SUPPLEMENTAL MATERIAL



Supplemental Figure 1: Lipid Profiles in ApoE^{-/-} Mice Undergoing Regression or Arrested Progression. Serum was collected from apoE^{-/-} mice after 12 weeks on Western diet (Before BMT) and from wildtype (WT), MΦLRP1^{-/-}, apoE^{-/-}, and apoE^{-/-}/MΦLRP1^{-/-} (DKO) recipient mice following 10 weeks on chow diet. Pooled serum subjected to FPLC as described in Materials and Methods. Area under the curve (AUC) analysis was performed for the determination of (A) VLDL, (B) IDL/LDL, and (C) HDL. Each data point reflects pooled serum from 2-3 mice. Kruskal-Wallis test with Dunn's post hoc analysis was used to determine differences among genotypes. **p<0.01, *p<0.05.



Supplemental Figure 2: Necrotic Core Size in Plaques During Halted Progression or Regression. (A) Sections of aortic sinus from baseline, WT and MΦLRP1^{-/-} recipient mice (n=6/group) were immunofluorescently stained for DAPI (blue), TUNEL (green) and Mac-2 (red). (B) Lesion apoptosis was determined via measuring total TUNEL area (mm2) and (C) Lesion efferocytosis was determined by TUNEL area within lesion Mac-2. Sections of aortic sinus from mice undergoing (D) arrested progression or (E) regression were stained with hematoxylin and eosin and the necrotic core area was quantified as area lacking cellularity. Kruskal-Wallis test with Dunn's post hoc analysis was performed to determine differences among genotypes.



Supplemental Figure 3: Analysis of Atherosclerosis in Arrested Progression. Atherosclerosis was developed in apoE^{-/-} mice as described in Materials and Methods. After lethal irradiation, mice underwent BMT from apoE^{-/-} or apoE^{-/-}/MΦLRP1^{-/-} (DKO) mice and switched to chow diet for 10 weeks. ApoE^{-/-} recipient mice were sacrificed after 2 weeks on chow diet and used as baseline. (A) Total serum cholesterol and (B) total serum triglycerides

from $apoE^{-/-}$ on Western diet for 12 weeks (Before BMT) and from $apoE^{-/-}$ and DKO recipient mice after 10 weeks on chow diet (mg/dL). (C) and (D) Cross-sections of aortic sinus were obtained and plaque area determined by Oil Red O staining (x10³ μ m²). One-way ANOVA with Bonferroni's post hoc tests were used to compare effects across genotypes.



Supplemental Figure 4: Plaque Characterization in Murine Model of Arrested **Progression.** Plaques from apoE^{-/-} and apoE^{-/-}/MΦLRP1^{-/-} (DKO) recipient mice were stained with DAPI (blue), CD68 (green) and arginase (red) for visualizing macrophages as described in Materials and Methods. Quantification of M2 and M1 macrophages reported as percent of total CD68⁺ cells. (A) and (D) M2 macrophages were identified by colocalization of Arg1⁺ and CD68⁺ staining. Scale bar = 100µm. (B) and (E) M1 macrophages were identified as Arg2⁺ and CD68⁺. (C) Quantification of CD68⁺ macrophage cell numbers reported as cells per section. Kruskal-Wallis test with Dunn's post hoc analysis was performed to determine differences among genotypes. (F) The ratio of M2 (Arg1⁺) to M1 (Arg2⁺) macrophages in plaques. Kruskal-Wallis test with Dunn's post hoc analysis was performed to determine differences among genotypes. One-way ANOVA with Bonferroni's post hoc test was used for comparison among all genotypes unless stated otherwise.



Supplemental Figure 5: Edu Labeled CCR7 Cells in Lymph Nodes. (A) Sequential gating (G1-G3) for single live cells. (B) Edu singled labeled cells (Pacific blue) to determine the gating for Edu positive cells. (C) PE single stained cells to determine the gating for CCR7 positive cells. (D) Unstained control cells. (E) Gating (G4) for Edu positive cells from lymph nodes. (F) Gating (G5) for pacific blue-Edu and PE-positive cells (Q2) mediastinal lymph nodes. (G) Pacific

blue-Edu and PE-positive cells (Q2) were barely detectable in superficial cervical lymph nodes, which were used as negative control.