

International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	THE IMPACT OF MATERNAL OBESITY ON PROCREATIVE CAPACITY, PREGNANCY, AND PARTURITION; A NARRATIVE REVIEW
DOI	https://doi.org/10.31435/ijitss.4(52).2026.6868
RECEIVED	05 September 2026
ACCEPTED	02 November 2026
PUBLISHED	10 November 2026
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

© The author(s) 2026.
This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

THE IMPACT OF MATERNAL OBESITY ON PROCREATIVE CAPACITY, PREGNANCY, AND PARTURITION; A NARRATIVE REVIEW

Wiktoria Jabłońska *(Corresponding Author, Email: wiktoriajablonska2001@gmail.com)*
Medical Univeristy of Warsaw, Poland
ORCID ID: 0009-0008-3472-3051

Kinga Janiszek
Medical Univeristy of Warsaw, Poland
ORCID ID: 0009-0008-4318-6681

Liwia Iwaniec
Medical Univeristy of Warsaw, Poland
ORCID ID: 0009-0006-3433-3884

Michał Uchański
Medical Univeristy of Warsaw, Poland
ORCID ID: 0009-0005-1847-8672

Iga Januszaniec
Medical Univeristy of Warsaw, Poland
ORCID ID: 0009-0009-9563-4789

Aleksander Mróz
Medical Univeristy of Warsaw, Poland
ORCID ID: 0009-0000-2702-4596

Alicja Bonder
Medical Univeristy of Warsaw, Poland
ORCID ID: 0009-0006-4893-9094

Michał Kiszka
Medical Univeristy of Warsaw, Poland
ORCID ID: 0009-0002-8490-632X

Julia Janicka
Medical Univeristy of Warsaw, Poland
ORCID ID: 0009-0001-4435-1430

Jolanta Janasik
Medical Univeristy of Warsaw, Poland
ORCID ID: 0009-0001-2303-5068

ABSTRACT

Objective: Obesity in women of reproductive age represents a growing public health problem, and excessive adipose tissue negatively impacts multiple aspects of reproductive health. This review aims to analyse the impact of maternal obesity on fertility, the course of pregnancy, and parturition, with particular emphasis on the pathological mechanisms - such as chronic inflammation, as well as endocrine and metabolic disorders—that lead to complications.
Methods: A narrative review of medical literature was conducted. Scientific databases (including PubMed and Google Scholar) were searched using combinations of keywords such as maternal obesity, fertility, insulin resistance, pregnancy complications, and obstetric anaesthesia. Peer-reviewed publications in English and Polish were included in the analysis, with a particular focus on studies featuring the latest pathophysiological findings.
Results: Obesity disrupts the functioning of the hypothalamic-pituitary-ovarian axis, impairing the quality of oocytes and embryos, which leads to conception difficulties and a higher risk of miscarriage. During pregnancy, a pathological intrauterine environment and generalised endothelial dysfunction drastically increase the risk of maternal gestational diabetes mellitus (GDM) and preeclampsia, as well as fetal macrosomia and congenital anomalies. In the peripartum period, obesity predisposes to uterine contractility disorders, a higher rate of obstetric trauma, surgical site infections, and poses significant anaesthetic challenges.
Conclusions: Maternal obesity exerts a multidirectional, detrimental impact on every stage of reproduction. The key strategy for preventing obstetric complications and long-term cardiometabolic consequences in the offspring is preconception counselling aimed at weight reduction prior to conception.

KEYWORDS

Maternal Obesity, Infertility, Pregnancy Complications, Insulin Resistance, Postpartum Period

CITATION

Jabłońska, W., Janiszek, K., Iwaniec, L., Uchański, M., Januszaniec, I., Mróz, A., Bonder, A., Kiszka, M., Janicka, J., & Janasik, J. (2026). The impact of maternal obesity on procreative capacity, pregnancy, and parturition; A narrative review. *International Journal of Innovative Technologies in Social Science*, *4*(52). https://doi.org/10.31435/ijitss.4(52).2026.6868

COPYRIGHT

© The author(s) 2026. This article is published as open access under the **Creative Commons Attribution 4.0 International License (CC BY 4.0)**, allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

Introduction

Obesity and being overweight currently represent a major global challenge. The WHO projects that by 2030, this problem will affect 2.93 billion individuals aged 20 and older, accounting for 50% of this population. Overweight individuals (BMI 25 - 30 kg/m²) will constitute 31%, individuals with class I obesity (BMI 30 - <35 kg/m²) will constitute 13%, and those with class II obesity and above (BMI ≥35 kg/m²) will constitute 6% (World Obesity Federation, 2025). Currently, the prevalence of overweightness and obesity among women of reproductive age (15–49 years) is 32% (Geberu et al., 2025), and by 2030 it is projected to reach 50%, with women with obesity (BMI ≥30 kg/m²) accounting for 20% of this group (World Obesity Federation, 2025).

Adipose tissue is an active organ that generates systemic inflammation, leading to multiple pathologies at cellular and tissue levels, including generalised insulin resistance, oxidative stress, and vascular endothelial damage. For women of reproductive age, this poses a reproductive challenge. The pathogenesis of fertility disorders in patients with obesity manifests long before a planned pregnancy. Women with excess body weight significantly more often experience hypothalamic-pituitary-ovarian (HPO) axis dysfunction, which presents as ovulatory disorders and irregular menstrual cycles (Polotsky & Doblado, 2015). As a result, a significantly prolonged time to pregnancy (TTP) and a significantly reduced efficacy of assisted reproductive technologies (ART, including in vitro fertilisation) are observed in this group of patients, which stems from decreased oocyte competence and impaired endometrial receptivity (Practice Committee of the American Society for Reproductive Medicine, 2021).

