Supporting Information

Chen et al. 10.1073/pnas.1401797111

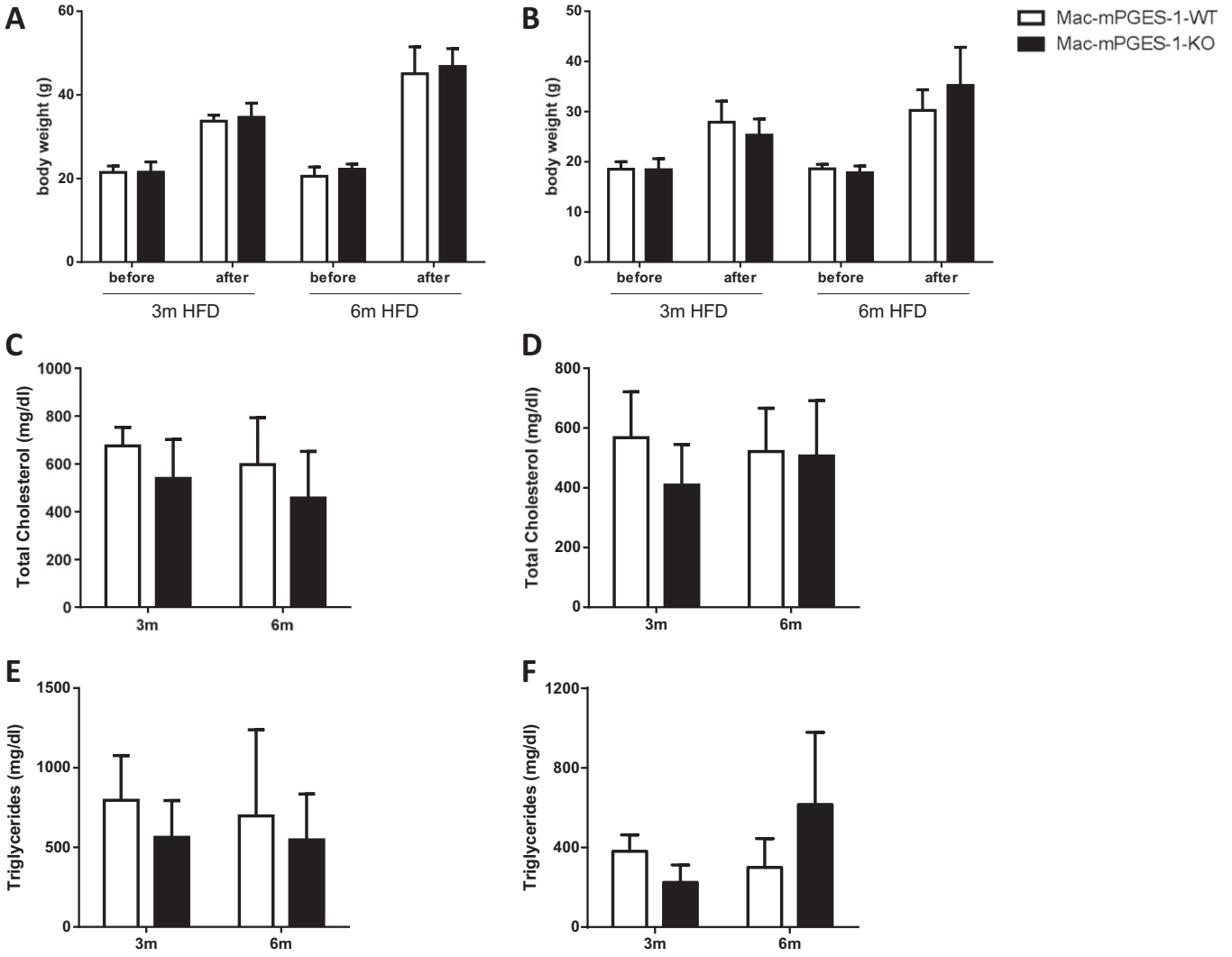

Fig. S1. Effects of myeloid cell microsomal prostaglandin E synthase 1 (mPGES-1) deletion on body weight and plasma lipid profile in hyperlipidemic mice. Body weight, plasma total cholesterol, and triglycerides were measured in Mac-mPGES-1-WT and Mac-mPGES-1-KO mice before and after 3 mo or 6 mo high-fat diet (HFD) feeding. No difference was detected between genotypes for either male or female mice. (*A*) Body weight, males; (*B*) body weight, females; (*C*) plasma total cholesterol, males; (*D*) plasma