Enhancement of choline kinase expression in implanted renal cancer : a mechanism of [^{11}C]choline accumulation

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Abstract

[methyl-^{11}C]choline (^{11}C-choline) is a radioligand potentially useful for oncological positron emission tomography (PET). To elucidate choline accumulation mechanism in cancer, the relationship between choline uptake, and the gene expression of choline kinase (CK) and CTP:phosphocholine cytidylytransferase (CCT), which catalyze the phosphorylation of choline to produce phosphocholine and CDP-choline respectively, within CDP-choline pathway, was examined by Northern blot and RT-PCR in a renal cancer implant model. RT-PCR were validated by demonstrating the RT-PCR products of expected sizes for CK and CCT mRNA in all tissues examined, including cancer tissues. The expression of CK and CCT in cancer tissues was higher than that of other tissues. Northern blot hybridization showed CK mRNA content increased approximately three-fold in renal cancer, whereas CCT mRNA did not change. These results suggest that CK expression plays an essential role in higher choline accumulation in renal tumor, leading to an increase of conversion of choline to phosphocholine, which is a stable neutral compound.