Even in the case of successful conception, a physiological pregnancy places an exceptional hemodynamic and metabolic burden on the maternal organism. The superimposition of natural adaptive processes on baseline pathologies associated with excess adipose tissue leads to a pathological failure of the body's compensatory mechanisms (American College of Obstetricians and Gynecologists, 2021). The

consequences of maternal obesity therefore encompass the entire reproductive continuum: starting from defects at the molecular level in the preconception phase, through impaired trophoblast invasion, to a cascade of severe obstetric complications, such as gestational diabetes mellitus, preeclampsia, and an elevated risk of peripartum complications and operative deliveries (American College of Obstetricians and Gynecologists, 2021; Gerede et al., 2025).

Methodology

The study was designed as a narrative literature review evaluating the impact of maternal obesity on reproductive health, the course of pregnancy, and parturition. Materials were gathered from PubMed and Google Scholar databases. Combinations of keywords related to obesity, infertility, insulin resistance, pregnancy complications, and obstetric anaesthesia were utilised. Peer-reviewed publications in English and Polish were included in the analysis, with a particular focus on the latest pathophysiological findings. The collected data were subjected to thematic analysis, categorising pathological mechanisms and complications into three main areas:

- Preconception alterations: hypothalamic-pituitary-ovarian (HPO) axis dysfunction, oocyte lipotoxicity, and embryo development.
- Pregnancy pathologies: gestational diabetes mellitus, preeclampsia, generalised endothelial dysfunction, and fetal macrosomia.
- Peripartum complications: uterine contractility disorders, obstetric trauma, wound infections, and anaesthetic challenges.

Results

1. The Impact of Obesity on Female Fertility

Pathomechanism of Ovulatory Dysfunction

Obesity significantly disrupts the function of the hypothalamic-pituitary-ovarian (HPO) axis. Adipose tissue aromatises androgens into oestrogens, leading to chronically elevated estrogen levels and negative feedback on the pituitary gland. This condition reduces the release of gonadotropins, particularly FSH, and consequently impairs normal ovarian follicle maturation (Polotsky & Doblado, 2015). Furthermore, insulin excess, primarily associated with visceral obesity, inhibits production of sex hormone-binding globulin (SHBG) in the liver, increasing circulating estrogen and androgen levels. Moreover, insulin stimulates the hypothalamus, increasing the pulse frequency of GnRH and LH secretion, and stimulates the ovaries to overproduce testosterone and androstenedione.

On the other hand, chronic excess of leptin, secreted by adipocytes, disrupts the process of folliculogenesis and blocks the sensitivity of ovarian cells to luteinizing hormone (Lonardo et al., 2024). Accumulated adipose tissue is also a source of proinflammatory cytokines. The resulting generalized inflammation impairs ovarian follicle function and directly contributes to chronic anovulation (Abir, 2010). The aforementioned dysfunctions exacerbate one another, ultimately leading to chronic anovulation, oligomenorrhea and irregular menstrual cycles, difficulties in conceiving, and the exacerbation of symptoms of polyglandular ovarian metabolic syndrome (POMS). Studies demonstrate that weight reduction in patients often reverses these detrimental mechanisms and restores normal HPO axis function (Polotsky & Doblado, 2015).

Oocyte Maturation Disorders

Elevated levels of insulin, fatty acids, and inflammatory markers in the follicular fluid of women with obesity alter the microenvironment in which the oocyte matures. This leads to alterations at the molecular, structural, and genetic levels, which directly translates into difficulties in conceiving and a higher risk of miscarriage (Broughton & Moley, 2017). An excess of free fatty acids damages the structures of the oocyte and the surrounding cumulus oophorus. This process, called lipotoxicity, drastically increases the production of deleterious reactive oxygen species (ROS) while concomitantly depleting glutathione. This leads to cell death (Wu et al., 2011). In cumulus-oocyte complexes, endoplasmic reticulum stress markers (ATF4 and GRP78) increase, exacerbating apoptosis in granulosa cells essential for proper oocyte development (Broughton & Moley, 2017). Mitochondria, which produce energy, sustain damage: they show altered

architecture (swelling, vacuolization, fewer cristae) and they cluster abnormally within the cytoplasm. The cell experiences metabolic stress and attempts to respond by increasing mitochondrial DNA copy number (Grindler & Moley, 2013).

Oocytes from obese women are less likely to reach maturity. They undergo severe disruptions during cell division (meiotic aneuploidies). Meiotic spindles become disorganised and fragmented, and chromosomes misalign, preventing proper development (Broughton & Moley, 2017). This is one of the primary reasons why oocytes from obese women fail to fertilise more frequently, and the resulting embryos harbour genetic defects, leading to early miscarriages (Broughton & Moley, 2017; Yong et al., 2023). Furthermore, clinical studies from in vitro fertilisation cycles indicate that mature oocytes retrieved from women with obesity are physically smaller - with a smaller diameter and a thinner zona pellucida—than oocytes from patients of normal BMI. This smaller size is associated with an overall inferior developmental potential (Atzmon et al., 2017).

