Effects of age, diet, and type 2 diabetes on the development and FDG uptake of atherosclerotic plaques.

This study investigated the effects of age, duration of a high-fat diet, and type 2 diabetes on atherosclerotic plaque development and uptake of (18)F-fluorodeoxyglucose ((18)F-FDG) in 2 mouse models. The animal's age and start time and duration of a high-fat diet have effects on plaque composition in atherosclerotic mice. The aortas of atherosclerotic low-density lipoprotein receptor deficient mice expressing only apolipoprotein B100 (LDLR(-/-)ApoB(100/100)) and atherosclerotic and diabetic mice overexpressing insulin-like growth factor II (IGF-II/LDLR(-/-)ApoB(100/100)) were investigated at 4, 6, and 12 months of age and older after varying durations of high-fat diet. C57BL/6N mice on normal chow served as controls. Plaque size (intima-to-media ratio), macrophage density (Mac-3 staining), and plaque uptake of (18)F-FDG were studied by means of in vivo positron emission tomography/computed tomography by ex vivo autoradiography and by histological and immunohistochemical methods. From the ages of 4 to 6 months and 12 months and older, the plaque size increased and the macrophage density decreased. Compared with the controls, the in vivo imaging showed increased aortic (18)F-FDG uptake at 4 and 6 months, but not at 12 months and older.
Autoradiography showed focal (18)F-FDG uptake in plaques at all time points (average plaque-to-normal vessel wall ratio: 2.4 ± 0.4, p < 0.001) with the highest uptake in plaques with high macrophage density. There were no differences in the plaque size, macrophage density, or uptake of (18)F-FDG between LDLR(-/-)ApoB(100/100) and IGF-II/LDLR(-/-)ApoB(100/100) mice at any time point. The 6-month-old LDLR(-/-)ApoB(100/100) and IGF-II/LDLR(-/-)ApoB(100/100) mice demonstrated highly inflamed, large, and extensive atherosclerotic plaques after 4 months of a high-fat diet, presenting a suitable model for studying the imaging of atherosclerotic plaque inflammation with (18)F-FDG. The presence of type 2 diabetes did not confound evaluation of plaque inflammation with (18)F-FDG.

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