

Supplementary figure legends

Figure S1 Synthesis reaction equation of C-TPP.

Figure S2 Basic properties of the fluorescent probe C-TPP-P12. C-TPP-P12 was synthesized by Fmoc-based on solid-phase peptide synthesis. It is green in acidic solution and red in alkaline or neutral solution. The molecular weight is 3091.7314. Maximum excitation wavelengths are 420–450, 530, 560 and 580 nm, and Maximum emission wavelengths are 650 and 720 nm.

Figure S3 CD spectra of L-4F and C-TPP-P12 in the water or 50% trifluoroethanol (TFE).

Figure S4 Net cholesterol efflux rates from 0 to 20 μM of apoA-I₂₂₁₋₂₄₀, P1–P17 and L-4F in RAW264.7 for 12 h.

Figure S5 Effects of different active peptides on cholesterol release in RAW264.7 cells. (a) RAW264.7 treated with the non-toxic or toxic concentration of peptides observed on the optical microscope. At toxic concentrations of peptide, the cells morphologies were changed, and even some cells were broken or differentiation. (b) Different dose-response curves of peptides with different cholesterol efflux activity. The dose-effect curve of P12 was an ideal. From 2 μM to 200 μM , P17 damaged the cell membrane lead to lots of cholesterol leak from RAW264.7, and its dose-effect curve was over the dose-effect curve of P12.

Figure S6 Hemolytic toxicity of the parent peptides apoA-I₂₂₁₋₂₄₀, P1–P17 and L-4F in C57BL/6J mice erythrocytes for 1 h.

Figure S7 Dose-climbing experiment result of P12 in two mice. n=6.

Figure S8 Body weight of the experimental mice in 16 weeks.

Figure S9 Four arterial typical pathological phenotypes of apoE^{-/-} atherosclerotic mice. (1-foam cells, 2-cholesterol crystal, 3-abnormally proliferating VSMCs and endothelium cells and 4-calcified plaque) apoE^{-/-} mice were fed high-fat diets for 4 months.

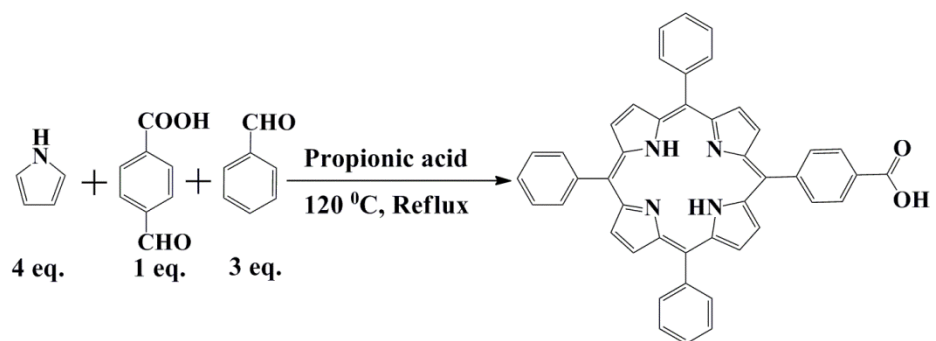


Figure S1 Synthesis reaction equation of C-TPP.

