Supplementary Figures

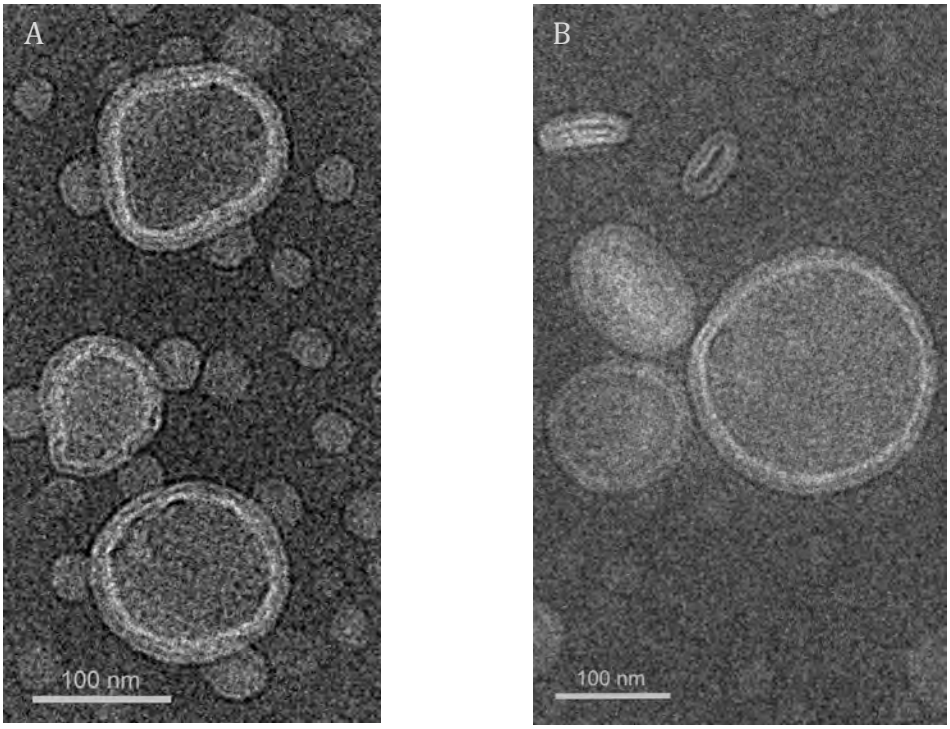

Fig. S1: (A) TEM Image of precursor carboxyl-terminated liposomes. Scale bar denotes 100 nm. (B) TEM Image of PPACK-Liposomes. Scale bar denotes 100 nm. Both samples were negatively stained with the vanadium-based stain NanoVan.

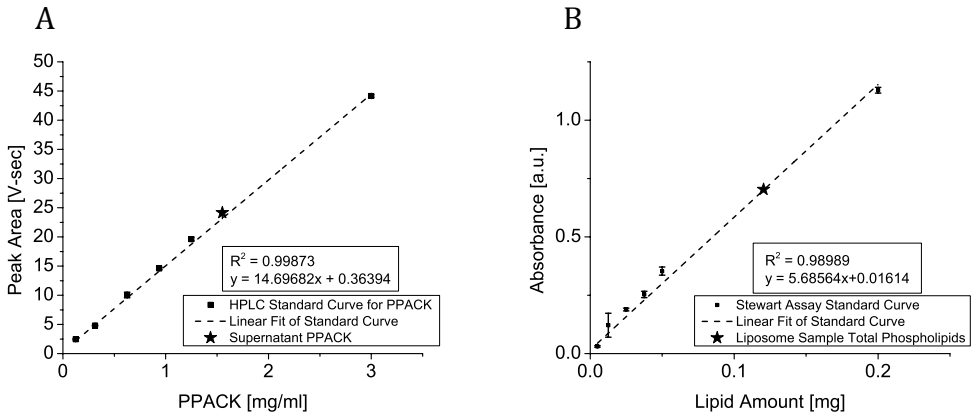

Fig. S2: (A) Standard curve of PPACK quantified by RP-HPLC. The standard curve was used to determine the amount of uncoupled PPACK left over after the conjugation step. (B) Stewart Assay standard curve to quantify the amount of phospholipids in the liposome sample. The Stewart Assay quantification was then used to determine the liposome concentration given the overall size of the liposome measured by dynamic light scattering. Both assays were then used in tandem to determine the number of PPACK molecules per liposome.

