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Narrative Review of Obesity and Fertility Outcomes

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ABSTRACT

Obesity has become one of the most significant public health challenges of the 21st century and is increasingly recognized as a major determinant of reproductive health. This narrative review synthesizes current evidence on the effects of obesity on both female and male fertility, highlighting the complex physiological, metabolic, and endocrine pathways through which excess adiposity impairs reproductive outcomes. In women, obesity disrupts the hypothalamic–pituitary gonadal axis, contributes to anovulation, prolongs time to pregnancy, increases miscarriage risk, and reduces the success of assisted reproductive technologies (ART). In men, obesity is associated with compromised semen parameters, hormonal imbalance, delayed conception, and potential adverse effects on embryonic development. Central adiposity, metabolic syndrome, and obesity-mediated comorbidities further contribute to subfertility in both sexes. Pregnancy complications, including gestational diabetes, hypertensive disorders, preterm birth, and cesarean delivery, occur in a dose-dependent relationship with rising maternal BMI. The review also evaluates current interventions, including lifestyle modification, pharmacotherapy, and bariatric surgery, and explores their respective impacts on fertility outcomes. Despite growing evidence, significant methodological limitations and heterogeneity in definitions of obesity and fertility hinder comparability across studies. Strengthening standardized measures, improving long-term population studies, and enhancing integrated clinical and public health strategies are essential for reducing obesity-related reproductive burdens. Overall, obesity substantially influences reproductive physiology and outcomes, underscoring the need for early preventive care and targeted interventions in reproductive-age populations.

Keywords: Obesity and Fertility, Reproductive Health, Anovulation and Time to Pregnancy, Assisted Reproductive Technology (ART), and Metabolic Syndrome and Reproductive Outcomes.

INTRODUCTION

Increasing rates of obesity pose a challenge across multiple sectors: personal, social, environmental, mental, and medical. Properly framed, obesity presents perhaps the greatest public health challenge of the 21st century [1]. Obesity is the most common disorder of the endocrine system in both developed and developing countries. High adiposity is a long-standing, unambiguous risk factor for diverse adverse health effects and outcomes, with an increase in duration and severity of exposure leading to a commensurate increase in risk of cardiovascular disease, diabetes, dyslipidemia, osteoarthritis, pregnancy complications, certain cancers, and mortality. Rising maternal obesity is responsible for worsening perinatal outcomes and undue suffering for both mothers and children [2]. Obesity disrupts normal physiologic processes such as reproduction across animal, human, and population studies. The situation is particularly serious in women of reproductive age, who are directly or indirectly counted as one of the top five reasons for seeking reproductive assistance globally [3]. Obesity represents a progressive condition, defined by an increase in duration, severity, and type of additional comorbidities, with subsequent aggravation to both the mother and the offspring.

Obesity and Reproductive Physiology

Obesity compromises reproductive health, with obesity-related endocrine dysfunction and metabolic derangements disrupting the hypothalamic-pituitary-gonadal (HPG) axis in women, impairing both ovarian and uterine functions, and disrupting spermatogenesis and steroidogenesis in men [5]. Obesity is characterized by an excess

of body fat associated with adverse health outcomes [4]. The WHO defines obesity as a body mass index (BMI) ≥30 kg/m2. However, determining obesity solely by BMI does not consider fat distribution and associated metabolic dysfunction [6]. For example, visceral fat accumulation is a major component of metabolic syndrome and increases cardiometabolic risk, while the ectopic accumulation of fat in non-adipose tissues, such as liver, muscle, and pancreas, leads to an increase in insulin resistance [7]. Thus, other clinical definitions of obesity include waist circumference (WC), abdominal circumference, waist–hip ratio, and metabolic syndrome components [7]. The International Federation of Gynecology and Obstetrics defines metabolic obesity as 1 of the following: a waist circumference >94 cm (European) or >90 cm (Asian) and/or 2 of the following components: (i) hypertension; (ii) hyperglycemia; and (iii) dyslipidemia [3]. Obesity has a profoundly negative impact on several aspects of human reproduction. On female fertility, infertility prevalence is reported to variably increase with BMI. Time to pregnancy (TTP) is prolonged, and the probability of spontaneous conception drops. The risk of miscarriage increases, particularly if obesity is accompanied by other comorbidities [4]. Diminished oocyte quality is noted in women with obesity, and the involvement of the gut microbiome in this process has been proposed [5]. In men, obesity is also linked to reduced fertility, although the molecular pathways remain less established than in females [2].