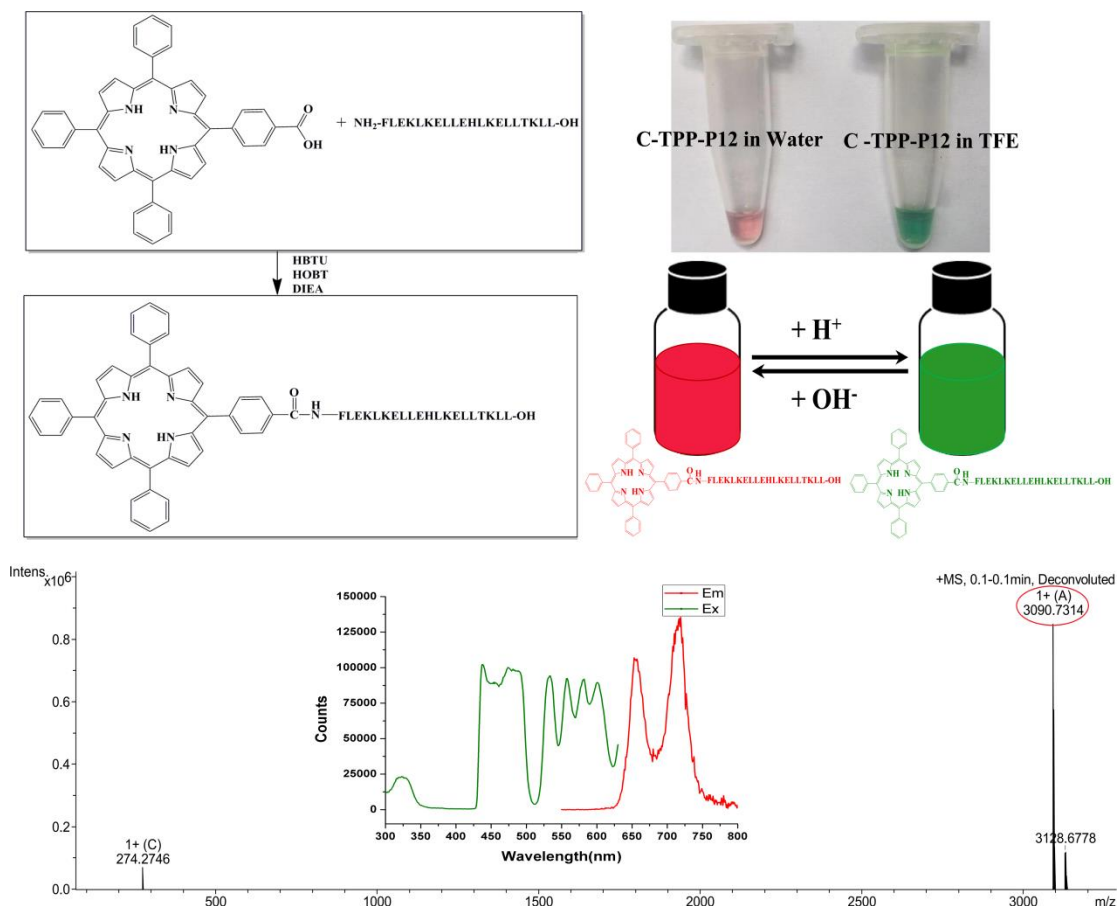


Figure S2 Synthesis and basic properties of the fluorescent probe C-TPP-P12. C-TPP-P12 was synthesized by Fmoc-based on solid-phase peptide synthesis. It is green in acidic solution and red in alkaline or neutral solution. The molecular weight is 3091.7314. Maximum excitation wavelengths are 420–450, 530, 560 and 580 nm, and Maximum emission wavelengths are 650 and 720nm.

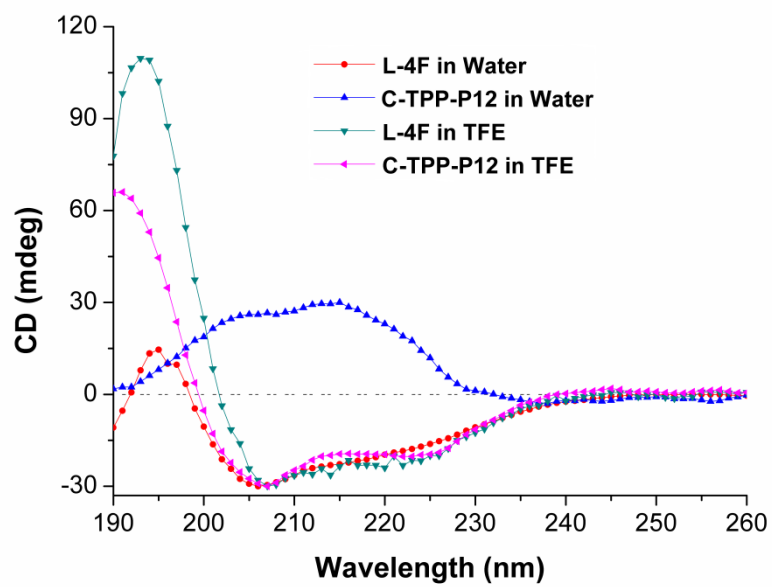


Figure S3 CD spectra of L-4F and C-TPP-P12 in the water or 50% trifluoroethanol (TFE)