total cholesterol, females; (*E*) plasma triglycerides, males; (*F*) plasma triglycerides, females (n = 8–11 for body weight and n = 6–8 for plasma total cholesterol and triglycerides).

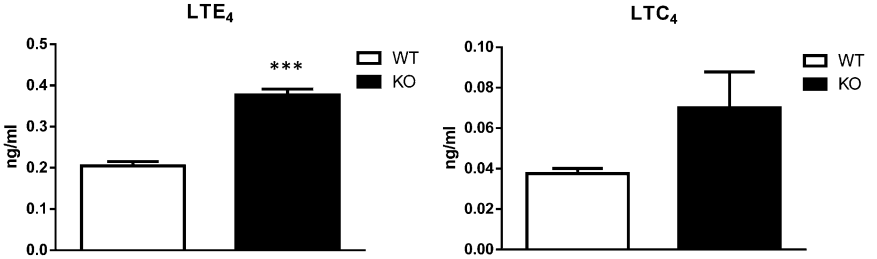

Fig. S2. Leukotriene profile in peritoneal macrophages. Peritoneal macrophages from LysMCre (WT) or LysMCre, mPGES-1$^{f/f}$ (KO) mice were cultured and stimulated with lipopolysaccharide (LPS) (5 μg/mL) for 24 h. Leukotriene (LT)E_4 and LTC_4 were quantified by mass spectrometry in the culture medium; LTB_4 and LTD_4 were under the determination limit (n = 4; ***P < 0.001).

