Supplemental Material



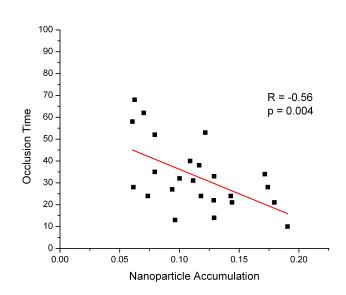
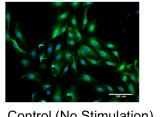
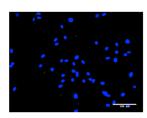


Figure I: Paired samples of aortic nanoparticle accumulation measurements and carotid occlusion times demonstrates a significant inverse correlation between the two metrics, confirming prior work¹ indicating a relationship between increased endothelial permeability and increased vessel hypercoagulability.

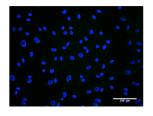
Figure II

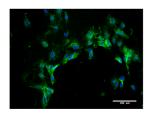


Control (No Stimulation)



Thrombin (1 U/ml)





Thrombin (1 U/ml) + Control NP Thrombin (1 U/ml) + PPACK-NP

Figure II: Immunocytochemistry for IkB on HAECs demonstrates diminished degradation of IkB in response to thrombin stimulation with PPACK-NP treatment, consistent with inhibited activation of NF-kB.



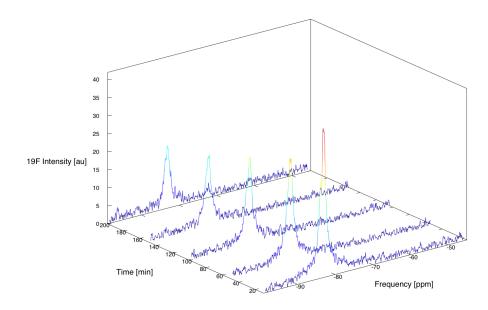


Figure III: Temporal series of ¹⁹F spectra for one mouse at increasing times after a bolus dose of PFC-NP. Signal decay can be monitored through decrease in the ¹⁹F signal intensity and fit to a biexponential model to estimate clearance half-life. Depicted spectra reflect an arbitrary scale for signal strength. Frequencies are given as PPM (parts per million) assuming a 190 MHz spectrometer frequency.

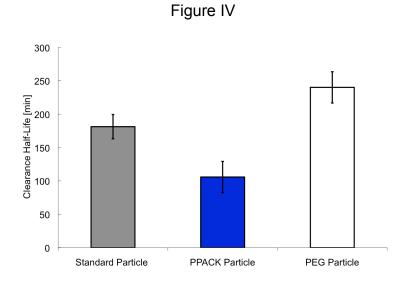


Figure IV: Clearance half-life for selected nanoparticles. ¹⁹F MRS determined clearance half-lives for PPACK-functionalized PFC nanoparticles (105.87±23.38 minutes), PFC nanoparticles with a surface-

conjugated carboxy-PEG spacer (240.16±23.42 minutes), and non-functionalized PFC nanoparticles (181.3±40.7 minutes).

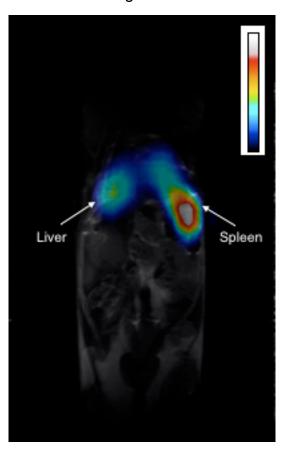




Figure V: ¹⁹F spin echo coronal projection image (false color) overlaid on a proton spin echo coronal slice image (grayscale). The image, demonstrating accumulation of nontargeted PFC-NP in the spleen and liver, was acquired post-mortem in a mouse sacrificed two hours after injection.

Figure VI

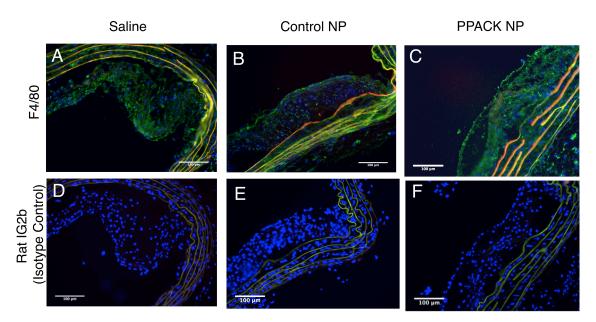


Figure VI: (A-C) Staining of mouse aortic arch sections for the macrophage F4/80 antigen provides secondary confirmation of the presence of macrophage populations in plaques. (D-F) Isotype control staining confirms lack of nonspecific binding of the MOMA-2 antibody (see Fig. 6A-D).

Work Cited

1. Palekar RU, Jallouk AP, Goette MJ, Chen J, Myerson JW, Allen JS, Akk A, Yang L, Tu Y, Miller MJ, Pham CTN, Wickline SA, Pan H. Quantifying progression and regression of thrombotic risk in experimental atherosclerosis. *FASEB J* 2015;29(7):3100–3109. doi:10.1096/fj.14-269084.