# Pittsburgh compound B

Pittsburgh compound B (PiB) is a radioactive analog of thioflavin T, which can be used in positron emission tomography scans to image beta-amyloid plaques in neuronal tissue. Due to this property, Pittsburgh compound B may be used in investigational studies of Alzheimer's disease.

Large prospective studies are required to verify whether [11C]PiB PET will be a diagnostic aid, particularly in early Alzheimer disease [1].

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To determine the relationship between cerebral amyloid plaque load and rates of cerebral atrophy in Alzheimer's disease. (11)C-PIB[2].

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A study of Rahayel et al., assessed the associations between cortical and subcortical 11 C-Pittsburgh Compound B (PiB) retention, namely, in the hippocampus, amygdala, putamen, caudate, pallidum, and thalamus, and subcortical morphology in cognitively normal individuals. They recruited 104 cognitive normal individuals who underwent extensive neuropsychological assessment, PiB-positron emission tomography (PET) scan, and 3-T magnetic resonance imaging (MRI) acquisition of T1-weighted images. Global, cortical, and subcortical regional PiB retention values were derived from each scan and subcortical morphology analyses were performed to investigate vertex-wise local surface and global volumes, including the hippocampal subfields volumes. They found that subcortical regional Aβ was associated with the surface of the hippocampus, thalamus, and pallidum, with changes being due to volume and shape. Hippocampal Aβ was marginally associated with volume of the whole hippocampus as well as with the CA1 subfield, subiculum, and molecular layer. Participants showing higher subcortical Aβ also showed worse cognitive performance and smaller hippocampal volumes. In contrast, global and cortical PiB uptake did not associate with any subcortical metrics. This study shows that subcortical Aβ is associated with subcortical surface morphology in cognitively normal individuals. This study highlights the importance of quantifying subcortical regional PiB retention values in these individuals [3].

1)
Leinonen V, Alafuzoff I, Aalto S, Suotunen T, Savolainen S, Någren K, Tapiola T, Pirttilä T, Rinne J, Jääskeläinen JE, Soininen H, Rinne JO. Assessment of beta-amyloid in a frontal cortical brain biopsy specimen and by positron emission tomography with carbon 11-labeled Pittsburgh Compound B. Arch Neurol. 2008 Oct;65(10):1304-9. doi: 10.1001/archneur.65.10.noc80013. Epub 2008 Aug 11. PubMed PMID: 18695050.
2)
11)C-6-OH benzothiazole)PET (positron emission tomography) findings were correlated with volumetric magnetic resonance imaging (MRI) measurements in nine subjects with mild to moderate AD. Analysis revealed a positive correlation between rates of whole brain atrophy and whole brain (p = 0.019) and regional (11)C-PIB uptake. This provides support for the central role of amyloid deposition in the pathogenesis of AD ((Archer HA, Edison P, Brooks DJ, Barnes J, Frost C, Yeatman T, Fox NC, Rossor MN. Amyloid load and cerebral atrophy in Alzheimer's disease: an 11C-PIB positron emission tomography study. Ann Neurol. 2006 Jul;60(1):145-7. PubMed PMID: 16802294.
3)
Rahayel S, Bocti C, Sévigny Dupont P, Joannette M, Lavallée MM, Nikelski J, Chertkow H, Joubert S. Subcortical amyloid load is associated with shape and volume in cognitively normal individuals. Hum

Brain Mapp. 2019 May 30. doi: 10.1002/hbm.24680. [Epub ahead of print] PubMed PMID: 31148327.