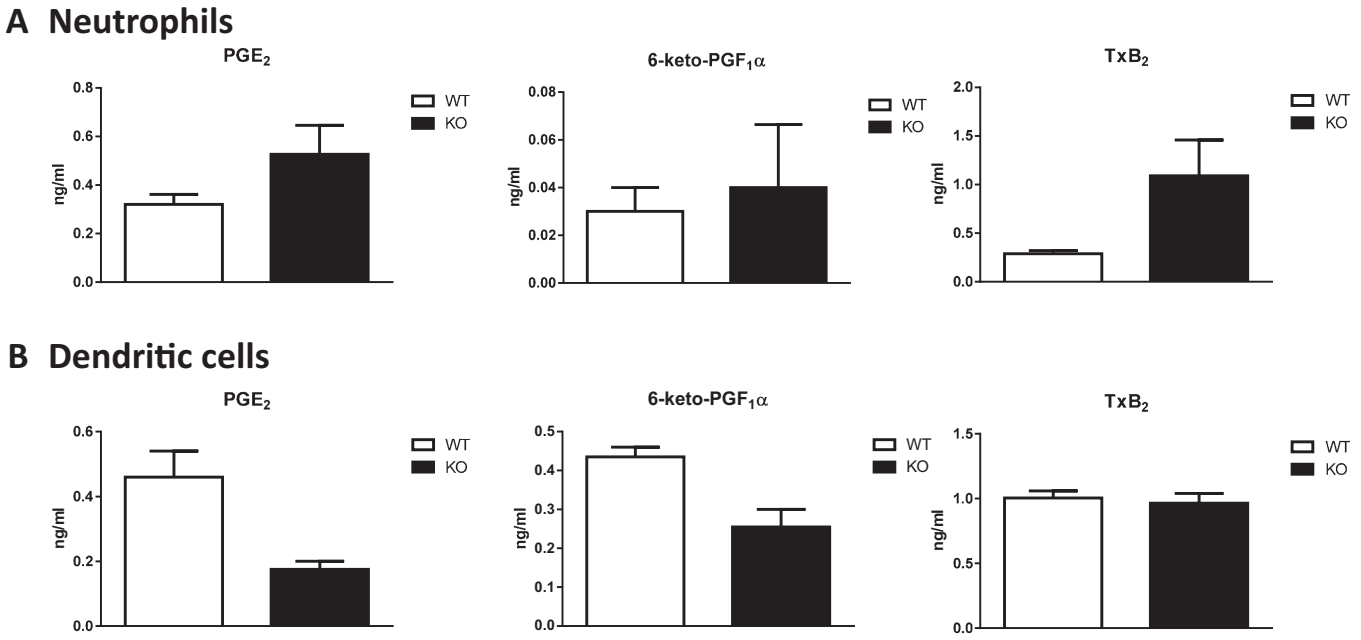

Fig. S3. (*A* and *B*) Prostanoid and leukotriene profiles in peritoneal neutrophils (*A*) and dendritic cells (*B*). Peritoneal neutrophils and splenic dendritic cells from LysMCre (WT) or LysMCre, mPGES-1$^{f/f}$ (KO) mice were cultured and stimulated with LPS (5 μg/mL) for 24 h and prostaglandin (PG)E_2, 6-keto-$PGF_{1\alpha}$, and thromboxane (Tx)B_2 were quantified by mass spectrometry in the culture medium. Leukotrienes were under the determination limit ($n = 3$ for neutrophils and $n = 2$ for dentritic cells).

