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Research Article

GLP-1 Receptor Agonists and Male Reproductive Health: Mechanisms Underlying Endocrine, Testicular, and Spermatogenic Modifications

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ABSTRACT

Glucagon-like peptide-1 receptor agonists (GLP-1 RAs)—including liraglutide, semaglutide, dulaglutide, and dual GLP-1/GIP receptor agonists such as tirzepatide—have transformed the clinical management of type 2 diabetes mellitus and clinical obesity [1-4]. Beyond metabolic and cardiovascular outcomes, accumulating preclinical models and human clinical evidence suggest that these agents exert profound, clinically relevant modifications across the male reproductive axis [1-3,5,6]. Obesity and metabolic dysfunction represent major drivers of male subfertility and secondary functional hypogonadism, impairing reproductive function through low-grade chronic inflammation, increased peripheral aromatization of testosterone to estradiol, testicular oxidative stress, altered metabolic signaling, and obesity-associated functional hypogonadism [7-11]. The reviewed evidence supports two broad explanatory pathways: indirect improvement through weight loss and metabolic restoration, and direct receptor-mediated signaling within central hypothalamic-pituitary networks and testicular microenvironments [1,6,12,13]. Reported effects include substantial improvement in endogenous testosterone production, preservation or restoration of gonadotropin pulse signaling, and favorable changes in selected semen parameters (concentration, total count, progressive motility), particularly in men with obesity or metabolic dysfunction [5,6,12,14-16]. However, definitive fertility outcomes, including natural pregnancy and live birth, remain insufficiently studied [1,5,17,18].

KEYWORDS

GLP-1 receptor agonists, Male reproductive health, Male infertility, Obesity, Testosterone, Hypogonadism, Spermatogenesis, Semen parameters, Liraglutide, Semaglutide, Tirzepatide, Dual GIP/GLP-1 agonists.

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Introduction

Male reproductive health is closely linked to metabolic status. Obesity and metabolic syndrome can adversely affect testosterone production, hypothalamic-pituitary-gonadal (HPG) axis function, semen quality, and sexual function, affecting millions of reproductive-aged men globally [7-11]. GLP-1 RAs—including liraglutide, semaglutide, dulaglutide, and dual GLP-1/GIP agonists such as tirzepatide—produce major improvements in glycemic regulation and body weight [1-4]. Their potential reproductive effects are therefore increasingly relevant because reversal of metabolic dysfunction may remove several established contributors to male reproductive impairment [1,5,17,18]. Understanding the interplay between incretin therapy and gonadal function is essential for clinicians managing reproductive-aged males with metabolic disorders [1,18].

The global rise in obesity over recent decades has run parallel to observed declines in population-level semen quality metrics and rising rates of secondary male hypogonadism. Visceral adiposity generates a complex pathophysiological cascade involving altered adipokine secretion, increased systemic inflammatory signaling, enhanced oxidative stress, and heightened aromatase expression in white adipose tissue. Consequently, therapeutic strategies that target underlying metabolic pathophysiology hold significant promise for restoring reproductive endocrine homeostasis.

GLP-1 Biology and Receptor Distribution in the Male Reproductive System

The reviewed literature describes GLP-1 receptor (GLP-1R)-related signaling across key components of the male reproductive axis, establishing a direct functional link between metabolic status and gonadal regulation [1,2,13]. Within central pathways, receptors have been reported in hypothalamic Kisspeptin-related (ARC/AVPV) and GnRH-associated networks and in anterior pituitary gonadotrope-related pathways, providing a possible direct link between incretin signaling and LH/FSH pulse frequency and regulation [1,12,13]. Activation of these central receptors directly influences the pulsatile secretion of luteinizing hormone (LH) and follicle-stimulating hormone (FSH), which are required for downstream androgenesis and spermatogenesis [1,6].

Within the testicular environment, robust GLP-1 receptor expression has been described in Leydig cells, which govern steroidogenesis, and Sertoli cells, which support spermatogenesis and maintain blood-testis barrier tight junction integrity [1,6,13,17,19]. Furthermore, functional GLP-1-related signaling and receptor expression have been reported in mature spermatozoa (head and midpiece), actively modulating cellular energy metabolism, mitochondrial membrane potential, progressive motility, and acrosome-related function [1,5,10,13,19] (Figure 1).

