^{18}F-FDG and ^{18}F-NaF PET/MR Imaging of the Musculoskeletal System

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Osteoarthritis (OA) is a disease of the entire joint that involves inflammation and bone remodelling such as subchondral sclerosis, cysts, and osteophytes. New PET/MR systems allow for simultaneous, sensitive, and quantitative assessments of early bone activity in OA[1]. ^{18}F-FDG PET shows areas of acute phase cellular response (neutrophils or PMNs) in the bone marrow, such as inflammation[2]. ^{18}F-NaF PET interrogates osteoblast activity in the bone[3]. This metabolic bone data from PET can be correlated with high-resolution quantitative MR methods[4] of other tissues to study the pathogenesis of OA.

Joint PET/MR Considerations: *PET Attenuation Correction:* In order to obtain accurate PET images, emission data recorded during a PET scan must be corrected for tissue and hardware attenuation. This is performed during reconstruction using an attenuation map (μ-map). Differences in PET attenuation between tissue fat and water as well as attenuation due to the PET/MR system is accounted for. Phantom and in vivo results showed that the flex coil effects on SUV values and lesion area appear to be negligible. Investigations are ongoing to correct for cortical bone attenuation.

Dose: MR knee and hip protocols (20-60 minutes) are considerably longer than the data collection time in one patient bed position in clinical PET-CT (3-4 minutes). As all of the MR scan time can be used to collect PET data, dose of the injected dose of radiotracer can be reduced by the equivalent increase in scan time (~5-15x). Our initial study uses a does of 5 mCi (~1/2 clinical dose).

Initial Results with PET-MR in OA: Initial results in subjects with knee OA showed more regions of increased tracer uptake on NaF PET than regions of increased FDG uptake or regions of bone abnormalities or cartilage damage on MRI[5]. Bone abnormalities observed on MRI consistently correlated with increased FDG and NaF PET tracer uptake. However, PET SUV in these regions was considerably higher with NaF than with FDG. Of significant interest, uptake on NaF did not always correspond to structural damage detected on MRI or increased FDG uptake.

The majority of abnormal regions of NaF uptake are seen in the subchondral bone, a region that is associated with the development of pain as well as cartilage degeneration. As NaF is related to osteoblast activity, areas of increased tracer uptake may also be signs of osteophyte formation. This suggests that metabolic abnormalities in the bone may occur prior to structural changes are seen on MRI. Work is ongoing to recruit more subjects to compare PET SUV in cartilage and subchondral regions to quantitative MR measures such as T2 and $T_{1\rho}$.

In a study of patients with hip OA and ^{18}F-FDG, normalized SUV_{mean} was highest in joint fluid, which may be a marker of increased ^{18}F-FDG diffusion or high glucose metabolism due to inflammation[6]. Normalized SUV_{mean} was elevated in both subchondral cysts and in bone marrow

lesions. Areas of OA damage identified on the MRI tended to co-localize with increased ^{18}F-FDG uptake.

Conclusions: PET/MR hybrid imaging of the knee and hip has potential to reveal insights about bone activity as well as other tissues in OA. Results suggest that PET/MR may detect knee and hip abnormalities unseen using MRI alone and is a promising tool for early detection of OA change in the bone.

References:

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