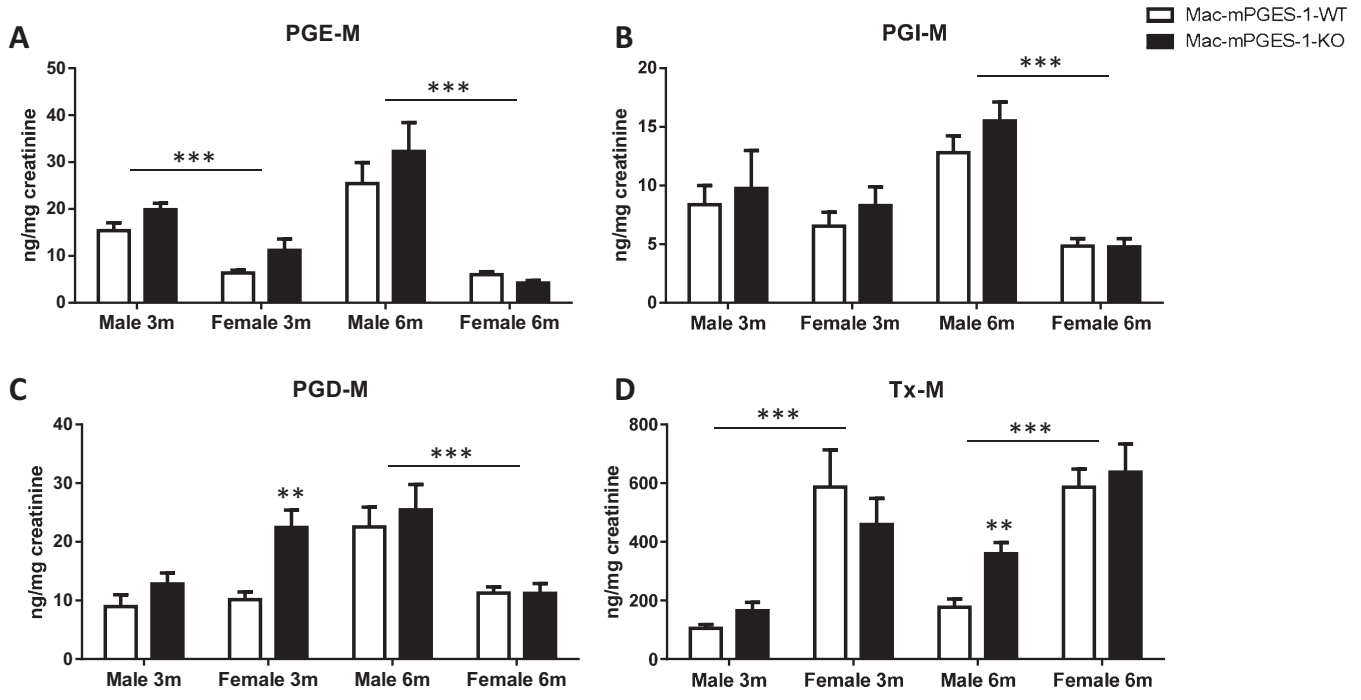

Fig. S4. Effects of myeloid cell mPGES-1 deletion on urinary prostanoid metabolites in hyperlipidemic mice. (*A*–*D*) PGE-M (*A*), PGI-M (*B*), PGD-M (*C*), and Tx-M (*D*) were examined in Mac-mPGES-1-WT and Mac-mPGES-1-KO mice after 3 mo or 6 mo HFD feeding. No difference was detected between genotypes for either male or female mice ($n = 4$–15, $^{**}P < 0.01$, $^{***}P < 0.001$).

