

## Comment on Hsiao et al.: Correlation of early-phase $^{18}\text{F}$ -florbetapir (AV-45/Amyvid) PET images to FDG images: preliminary studies

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Dear Sir,

I read with interest the paper by Hsiao et al. [1] in which they compared the early regional cerebral distribution of the putative amyloid imaging agent  $^{18}\text{F}$ -florbetapir with that of  $^{18}\text{F}$ -FDG. It has long been recognized that the early cerebral distribution of lipophilic tracers reflects regional perfusion [2–4] and indeed it was suggested many years ago that a single injection might provide both perfusion and receptor binding information by imaging at two time points [5, 6].

However, it is also recognized that perfusion and metabolism may be coupled in the normal state [7, 8] but are often disparate under activation or pathological processes, and thus perfusion is an imperfect substitute for metabolic imaging [9, 10]. For this reason, diffusion MRI has not replaced  $^{18}\text{F}$ -FDG PET of the brain.

Nonetheless, the authors are correct in suggesting that utilization of the early perfusion image obtained with florbetapir may increase the diagnostic power of florbetapir imaging for detection of Alzheimer's disease by combining the well-known characteristic perfusion pattern of bilateral temporoparietal deficits with the emerging patterns of amyloid deposition [11].

In conclusion, I feel it is both scientifically incorrect and misleading to describe the early distribution of florbetapir as resembling a glucose map.

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A response to this letter can be found at doi:10.1007/s00259-012-2137-5

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