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J Clin Endocrinol Metab. 1992 Apr;74(4):757-65.

Augmented growth hormone (GH) secretory burst frequency and amplitude mediate enhanced GH secretion during a two-day fast in normal men.

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Abstract

Serum GH concentrations are increased in fasted or malnourished human subjects. We investigated the dynamic mechanisms underlying this phenomenon in nine normal men by analyzing serum GH concentrations measured in blood obtained at 5-min intervals over 24 h on a control (fed) day and on the second day of a fast with a multiple-parameter deconvolution method to simultaneously resolve endogenous GH secretory and clearance rates. Two days of fasting induced a 5-fold increase in the 24-h endogenous GH production rate [78 +/- 12 vs. 371 +/- 57 micrograms/Lv (Lv, liter of distribution volume) or 0.24 +/- 0.038 vs. 1.1 +/- 0.16 mg/m2 (assuming a distribution volume of 7.9% body weight), P = 0.0001]. This enhanced GH production rate was accounted for by 2-fold increases in the number of GH secretory bursts per 24 h (14 +/- 2.3 vs. 32 +/- 2.4, P = 0.0006) and the mass of GH secreted per burst (6.3 +/- 1.2 vs. 11 +/- 1.6 micrograms/Lv, P = 0.002). The latter was a result of increased secretory-event amplitudes (maximal rates of GH release attained within a burst) with unchanged secretory burst durations. GH was secreted in complex volleys composed of multiple discrete secretory bursts. These secretory volleys were separated by shorter intervals of secretory quiescence in the fasted than fed state (respectively, 88 +/- 4.2 vs. 143 +/- 14 min, P = 0.0001). Similarly, within volleys of GH release, constituent individual secretory bursts occurred more frequently during the fast [every 33 +/- 0.64 (fasted) vs. every 44 +/- 2.0 min (fed), P = 0.0001]. The t1/2 of endogenous GH was not significantly altered by fasting [18 +/- 2.2 (fasted) vs. 20 +/- 1.5 min (fed), P = 0.47]. Serum insulin-like growth factor I concentrations were unchanged after 56 h of fasting. In conclusion, the present data suggest that starvation-induced enhancement of GH secretion is mediated by an increased frequency of GHRH release, and longer and more pronounced periods of somatostatin withdrawal.

PMID: 1548337 DOI: 10.1210/jcem.74.4.1548337

[Indexed for MEDLINE]