Embryo Development and Epigenetic Programming

Beyond its direct impact on oocyte quality, maternal obesity exerts deleterious effects on the embryo. Embryos of women with obesity develop at a different rate than embryos of women with normal BMI. Importantly, studies demonstrate that up to day 3 of development (the cleavage stage), embryos may appear morphologically normal. However, oocyte-derived metabolic defects manifest later. - these embryos have a significantly lower capacity to reach the blastocyst stage on day 5. Furthermore, the blastocysts that do develop often exhibit an atypical cleavage rate and have a fewer number of trophectoderm (TE) cells, which drastically diminishes their potential for proper uterine implantation and the formation of a healthy placenta (Bellver et al., 2021; Comstock et al., 2015). These embryos exhibit impaired metabolism: they absorb glucose less efficiently while accumulating excess triglycerides. They also show reduced sensitivity to insulin and leptin, which, at a critical stage of development, deprives them of essential building blocks and inhibits their growth (Broughton & Moley, 2017). Embryonic exposure to excess free fatty acids inhibits cell division, decreases the number of nuclei within cells, and exacerbates apoptosis. There is also evidence that an imbalance of fatty acids (an excess of omega-3 relative to omega-6) in women undergoing in vitro fertilization reduces the chances of pregnancy (Jungheim et al., 2013).

Maternal obesity affects the embryo at the level of epigenetic modifications, the mechanisms controlling how genes are expressed (including through DNA methylation and histone modifications). An abnormal metabolic environment during the so-called maternal-to-zygotic transition (MZT), or zygotic genome activation, can disrupt this process. This means that obesity not only affects the embryo's chances of survival, but also permanently alters the metabolic profile of embryos that successfully develop into a pregnancy. This phenomenon causes children born from these pregnancies to be genetically and metabolically predisposed to obesity, insulin resistance, and cardiovascular diseases in adulthood (Şanlı & Kabaran, 2019).

Endometrial Receptivity and the Implantation Process

Equally destructive is the impact of obesity on the endometrium, which is the foundation for embryo implantation and the maintenance of pregnancy. Under conditions of chronic inflammation and metabolic disturbances, the uterus loses its natural receptivity, and the insulin resistance accompanying obesity results in constantly elevated doses of insulin reaching the uterus. Insulin, in turn, impairs glucose transport efficiency within the endometrial mucosa. At the same time, by stimulating growth receptors in an environment saturated with peripheral estrogens derived from adipose tissue, it induces hyperplasia. Such endometrium is non-functional from a reproductive standpoint. Furthermore, gene expression of the uterine mucosa during the implantation window is altered. Genetic studies demonstrate that in cases of implantation failure in patients with obesity, pathological activation of the immune system occurs within the endometrium; the resulting severe, local inflammation directly blocks the implantation process (McGrice & Porter, 2017). To maintain early pregnancy, the endometrium must develop an optimal vascular network. Impaired nitric oxide synthesis and calcium signaling compromise endothelial function in the spiral arteries supplying the uterus. These arteries become narrower and lose their elasticity, resulting in profound hypoxia of the implantation environment and the deprivation of the embryo of essential nutrients (Šišljagić et al., 2024). Inflammation-related implantation issues disrupt placentation. Endothelial dysfunction leads to severe complications in the later stages of pregnancy, such as pregnancy-induced hypertension or stillbirths (Broughton & Moley, 2017).

Obesity therefore radically decreases the chances of spontaneous conception and reduces the efficacy of in vitro techniques (Broughton & Moley, 2017). Understanding these mechanisms, however, carries a positive therapeutic message: weight reduction, often by a mere 5–10%, particularly in patients with POMS, naturally restores ovulation, improves the metabolic environment of the uterus, and increases live birth rate (Practice Committee of the American Society for Reproductive Medicine, 2021).

2. The Impact of Obesity on the Course of Pregnancy

Miscarriage and Early Pregnancy Loss

Due to the earlier-mentioned abnormalities, the risk of miscarriage is elevated in women with obesity. An observational cohort study of women who experienced recurrent early pregnancy loss demonstrated that the risk of miscarriage in women with obesity is 58%, compared to 37% in women of normal BMI, with obesity significantly increasing the risk of euploid miscarriages (Faruga-Lewicka et al., 2023). This indicates that early pregnancy loss in these patients is not only a consequence of the previously mentioned aneuploidies and oocyte defects but rather stems from a pathological intrauterine environment that destroys a healthy embryo (Lee et al., 2020).

The process of angiogenesis is also disrupted. Pathophysiological analyses specify that in pregnant women with obesity, there is an excessive influx of macrophages and CD163+ cells into the decidua and placental villi. These macrophages adopt a highly proinflammatory profile, leading to a local, toxic overproduction of cytokines, such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α). Furthermore, a high-fat diet and obesity decrease the expression of interferon-gamma (IFN-γ) in natural killer (NK) cells present in the uterus, which directly impairs the process of angiogenesis in early pregnancy. The absence of a dense and healthy vascular network precludes proper nourishment of the implanting embryo, leading to its hypoxia and subsequent demise (Wang et al., 2025). In early pregnancy, placental tissues possess insulin receptors on their surface that are significantly more numerous and active than at later stages of gestation. Hyperinsulinemia disrupts physiological signaling pathways within the early trophoblast, causing the cells to prematurely arrest their division and migration deep into the uterine tissues (Catalano & Shankar, 2017).