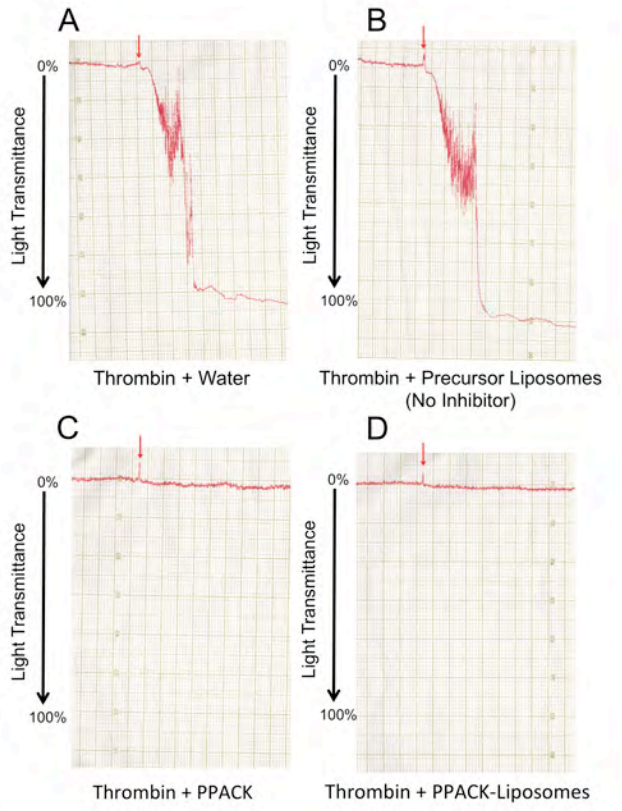

Fig. S3: The effect of PPACK-Liposomes on thrombin-mediated aggregation of human platelets was evaluated using platelet aggregometry, where 100% light transmittance represents 100% aggregation. Platelet-rich plasma (PRP) containing either (A) water, (B) Precursor Liposomes, (C) free PPACK, (D) PPACK-Liposomes was exposed to thrombin and allowed to aggregate. The red arrow indicates the addition of thrombin to the PRP. The presence of PPACK or PPACK-Liposomes in the PRP abolishes thrombin-mediated activation of platelets.

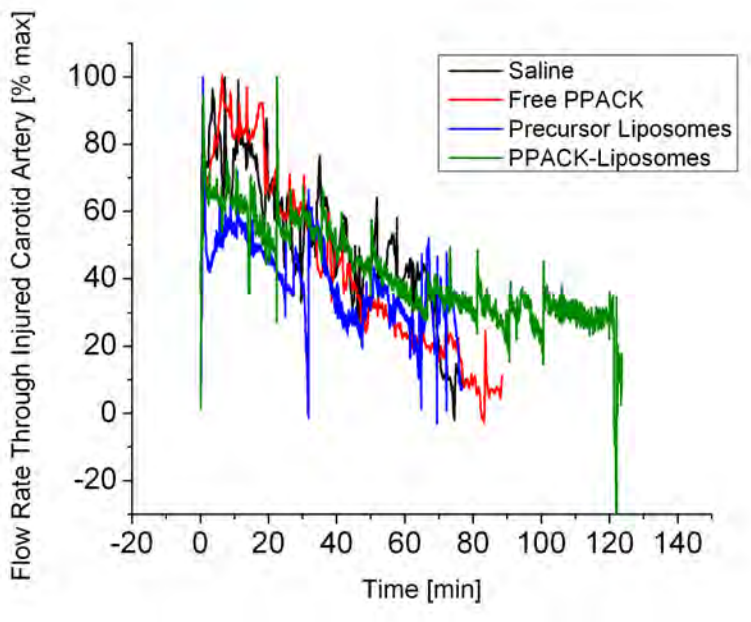

Fig. S4: Representative measurements of flow rate through the injured carotid artery throughout the duration of the in vivo photochemically-induced thrombus formation model. The formation of the thrombus continues over the entire course of the experiment—a process that is gradually delayed with PPACK-Liposome treatment. The time course for thrombus formation was largely unchanged for saline, Free PPACK and Precursor Liposomes.

