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OBESITY IN WOMEN NOT ONLY AFFECTS HER METABOLISM BUT ALSO HER REPRODUCTIVE HEALTH

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Obesity has progressed from a significant health risk to an epidemic in nowadays. Obesity is defined as body mass index (BMI) greater than 30 kg/m². Reproductive age women (20–39 years) have similar obesity rates (36.5%) compared to the general population. Obesity is characterized by increased lipid storage in adipose tissue and other metabolic organs, which leads to cellular lipid toxicity, inflammation and oxidative stress. The result is the development of metabolic dysfunctions like type II diabetes, cardiovascular disease and ultimately, reduced quality and quantity of life. Importantly, the percentages of obese and overweight adults are expected to rise to 50% by 2030.

Obesity in women not only affects her metabolism but also her reproductive health. Specifically, obese women are at increased risk for ovulatory subfertility and anovulatory infertility compared to age-matched lean women. While anovulation can be overcome with ovarian stimulation, obese women have decreased responsiveness to gonadotropins, decreased oocyte retrieval, decreased oocyte quality, reduced rates of pre-implantation embryo development and increased risk for miscarriage compared to their lean counterparts. Current research aims to define obesity-dependent mechanisms that cause these phenotypes in order to prevent or reverse female infertility.

Acute inflammation, which is triggered by tissue damage as a result of an invading pathogen or trauma, activates the release of chemokines by resident innate immune cells. These chemokines attract additional innate immune cells from the systemic circulation. At the same time, resident and infiltrating innate immune cells produce pro-inflammatory cytokines. The cytokines initiate signaling pathways at the cellular level to stimulate the expression of chemokines and cytokines as well as genes that regulate cell death, senescence and survival. The end result is phagocytosis of damaged tissue and subsequent secretion of anti-inflammatory cytokines that regulate wound repair and resolution of the inflammatory response. Chronic inflammation is defined as unregulated and persistent chemokine and cytokine synthesis and secretion. This can be caused by unresolved inflammation after tissue damage. Alternatively, environmental pressures (e.g. allergens), abnormal metabolism (e.g. microbiome changes) or persistent necrotic cell death within a tissue (e.g. obese adipocyte) can induce *de novo* inflammatory responses.

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NEW METHODS OF EARLY PREVENTION OF TOXICOSIS IN PREGNANT WOMEN

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Late toxicosis is one of the most common complications of pregnancy and is accompanied by significant hemodynamic and metabolic disorders, which are largely determined by changes in renal function and water-salt homeostasis. The study of pathogenesis and development of new methods of corrective therapy for late toxicosis of pregnant women is the most important task of modern obstetrics.

Conducted the study of the kidneys function, water and mineral balance in pregnant depends on the form of late toxicosis, and those transformations they undergo under the influence held the therapy. In our study, we examined 377 pregnant women with late toxicosis aged 18-48 years, 221 of which were first-time mothers, and 156 – second-time motherhood. All pregnant women, depending on the lane Eden therapy were divided into two groups: the control assigned 161 pregnant women, who treated the classical scheme, the main - 216 pregnant women, among whom was anomalies targeted to corrective therapy in 153, and maintenance in 63. A study of these indicators was also conducted in 40 virtually healthy non-pregnant women and 48 pregnant women. It was also found that after the main course of effective therapy until discharge from the hospital remained reduced glomerular filtration in the kidneys (84.84 ± 3.34 ml / min), their