

# Inappropriate gonadotropin secretion in polycystic ovary syndrome

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摘要

## Abstract

In recent years, there has been uncertainty concerning the association of inappropriate gonadotropin secretion (high LH and normal FSH) and the polycystic ovary syndrome (PCOS). In the present study, we ascertained the influence of body composition on LH pulsatile parameters in 33 PCOS and 32 normal cycling (NC) women across a wide range of body mass index (BMI, 19 – 42 kg/m<sup>2</sup>). Twenty four-hour pulsatile parameters for serum LH (10-min sampling) and pituitary gonadotropin responses to iv bolus GnRH (10 µg) were evaluated. Fasting (0800 h) FSH and steroid hormone concentrations and 24-h mean insulin levels were determined. Insulin sensitivity (SI) was assessed by rapid iv glucose tolerance test in a subset of 28 PCOS and 29 NC subjects.

Our results showed that BMI, an indicator of relative adiposity, had a significant negative impact on 24-h mean LH pulse amplitude ( $r = -0.63$ ,  $P < 0.001$ ) and the peak increment of LH in response to GnRH stimulation ( $r = -0.41$ ;  $P = 0.02$ ) for PCOS but not NC women. In contrast, 24-h LH pulse frequency was uniformly increased (40%) in PCOS as compared with NC women independent of BMI. In PCOS women, the blunting of pulse amplitude with increasing BMI resulted in a decline in 24-h mean LH levels ( $r = -0.63$ ,  $P < 0.001$ ) and the ratio of LH/FSH ( $r = -0.44$ ,  $P = 0.02$ ) not seen in NC. With BMI  $< 30$  kg/m<sup>2</sup>, 24-h mean LH values for PCOS women were greater than the normal range for NC in 95% (18/19) of cases, whereas 24-h LH levels failed to discriminate PCOS from NC women in 43% (6/14) of obese (BMI  $> 30$  kg/m<sup>2</sup>) PCOS women. Thus, the diagnostic value of LH determinations is retained for PCOS women with BMI  $< 30$  kg/m<sup>2</sup>. For screening purposes, the mean of two LH values in samples collected at 30-min intervals was found to have a discriminatory power equal to that of the 24-h mean.

These findings suggest that 1) BMI negatively influences LH pulse amplitude in PCOS women principally by an effect at the pituitary level; 2) accelerated LH pulse frequency in PCOS women is not influenced by BMI and represents a basic component of hypothalamic dysfunction in PCOS women; and 3) BMI does not influence gonadotropin secretion in

normal cycling women. Thus assessments of basal LH levels and the LH/FSH ratio in hyperandrogenic anovulatory women are clinically meaningful when BMI is taken into account. Investigations to define the factor(s) that link adiposity and the attenuation of LH pulse amplitude in PCOS women would add further understanding of this complex neuroendocrine-metabolic disorder.