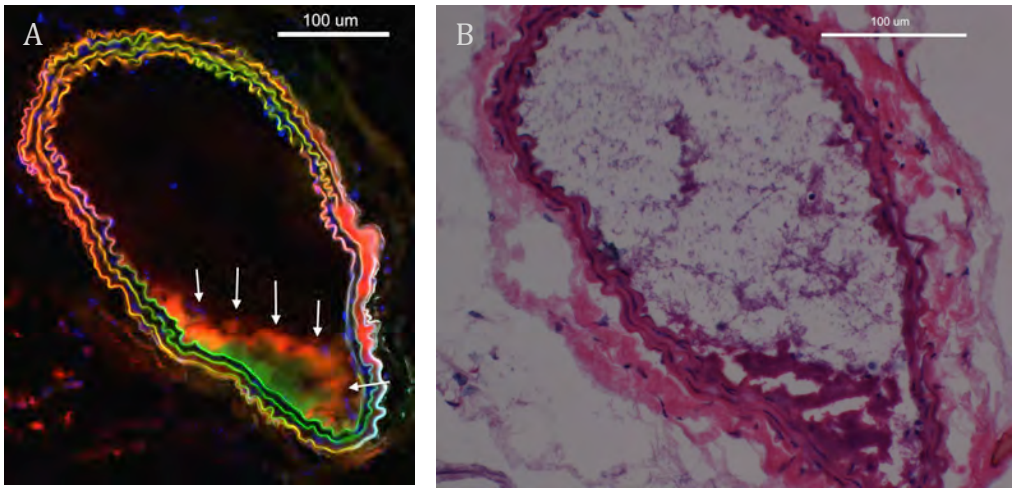

Fig. S5: Sections of carotid artery after partial formation of thrombus. (A) Localization of PPACK-Liposomes (white arrows) at site of initial clot formation. (B) H&E stain of neighboring section showing initial formation of clot and endothelial damage. Scale bars represent 100 µm.

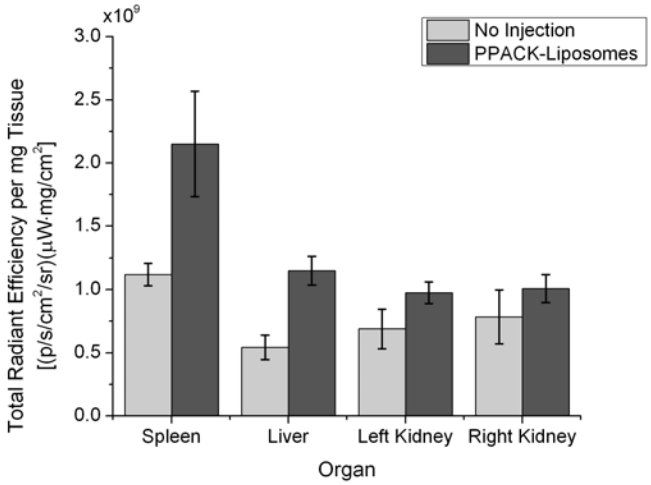

Fig. S6: Biodistribution of PPACK-Liposomes after 2 hours of circulation time. The light grey bars represent baseline organ fluorescence from mice that received no injections. The clearance of PPACK-Liposomes from the circulation appears to be mediated by the liver and spleen.