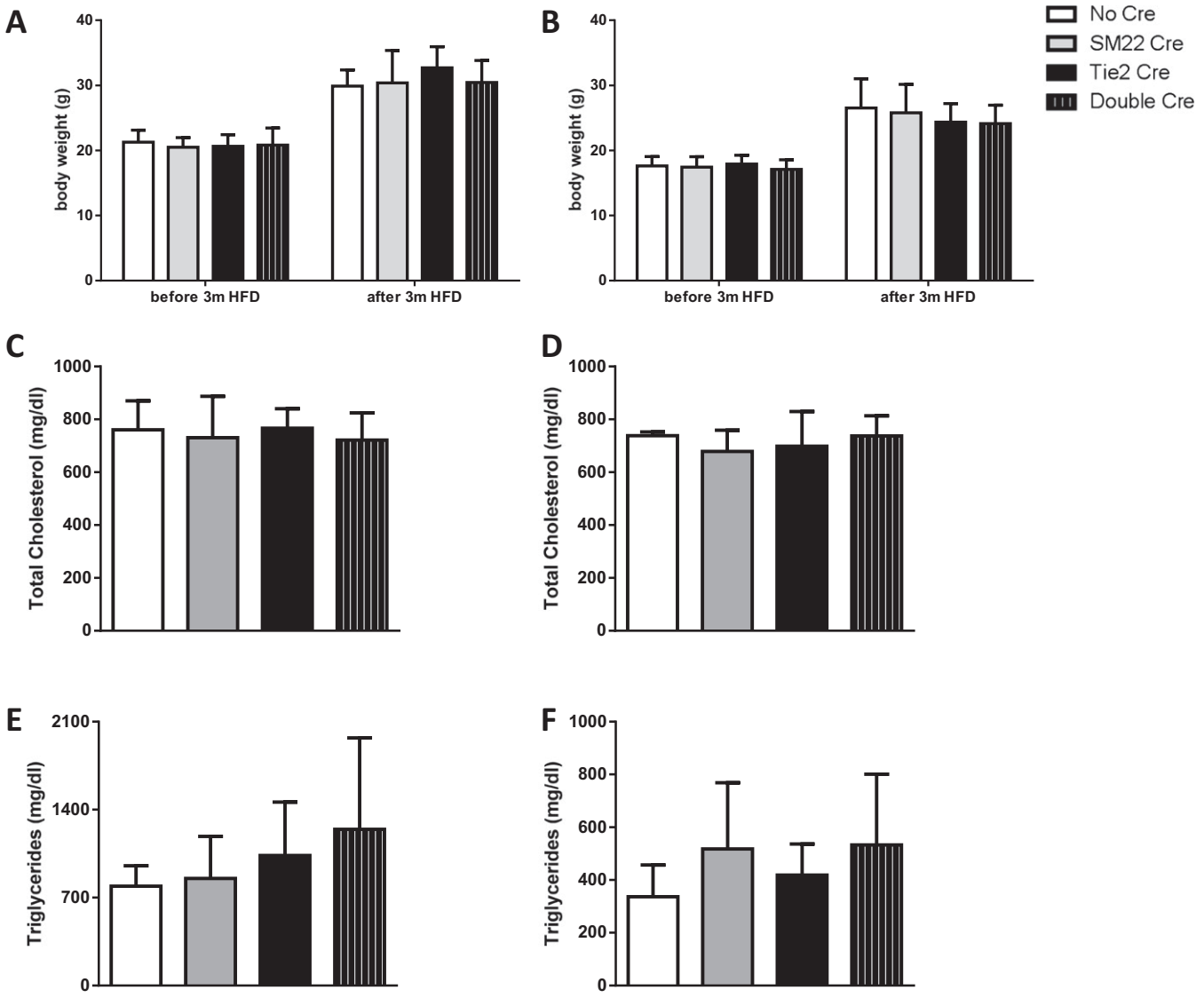

Fig. S5. Effects of vascular cell mPGES-1 deletion on body weight and plasma lipid profile in hyperlipidemic mice. Body weight, plasma total cholesterol, and triglycerides were measured in vascular cell mPGES-1–deficient mice before and after 3 mo HFD feeding. No difference was detected between genotypes for either male or female mice. (*A*) body weight, males; (*B*) body weight, females; (*C*) plasma total cholesterol, males; (*D*) plasma total cholesterol, females; (*E*) plasma triglycerides, males; (*F*) plasma triglycerides, females (n = 6–12 for body weight and n = 7–9 for plasma total cholesterol and triglycerides).