Anatomical Mapping of Incretin Targets in Male Gonadal Tissues

GLP-1 receptor (GLP-1R) expression spans multiple anatomical locations across the male reproductive axis to exert distinct physiological roles [1-5]. Within the hypothalamus and pituitary, GLP-1R is expressed in kisspeptin neurons, GnRH projections, and pituitary gonadotropes, where it restores pulsatile GnRH secretion and regulates baseline and peak LH/FSH pulse frequency [1-3,5,13]. In the testicular interstitium, GLP-1R target cells—specifically Leydig cells—stimulate steroidogenic acute regulatory (StAR) protein expression and endogenous

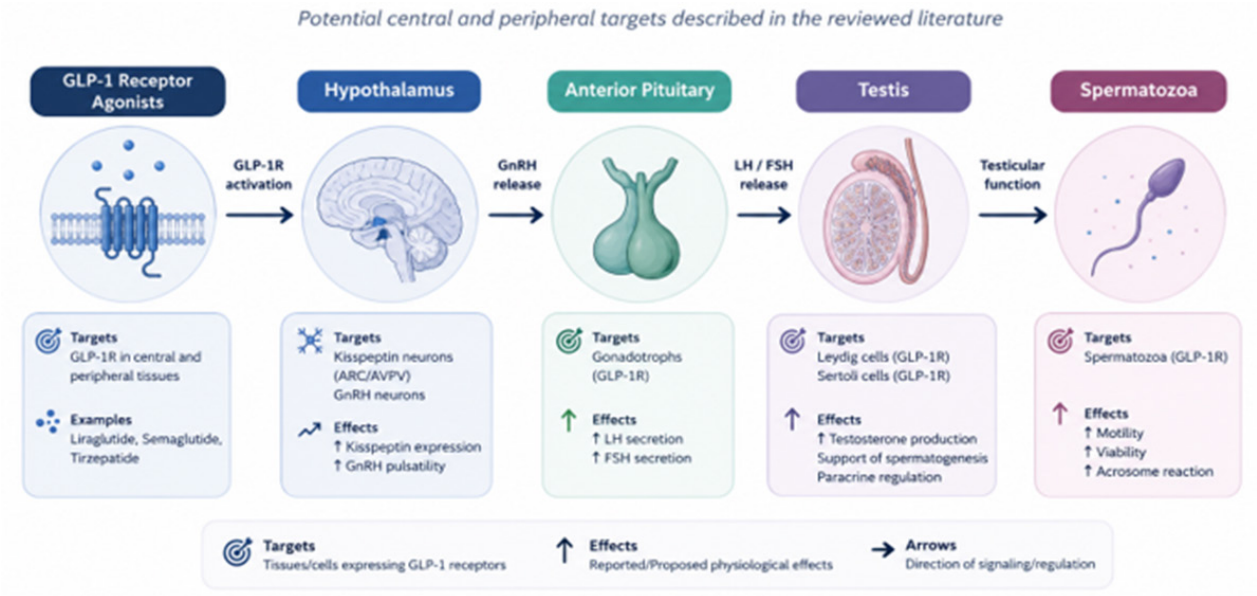

Figure 1: Proposed GLP-1 signaling across the male reproductive axis. Receptors expressed centrally in hypothalamic ARC/AVPV kisspeptin neurons and anterior pituitary gonadotrophs regulate GnRH and gonadotropin pulsatility, while peripheral GLP-1Rs in Leydig cells, Sertoli cells, and mature spermatozoa modulate steroidogenesis, blood-testis barrier dynamics, and motility.

testosterone synthesis [1,5,8,17]. At the level of the seminiferous tubules, GLP-1R expression in Sertoli cells and the blood-testis barrier upregulates GLUT8 glucose transporters, enhances lactate production, and maintains tight junction integrity [1,5,10,17,19]. Finally, in ejaculated spermatozoa, GLP-1R localized to the mature sperm head and midpiece enhances mitochondrial membrane potential and drives ATP generation essential for flagellar motility [1,5,14,16,17] (Table 1).

Table 1: Anatomical mapping and physiological actions of GLP-1 receptor (GLP-1R) expression across the male reproductive axis.

Anatomical Location	Target Cells Expressing GLP-1R	Primary Physiological Role
Hypothalamus & Pituitary	Kisspeptin Neurons, GnRH projections, Pituitary Gonadotropes	Restores pulsatile GnRH secretion; regulates baseline and peak LH/FSH pulse frequency.
Testicular Interstitium	Leydig Cells	Stimulates StAR protein expression and endogenous testosterone synthesis.
Seminiferous Tubules	Sertoli Cells & Blood-Testis Barrier	Upregulates GLUT8 glucose transporters, enhances lactate production, maintains tight junction integrity.
Ejaculated Spermatozoa	Mature Sperm Head & Midpiece	Enhances mitochondrial membrane potential, drives ATP generation for flagellar motility.

Obesity, Metabolic Dysfunction, and Male Reproductive Impairment

Visceral adiposity contributes to an endocrine environment highly unfavorable for male reproductive function. Increased adipose tissue promotes peripheral aromatization of androgens into estrogens (such as estradiol) via heightened aromatase activity, contributing to feedback suppression of the hypothalamic-pituitary-gonadal axis [7,8,11,12,20]. Hyperinsulinemia, hepatic metabolic dysfunction (such as steatosis), systemic low-grade inflammation driven by circulating cytokines (e.g., TNF-α, IL-6), altered leptin/ghrelin dynamics, and testicular oxidative stress further impair Leydig-cell steroidogenesis, Sertoli-cell support, germ-cell development, and sperm integrity [7-11] (Table 1). Accordingly, therapies that improve metabolic health and clear metabolic dysfunction may improve reproductive physiology by acting on several convergent pathways [1-3].

