Supplementary Figure 1

Supplementary figure 1: Western diet exacerbates dyslipidaemia in apoE2-KI mice.

Statistically significant differences are indicated (Mann&Whitney test, *** p<0.001).

TC: total cholesterol, HDL-C: HDL-cholesterol, N-HDL-C: non-HDL-cholesterol, TG: triglycerides
Supplementary figure 2: MP cell origin distribution on apoE2-KI mice fed a Western diet

Distribution of MPs was measured in atherosclerotic lesions, liver and plasma using specific markers of cell origin, i.e. F4/80 (macrophages), CD144 (endothelial cells), CD41 (platelets) and TER119 (erythrocytes).
Supplementary figure 3: Long-term fenofibrate treatment increases hepatic ACO gene expression and improves diet induced-dyslipidaemia in apoE2-KI mice.

A: ACO mRNA level in the liver.

B: Plasma lipid concentrations.

Statistically significant differences are indicated (Mann-Whitney test, * p<0.05, *** p<0.001).

ACO, Acyl CoA oxidase; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; N-HDL-C, non-HDL-C; TG, triglycerides.
Supplementary Figure 4

Supplementary figure 4: Short-term fenofibrate treatment after long-time Western diet increases hepatic ACO gene expression and improves diet induced-dyslipidaemia in apoE2-KI mice.

A: ACO mRNA level in the liver.

B: Plasma lipid concentrations.

Statistically significant differences are indicated (Mann & Whitney test, *** p<0.001).

ACO, Acyl CoA oxidase; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; N-HDL-C, non-HDL-C; TG, triglycerides.
Supplementary figure 5: Short-term fenofibrate treatment increases hepatic ACO gene expression and improves diet induced-dyslipidaemia in apoE2-KI mice.

A: ACO mRNA level in the liver.
B: Plasma lipid concentrations.

Statistically significant differences are indicated (Mann&Whitney test, ** p<0.01, *** p<0.001).

ACO, Acyl CoA oxidase; TC, total cholesterol; HDL-C, high-density lipoprotein cholesterol; N-HDL-C, non-HDL-C; TG, triglycerides.