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IN VIVO EVIDENCE FOR IMPAIRED PERIPHERAL FATTY ACID TRAPPING IN HIV-1 LIPOATROPHY

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BACKGROUND: The use of highly active antiretroviral therapy in HIV-infected patients has been associated with changes in fat distribution (lipodystrophy), insulin resistance and dyslipidaemia and increases the risk for atherosclerosis. Lipodystrophy may result from impaired peripheral free fatty acid (FFA) trapping, which may cause enhanced hepatic FFA flux and consequently VLDL overproduction and exaggerated postprandial lipaemia.

METHODS: We have investigated FFA, hydroxybutyric acid (HBA, a marker of hepatic FFA oxidation) and triglyceride (TG) changes after a single oral fat challenge (10 h, 50 g/m²) in HAART-treated HIV-infected male patients with (LIPO, *n*=26) and without lipodystrophy (NON-LIPO, *n*=12) and in healthy normolipidaemic controls (*n*=35). Immunological parameters and duration of HIV and HAART were similar between the LIPO and NON-LIPO group. Area under the curves (AUCs) for FFA and TG were significantly higher in the LIPO group (7.9 ±2.1 and 45.0 ±12.9 mmol·h/l, respectively) compared with the NON-LIPO group (6.1 ±1.1 and 22.7 ±10.7 mmol·h/l, respectively) and healthy controls (5.9 ±1.2 and 17.0 ±3.9 mmol·h/l, respectively). The postprandial increase in ketone bodies was two times higher in the LIPO group (1954 ±1123 μmol·h/l) compared with the NON-LIPO group and healthy controls (1188 ±567 and 1067 ±501 μmol·h/l, respectively, *P*<0.05 for each). There was no difference in HBA-AUC and FFA-AUC between the NON-LIPO group and healthy controls. In HIV-infected patients, HBA-AUC was negatively associated with BMI (*r*= -0.50, *P*<0.05), total body fat mass (*r*= -0.40, *P*<0.05) and hip circumference (*r*= -0.38, *P*<0.05) and positively associated with incremental FFA-AUC.

CONCLUSIONS: The present data suggest elevated hepatic FFA flux in HIV lipotrophy, most likely as a result of inadequate incorporation of FFA into TG in adipocytes. Impaired peripheral postprandial FFA uptake may lead to increased hepatic FFA flux, VLDL overproduction and increased concentrations of atherogenic postprandial TG-rich lipoproteins in these patients.

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