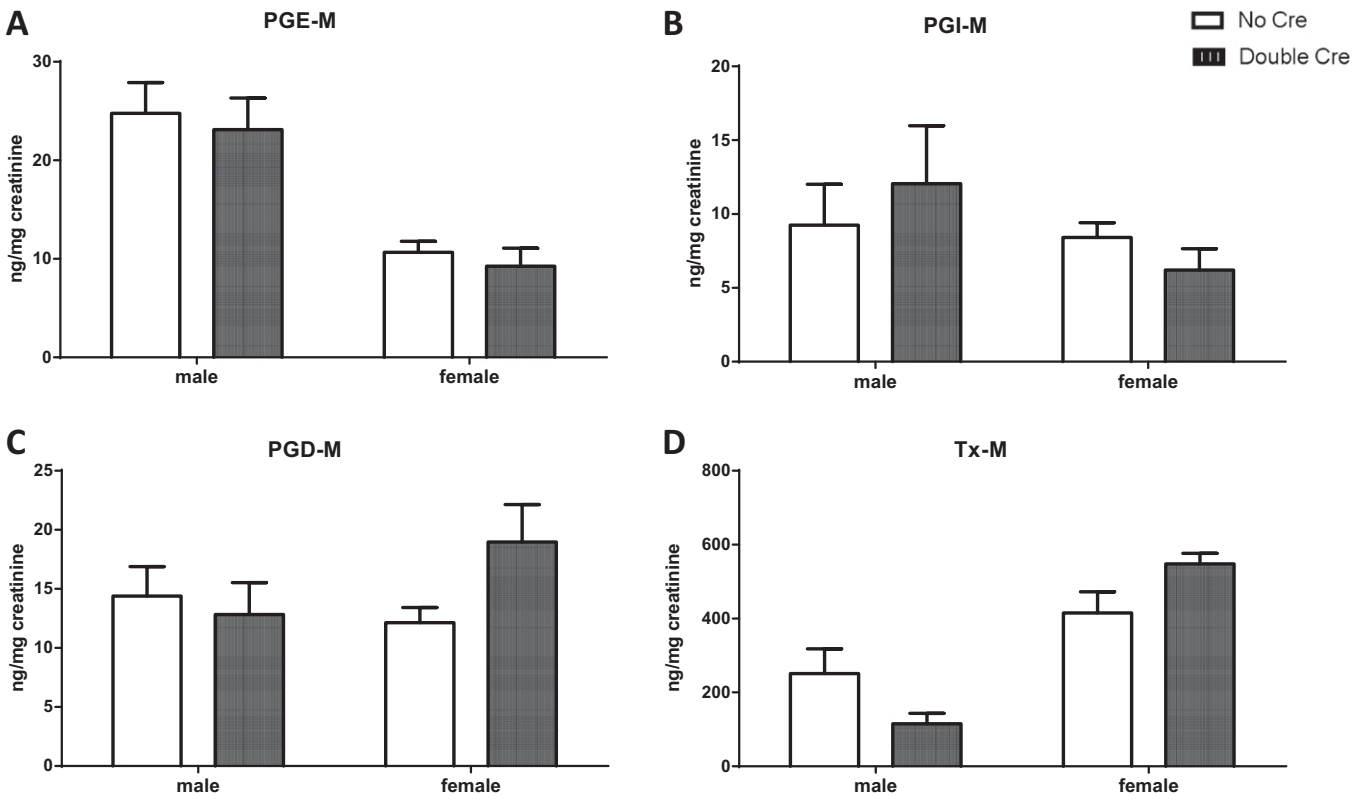

Fig. S6. Effects of vascular cell mPGES-1 deletion on urinary production of prostanoids metabolites in hyperlipidemic mice. (*A*–*D*) PGE-M (*A*), PGI-M (*B*), PGD-M (*C*), and Tx-M (*D*) were examined in vascular cell mPGES-1–deficient mice after 3 mo HFD feeding. No difference was detected between genotypes for either male or female mice (n = 4–8).

