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ASSESSMENT OF GROWTH HORMONE PHYSIOLOGY IN THE HIV LIPODYSTROPHY SYNDROME

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P Rietschel, C Hadigan, C Corcoran, T Stanley, J Gertner and S Grinspoon
Massachusetts General Hospital and Harvard Medical School, Boston, Mass.; and Serono Laboratories, Norwell, Mass., USA

BACKGROUND: The HIV lipodystrophy syndrome is characterized by increased visceral adiposity, dyslipidemia and insulin resistance.

OBJECTIVES: Reduced GH concentrations are seen in other populations of viscerally obese patients, but previous studies have not characterized GH physiology in the HIV lipodystrophy syndrome.

METHODS: Twenty-one subjects with the HIV lipodystrophy syndrome (LIPO), 20 HIV-infected non-lipodystrophy subjects (NONLIPO) and 20 control (C) subjects were recruited. Subjects in the three groups were age [44.3±1.5(LIPO), 41.0±1.3 (NONLIPO) and 42.8±1.8 (C) years] and BMI-matched [24.8±0.7 (LIPO), 24.6±0.5 (NONLIPO) and 24.6±0.5 kg/m² (C)] ($P>0.05$ for all comparisons).

RESULTS: Visceral abdominal fat [15 470±1193 (LIPO), 8518±950 (NONLIPO), and 9243±924 mm² (C), $P<0.01$ LIPO versus NONLIPO and LIPO versus C] and the ratio of visceral abdominal fat to subcutaneous abdominal fat [2.05±0.48 (LIPO) versus 0.63±0.05 (NONLIPO) versus 0.59±0.06 (C), $P<0.01$ LIPO versus NONLIPO and LIPO versus C] were significantly increased in the LIPO subjects. The mean overnight GH concentration determined from frequent sampling every 20 min from 20.00 h to 08.00 h was decreased in the LIPO subjects [0.58±0.13 (LIPO) versus 1.02±0.14(NONLIPO) versus 0.98±0.19 ng/ml (C), $P<0.05$ LIPO versus NONLIPO and LIPO versus C]. Pulse analysis demonstrated decreased baseline GH [0.16±0.03 (LIPO) versus 0.25±0.04 (NONLIPO) versus 0.29±0.06 ng/ml (C), $P<0.05$ LIPO versus NONLIPO and LIPO versus C] and GH peak amplitude [1.62±0.38 (LIPO) versus 2.59±0.31 (NONLIPO) and 2.70±0.40 ng/ml (C), $P<0.05$ LIPO versus NONLIPO and LIPO versus C], but no

significant difference in pulse frequency. Total body fat ($R=-0.40$, $P=0.01$) and visceral fat ($R=-0.58$, $P=0.0001$) correlated inversely with mean overnight GH concentrations. In a multivariate regression model, controlling for age, BMI, body fat and visceral fat, only visceral fat was a significant predictor of mean GH concentrations ($P=0.0036$, $R^2=0.40$).

CONCLUSIONS: These data demonstrate that increased visceral abdominal fat strongly predicts reduced GH concentrations in patients with the HIV lipodystrophy syndrome. Further studies are necessary to determine the physiological relevance of reduced GH in patients with the HIV lipodystrophy syndrome.

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