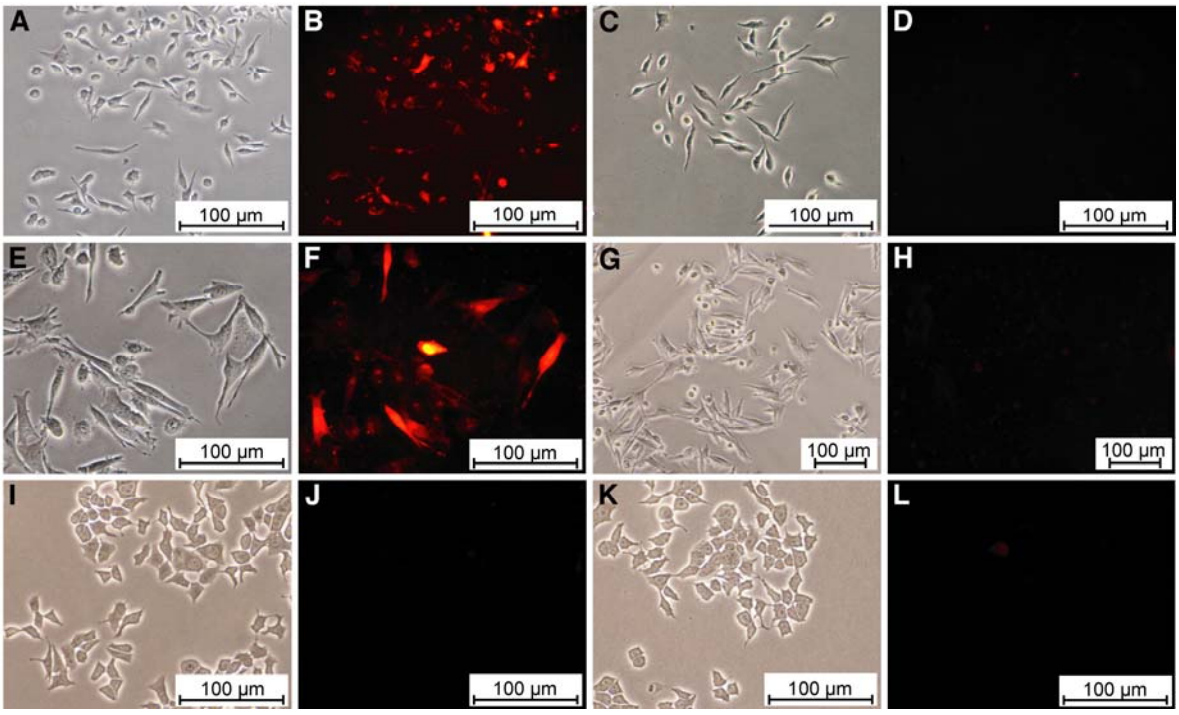
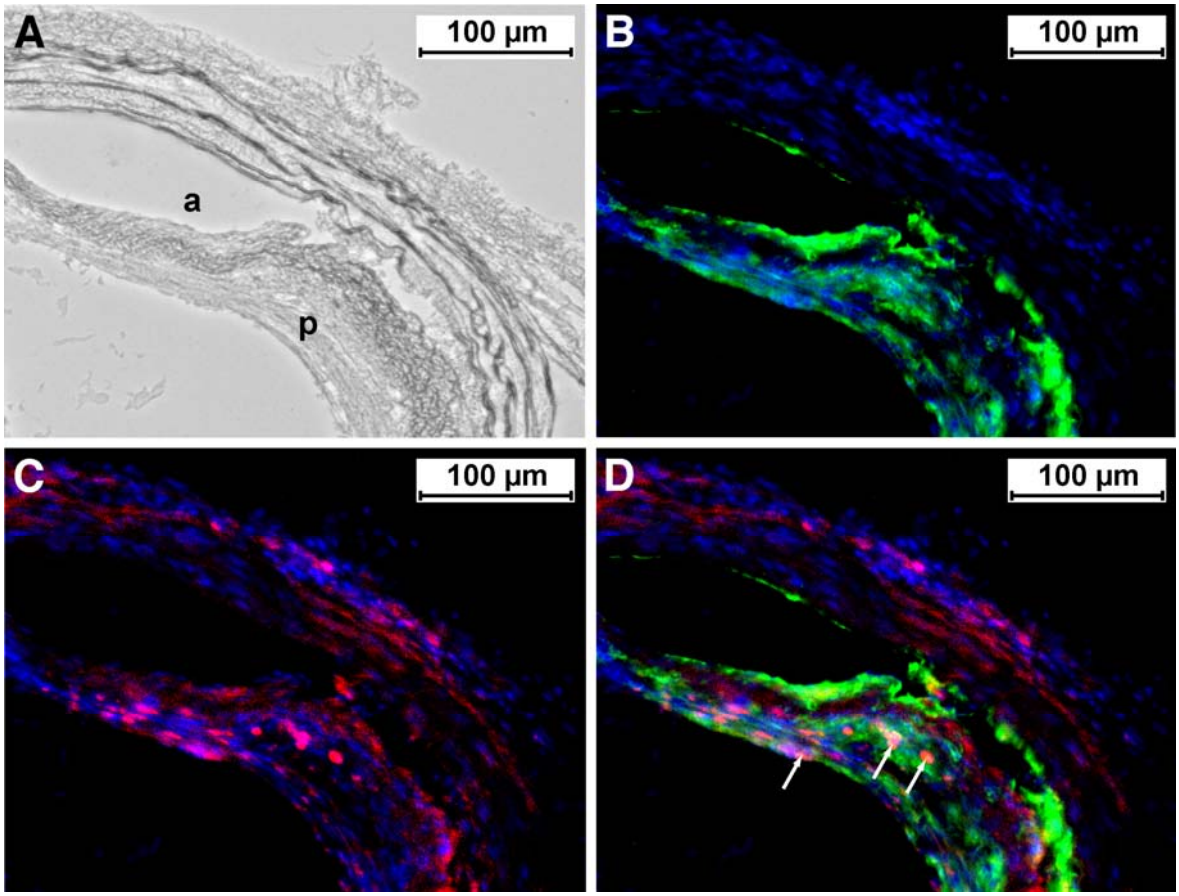