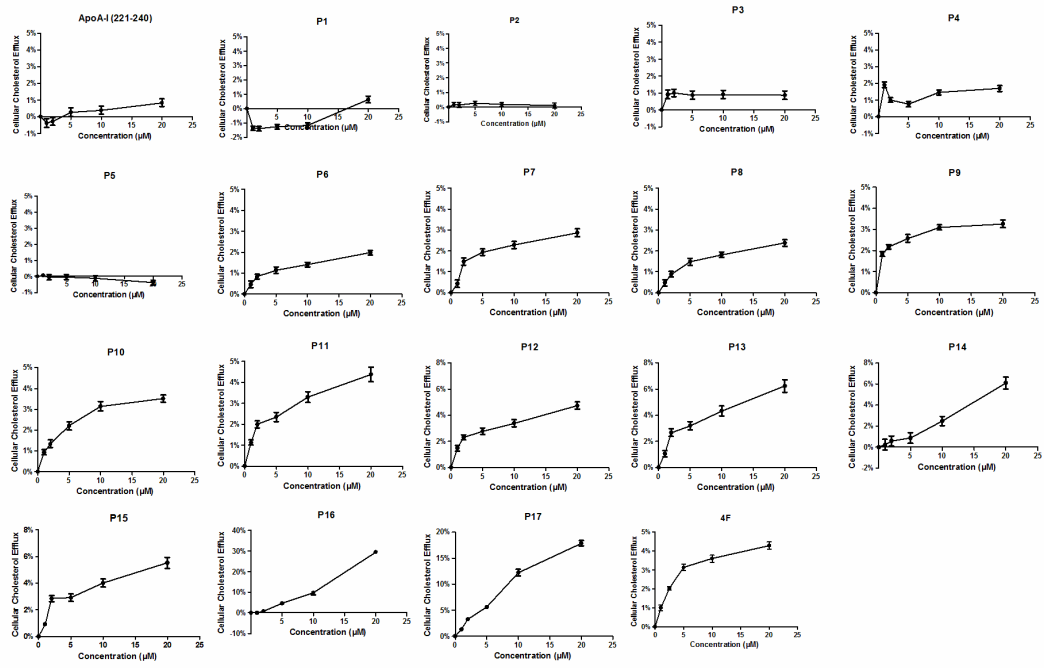
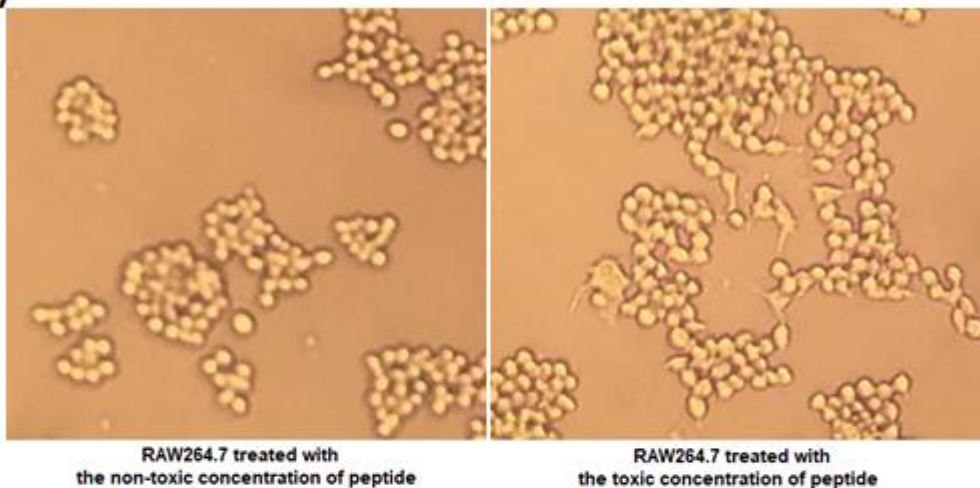