Obesity and Female Fertility Outcomes

Obesity affects up to one-third of reproductive-aged women worldwide and has become a significant health concern. Studies indicate that obesity reduces female fertility and adversely influences assisted reproductive technology (ART) outcomes [2]. Obesity negatively impacts the physiological processes required for reproduction, including gametogenesis, fertilization, embryonic development, and gestational physiology. Several adiposity measures better convey the impact of obesity, such as body mass index (BMI), central adiposity, and metabolic syndrome. Definitions of obesity vary across studies [7]. Anovulation, impaired oocyte quality, and embryo implantation failure are the most significant reproductive disorders related to obesity [3]. Thresholds for these dysfunctions vary across populations. Guidelines recommended a BMI threshold of 30 kg/m^2 and/or a waist circumference criterion of 80 cm for assessing obesity-induced reproductive alterations in a European cohort. Although obesity is often associated with irregular menstrual cycles, ~25% of affected individuals still exhibit amenorrhea [8]. Using data from large cohorts, several studies characterized FSH, LH, estradiol, and ovarian antrum dynamics in the menstrual cycle and analyzed hormonal status before and after successful weight-loss interventions. Neither reproductive hormone characteristics nor ovulation presence significantly changed in obese individuals relative to non-obese counterparts, suggesting that ovulatory disorders may not represent a general population affliction [4]. Despite observations of early cycle dysfunction, impaired ovulatory recovery was uncommon before and following interventions [4]. The majority of overweight or obese women achieve natural conception without ART, underscoring the importance of these disturbances in untreated subfertility.

Anovulation and Menstrual Function

Increasing body mass index (BMI) and abdominal obesity decrease the likelihood of regular menstrual cycles. Ganer et al. specifically identified a central waist circumference threshold of 81 cm in women of childbearing age that predicts the impairment of menstrual cycles, beyond which central adiposity is highly likely to negatively interfere with reproductive health. Chen et al. found that most menstrual cycles become irregular as soon as age reaches 35 years; however, women with a central waist circumference of 83 cm or higher are likely to have menstrual cycles become irregular at age [25]. Conversely, women with very low BMI, regardless of age, were less likely to report irregular menstrual cycles [6]. Obesity influences ovulatory function and progesterone levels, making weight management efficacious in women with obesity who have anovulatory cycles or polycystic ovarian syndrome (PCOS). Studies reported that nearly 70% of anovulatory women resume ovulation after weight loss [13]. Obesity is a prevalent causal factor of anovulation and menstrual irregularity in the general population, with a potential impact of population composition in this regard: in a cross-sectional survey of more than 4,000 Chinese women aged 20–49 years, with a population prevalence of normal-weight 63% and 29% of total obesity, overweight, and obesity contributed to anovulation [10]. Overweight women had less extreme anovulatory cycles than with other conditions, such as primary ovarian insufficiency (POI) or hormonal treatments [4]. Forty to sixty percent of the population with a BMI exceeding 35 kg/m2 possesses anovulatory cycles. PCOS diagnosis includes oligo-, amenorrhea, and anovulation as symptoms. Patient population with primary anovulation may contain a high prevalence of PCOS, due to its influence on the population at large, making the association between BMI and overall anovulation difficult to ascertain [6]. Analysis of "pathophysiological consequences of obesity" in 577 overweight-anovulatory women confirmed other conditions as the major contributors to irregularity, establishing a still significant link between anovulation and obesity [6].

Natural Conception and Time to Pregnancy

Maternal obesity is a major concern for public health and reproductive medicine [2]. Obese women face longer times to pregnancy, affecting the chance of natural conception. Evidence shows that female patients experiencing

obesity and same-sex relationships present longer time frames to pregnancy, indicating that clinical endpoints are relevant for this specific population [11]. Women with obesity face an extended time to pregnancy and lower natural conception rates. Data on time to pregnancy within the defining characteristics of natural conception confirm these findings [10]. The effect of maternal adiposity on time to pregnancy is typically reported as a median extension. Combinations of these factors account for 70%–90% of the impact. All available evidence highlights a clear dose–response association between increasing body mass index (BMI) and extended time to pregnancy [9].

