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ADIPOSITY AND OVULATION, THE COMPLEX INTERACTIONS

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Central body adiposity results from fat deposited in the abdominal wall and visceral locations. This fat distribution is associated with a metabolic profile of hyperinsulinemia, impaired glucose tolerance, and dyslipidemia as well as hyperandrogenemia.
Polycystic ovary syndrome (PCOS) has long been hypothesized to result from functional ovarian hyperandrogenism due to dysregulation of androgen secretion/effect. When defined as otherwise unexplained hyperandrogenic oligoanovulation, the majority of PCOS cases have 17-hydroxyprogesterone hyperresponsiveness to gonadotropin stimulation, testosterone elevation after suppression of adrenal androgen production. Most PCOS cases who lack pure evidence of androgen secretory abnormalities are obese. The metabolic syndrome of obesity-related insulin resistance and the compensatory hyperinsulinism has tissue-selective effects, which include aggravation of hyperandrogenism and impaired ovulation.
PCOS seems to arise as a complex trait that results from the interaction of diverse genetic and environmental factors. Heritable factors include polycystic ovarian morphology, hyperandrogenemia, insulin resistance, and insulin secretory defects. Environmental factors include prenatal androgen exposure and poor fetal growth, whereas acquired obesity is a major postnatal factor. The variety of pathways involved which still lack of a common thread attests to the multifactorial nature and heterogeneity of the syndrome.

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