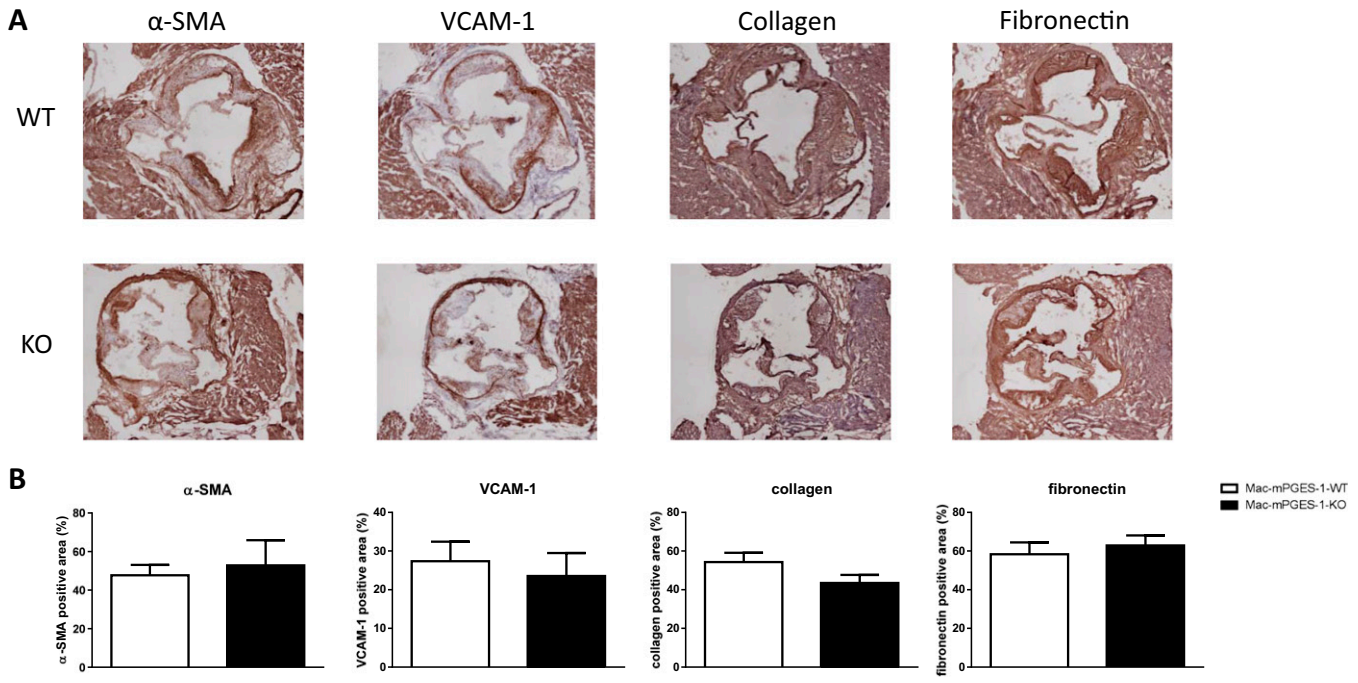

Fig. S7. Lesion morphology analysis in aortic roots of Mac-mPGES-1-WT and Mac-mPGES-1-KO mice on a HFD for 3 mo. (*A*) Representative images for α-SMA, VCAM-1, collagen, and fibronectin staining. (*B*) Quantification of immunohistochemistry staining of α-SMA, VCAM-1, collagen, and fibronectin (n = 4–5).

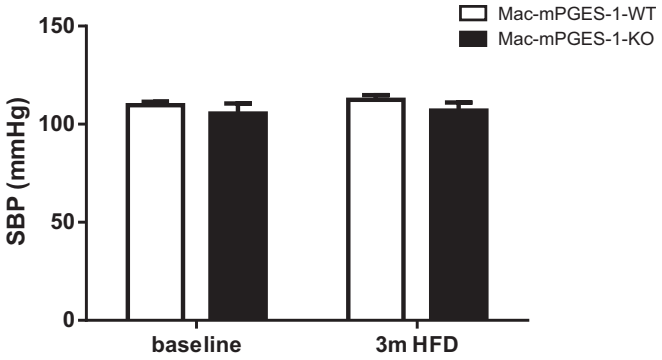

Fig. S8. Effects of myeloid cell mPGES-1 deletion on blood pressure regulation in hyperlipidemic mice. Systolic blood pressure was similarly irrespective of genotype either before or after 3 mo HFD feeding (n = 7–10).

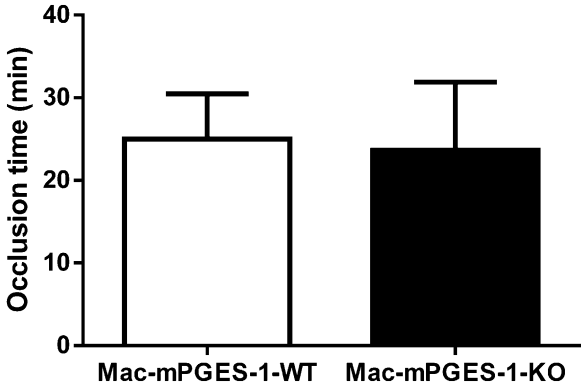

Fig. S9. Effect of myeloid cell mPGES-1 deletion on thrombogenesis in hyperlipidemic mice. The time to thrombotic carotid artery occlusion after photochemical injury was unaltered in myeloid cell mPGES-1–deficient mice after 3 mo HFD feeding (n = 3–4).