Alzheimer's disease (AD) is a neurodegenerative disease characterized by dementia, cognitive impairment, and memory loss. Postmortem brains of AD patients reveal the presence of beta-amyloid plaques and neurofibrillary tangles, combined with gliosis and neuronal loss. Increases in the levels of soluble Abeta peptides in the course of the disease lead to gradual increase of beta-amyloid plaques, which are thought to be an early event in the neuropathology of AD, and may occur years before the onset of clinical symptoms. Therefore, the development of molecular imaging techniques for in vivo assessment of beta-amyloid accumulation in the ageing brain is an important and active area of research. Up to now, only very limited attempts have been made to target other characteristics of AD, such as microglial activation. As for MRI-based contrast agents, numerous ligands have been developed with affinity for beta-amyloid. Several groups have taken the approach of targeting plaques using beta-amyloid fragments labeled with gadolinium or iron oxide nanoparticles (Poduslo et al. 2002; Wadghiri et al. 2003). Alternative approaches are to use the F(ab')2 fragment of a monoclonal antibody against beta-amyloid, or small peptides with high affinity for amyloid plaques (Ramakrishnan et al. 2008, Larbanoix et al. 2008, Sirgurdsson et al. 2008). To functionalize these compounds for MRI detection in mouse models, they were again linked to relaxation agents, either iron oxide particles or gadolinium chelates. However, to target amyloid plaques, the compound has to be able to cross the blood-brain-barrier (BBB), and all compounds above failed to do so without artificial opening of the BBB. Several groups have worked on modifying the BBB-penetrating properties, e.g. Poduslo et al. (2004), who attempted to improve BBB penetration by linking the contrast agent to several putrescine groups, which enhances both blood-brain barrier (BBB) permeability and binding to plaques themselves. In 2005, Higuchi and others reported successfully labeling amyloid plaques in transgenic AD mice with small aromatic molecule containing a $^{19}$F atom. This approach, using $^{19}$F MRI, has also been called ‘hot spot’ imaging, because of its similarities to nuclear techniques: uptake of the compound should appear as the only signal in the brain without any background, since there is virtually no endogenous $^{19}$F present in the body. All these compounds show promising results in animal models, but further development is necessary to improve sensitivity and brain penetration. Up to now, the only compounds that are in use for clinical imaging are nuclear medicine-based amyloid targeting tracers. Many of those are based on small aromatic molecules that have been in use as traditional histological markers for beta-sheet amyloid. The most promising one in Phase II clinical trial is Pittsburgh compound-B ($^{18}$F-PIB), which was originally developed at the University of Pittsburgh. One other PET compound, $^{18}$F-FDDNP, is worth mentioning, as recent evidence is indicating that this tracer recognizes both beta-amyloid plaques and tau tangles.