Placental Insufficiency and Intrauterine Fetal Demise

The consequences of intrauterine defects from the first weeks of pregnancy, particularly impaired trophoblast invasion, manifest in the later stages of fetal development. An abnormally developed placenta becomes insufficient, which, coupled with exacerbating maternal vascular and metabolic complications, drastically increases the risk of intrauterine fetal demise in the second and third trimesters (Aune et al., 2014; Catalano & Shankar, 2017).

In the later stages of pregnancy, an insufficient placenta is unable to meet the fetal demand for oxygen and nutrients. The fetus experiences progressive hypoxia and growth restriction, which in extreme cases leads to its demise in advanced gestation. Additionally, obesity increases the risk of obstructive sleep apnea. Nocturnal episodes of breathing cessation induce sudden drops in maternal oxygen saturation. For the developing fetus, this translates into recurrent, drastic interruptions in oxygen supply (Catalano & Shankar, 2017).

Fetal Congenital Anomalies and Diagnostic Challenges

As a consequence of exposure to high levels of glucose and free fatty acids, there is an increased risk of fetal congenital anomalies, such as neural tube defects and congenital heart defects, including ventricular septal defects, aortic arch defects, patent ductus arteriosus, congestive defects, as well as left and right ventricular outflow tract defects (Faruga-Lewicka et al., 2023). Embryos with such anomalies often survive the initial stage of pregnancy; however, critical structural defects manifest as the fetus grows, leading to late miscarriage or demise in the second trimester (Catalano & Shankar, 2017).

Studies also highlight a purely mechanical and diagnostic aspect. A thick layer of maternal adipose tissue attenuates ultrasound waves, which diminishes the clarity of the ultrasound image, delaying the diagnosis of the aforementioned congenital anomalies. Furthermore, in the third trimester, obesity impedes accurate cardiotocographic monitoring and causes the mother to perceive weakening fetal movements significantly less intensely and at a later stage. When the fetus begins to manifest signs of hypoxia, medical intervention is occasionally implemented with a delay (Haq et al., 2024).

3. Maternal Metabolic and Vascular Complications

Intrauterine disorders and fetal complications represent just one part of the problem. Obesity exerts an equally destructive impact on the mother's organism, exacerbating the physiological burdens associated with pregnancy. A normally progressing pregnancy is a state of exceptional challenge for the body - it is a period of physiological insulin resistance, increased cardiac output, and a heightened prothrombotic state. In normal-weight women, compensatory mechanisms smoothly adapt to these changes. However, in patients with overweight and obesity, baseline abnormalities—such as chronic inflammation of adipose tissue, pre-existing

insulin resistance, and vascular endothelial damage—are superimposed on the physiology of pregnancy, leading to their pathological failure. Consequently, obesity becomes the primary catalyst for the development of the most severe systemic maternal complications, which primarily include gestational diabetes mellitus, hypertensive disorders including preeclampsia, and venous thromboembolism.

Diabetes

From the second trimester of pregnancy, the placenta begins to produce hormones with diabetogenic activity, primarily placental lactogen, placental growth hormone, corticotropin-releasing hormone, and progesterone. Their function is to decrease maternal tissue sensitivity to insulin to ensure a constant, uninterrupted transport of glucose to the developing fetus. In a healthy woman, the pancreas compensates for this by increasing insulin production two- or even threefold, maintaining maternal glycemia within the normal limits (Haq et al., 2024). In pregnant women with obesity, these physiological adaptive mechanisms decompensate. Excessive accumulation of adipose tissue, particularly visceral fat, induces systemic insulin resistance as early as the preconception period. This phenomenon is driven by the pathological dysfunction of hypertrophic adipocytes, which exhibit increased secretion of free fatty acids (FFAs) and proinflammatory cytokines (including TNF-alpha and interleukin-6), with a concurrent decrease in the synthesis of adiponectin—a key adipokine that sensitizes tissues to insulin. The impact of placental hormones on pre-existing insulin resistance further intensifies the insensitivity of peripheral tissues to insulin. Under conditions of such severe tissue resistance, the compensatory mechanisms of the pancreas fail (leading to the so-called exhaustion of pancreatic islet β-cells), resulting in a relative insulin deficit. Consequently, this leads to impaired cellular glucose uptake, the development of chronic hyperglycemia, and ultimately—the clinical manifestation of gestational diabetes mellitus (Catalano & Shankar, 2017).

Venous Thromboembolism

Pregnancy is a physiological state of hypercoagulability, which prepares the organism to minimize blood loss during delivery. In pregnant women of normal BMI, this rarely leads to thromboembolic complications. However, in women with obesity, pathological vascular changes induced by excess adipose tissue increase the risk of these complications. Statistics indicate that 57% of fatal pulmonary embolism cases among pregnant women in the United Kingdom involved women diagnosed with obesity (Haq et al., 2024).

The elevated risk stems from the increased exacerbation of the three components of Virchow's triad. Venous stasis is caused by the uterus compressing the inferior vena cava and the iliac veins. This effect is amplified by a large volume of visceral adipose tissue. Additionally, in advanced pregnancy, women with obesity are characterized by statistically lower mobility and physical activity, which directly impedes venous return from the lower extremities (Haq et al., 2024). Chronic inflammation and frequently co-occurring arterial hypertension lead to vascular endothelial damage and, consequently, thrombus formation. In addition to proinflammatory cytokines, adipose tissue also produces plasminogen activator inhibitor, and its high concentration blocks the dissolution of small thrombi forming in the veins (Catalano & Shankar, 2017).

