

**PATHOPHYSIOLOGICAL CONNECTION BETWEEN
OBESITY AND OVARIAN POLYCYSTOSIS SYNDROME**

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PCOS is a clinical syndrome associated with chronic anovulation, menstrual cycle disorders, and ovarian hyperandrogenism in the absence of other causes of androgen overproduction.

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The prevalence of PCOS among women of reproductive age reaches 21%. The disease is the most common cause of infertility in this age group.

The most important pathogenetic links of anovulation in PCOS are IR and obesity that potentiates it. PCOS is an independent factor that reduces tissue susceptibility to insulin, and this effect is observed regardless of body weight. However, obesity has an independent negative effect, potentiating IR.

The severity of IR in women with PCOS with obesity is higher than in women with PCOS and normal body weight. Additionally, the frequency and

severity of IR increase as the total body fat mass increases, especially in the visceral region.

Decreased sensitivity of peripheral tissues to normal insulin concentrations leads to concomitant chronic compensatory hyperinsulinemia - a condition typical of PCOS patients. IR and hyperinsulinemia underlie all manifestations of PCOS: hyperandrogenism, reproductive disorders, acne, hirsutism, and metabolic disorders.

Insulin resistance and hyperinsulinemia stimulate increased hormonal secretion of the ovaries and adrenal glands, inhibit the production of globulin, which binds sex hormones, and thereby increase the activity of testosterone. High levels of testosterone contribute to abdominal obesity, which in turn exacerbates insulin resistance, closing the vicious circle.

The pronounced damaging effect of abdominal obesity is associated with the greater endocrine activity of visceral fat, which produces various peptide products - adipokines. Of these, leptin, adiponektin, grelin, and tumor necrosis factor α (TNF- α) play the most significant role in the pathogenesis of reproductive dysfunctions.

Metabolic syndrome in combination with obesity and PCOS is associated with ovulatory dysfunction, menstrual cycle disorders, decreased effectiveness of infertility treatment and ovulation induction, increased frequency of spontaneous pregnancy termination, preeclampsia, fetal macrosomia, and perinatal morbidity.