Assisted Reproductive Technology Outcomes

Obesity has been linked to a range of health and fertility conditions, including lower chances of spontaneous pregnancy and ART success, longer time to pregnancy, and poorer outcomes once pregnancy is achieved [5]. The characteristics of the female reproductive axis are crucial in the increasingly common assisted reproductive technologies (ART) used to aid conception; as evidence surrounding the effect of obesity on ART remains scarce, this section aims to highlight the fundamental information governing female reproduction alongside the impact of obesity on the parameters of ART[9].

Pregnancy Complications Linked To Maternal Obesity

Maternal obesity is consistently associated with myriad pregnancy complications encompassing gestational diabetes mellitus (GDM), hypertensive disorders, cesarean delivery, and preterm birth [2]. Such complications emerge in a dose-dependent fashion for both body mass index (BMI) [7] and waist circumference, aggravating fetal, neonatal, and maternal outcomes [8]. Maternal obesity is additionally linked with obstetric procedures (e.g., prolonged labor, induction of labor, vacuum delivery, and episiotomy), delivery Trauma (e.g., maternal lacerations, perineal injury, and infant brachial plexus injury), and excess perinatal mortality. Intergenerational transmission is evident, with both maternal and paternal obesity implicated; the effect of paternal obesity on offspring is hypothesized to act through conditioning effects on oocytes [6]. Early guidelines recommended categorizing maternal obesity as class I (BMI 30.0–34.9), class II (35.0–39.9), and class III (≥40.0) on the basis of perinatal risk factors and excess cesarean delivery are increased in classes II–III; fetal macrosomia and preterm birth are elevated across classes, and neonatal effects appear accentuated for fathers with class II maternal obesity and class I–II maternal obesity [8].

Obesity and Male Fertility Outcomes

Obesity is recognized as an important risk factor affecting human fertility, but the nature of this relationship varies significantly between men and women [5]. In female physiology, the primary pathway linking obesity to diminished reproductive capacity is through disruption of the hypothalamic-pituitary-gonadal axis, supported by extensive evidence from both preclinical and clinical studies. In contrast, evidence linking obesity with impaired male fertility is less consistently observed, and reports on the precise mechanisms involved are still incomplete [6]. Obesity is associated with compromised semen quality and function and extends time to pregnancy, although the effect appears modest and less severe than typically observed in female obesity [6]. Preclinical evidence also indicates that excessive weight in males can adversely influence gamete quality, fertilization rates, and early embryonic development, suggesting that paternal obesity may affect both conception and subsequent pregnancy outcomes [7]. Thus, although the diverse manifestations of increased adiposity and their reproductive consequences in females may not apply directly to males, a comprehensive understanding of the impact of obesity on male fertility is fundamental for evaluating the extent to which these conditions are causally related [7].

Semen Quality and Sperm Function

Obesity, defined as a body mass index (BMI) greater than 30 kg/m2, has been linked with various deteriorations in semen quality and sperm function [8]. Several studies indicate that obesity also adversely affects the time needed to conceive and therefore couples planning a family [3]. Total sperm count, progressive motility, and normal morphology are frequently reported to be lower in obese men. Nevertheless, definitions of male obesity vary among the different obesity categories of metabolic syndrome [8]. Thus, it is still unclear whether fertility rates in obese men seeking stabilization results differ from those obtained through other ovulating stimulants. Furthermore, establishing the impact of obesity on male fertility remains challenging. Lifestyle factors that increase the possibility of infertility, such as smoking and drinking, are more evident in obese than in non-obese men [7]. Obesity decreases semen quantity (e.g., a study on Hispanic men shows a reduced number of motile sperm) [6], and onward shipment of these reduced motile sperm also decreases pregnancy rate in the partner [8]. Therefore, fathering children can be delayed [8].

Time to Pregnancy and Paternity Outcomes

Obese women experience longer normal conception times than non-obese women. Although the majority of women conceive within a year of regular unprotected intercourse, the probability of conception decreases as time progresses [5]. Using data from women with spontaneous conceptions, such as those in the European multicenter study, average time-to-pregnancy curves could be fitted by a two-stage process: a rapid initial velocity declining to

zero at a later stage. Based on a literature survey, conception probabilities and modelling curves were replotted for different ranges of body mass index, waist circumference, and metabolic health [2]. Maternal obesity generally extends the time to pregnancy [10]. Potentially confounding factors have been identified, but the literature is still insufficient to determine whether maternal excess weight systematically reduces female fecundity [7]. A weighted mean of studies on normally ovulating populations indicates a typical increase of two to three months. Weight-related obstacles to preconception also differ across settings, posing questions about the presumed directness of the association [6]. Time-to-pregnancy studies confirm that female fertility diminishes with excess adiposity. Age interactions have been noted, as have partial to near-complete attenuation of enlargement effects on central obesity or metabolic dysregulation [9]. Concordance across diverse cohorts strengthens external validity, evidence from non-obese controls indicates. A systematic review identified no studies examining links between paternal adiposity, intercourse frequency, and time to conception, highlighting a substantial lack of population-based paternity research. Triads consisting of father, mother, and child have seldom been investigated [5]. Existing studies about parental obesity or overweight provide inconsistent indications concerning paternal body mass index and paternity outcomes in Western countries [11].