Furthermore, medical interventions, such as prolonged antepartum hospitalization and cesarean section, which are required more frequently in patients with obesity than in those with normal BMI, contribute to an increase in thrombotic risk.

Preeclampsia

Preeclampsia is one of the most severe perinatal complications, posing a direct threat to the life of the mother and the fetus. It typically develops after the 20th week of gestation and occurs in 2–4% of pregnancies (Schiavone et al., 2024). The key defect underlying preeclampsia is abnormal trophoblast invasion in the first trimester of pregnancy. This process is impaired in women with obesity, as described above. In the second and third trimesters, when fetal oxygen demand increases drastically, chronic ischemia and hypoxia of the placental tissue occur. The hypoxic placenta begins to produce potent inflammatory and antiangiogenic factors (such as sFlt-1), which, in patients with excess body weight, is superimposed upon the pre-existing, chronic systemic inflammation. Consequently, the inflammatory response originating from adipose tissue exacerbates the response of the hypoxic placenta, stimulating a robust expression of antiangiogenic factors and leading to generalized endothelial dysfunction. Additionally, low adiponectin levels, caused by adipokine dysregulation, alongside increased concentrations of leptin and resistin, lead to vascular endothelial damage. The pivotal process that ultimately triggers the symptoms of preeclampsia is oxidative stress. An excess of free radicals also damages the endothelium and drastically reduces the bioavailability of nitric oxide (NO)—the primary

vasodilator. Disruption of the L-arginine/NO pathway precludes blood pressure regulation. As a result, maternal arteries lose their capacity to adapt physiologically to the increased stroke volume during pregnancy. This results in generalized vasoconstriction, proteinuria, and the development of severe arterial hypertension (Schiavone et al., 2024).

4. Fetal and Neonatal Complications

Macrosomia

Maternal obesity has significant consequences for fetal development. As a consequence of decreased maternal adiponectin concentrations and hyperglycemia, there is an increased transplacental delivery of nutrients, resulting in fetal macrosomia (Faruga-Lewicka et al., 2023). This phenomenon also stems from fetal insulin excess, which acts as a growth hormone (Catalano & Shankar, 2017).

Neonatal Metabolic and Electrolyte Disturbances

Infants of mothers with obesity are born with elevated umbilical cord blood insulin and glucose levels. In the first 24 hours of life, these infants exhibit insulin resistance, which programs their organism to accumulate adipose tissue. Such children have an increased susceptibility to the early development of metabolic syndrome and diabetes later in life (Hajagos-Tóth et al., 2017). Due to abnormal insulin and glucose concentrations, normal parathyroid hormone secretion is inhibited. Consequently, this leads to disturbances in calcium and magnesium homeostasis, which clinically poses a risk of neuromuscular hyperexcitability, tremors, and seizures (Aune et al., 2014).

As a result of chronic intrauterine hypoxia, neonatal polycythemia occurs, increasing the risk of thrombosis and pathological jaundice (Aune et al., 2014).

Infant Mortality

There is a proven linear correlation between maternal BMI and infant mortality - this risk is lowest in normal-weight mothers and increases with each class of obesity (Faruga-Lewicka et al., 2023). This elevated neonatal and infant mortality is the result of multiple overlapping pathomechanisms. Fetal macrosomia increases the risk of shoulder dystocia and difficult, prolonged labor. These complications result in profound infant hypoxia (hypoxic-ischemic encephalopathy) and translate into mortality during the first days of life (Aune et al., 2014; Johansson et al., 2014). The chances of survival are further diminished by iatrogenic prematurity, resulting from the necessity of an emergency, premature delivery due to life-threatening maternal complications, such as severe preeclampsia (Johansson et al., 2014). Extreme organ immaturity in such preterm infants is frequently accompanied by respiratory distress syndrome (RDS), as maternal insulin resistance stimulates insulin overproduction by the fetal pancreas, which directly inhibits the synthesis of surfactant necessary for alveolar expansion (Aune et al., 2014). Even after the early perinatal risk resolves, the threat does not subside, as long-term exposure to a toxic, inflammatory intrauterine environment leads to subtle cardiometabolic damage and neurological defects in the infant. These disrupt the central regulation of respiration, which explains the proven, maternal BMI-dependent correlation with an increased risk of Sudden Infant Death Syndrome (SIDS) (Johansson et al., 2014).

5. Labor and the Postpartum Period

Obesity is one of the primary risk factors disrupting the course of labor and recovery during the puerperium. Excess adipose tissue and the associated metabolic disorders directly affect the mechanics and dynamics of labor. The consequences of these abnormalities are frequently preterm birth and cesarean section.

Uterine Contractility Disorders

In pregnant women with obesity, elevated blood cholesterol concentrations, through their effect on cell membranes, cause the disruption of signaling pathways in smooth muscle cells, including those of the uterus. Concurrently, a high-cholesterol and high-fat diet decreases the expression of proteins crucial for uterine contractions and elevates cholesterol levels in late pregnancy, which exerts an inhibitory effect on the myometrium. Elevated leptin levels inhibit uterine contractions and stimulate the release of prostaglandin E by adipose tissue and the placenta. This leads to impaired collagen degradation in late pregnancy, which impedes cervical dilation, significantly delaying labor (Hajagos-Tóth et al., 2017).

