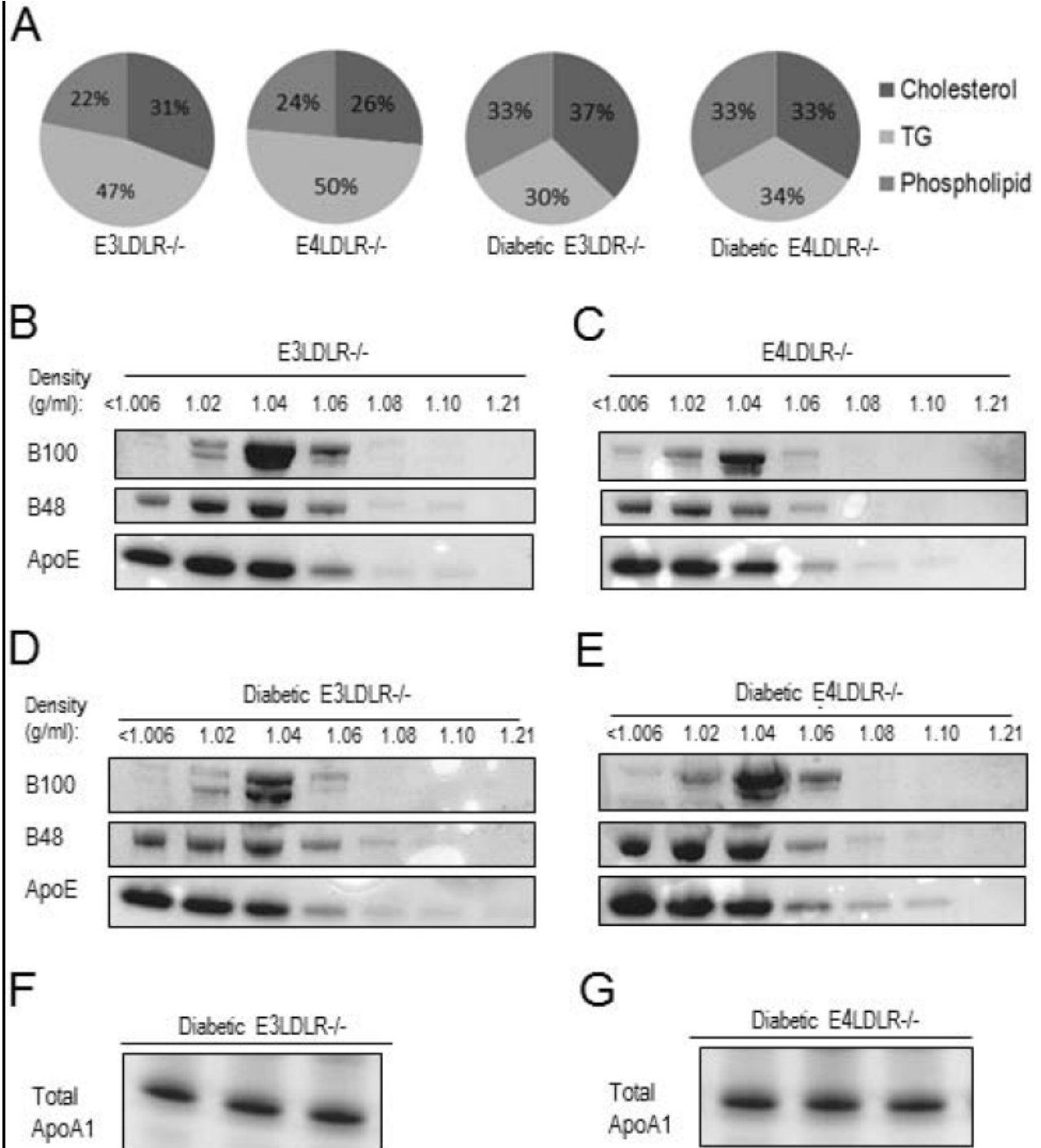