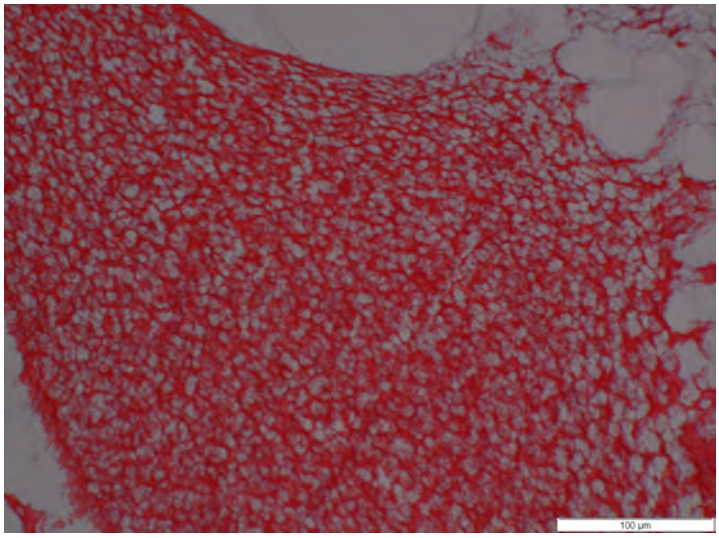

Fig. S7: Carstair's stain of clot formed in vitro for clot growth experiment. In vitro clots are primarily composed of fibrin strands (bright red). Scale bar represents 100 µm.

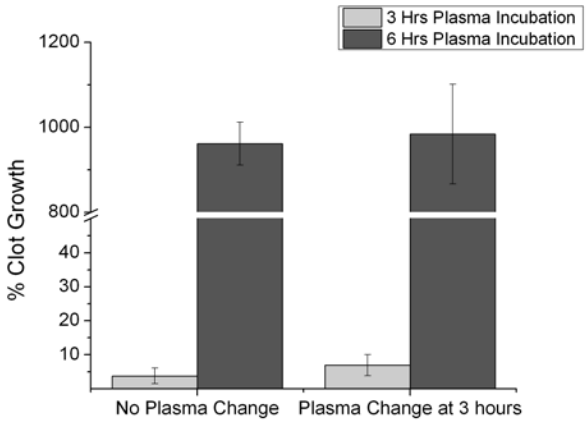

Fig. S8: The in vitro clot growth experiment was extended for PPACK-Liposome treated clots to 6 hours of total plasma incubation time. Clots were weighed after 3 hours and either placed back into the same plasma (N=3) or transferred to a new well with fresh plasma for another 3 hours (N=3).

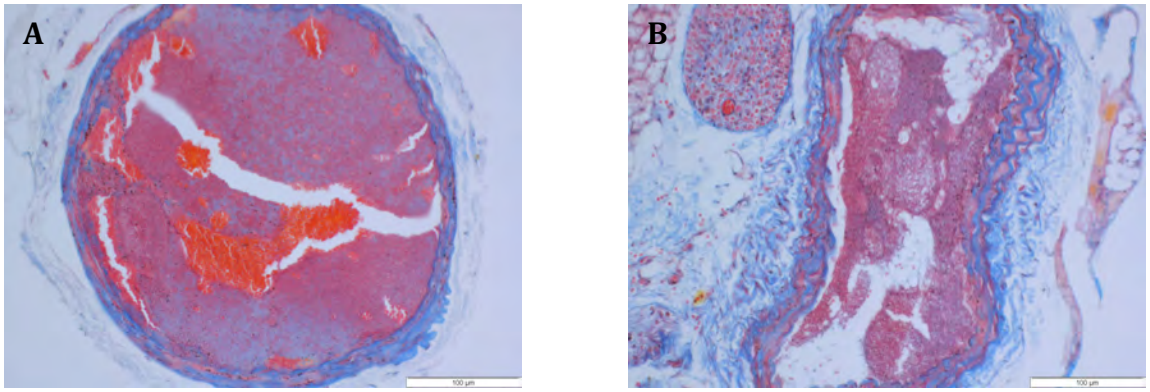

Fig. S9: Carstair's staining of injured carotid arteries given (A) Precursor Liposomes or (B) PPACK-Liposomes. Platelet staining (grey-blue in clot interior) was quantified using the NIH ImageJ software indicating less platelet accumulation in the PPACK-Liposome group. (A) Precursor Liposome treatment resulted in 44.24% of the thrombus area occupied by platelets. (B) PPACK-Liposome treatment resulted in 9.44% of the thrombus area occupied by platelets. Scale bars represent 100 µm.