Obstetric Trauma

Increased fetal weight and the occurrence of shoulder dystocia prolong the duration of labor and increase the risk of maternal trauma. In deliveries complicated by dystocia, the risk of a ≥3rd-degree perineal laceration is 16.1%, compared to 5.7% in deliveries without dystocia (Jeppegaard et al., 2024). Fetal macrosomia frequently demands the application of operative maneuvers (e.g., the McRoberts maneuver, forceps, vacuum extraction) and episiotomy, which, under difficult anatomical conditions, increase the risk of third- and fourth-degree perineal lacerations, cervical injuries, and transient peripheral neuralgia. In extreme cases, symphysis pubis diastasis and uterine rupture may occur. Furthermore, uterine overdistension by a large fetus and prolonged labor lead to postpartum uterine atony, which is a direct cause of hemorrhage (Tsikouras et al., 2024).

Wound Healing and Infections

Perineal and cesarean section wounds heal significantly slower and face markedly higher complication rates in patients with obesity. Poor vascularity of adipose tissue is associated with restricted delivery of oxygen, nutrients, and cellular elements. Immune system impairment in obesity limits the capacity to combat bacterial infections. The lack of an immediate and continuous supply of nutrients to the wound area directly inhibits the healing process, creating favorable conditions for the development of infection (Sutardji'in et al., 2025). Studies indicate that women with class II and class III obesity have over a 50% higher risk of infection compared to normal-weight women during an 8-week follow-up (Axelsson et al., 2023). These infections involved the endometrium (endometritis) and cesarean section wounds. Additionally, the risk of wound infection increases in the case of an emergency cesarean section (Sutardji'in et al., 2025). Unplanned surgery is associated with compromised preparation of the surgical field, frequent premature rupture of membranes, and prolonged labor. However, elevated maternal BMI was not associated with an increased risk of perineal wound infection. A key determinant of perineal tissue infections is obstetric anal sphincter injuries (OASIS). Women with class II and class III obesity were characterized by a lower incidence of OASIS compared to normal-weight patients (Axelsson et al., 2023).

Anesthetic Challenges

Adipose tissue accumulation directly affects respiratory and cardiovascular dynamics. A decrease in functional residual capacity (FRC) entails a reduction in vital capacity (VC) and expiratory volume in a patient with obesity assuming the supine position or undergoing spinal anesthesia. This results in highly rapid oxygen desaturation during periods of apnea or sedation. Furthermore, obstructive sleep apnea poses a risk of acute ventilatory disturbances. Compression of the inferior vena cava by adipose tissue predisposes to inferior vena cava compression syndrome (supine hypotensive syndrome), which manifests as hypotension in the supine position and sudden drops in cardiac output (Ismail & Ismail, 2025).

Abundant adipose tissue complicates the administration of neuraxial anesthesia. This is associated with difficulties in identifying anatomical landmarks, resulting in a higher number of puncture attempts and needle position adjustments. As the BMI increases, so does the risk of an incomplete block, technical failure, and delayed recognition of epidural catheter failure (Ismail & Ismail, 2025). In situations where the administration of neuraxial anesthesia is not feasible, conversion to emergency general anesthesia is frequently required. Consequently, the risks associated with intubation and ventilation arise. Due to elevated intra-abdominal pressure and frequent gastroesophageal reflux, patients with obesity undergoing general anesthesia are particularly susceptible to aspiration pneumonia (Patel & Habib, 2021).

The consequences of these challenges include postpartum respiratory complications, presenting as episodes of hypoventilation and desaturation, prolonged duration of procedures and surgeries, and a higher risk of thromboembolic complications (Bomba-Opon et al., 2023). Moreover, obesity constitutes one of the most significant and consistent predictors of mortality associated with obstetric anesthesia (Ismail & Ismail, 2025).

Discussion

The conducted literature review unequivocally indicates that maternal obesity is not merely a mechanical burden on the organism, but a chronic meta-inflammatory state that impairs the functioning of the reproductive system at every stage. The analysis of pathomechanisms reveals a common denominator for seemingly disparate complications: chronic inflammation, insulin resistance, oxidative stress, and vascular endothelial dysfunction. The very same processes that, in the preconception phase, induce oocyte lipotoxicity and impede embryo implantation, lead to metabolic (gestational diabetes mellitus) and vascular (preeclampsia) decompensation in the second and third trimesters of pregnancy.

Contemporary literature strongly emphasizes that advances in the field of assisted reproductive technologies (ART) do not provide a definitive solution to the problem of obesity-related infertility. As demonstrated, maternal obesity induces molecular defects in oocytes and alters endometrial immunity, even the transfer of a morphologically normal embryo carries a higher risk of implantation failure or miscarriage. This phenomenon highlights the need for a paradigm shift in treatment: instead of bypassing the problem through the rapid qualification of patients with obesity for in vitro procedures, reproductive medicine should focus on optimizing the metabolic environment prior to conception.

Recent studies have drawn particular attention to the phenomenon of epigenetic programming. Maternal obesity ceases to be treated solely as a risk factor for perinatal complications and becomes a vector for the intergenerational transmission of metabolic diseases. The exposure of the developing fetus to hyperglycemia, hyperinsulinemia, and an excess of free fatty acids permanently alters its metabolism, thereby perpetuating the "vicious cycle" of the obesity epidemic in subsequent generations.

Conclusions

Maternal obesity exerts a multidirectional, detrimental impact on every stage of reproduction. The key strategy for preventing obstetric complications and long-term cardiometabolic consequences in the offspring is preconception counseling aimed at weight reduction prior to conception. The direction of future research should focus on the search for targeted therapies shielding oocyte mitochondria from lipotoxicity and pharmacological agents improving trophoblast invasion in patients with a high BMI. Nevertheless, at the current stage of medical knowledge, the only fully effective management remains the patient's weight reduction.