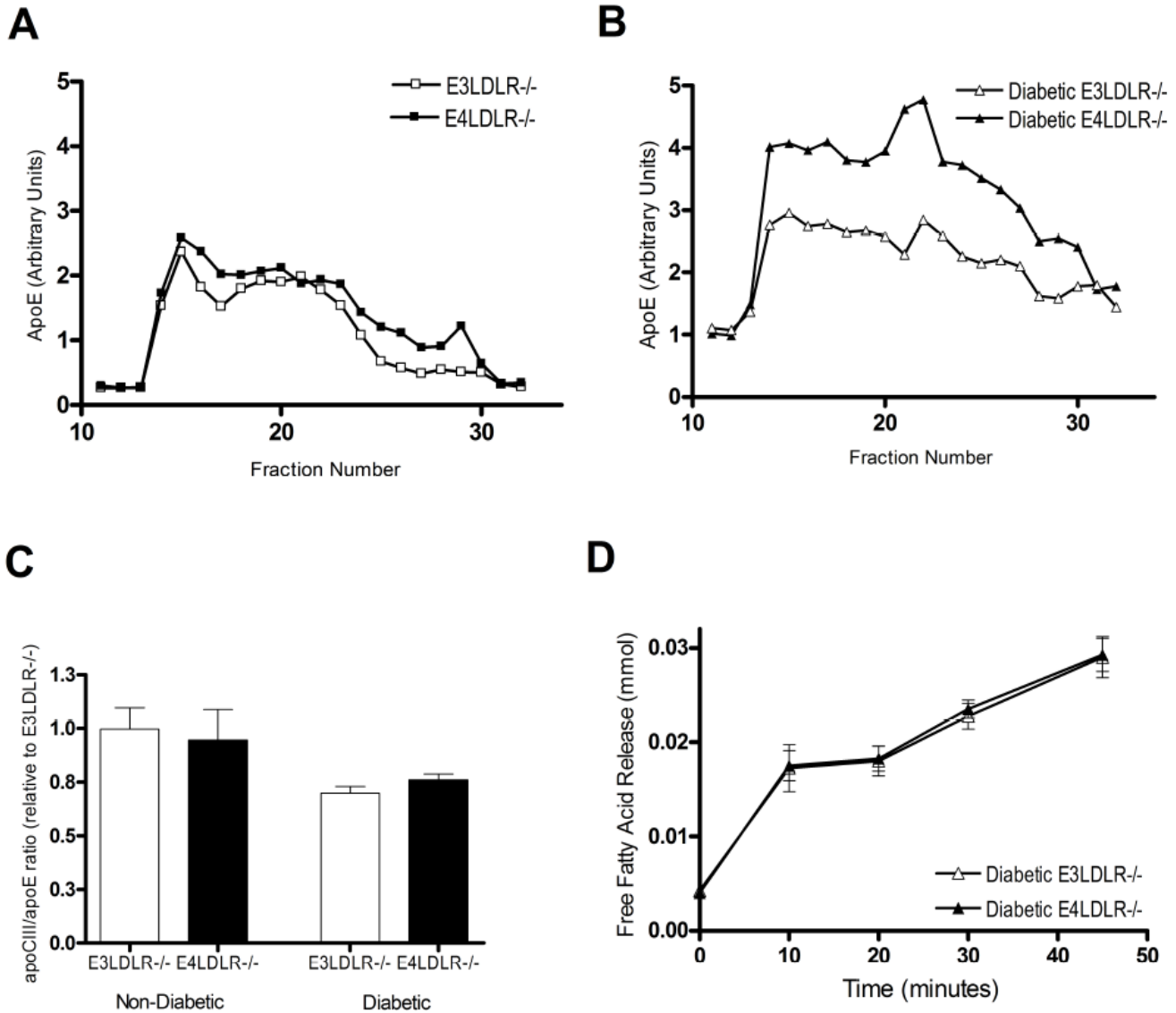


