

Comparison of ^{18}F -Fluorethyltyrosin (FET) and ^{18}F -Fluorodeoxyglucose (FDG) transport kinetics with radio sensitivity of different carcinoma cells *in vitro*

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Aim:

It is well known that fast tumor progression often correlates with radio sensitivity. Furthermore, metabolic activity of tumors can be quantified using uptake of PET-tracers as FET or FDG that mirror amino acid or glucose metabolism, respectively. However, prediction of radio sensitivity in early tumor stages is difficult. For this reason, we established cell culture models to investigate correlations between tracer uptake and radio sensitivity of different tumor entities.

Methods:

Human cell lines derived from squamous cell carcinomas PCI13, A549 and melanoma Mel624 were cultured. Confluent cells were radiated with doses of 25, 50, 75, and 100 Gy. Following incubation for 48 hr total protein concentration of survived cells was determined. Tracer uptake of untreated cells was measured by gamma counting after incubation with 0.5 MBq/ml FET or FDG in the supernatant for 1, 3, 8, 15, 30 or 5, 10, 20, 30, 60 min, respectively, and standardized to identical protein concentration. Specific uptake inhibitors served as negative controls.

Results:

Radio sensitivity was cell type specific and correlated to the dose. Survival after 48 hr following 100 Gy was 23, 68 and 90% for PCI13, Mel624, and A594, respectively. For both tracer, all tested cell lines has a specific uptake that followed an exponential kinetic and depended on the cell number. For FET A549 had maximum uptake (arbitrarily set to 100%) followed by Mel624 (44%), and PCI13 (43%). In contrast, order of FDG tracer uptake was PCI13 (100%), Mel624 (52%), and A549 (16%).

Conclusion:

For the investigated cell lines, uptake kinetics for amino acid and glucose metabolism showed no correlation. High FDG uptake paralleled with high radio sensitivity whereas FET showed no concordance. Taken together, our data may suggest that glucose is a better marker for radio sensitivity compared to amino acid metabolism.

