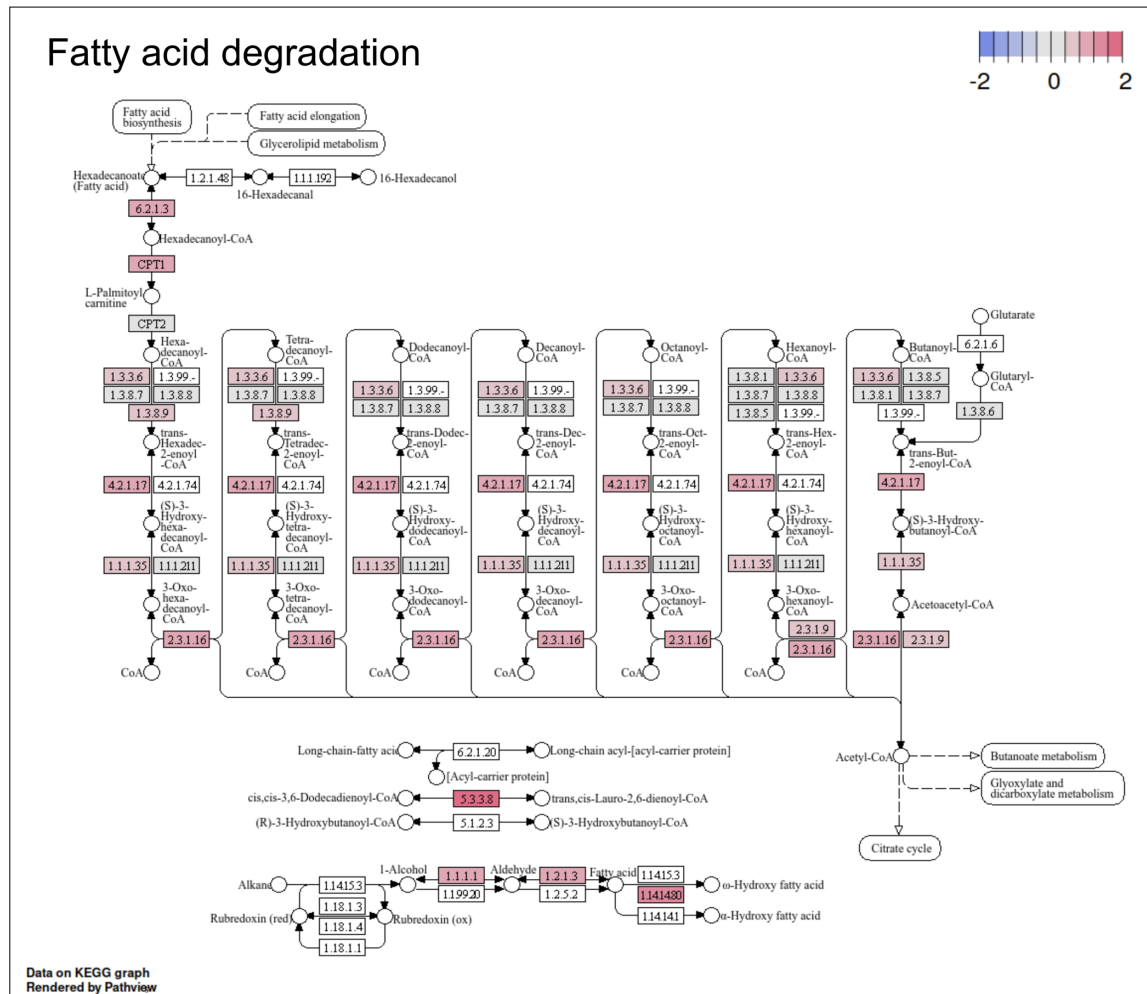
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Supplementary Figure 1. Comparison between WT and muFHR1^{-/-} mice. (A) The body weight of muFHR1^{-/-} mice (n=8), as well as (B) the weight of various organs, were similar to that of wild-type (WT) animals (unpaired Student's t-test, n=4). (C) There was no difference in serum levels of factor H and (D) complement C3 between muFHR1^{-/-} mice and WT mice, as measured by ELISA (unpaired Student's t-test, n=5). (E – F) muFHR1^{-/-} mice show no significant differences in percentages of myeloid cells (CD11b⁺) of CD45⁺ leucocytes, of dendritic cells (CD11c⁺), of monocytes (Ly6C⁺CD11c⁻) and macrophages (F4/80⁺CD11c⁻). (I – L) There was also no significant difference in the absolute number of cells measured.



Supplementary Figure 2: The muFHR1^{+/+}ApoE^{-/-} mouse displays atherosclerotic deposits in the heart Cross sections of the heart of 40 weeks old muFHR1^{-/-}ApoE^{-/-} mouse stained with Masson-Goldner-Elastica shows (A) fatty deposits on the aortic wall with foam cells (triangle) and (B) a plaque close to the aortic valve containing cholesterol crystals (triangles, magnification x 400).



Supplementary Figure 3: Influence of muFHR1 on fatty acid degradation. muFHR1 deficiency enhances cytochrome P450 oxidoreductases Cyp3a16, Cyp2c29 and Cyp2a22 which are involved in fatty acid degradation.