Ethical Considerations: As this study is a narrative review of existing literature, ethics committee approval was not required. No conflicts of interest to declare.

REFERENCES

1. Abir, R. (2010). Workshop 7: Obesity and reproduction. Effects of obesity on ovarian function. *European Journal of Clinical Investigation, 40*, 76. https://www.ovid.com/journals/ejci/abstract/00003653-201003001-00222~effects-of-obesity-on-ovarian-function-705
2. American College of Obstetricians and Gynecologists. (2021). ACOG committee opinion, number 819: Informed consent and shared decision making in obstetrics and gynecology. *Obstetrics & Gynecology*, *137*(2), e34-e41. https://doi.org/10.1097/AOG.0000000000004247
3. Atzmon, Y., Shoshan-Karchovsky, E., Michaeli, M., Aslih, N., Shrem, G., Ellenbogen, A., & Shalom-Paz, E. (2017). Obesity results with smaller oocyte in in vitro fertilization/intracytoplasmic sperm injection cycles-a prospective study. *Journal of Assisted Reproduction and Genetics*, *34*(9), 1145-1151. https://doi.org/10.1007/s10815-017-0975-6
4. Aune, D., Saugstad, O. D., Henriksen, T., & Tonstad, S. (2014). Maternal body mass index and the risk of fetal death, stillbirth, and infant death: A systematic review and meta-analysis. *JAMA*, *311*(15), 1536-1546. https://doi.org/10.1001/jama.2014.2269
5. Axelsson, D., Brynhildsen, J., & Blomberg, M. (2023). Maternal obesity and the risk of postpartum infections according to mode of delivery. *The Journal of Maternal-Fetal & Neonatal Medicine*, *36*(2), 2245102. https://doi.org/10.1080/14767058.2023.2245102

6. Bellver, J., Brandão, P., Alegre, L., & Meseguer, M. (2021). Blastocyst formation is similar in obese and normal weight women: A morphokinetic study. *Human Reproduction*, *36*(12), 3062-3073. https://doi.org/10.1093/humrep/deab212
7. Bomba-Opon, D., Gutaj, P., Kedzia, M., Leszczynska-Gorzelak, B., Majewska, A., Radzicka-Mularczyk, S., Stanirowski, P., & Wender-Ozegowska, E. (2023). Guidelines of the Polish Society of Gynecologists and Obstetricians on the obstetric care of women with obesity. *Ginekologia Polska*, *94*(12), 1011-1029. https://doi.org/10.5603/gpl.97361
8. Broughton, D. E., & Moley, K. H. (2017). Obesity and female infertility: Potential mediators of obesity's impact. *Fertility and Sterility*, *107*(4), 840-847. https://doi.org/10.1016/j.fertnstert.2017.01.017
9. Catalano, P. M., & Shankar, K. (2017). Obesity and pregnancy: Mechanisms of short term and long term adverse consequences for mother and child. *BMJ*, *356*, j1. https://doi.org/10.1136/bmj.j1
10. Comstock, I. A., Kim, S., Behr, B., & Lathi, R. B. (2015). Increased body mass index negatively impacts blastocyst formation rate in normal responders undergoing in vitro fertilization. *Journal of Assisted Reproduction and Genetics*, *32*(9), 1299-1304. https://doi.org/10.1007/s10815-015-0515-1
11. Faruga-Lewicka, W., Pietraszko, Z., Staśkiewicz, W., Kardas, M., & Kardas, J. (2023). Eating habits of football players training in sports clubs in the Żywiec district. *Journal of Education, Health and Sport*, *13*(4), 256-265. https://apcz.umk.pl/JEHS/article/view/42667
12. Geberu, D. M., Abera, K. M., Tsega, Y., et al. (2025). Pooled prevalence and factors of overweight/obesity among women of reproductive age in low and middle-income countries with high maternal mortality: A multi-level analysis of recent demographic and health surveys. *PLoS One*, *20*(10), e0316962. https://doi.org/10.1371/journal.pone.0316962
13. Gerede, A., Danavasi, M., Stavros, S., Potiris, A., Zikopoulos, A., Moustakli, E., Skentou, C., Domali, E., Nikolettos, N., & Eleftheriades, M. (2025). Obesity and pregnancy: Impact on childbirth timing, delivery mode, and maternal recovery: An update. *Medical Sciences*, *13*(3), 182. https://doi.org/10.3390/medsci13030182
14. Grindler, N. M., & Moley, K. H. (2013). Maternal obesity, infertility and mitochondrial dysfunction: Potential mechanisms emerging from mouse model systems. *Molecular Human Reproduction*, *19*(8), 486-494. https://doi.org/10.1093/molehr/gat026
15. Hajagos-Tóth, J., Ducza, E., Samavati, R., Vari, S. G., & Gaspar, R. (2017). Obesity in pregnancy: A novel concept on the roles of adipokines in uterine contractility. *Croatian Medical Journal*, *58*(2), 96-104. https://doi.org/10.3325/cmj.2017.58.96
16. Haq, N., Chamarthi, V. S., & Ayabe, A. (2024). Obesity in pregnancy. In *StatPearls*. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK572113/
17. Ismail, N., & Ismail, M. F. (2025). Maternal obesity and obstetric anesthesia: A comprehensive review of challenges and clinical strategies. *Asian Journal of Medicine and Health*, *23*(3), 105-111. https://journalajmah.com/index.php/AJMAH/article/view/1193
18. Jeppegaard, M., Larsen, M. H., Thams, A. B., et al. (2024). Incidence of shoulder dystocia and risk factors for recurrence in the subsequent pregnancy—A historical register-based cohort study. *Acta Obstetricia et Gynecologica Scandinavica*, *103*(10), 1975-1984. https://doi.org/10.1111/aogs.14784
19. Johansson, S., Villamor, E., Altman, M., Bonamy, A. K., Granath, F., & Cnattingius, S. (2014). Maternal overweight and obesity in early pregnancy and risk of infant mortality: A population based cohort study in Sweden. *BMJ*, *349*, g6572. https://doi.org/10.1136/bmj.g6572
20. Jungheim, E. S., Frolova, A. I., Jiang, H., & Riley, J. K. (2013). Relationship between serum polyunsaturated fatty acids and pregnancy in women undergoing in vitro fertilization. *The Journal of Clinical Endocrinology & Metabolism*, *98*(8), E1364-E1368. https://doi.org/10.1210/jc.2012-4115
21. Lee, J. C., Bernardi, L. A., & Boots, C. E. (2020). The association of euploid miscarriage with obesity. *F&S Reports*, *1*(2), 142-148. https://doi.org/10.1016/j.xfre.2020.05.011
22. Lonardo, M. S., Cacciapuoti, N., Guida, B., Di Lorenzo, M., Chiurazzi, M., Damiano, S., & Menale, C. (2024). Hypothalamic-Ovarian axis and adiposity relationship in polycystic ovary syndrome: Physiopathology and therapeutic options for the management of metabolic and inflammatory aspects. *Current Obesity Reports*, *13*(1), 51-70. https://doi.org/10.1007/s13679-023-00531-2
23. Sutardji'in, S., Setiowati, D., & Famaningrum, W. (2025). Maternal obesity and cesarean surgical wound infection: An evidence-based reassessment of risk. *MIN*, *8*(8), 1058-1064. https://doi.org/10.33024/minh.v8i8.1572
24. McGrice, M., & Porter, J. (2017). The effect of low carbohydrate diets on fertility hormones and outcomes in overweight and obese women: A systematic review. *Nutrients*, *9*(3), 204. https://doi.org/10.3390/nu9030204

