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Recent Development in Imaging of COX

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Perspective

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Recent development in visualization of cyclooxygenase type2 (COX-2) expression using positron emission tomography (PET) has attracted a lot of attention. Cyclooxygenase catalyzes the conversion of arachidonic acid to form prostaglandins through functionally cooperative COX and peroxides (POX) enzymes. Comparing with the house keeping role played by homeostatic COX-1, COX-2 expression has been associated with the tumor progression and inflammation. COX-2 selective inhibitors such as the class of Coxibs exemplifying as celecoxib and valecoxib have been labeled with positron emitters such as fluorine-18 and C-11 for visualization through PET. The unique quantitative imaging modality enables a rapid and direct assessment of enzyme expression and receptor binding determination in both *in vitro* and *in vivo* models. Although most of the COX inhibitors exert COX-2 inhibition potencies in nano molarities *in vitro*, visualization of their sufficient accumulation in COX-2 related tumor models were not successful excepting inflammation associated animal models and xenograft tumor models. For example, Uddin and coworkers have developed COX-2 radiotracer for visualization of carrageenan-induced inflammation in the rat inflammation. COX-1 selective probe ^{11}C-ketoprofen methyl ester developed by Onoe and coworkers proved to be reliable in imaging of the inflammation induced rat models. But, the main role played in tumor progression, COX-2, is still not easily to localize. Especially, when these probes are designed for imaging COX-2 expression in brain tumors, the brain related barriers are needed to penetrate. Lipophilicity need to be enhanced through structural modification. This may, however, increase the nonspecific binding to other nontarget enzymes. Nonspecific binding can hamper the binding profile such as the higher background signal due to binding to various carbonic anhydrase enzymes pointed out recently by Wuest and coworkers. They have recently addressed the insufficient blockade of ^{18}F-celecoxib in the presence of a number of Coxibs. Either the unexpected binding to nontarget enzymes or inadequate tumor models limit current application to COX-2 visualization. It remains a bottom neck for the PET tracer development. Future development of the drugs for COX-2 expression may need to screen a compound library by assaying their activities toward a range of enzymes to find out the most potent and selective candidate lead compounds. Drug development for intracellular targeting approach has to overcome more barriers than that by drug development for targeting membrane-related receptors.