Figure S4 Net cholesterol efflux rates from 0 to 20 μM of apoA-I₂₂₁₋₂₄₀, P1–P17 and L-4F in RAW264.7 for 12 h.

(a)



(b)

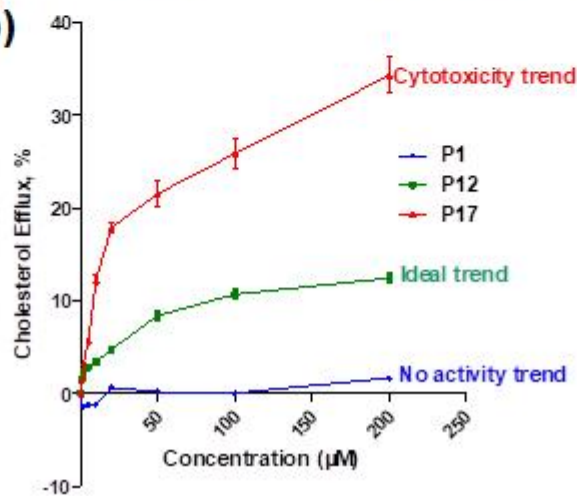


Figure S5 Effects of different active peptides on cholesterol release in RAW264.7 cells. (a) RAW264.7 treated with the non-toxic or toxic concentration of peptides observed on the optical microscope. At toxic concentrations of peptide, the cells morphologies were changed, and even some cells were broken or differentiation. (b) Different dose-response curves of peptides with different cholesterol efflux activity. The dose-effect curve of P12 was an ideal. From 2 μM to 200 μM , P17 damaged the cell membrane lead to lots of cholesterol leak from RAW264.7, and its dose-effect curve was over the dose-effect curve of P12.

