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Deriving the Point Spread Function of MR Pulse Sequences for Molecular Imaging through a Realistic Simulator

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To evaluate performance, sensitivity, and specificity of MR acquisition methods for molecular imaging, we extended SIMRI simulator by modifying some of source-codes to model an object with microscopic resolution while keeping image resolution in practical sizes and simulating arbitrary pulse sequences such as EPI. This simulator is also capable of modeling tissue in microscopic level to study effect of spatial distribution and concentration of nano-particles. Apparent change in T2* of environment containing Super-Paramagnetic-Iron-Oxide (SPIO) enables T2*-sensitive pulse sequences to detect them. In this paper, using this tool we studied EPI, spin-echo (SE), gradient-echo (GE), and True-FISP for imaging 20 pgs of SPIOs in the center of a cell in which $\Delta\chi$=20 ppm. We constructed a digital model of the object (a container of water) in microscopic level using 512×512×512 voxels. Each point of the object (an isotropic cube of 5 micron length) was modeled via its physical parameters (T1, T2, proton density) and field inhomogeneity. Using the modified simulator, we then applied various pulse sequences to construct a 64×64 image from this phantom, with slice thickness of 40 micron. We added a zero mean Gaussian noise with standard deviation equal to 7% of (K-space) mean to K-space. We were able to quantify performance of pulse sequences via their sensitivity and FWHM of their PSF. In terms of detection sensitivity, they were ordered as: True-FISP, EPI, GE, and SE. However, True-FISP and EPI had smoother PSF which lead to lower specificities. Fig. 1 shows images obtained by various pulse sequences and the intensity variation of them in the central horizontal line.

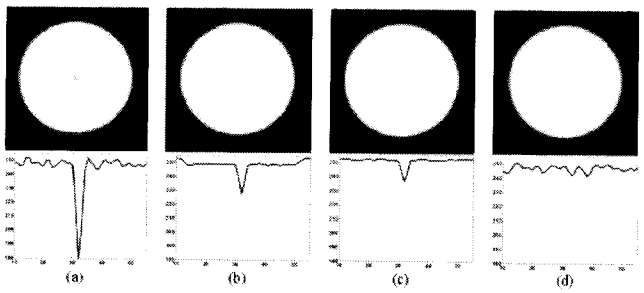

Figure 1: Top row: images obtained by: (a) True-FISP, (b) EPI, (c) GE, and (d) SE; Bottom row: intensity variation of top images along their central horizontal line.

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In vivo Tracking of Intravenously Injected ^{99m}Tc-HMPAO Labeled Mesenchymal Stem Cells in Brain Trauma Model

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Purpose: The aim of this study was to evaluate an *in vivo* tracking method using radiolabeled, intravenously injected bone marrow mesenchymal stem cells (rBMSCs) in brain trauma model. Methods: rBMSCs were isolated from the femur of male rats and labeled with 370 -1,480 MBq ^{99m}Tc-HMPAO. ^{99m}Tc-labeled rBMSCs were injected via lateral tail vein in normal rats (n=3), trauma models (n=3) and sham-operated controls (n=2). *In vitro* labeling efficiency and retention rate were measured, and cell viability at 48 hours and growth curves until 14 days after radiolabeling were evaluated. Dynamic and static images were acquired at 2, 24, and 48 hours after injection and organs were removed at 24 hours for biodistribution study. Results: Average labeling efficiency of ^{99m}Tc-labeled rBMSCs was 61 ± 15 %. Retention rates were 72 %, 45 %, 31 %, and 16 % at 1, 3, 6, and 24 hours, respectively. At 48 hour after radiolabeling, number of viable cells in labeled rBMSC was not different from that of control by XTT assay. Growth of labeled rBMSC was inhibited after 3rd day compared to control, however it reach to a confluency after 10th day of labeling. In animal study, gamma camera imaging of ^{99m}Tc-labeled rBMSCs was adequate for tracking until 24 hour after injection. Meanwhile, 48 hour images were also available, but not sufficient for localization of rBMSC because of radioactivity decay. Immediately after injection, ^{99m}Tc-labeled rBMSCs were distributed mostly in the lungs and kidneys, and it migrated to liver and spleen at 24 hour and did not change until 48 hour. In all animals, ^{99m}Tc-labeled rBMSCs were not visualized in brain. Conclusion: ^{99m}Tc-HMPAO was easily labeled to rBMSCs with sufficient labeling efficiency and sensitivity. Therefore, radiolabeling method with ^{99m}Tc-HMPAO can be used for *in vivo* tracking of rBMSC at early phase of transplantation.

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Evaluation of new immunotherapies for breast cancer with 18F-FDG-labeled NK cells

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The purpose of this study was to label genetically engineered, anti-HER2/neu directed natural killer (NK) cells with 18F-FDG and to track the labeled NK cells to HER2/neu positive mammary tumors. NK-92 cells were genetically modified to NK-92-scFv(FRP5)-zeta cells, which express a chimeric antigen receptor specific to the tumor-associated ErbB2 (HER2/neu) antigen. These NK-92-scFv(FRP5)-zeta cells were labeled with 18F-FDG by simple incubation. The labeling efficiency was evaluated by a gamma-counter. Differences between different cells were tested for significance with analysis of variance ($p<0.05$). Subsequently, 18F-FDG-labeled NK-92-scFv(FRP5)-zeta (n=6) or parental NK-92 control cells (n=3) were intravenously injected into the tail vein of mice with implanted HER2/neu positive NIH/3T3 tumors. The radioactivity in the tumors, which apparently corresponded to the tumor-accumulation of 18F-FDG labeled cells was quantified by digital autoradiography and correlated with histopathology. Both, parental NK-92 and genetically modified NK-92-scFv(FRP5)-zeta cells could be efficiently labeled with 18F-FDG by simple incubation. Optimal labeling efficiencies (80%) were achieved using an incubation period of 60 min and additional insulin (10 IU/ml). After intravenous injection of 5*106 18F-FDG labeled NK-92-scFv(FRP5)-zeta cells into tumor bearing mice, digital autoradiography showed an increased uptake of