



CARBOHYDRATE AND LIPID METABOLISM
DISORDERS IN REPRODUCTIVE-AGED WOMEN
WITH OBESITY

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ABSTRACT

The article analyzes the pathogenetic mechanisms underlying carbohydrate and lipid metabolism disorders in reproductive-aged women with obesity and their impact on reproductive health. The roles of insulin resistance, hyperinsulinemia, dyslipidemia, and chronic low-grade systemic inflammation in hormonal imbalance, impaired folliculogenesis, ovulatory dysfunction, and reduced fertility are discussed. The association of obesity and metabolic dysfunction with polycystic ovary syndrome, anovulation, and infertility is also considered.

The functioning of the reproductive system is closely interconnected with the body's energy and metabolic processes. Glucose and lipid metabolism are functionally associated with ovarian steroidogenesis, folliculogenesis, ovulation, the functional state of the endometrium, and the processes involved in preparing the body for pregnancy. Therefore, alterations in metabolic homeostasis may directly or indirectly affect certain components of reproductive function.

In obesity, an increase in adipose tissue mass is accompanied by changes in its metabolic and endocrine activity. In particular, an increase in visceral adipose tissue may be associated



with insulin resistance, compensatory hyperinsulinemia, impaired lipid metabolism, and chronic low-grade inflammation. The simultaneous development of these processes creates a metabolic environment that may affect hormonal homeostasis, ovarian function, and the functional characteristics of the endometrium [5].

The clinical significance of obesity in reproductive-aged women is not limited to increased body weight. Menstrual irregularities, anovulatory cycles, hyperandrogenemia, polycystic ovary syndrome (PCOS), reduced fertility, and an increased risk of certain metabolic disorders may occur in association with obesity. However, it would be inappropriate to explain these associations by a single pathogenetic mechanism in all patients. The degree of obesity, distribution of adipose tissue, insulin sensitivity, hormonal status, and comorbidities may influence the development of the reproductive phenotype in different ways.

In this regard, studying carbohydrate and lipid metabolism parameters together with reproductive status may provide a more comprehensive understanding of the pathophysiological basis of reproductive dysfunction associated with obesity.

One of the most common components of metabolic disorders associated with obesity is insulin resistance. Insulin resistance is characterized by reduced sensitivity of peripheral tissues to the effects of insulin. Under these conditions, pancreatic β -cells increase insulin secretion in order to maintain glucose homeostasis for a certain period of time. As a result, compensatory hyperinsulinemia may develop. If this compensatory mechanism persists over a prolonged period, conditions may arise that contribute to impaired metabolic control and clinical alterations in glucose metabolism.

In the literature, elevated HOMA-IR values are frequently reported among women with obesity. Some studies have shown that HOMA-IR values above 2.0 were observed in 50–70% of the study groups [9]. However, this value cannot be regarded as a universal diagnostic threshold. The distribution of HOMA-IR varies depending on the population, age, body composition, laboratory methods, and metabolic characteristics. Therefore, HOMA-IR should preferably be interpreted in conjunction with clinical and laboratory findings.

Hyperinsulinemia may affect the reproductive endocrine system through several mechanisms. Insulin may stimulate androgen synthesis in the ovaries and may be associated with reduced hepatic production of sex hormone-binding globulin (SHBG). Consequently, the proportion of biologically active free androgens may increase [9]. A hyperandrogenic environment may interfere with normal follicular maturation and may be associated with disturbances of the ovulatory cycle, oligomenorrhea, amenorrhea, and anovulation.

This pathogenetic pathway is particularly relevant in patients with PCOS. Insulin resistance may affect ovarian steroidogenesis, while hyperandrogenemia may contribute to impaired folliculogenesis. However, the pathogenesis of PCOS is multifactorial and cannot be adequately explained by insulin resistance or obesity alone.

The biological activity of adipose tissue should also be taken into account in the interaction between obesity and insulin resistance. In addition to serving as an energy reserve, adipose tissue produces various biologically active mediators. Increased visceral adipose tissue may be accompanied by enhanced secretion of pro-inflammatory cytokines such as $\text{TNF-}\alpha$ and IL-6 [7].

These mediators may affect post-receptor insulin signaling pathways and contribute to reduced insulin sensitivity in peripheral tissues. Increased hepatic synthesis of C-reactive protein under the influence of IL-6 may be observed as one of the laboratory markers of low-



grade systemic inflammation. TNF- α may impair certain components of insulin signaling and thereby reduce glucose utilization by tissues [7].