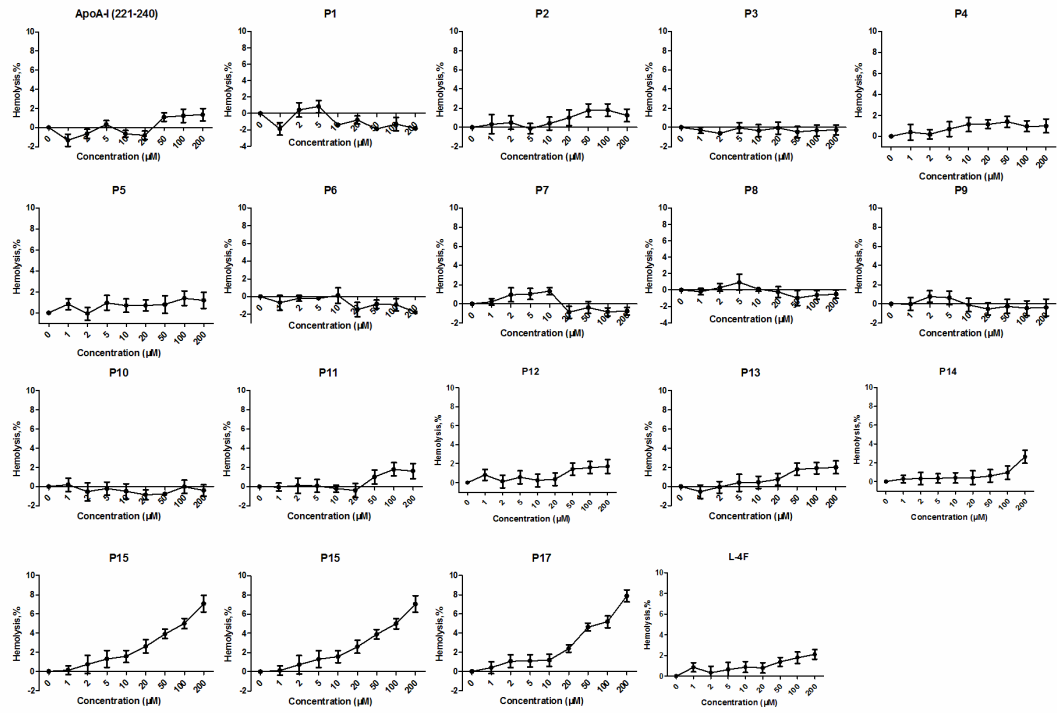


Figure S6 Hemolytic toxicity of the parent peptides apoA-I₂₂₁₋₂₄₀, P1–P17 and L-4F in C57BL/6J mice erythrocytes for 1 h.

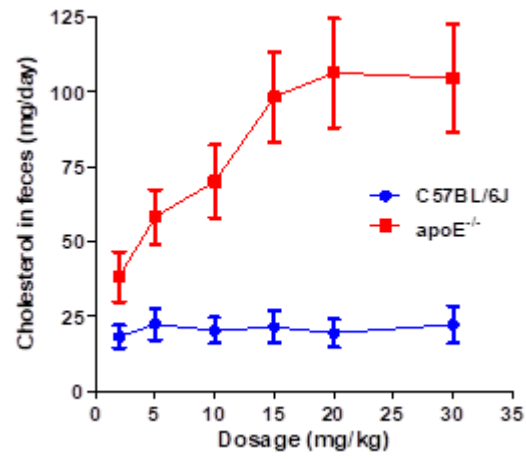


Figure S7 Dose-climbing experiment result of P12 in two mice. n=6.

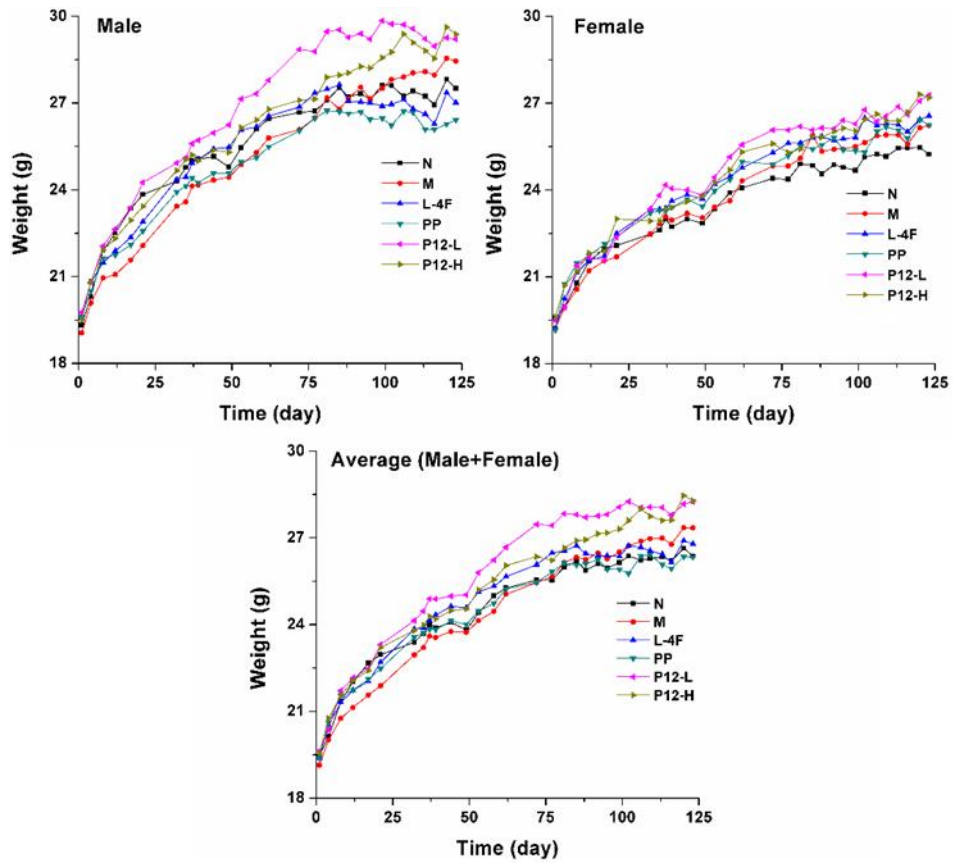


Figure S8 Body weight of the experimental mice in 16 weeks.

