Many brain diseases have been linked to abnormal oxygen metabolism and blood perfusion; nevertheless, there is still a lack of robust diagnostic tools for directly imaging cerebral metabolic rate of oxygen (CMRO2) and cerebral blood flow (CBF), as well as the oxygen extraction fraction (OEF) that reflects the balance between CMRO(2) and CBF.

Measurement of brain tissue oxygen extraction fraction (OEF) in both baseline and functionally activated states can provide important information on brain functioning in health and disease.

MR-rOEF was determined from separate measurements of T2 , T2 * and relative cerebral blood volume (rCBV) employing a multi-parametric approach for quantification of the blood-oxygenation-level-dependent (BOLD) effect. With respect to 18F fluoromisonidazole positron emission tomography, besides the commonly used late uptake between 120 and 130 min ([18 F]-FMISO120-130 min), we also analyzed the hypoxia specific uptake rate [18 F]-FMISO-k3 , as obtained by pharmacokinetic modeling of dynamic uptake data. Since pharmacokinetic modeling of partially acquired dynamic [18 F]-FMISO data was sensitive to a low signal-to-noise-ratio, analysis was restricted to high-uptake tumor regions. Individual spatial analyses of deoxygenation and hypoxia-related parameter maps revealed that high MR-rOEF values clustered in (edematous) peritumoral tissue, while areas with high [18 F]-FMISO120-130 min concentrated in and around active tumor with disrupted blood-brain barrier, i.e. contrast enhancement in T1 -weighted MRI. Volume-of-interest-based correlations between MR-rOEF and [18 F]-FMISO120-130 min as well as [18 F]-FMISO-k3 , and voxel-wise analyses in individual patients, yielded limited correlations, supporting the notion that [18 F]-FMISO uptake, even after 2 h, might still be influenced by perfusion while [18 F]-FMISO-k3 was severely hampered by noise. According to these results, vascular deoxygenation, as measured by MR-rOEF, and severe tissue hypoxia, as measured by [18 F]-FMISO, show a poor spatial correspondence. Overall, the two methods appear to rather provide complementary than redundant information about high-grade glioma biology [1].

1)
Preibisch C, Shi K, Kluge A, Lukas M, Wiestler B, Göttler J, Gempt J, Ringel F, Al Jaberi M, Schlegel J, Meyer B, Zimmer C, Pyka T, Förster S. Characterizing hypoxia in human glioma: A simultaneous multimodal MRI and PET study. NMR Biomed. 2017 Aug 14. doi: 10.1002/nbm.3775. [Epub ahead of print] PubMed PMID: 28805936.