Thus, metabolic and inflammatory processes in obesity may develop through mutually reinforcing mechanisms. Increased adipose tissue mass creates conditions for the release of pro-inflammatory mediators; inflammation contributes to reduced insulin sensitivity; and insulin resistance may persist alongside increased lipid mobilization and metabolic dysfunction. It is therefore more accurate to view these relationships not as a single linear cause-and-effect chain, but as a complex network of interacting pathophysiological processes.

Increased lipolysis in visceral adipose tissue may lead to elevated circulating levels of free fatty acids. Excessive exposure to free fatty acids may impair insulin signaling in the liver and skeletal muscles and may exert adverse metabolic effects on pancreatic β -cell function. Prolonged metabolic stress may also affect the physiological regulation of insulin secretion.

Changes in carbohydrate metabolism are often accompanied by disturbances in lipid metabolism. Altered insulin action affects the regulation of hepatic lipid synthesis and lipolysis. As a result, some patients may develop a dyslipidemic profile characterized by increased triglyceride and atherogenic lipoprotein levels and reduced high-density lipoprotein (HDL) levels.

According to the literature reviewed in this article, increased triglyceride levels were reported in 45–60% of certain groups of women with obesity, increased low-density lipoprotein (LDL) concentrations in 50–70%, and reduced HDL levels in 40–50% [5]. These figures should not be generalized to all reproductive-aged women with obesity, as the distribution of lipid profiles depends on the study population, degree of obesity, dietary patterns, physical activity, and accompanying metabolic conditions.

Dyslipidemia may also occur in association with reproductive disorders. Increased LDL and triglyceride concentrations have been associated with alterations in ovarian steroidogenesis in the context of overall metabolic dysfunction. Reduced HDL levels are often accompanied by insulin resistance and markers of cardiometabolic risk. Based on the available evidence, there is insufficient basis to consider dyslipidemia an independent and universal cause of reproductive dysfunction; a more scientifically cautious approach is to interpret it as part of the overall metabolic phenotype associated with obesity.

The effects of obesity on the reproductive system may extend beyond the ovaries. Excess adipose tissue, hyperinsulinemia, hormonal imbalance, and inflammatory mediators may affect the functional characteristics of the endometrium. Some studies have reported associations between such alterations and endometrial receptivity and the physiological conditions required for embryo implantation [5]. This mechanism may therefore represent one of the potential components contributing to the multifactorial nature of infertility associated with obesity.

PCOS is one of the most extensively studied clinical conditions in the context of the relationship between obesity and metabolic dysfunction. Insulin resistance, hyperandrogenemia, and ovulatory dysfunction may coexist within the syndrome. Some of the literature reviewed reports that the frequency of PCOS may reach 50–70% in studied groups of women with obesity [4]. Because this figure is directly dependent on the diagnostic criteria and the population selected, it should not be interpreted as a general epidemiological estimate.

When PCOS and obesity coexist, insulin resistance, dyslipidemia, and hyperandrogenemia may be more pronounced. In these patients, disturbances of folliculogenesis and ovulatory



function may be more likely to be accompanied by reduced fertility. Therefore, assessment of metabolic status is of particular clinical importance when planning pregnancy [4].

The clinical manifestations of obesity-associated metabolic disorders are often nonspecific. Chronic fatigue, reduced physical activity, exertional dyspnea, joint discomfort, and increased body weight are not specific to metabolic dysfunction but may nevertheless be considered during clinical assessment. From the reproductive perspective, oligomenorrhea, amenorrhea, menstrual irregularities, and ovulatory dysfunction may occur [1].

In such clinical situations, body mass index alone does not fully characterize metabolic status. Fasting blood glucose is one of the initial screening parameters used to assess carbohydrate metabolism. The reviewed sources indicate that a fasting glucose concentration of 5.6 mmol/L or higher may provide a basis for further evaluation [3]. Fasting insulin levels and the HOMA-IR index are used to indirectly characterize insulin resistance. However, it should be recognized that HOMA-IR thresholds vary depending on the laboratory and population.