Supplementary Figure 1. Lipoprotein lipid composition and apolipoprotein distribution.

A: Composition of cholesterol, triglycerides, and phospholipids in VLDL isolated by ultracentrifugation of plasma from non-diabetic and diabetic E3LDLR^{-/-} and E4LDLR^{-/-} mice. B-E: Lipoprotein were separated by sequential ultracentrifugation from 800 I pooled (n = 6) plasma into density fractions indicated. Equal volume of fractions were loaded onto 4-20% SDS-PAGE. F-G: Total plasma ApoA1 from individual diabetic E3LDLR^{-/-} (F) and diabetic E4LDLR^{-/-} (G) mice was estimated by loading all lipoprotein fractions (density <1.21 g/ml) onto 4-20% SDS-PAGE.

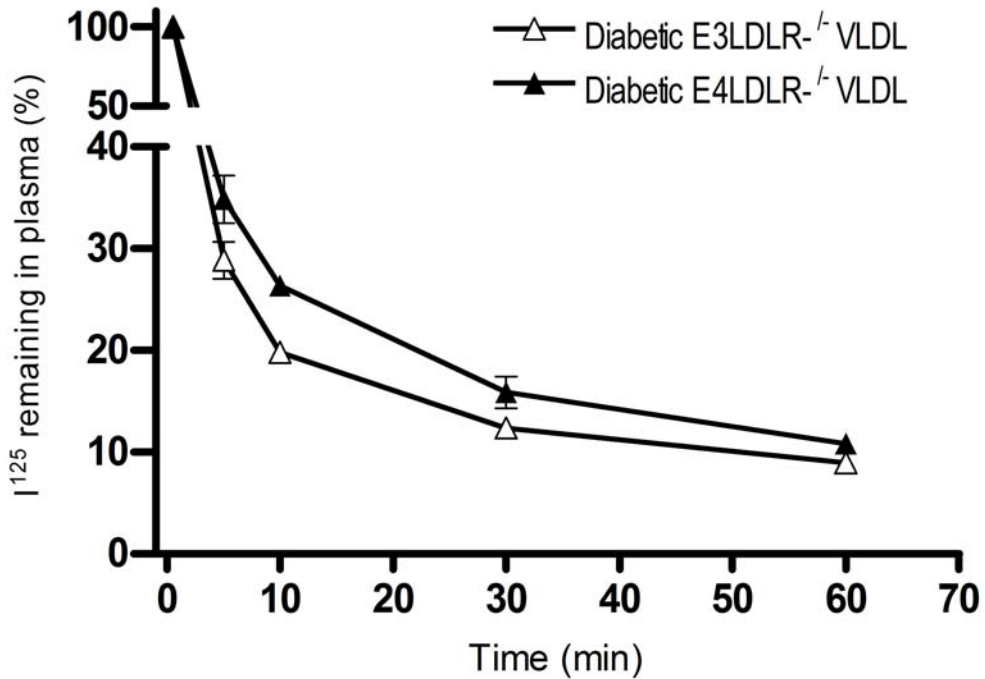


Supplementary Figure 2. Apolipoprotein E distribution, ApoCIII/ApoE ratio and VLDL lipolysis. A-B: ApoE protein was determined by ELISA in lipoprotein fractions separated by FPLC from the pooled plasma (n = 6) of diabetic E3LDLR^{-/-} (A) and diabetic E4LDLR^{-/-} (B) mice. C: Plasma Apolipoprotein CIII and Apolipoprotein E protein was measured by ELISA. n = 4-8. Data is expressed as the ratio of ApoCIII/ApoE relative to the mean of non-diabetic E3LDLR^{-/-} mice as 1.0. D: VLDL was isolated from diabetic E3LDLR^{-/-} (white triangles) or diabetic E4LDLR^{-/-} (black triangles) mice and incubated at 37°C with bovine lipoprotein lipase. The reaction was stopped at timepoints indicated by adding 5M NaCl, and FFA release (FA_{timepoint} - FA₀) was measured. n = 4-6.