Intersections with Comorbidities and Metabolic Health

A high prevalence of obesity-related morbidity and mortality in reproductive-age women raises concerns about the reproductive health of an increasing number of individuals [5]. Obesity poses a significant threat to women's reproductive health, with obesity and body weight being associated with a wide spectrum of reproductive complications, from menstrual irregularities to infertility, poor in vitro fertilization (IVF) outcomes, and adverse pregnancy events [8]. These effects have been attributed to the multiple effects of obesity on the hypothalamic-pituitary-ovarian axis, systemic inflammation, and metabolic dysfunction [8]. Many studies show a lower risk of metabolic syndrome and its individual components in women with high body mass index (BMI) but high waist circumference compared to women with a normal BMI and similar waist circumference [8]. There is also evidence that a high prevalence of obesity-mediated, multi-systemic pathologies can be observed in reproductive-age women who appear lean yet develop abdominal obesity, central adiposity, and visceral fat accumulation from a young age [3]. Furthermore, excess central and visceral fat limits access to fertility care among women with high obesity prevalence. Women with PCOS appear to be overrepresented among obese IVF patients, who are often referred for treatment after numerous failed cycles [6]. Although Metabolically Associated Fatty Liver Disease (MAFLD) is more prevalent among nonalcoholic fatty liver disease (NAFLD) phenotypes among women with obesity and metabolic disorders, it is less commonly investigated in the context of infertility [7]. Obesity remains associated with impairments in the male partner's semen quality, sperm DNA integrity, and fertility, confounding reproductive outcomes further. Consequently, a moderate BMI and other metabolic health-related body metrics have assumed significant importance in obesity-affected populations seeking to establish a family [2].

Interventions and Their Impact on Fertility Outcomes

A narrative review of the evidence regarding the effects of obesity on fertility identifies a broad range of common physiological mechanisms through which the pathophysiology of obesity might have direct effects on fertility. Evidence linking obesity directly to several fertility-related endpoints, including anovulation [3], natural conception and time to pregnancy [4], Assisted Reproductive Technology (ART) outcomes [5], and several pregnancy complications, is synthesised accordingly. Heterogeneity in the relationship between obesity and these endpoints based on metabolic health, age, ethnicity, and the nature of the underlying data is also specified [5]. Obesity continues to increase worldwide and, through its effect on fertility, poses a public health challenge [1]. While conventional approaches to weight management are expected to alter reproductive physiology and restore fertility-related endpoints toward healthier levels, evidence supporting a direct connection between obesity and these endpoints is clearer than for any of the endpoints yet dealt with has been systematically reviewed [5]. A narrative review of the evidence regarding the impact of obesity on fertility has also been published; the principal conclusions from that review are incorporated throughout [4].

Lifestyle Modification and Weight Management

Lifestyle modification is crucial for fertility restoration in obese individuals [4]. To cope with the metabolism and fertility restoration, different approaches of treatment have been proposed [8]. Lifestyle intervention is the first line of multitargeted intervention, taking into account amelioration of obesity condition and metabolic disorder in cardiovascular health [5]. Attempts in weight-loss should therefore carefully balance caloric, exercise, and behavior modification credentials among each individual, requiring a large gathering of experience and scientific evidence to reinforce, and an appreciation of fertility treatment in working adult are needed [8].

Bariatric Surgery and Fertility Considerations

Timing of bariatric procedures in relation to future pregnancy is clinically relevant. Pregnancy during the first year after surgical intervention is discouraged because of accelerated weight loss, potential deficiency of micronutrients, and fetal growth issues [8]. However, pregnancy may be safely attempted 12–18 months after

surgery, and a second surgery is sometimes performed prior to conception [5]. Regardless of type, adverse pregnancy outcomes related to bariatric interventions are low when pregnancies are undertaken after a year. Such interventions remain associated with reduced gestational diabetes and hypertension risk and decreased cesarean delivery through the second year after surgery [9]. Infants are also at lower risk of being large for gestational age; however, growth restriction, preterm birth, and other complications are more frequent. Long-term maternal, neonatal, and childhood outcomes require further investigation [10].