For assessment of lipid metabolism, measurement of triglycerides, total cholesterol, LDL, and HDL is appropriate. These parameters help characterize the metabolic phenotype associated with obesity in addition to assessing cardiometabolic risk. When clinically indicated, inflammatory markers such as C-reactive protein may provide additional information. However, their alterations should not be interpreted as specific diagnostic markers of obesity.

Management of metabolic disorders associated with obesity includes weight control, a balanced diet based on energy balance, regular physical activity, and dynamic monitoring of metabolic parameters. Weight reduction may have a beneficial effect on insulin sensitivity, hyperinsulinemia, and the lipid profile. Improvement in metabolic status may be accompanied by restoration of ovulatory function and improved reproductive outcomes in some patients. However, the extent of this response depends on the patient's metabolic phenotype, presence of PCOS, degree of obesity, and other reproductive factors.

Conclusions

Analysis of the available literature indicates that carbohydrate and lipid metabolism disorders in reproductive-aged women with obesity develop through several interrelated pathophysiological mechanisms. Insulin resistance and compensatory hyperinsulinemia are common components of this metabolic phenotype and may coexist with alterations in lipid metabolism, hyperandrogenemia, and low-grade systemic inflammation.

Hyperinsulinemia may affect ovarian steroidogenesis and sex hormone-binding globulin levels, thereby contributing to an increased proportion of free androgens. A hyperandrogenic environment may, in turn, contribute to impaired folliculogenesis and ovulatory dysfunction. This mechanism may be particularly relevant in women with obesity and concomitant polycystic ovary syndrome (PCOS).

Pro-inflammatory mediators and free fatty acids released from visceral adipose tissue may contribute to reduced insulin sensitivity and the persistence of metabolic dysfunction. Therefore, obesity should be regarded not merely as excess body weight, but as a complex condition involving interacting metabolic, endocrine, and inflammatory components.

Although lipid metabolism disorders are associated with reproductive dysfunction, the available literature does not provide sufficient evidence to regard dyslipidemia as an independent and universal cause of reduced fertility. Rather, dyslipidemia should be interpreted within the broader metabolic phenotype associated with insulin resistance and obesity.



The reproductive consequences of obesity may extend beyond ovarian function. Alterations in endometrial receptivity, ovulatory dysfunction, hormonal imbalance, and chronic low-grade inflammation may collectively contribute to impaired fertility, highlighting the multifactorial nature of obesity-associated reproductive disorders.

Scientific Conclusions

1. In reproductive-aged women with obesity, insulin resistance should be considered not as an isolated disorder of carbohydrate metabolism, but as a component of a metabolic phenotype interacting with hyperinsulinemia, dyslipidemia, and low-grade systemic inflammation.

2. Hyperinsulinemia may contribute to an increased proportion of biologically active androgens through stimulation of ovarian androgen synthesis and reduction of sex hormone-binding globulin levels. This mechanism is pathogenetically associated with impaired folliculogenesis and ovulatory function.

3. Increased endocrine activity of visceral adipose tissue in obesity contributes to the persistence of metabolic dysfunction. Increased levels of TNF- α , IL-6, and free fatty acids represent potential mechanisms affecting insulin signaling and peripheral insulin sensitivity.

4. Dyslipidemia accompanying obesity should be interpreted not as an independent cause of reproductive dysfunction, but as a process occurring alongside insulin resistance and broader cardiometabolic disturbances.

5. When PCOS and obesity coexist, the interaction between insulin resistance, hyperandrogenemia, and ovulatory dysfunction may become more pronounced. However, reported percentages of PCOS prevalence depend on diagnostic criteria and the population studied and therefore should not be applied as universal epidemiological estimates.

6. The pathogenesis of obesity-associated reproductive disorders is not limited to ovarian alterations. Changes in endometrial receptivity, chronic inflammation, and metabolic-hormonal imbalance may also contribute to impaired fertility.

7. Clinical assessment of reproductive-aged women with obesity should not rely solely on body mass index. Joint assessment of glucose metabolism, insulin resistance, lipid profile, menstrual cycle, and ovulatory function provides a more comprehensive characterization of the metabolic and reproductive phenotype.

8. The use of a single universal diagnostic threshold for HOMA-IR is not scientifically justified. Its interpretation should take into account the population, laboratory method, and clinical context.

9. Early detection and individualized management of metabolic abnormalities may contribute to reducing obesity-associated cardiometabolic risk. Their effects on reproductive outcomes may vary depending on the patient's baseline metabolic and hormonal phenotype.

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