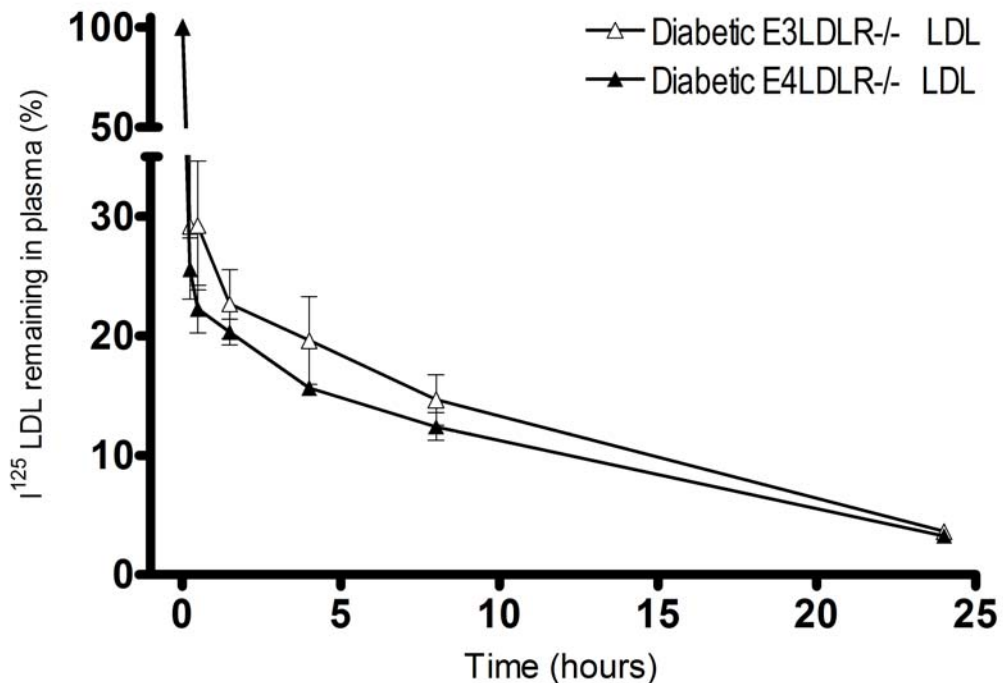


Supplementary Figure 3. Lipoprotein clearance. A-B: VLDL (A) or LDL (B) isolated from diabetic E3LDLR^{-/-} or diabetic E4LDLR^{-/-} mice was labeled with ¹²⁵I and re-injected via tail vein into non-diabetic LDLR^{-/-} recipients. Plasma was collected at the listed timepoints and measured for radioactivity. All values are normalized to values 2 minutes post-injection.