25. Patel, S. D., & Habib, A. S. (2021). Anaesthesia for the parturient with obesity. *BJA Education*, *21*(5), 180-186. https://doi.org/10.1016/j.bjae.2020.12.007
26. Polotsky, A. J., & Doblado, M. A. (2015). Obesity and the HPO axis. In E. S. Jungheim (Ed.), *Obesity and fertility*. Springer. https://doi.org/10.1007/978-1-4939-2611-4_2
27. Practice Committee of the American Society for Reproductive Medicine. (2021). Obesity and reproduction: A committee opinion. *Fertility and Sterility*, *116*(5), 1266-1285. https://doi.org/10.1016/j.fertnstert.2021.08.018
28. Şanlı, E., & Kabaran, S. (2019). Maternal obesity, maternal overnutrition and fetal programming: Effects of epigenetic mechanisms on the development of metabolic disorders. *Current Genomics*, *20*(6), 419-427. https://doi.org/10.2174/1389202920666191030092225
29. Schiavone, M. J., Pérez, M. P., Aquieri, A., Nosetto, D., Pronotti, M. V., Mazzei, M., Kudrle, C., & Avaca, H. (2024). The role of obesity in the development of preeclampsia. *Current Hypertension Reports*, *26*(6), 247-258. https://doi.org/10.1007/s11906-024-01299-z
30. Šišljagić, D., Blažetić, S., Heffer, M., Vranješ Delać, M., & Muller, A. (2024). The interplay of uterine health and obesity: A comprehensive review. *Biomedicines*, *12*(12), 2801. https://doi.org/10.3390/biomedicines12122801
31. Tsikouras, P., Kotanidou, S., Nikolettos, K., Kritsotaki, N., Bothou, A., Andreou, S., Nalmpanti, T., Chalkia, K., Spanakis, V., Peitsidis, P., Iatrakis, G., & Nikolettos, N. (2024). Shoulder dystocia: A comprehensive literature review on diagnosis, prevention, complications, prognosis, and management. *Journal of Personalized Medicine*, *14*(6), 586. https://doi.org/10.3390/jpm14060586
32. Wang, R. Q., Deng, Z. M., Chen, G. T., Dai, F. F., & Xia, L. B. (2025). Obesity and recurrent spontaneous abortion: The crucial role of weight management in pregnancy. *Reproductive Biology and Endocrinology*, *23*(1), 10. https://doi.org/10.1186/s12958-024-01326-3
33. World Obesity Federation. (2025). *World obesity atlas*. Retrieved from https://www.worldobesity.org/
34. Wu, L. L., Norman, R. J., & Robker, R. L. (2011). The impact of obesity on oocytes: Evidence for lipotoxicity mechanisms. *Reproduction, Fertility and Development*, *24*(1), 29-34. https://doi.org/10.1071/RD11904
35. Yong, W., Wang, J., Leng, Y., Li, L., & Wang, H. (2023). Role of obesity in female reproduction. *International Journal of Medical Sciences*, *20*(3), 366-375. https://doi.org/10.7150/ijms.80189