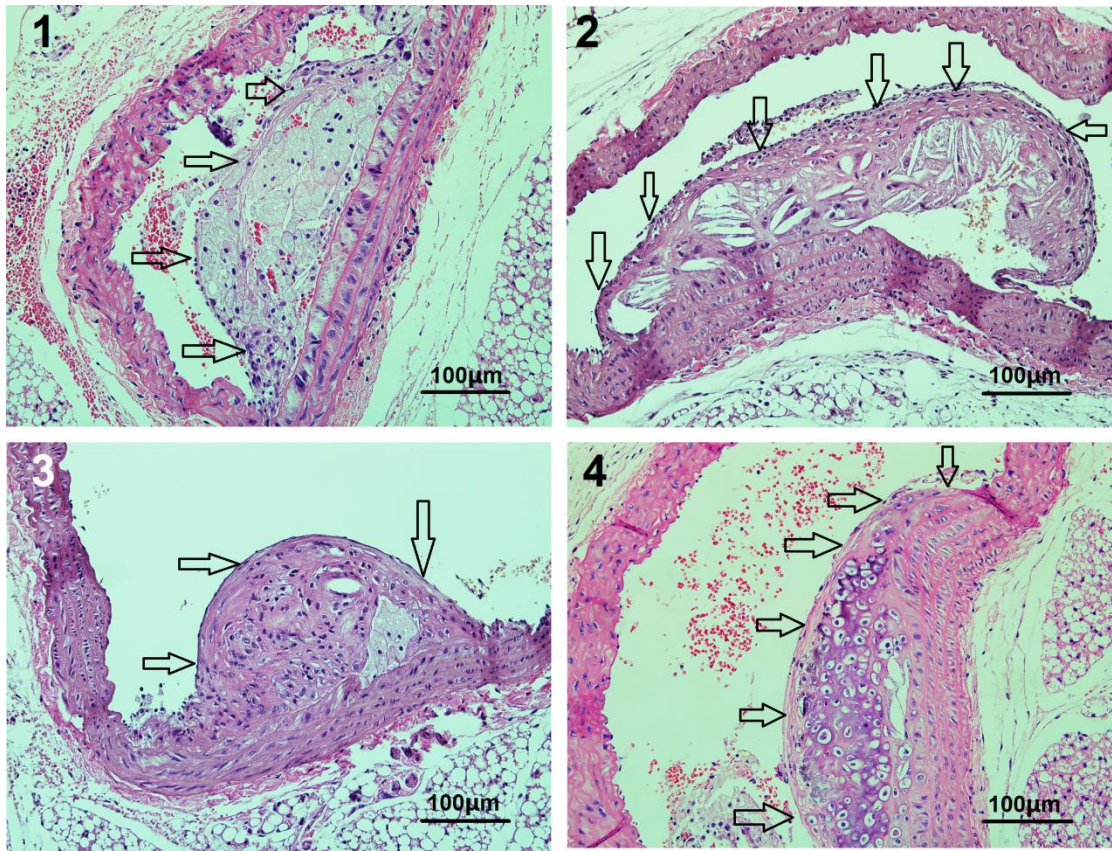


Figure S9 Four arterial typical pathological phenotypes of apoE^{-/-} atherosclerotic mice. (1-foam cells, 2-cholesterol crystal, 3-abnormally proliferating VSMCs and endothelium cells and 4-calcified plaque) apoE^{-/-} mice were fed high-fat diets for 4 months.