A

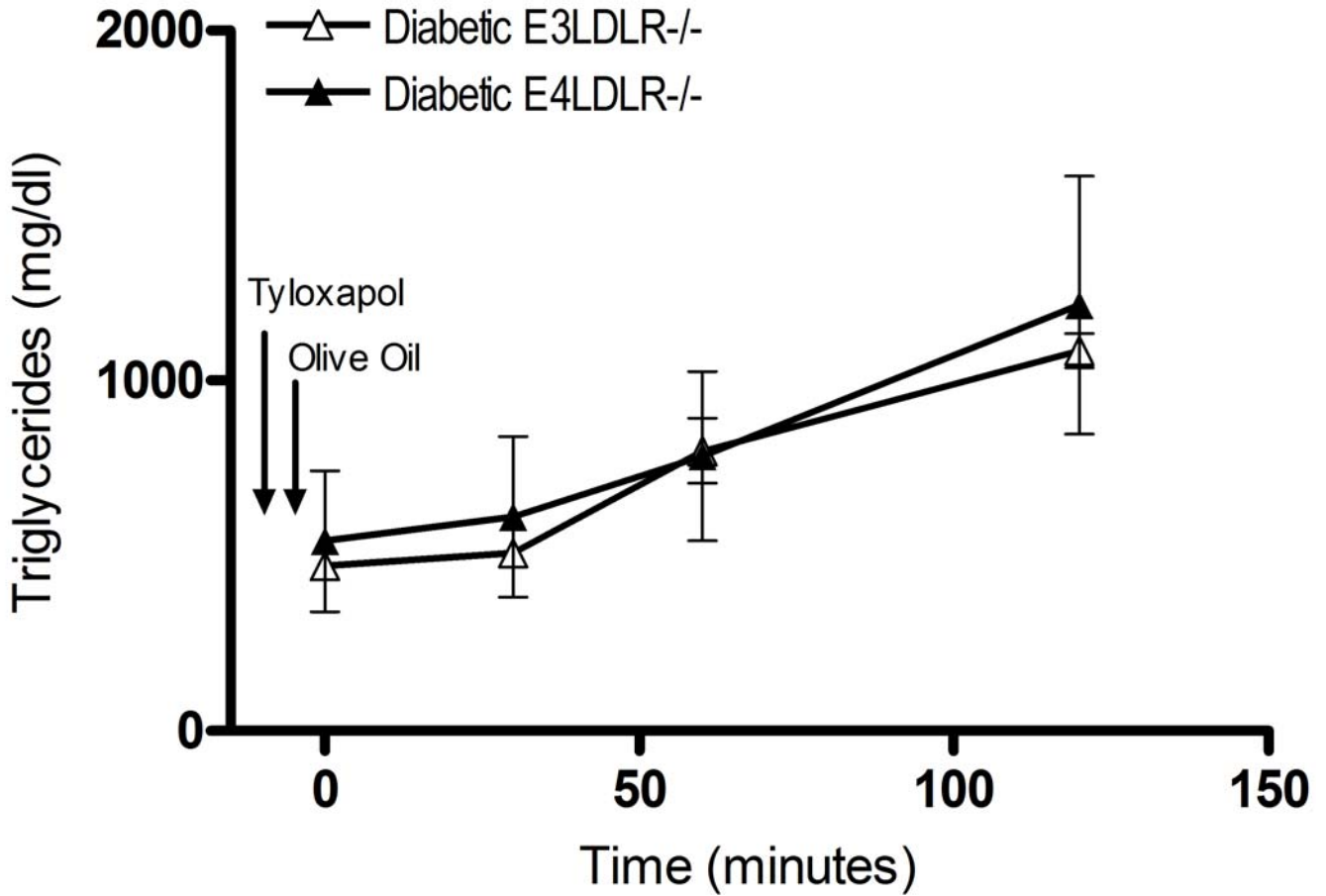


B



SUPPLEMENTARY DATA

Supplementary Figure 4. Dietary lipid absorption. Diabetic E3LDLR^{-/-} (white triangles) and diabetic E4LDLR^{-/-} (black triangles) mice were given an oral gavage of olive oil (200 μ l) 5 minutes after tail vein injection of Tyloxapol (0.7 mg/g BW). Plasma triglycerides were measured 0, 30, 60, and 120 minutes post-gavage. n = 3-4.



SUPPLEMENTARY DATA

Supplementary Figure 5. Apoptosis and macrophage infiltration in the atherosclerotic plaque. A-B: Macrophages were detected using MOMA-2 antibodies followed by a secondary conjugated to Cy5. Plaques from diabetic E3LDLR^{-/-} (A) and diabetic E4LDLR^{-/-} (B) mice are shown in Brightfield (upper image) and Cy5 (blue, lower image). Magnifications are 20X. C-D: DNA fragmentation was detected in 8 μm frozen sections of the aortic root using ApopTag Fluorescein *in situ* staining. Pictured are plaques bordering the aortic valves (V-shaped structures at the center of the image) of diabetic E3LDLR^{-/-} (C) and diabetic E4LDLR^{-/-} (D) mice. A 1% methyl-green solution was used to delineate ultrastructure. Magnifications are 30x.

