reactive astrocytosis but not in macrophage infiltration or activated microglia. In humans, the histologic finding of pronounced reactive astrocytosis was confirmed in different nonneoplastic lesions that exhibited increased $^{18}$F-FET uptake (*7,8*). Thus, according to the current knowledge, a high incidental uptake of $^{18}$F-FET in benign brain lesions is most likely due to reactive astrocytosis.

Furthermore, in a clinical study we already addressed the problem of nonspecific brain lesions on MR imaging with low $^{18}$F-FET uptake (*9*). We observed that normal or low $^{18}$F-FET uptake is a strong predictor for a benign course, with the eventual development of a low-grade glioma.

We would like to emphasize that the data on lesion-to-brain ratios of $^{18}$F-FET uptake in different brain lesions at initial diagnosis may be helpful for decision making but that the additional value of $^{18}$F-FET PET lies in defining an optimal site for biopsy and determining the extent of metabolically active tumor for treatment planning.

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**Erratum**

In the article "Assessment of Cellular Proliferation in Tumors by PET Using $^{18}$F-ISO-1," by Dehdashti et al. (*J Nucl Med.* 2013;54:350–357), the name of the tenth author in the byline was misspelled. The correct name is Nina Wagner-Johnston. The authors regret the error.