Supplemental Figure 1. Binding affinity of RGD-Cy 5.5 to macrophages and tumor cell lines with different $\alpha_v\beta_3$ expression levels.

Murine macrophages (RAW 264.7; A-D), highly $\alpha_v\beta_3$-positive M21 melanoma (positive control; E-H), and $\alpha_v\beta_3$ integrin-negative MCF-7 adenocarcinoma cells (negative control; I-L) were incubated with 14 µM RGD-Cy 5.5 to evaluate the target affinity of the probe. A strong cellular $\alpha_v\beta_3$-mediated fluorescence could be observed for RAW 264.7 (B; A: phase contrast image) and M21 (F; E: phase contrast image) cells, which could be selectively blocked by predosing with the unlabeled RDG-peptide (D: RAW 264.7, H: M21). MCF-7 cells, which lack $\alpha_v\beta_3$-expression, did not bind the targeted fluorochrome (I, K: phase contrast; J, L: fluorescence microscopy). Scale bars are indicated.



Supplemental Figure 2. Immunohistochemistry in murine atherosclerotic lesions.

Frozen tissue atherosclerotic plaquelike lesion (p) sections from carotid arteries of ApoE$^{-/-}$ mice (A, white light) were incubated with CD68 antibody (B, D; green signal), directed against macrophages or RGD-Cy 5.5 (C, D; red signal). Nuclei were counterstained with 4′,6-diamidino-2-phenylindole (DAPI; B, C, D; blue signal). Merging the 3 fluorescence stainings (D) visualized macrophage rich areas (white arrows) in the plaquelike lesions (p) that revealed homing/binding of the RGD-Cy 5.5 probe. Note the considerable background signal through binding of RGD-Cy 5.5 to the elastin rich fibers of the medium and the vascular SMCs. Scale bars are indicated. Fixation artifact of the tissue (a).