Pharmacologic and Medical Approaches

Recent years have seen increasing interest in weight loss as a means of improving fertility in individuals with obesity. Starting in the 2020s, pharmaceuticals targeting weight reduction began to proliferate [8]. The rapidly expanding class of glucose-dependent insulinotropic-peptide (GLP-1) receptor agonists offers potential for managing obesity in the fertility setting; however, research on the impact of GLP-1 agonists on fertility is limited[10]. The safety of administration throughout conception, pregnancy, and lactation remains uncertain [9]. Similarly, metformin is frequently prescribed to individuals with obesity, particularly those with polycystic ovary syndrome (PCOS) and insulin resistance, but whether metformin influences fertility outcomes remains debated. The appetite-suppressing agent orlistat, which inhibits the gastrointestinal absorption of fats, shows potential in the management of obesity, but its effect on fertility has received minimal investigation [5, 9, 10].

Methodological Considerations and Gaps in Evidence

Research examining how obesity affects fertility is hindered by methodological limitations; inconsistent definitions of obesity phenotypes and fertility outcomes contribute to this impediment. Moreover, the influence of behavioral factors, including smoking, drinking, exercise, and diet, remains notoriously difficult to measure and even harder to control [9]. A focus on long-term, high-quality longitudinal studies and consistent, widely accepted definitions of key measures would greatly enhance the robustness of the field [7]. BMI, waist circumference, and the presence of metabolic disease are three biomarkers employed to characterize obesity; harmonization of the definitions and categorization of these three, especially central adiposity, would represent a major advance [5]. In fertility research, common measures include time to pregnancy, conception rate, and a variety of obstetric outcomes; standardized definitions of these desired endpoints would facilitate better comparison of data between studies [2].

Implications for Clinical Practice and Public Health

Interpreting the body of evidence with clinical, public health, and policy perspectives, obesity emerges as an antecedent condition that can root fertility difficulties across the life course, and broadcasting these connections delivers clinically actionable frameworks [9-12]. When the persistence of reproductive-age adiposity develops, clinicians can choose to screen for obesity-related fertility conditions in addition to those that have more specific symptomology or are proximal to reproductive intent [13-15]. Weight management, whether by lifestyle, surgical, or pharmacological approaches, would benefit fertility-related results of those intending to conceive, regardless of whether the threshold required for a formal intervention is met. Referring back to overarching principles of establishment and prioritizing maternal health before conception remains paramount as policies evolve to reduce the obesity-related fertility burden and improve perinatal outcomes [16, 17].

CONCLUSION

Obesity exerts profound and multifactorial impacts on reproductive health, affecting both female and male fertility through metabolic, endocrine, and physiological pathways. In women, excess adiposity contributes to anovulation, menstrual irregularities, impaired oocyte and embryo quality, prolonged time to pregnancy, and decreased ART success. Men with obesity demonstrate reduced sperm quality, altered hormonal profiles, compromised gamete function, and potential negative influences on early embryonic development. These reproductive impairments occur alongside significant pregnancy complications, including gestational diabetes, hypertensive disorders, fetal macrosomia, and preterm birth, highlighting the intergenerational consequences of obesity. Central adiposity and metabolic syndrome emerge as critical determinants of reproductive dysfunction, often exerting effects independent of BMI. The heterogeneity of obesity phenotypes and the inconsistency of fertility outcome measures pose major methodological challenges, underscoring the need for standardized definitions, high-quality longitudinal studies, and improved characterization of metabolic health. Interventions such as lifestyle modification, targeted medical therapy, and bariatric surgery show promise in restoring fertility, yet evidence remains variable, particularly for emerging pharmacologic agents such as GLP-1 receptor agonists. Optimizing reproductive outcomes requires a multifaceted approach that integrates clinical management with broader public health efforts aimed at reducing obesity prevalence among individuals of reproductive age. Ultimately, addressing obesity before conception, not only for fertility restoration but for overall maternal, paternal, and offspring health, must remain central to clinical practice and public health policy. Strengthening preventive strategies, refining treatment algorithms, and advancing research into obesity-related reproductive pathways will be essential to mitigating the growing global burden of obesity-associated infertility.

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