Errata: Noninvasive imaging of focal atherosclerotic lesions using fluorescence molecular tomography

Dolonchampa Maji
Metasebya Solomon
Annie Nguyen
Richard A. Pierce
Pamela K. Woodard
Walter J. Akers
Samuel Achilefu
Joseph P. Culver
Dana R. Abendschein
Monica Shokeen
Errata: Noninvasive imaging of focal atherosclerotic lesions using fluorescence molecular tomography


Washington University, Department of Biomedical Engineering, One Brookings Drive, St. Louis, Missouri 63110, United States
Washington University School of Medicine, Department of Radiology, 4525 Scott Avenue, St. Louis, Missouri 63110, United States
Washington University School of Medicine, Center for Cardiovascular Research, Department of Internal Medicine, 660 S. Euclid Avenue, St. Louis, Missouri 63110, United States

[DOI: 10.1117/1.JBO.19.11.119801]

This article [J. Biomed. Opt. 19(11), 110501 (2014)] was originally published online on 12 November 2014 with errors in the three figure captions. The figures and corrected captions are republished below.

This article was corrected online on 14 November 2014. It appears correctly in print.

Fig. 1 (a) Absorption and fluorescence spectra of LS668 in dimethylsulfoxide. Fluorescence microscopy images showing cellular internalization of LS668 (b) in NPR-C transfected cells, (c) inhibition of internalization in presence of excess C-ANF peptide, and (d) absence of internalization in NPR-A transfected cells. Blue (DAPI, nuclear stain) and red (LS668). Scale: 100 μm.
Fig. 2 (a) Coronal (depth = 7 mm), sagittal and transverse sections of reconstructed fluorescence molecular tomography (FMT) signal from injured artery and corresponding control artery from a representative animal (rabbit 1). White lines indicate the position of the respective sagittal and transverse sections. (b) Schematic showing the relationship between the FMT images displayed to their orientation with respect to the tissue volume. (c) Time dependent changes in integrated fluorescence signal (mean ± SD, n = 3) for injured and control arteries (*P = 0.0283; **P = 0.0282). (d) Mean (n = 2) fluorescence intensity obtained from the ex vivo injured artery containing the lesion and the control artery. Adjoining figure (inset) shows the fluorescence images (excitation/emission: 785 nm / > 800 nm) of the injured artery containing the lesion (top) and the control artery (bottom).

Fig. 3 Ex vivo studies on the paraffin fixed sections of injured (top row) and control artery (bottom row) sections obtained at 8 weeks post-surgery. (a) Bright field images showing IEL, internal elastic lamina; A, adventitia; M, media; 1 deg NEO: primary neointima. Scale: 500 μm. (b) Corresponding fluorescence images (excitation/emission: 710 ± 75 nm/810 ± 90 nm) after ex vivo staining with LS668. Scale: 500 μm. (c) Immunohistochemistry on tissue sections with clone RAM11 antibody (1: 100 dilution; blue) for macrophages and counterstained with nuclear fast red. Scale: 250 μm.