In addition, visceral fat accumulation raises scrotal temperature, further exacerbating oxidative damage in developing germ cells. Hyperinsulinemia suppresses liver production of sex hormone-binding globulin (SHBG), altering the bioavailability of circulating testosterone and accelerating renal clearance of unbound steroids.

Mechanisms by Which GLP-1 Agonists May Modify the HPG Axis

A central question in reproductive endocrinology is whether reproductive improvement is mediated entirely by weight loss or also involves direct pharmacological receptor actions [1,5,13].

- Indirect / Weight Loss-Mediated Pathways: Major mechanisms include substantial reduction of visceral adiposity, decreased systemic aromatase activity and peripheral estradiol levels, reversal of hepatic steatosis, normalization of liver-derived SHBG synthesis, improved insulin sensitivity, and reduction of systemic circulating inflammatory cytokines (TNF-α, IL-6) and testicular oxidative stress [3,7-9,12].
- Direct / Receptor-Mediated Pathways: Proposed direct mechanisms include modulation of hypothalamic Kisspeptin/GnRH pathways, direct support of gonadotrope LH and FSH pulse secretion, Leydig-cell steroidogenic activity (including StAR protein expression), Sertoli-cell metabolic support (GLUT8 upregulation and lactate yield), and direct enhancement of sperm mitochondrial potential and ATP generation [1,6,13].

Clinical and experimental evidence indicates that in healthy, normosmic men, short-term GLP-1 administration produces minimal shifts in baseline gonadotropins or testosterone, indicating that direct signaling plays a secondary role in the absence of metabolic impairment [1,2,13]. However, in obese or diabetic men, direct central/peripheral activation acts in synergy with major weight-loss-mediated reductions in peripheral estrogen, hyperinsulinemia, and systemic inflammation [1,6,12,18]. The current evidence is most consistent with a synergistic model in which direct signaling occurs within a substantially improved systemic metabolic environment [1,5,17] (Figure 2).

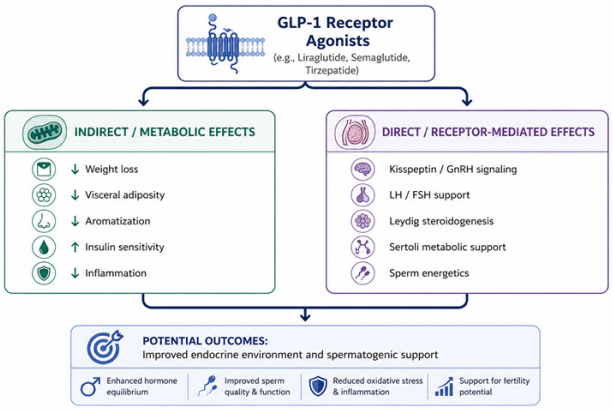

Figure 2: Proposed indirect metabolic and direct receptor-mediated mechanisms linking GLP-1 therapy to male reproductive modification. The model illustrates how visceral adiposity reduction acts in concert with direct receptor activation across hypothalamic, gonadotropic, and testicular targets.

Effects on Testosterone, LH, FSH, SHBG, and Estradiol

Clinical studies summarized in the review report consistent increases in total testosterone following GLP-1-based therapy, particularly among men with obesity, low baseline testosterone,

or metabolic dysfunction [6,12,13,18]. Free testosterone remains stable or increases moderately, while sex hormone-binding globulin (SHBG) increases significantly as reversal of hepatic steatosis and hyperinsulinemia normalizes hepatic SHBG gene expression and synthesis [6,12,13]. Estradiol decreases markedly as visceral adiposity and peripheral aromatization are curtailed [7,8,12].

Exogenous TRT vs. GLP-1 receptor agonists

Exogenous testosterone produces negative feedback suppression of LH and FSH, reducing intratesticular testosterone and potentially causing profound impairment of spermatogenesis, including azoospermia. In contrast, GLP-1 receptor agonists do not directly suppress gonadotropin secretion in the manner of exogenous testosterone. In men with obesity-associated functional hypogonadism, GLP-1–mediated weight loss and metabolic improvement may help restore endogenous hypothalamic–pituitary–gonadal function and serum testosterone. Thus, GLP-1 RAs may represent a fertility-compatible approach in appropriately selected men, although their direct effects on LH/FSH and male fertility require further study.

Systematic Overview of Endocrine Parameters Under Incretin Therapy

Incretin therapy induces significant modifications across the male endocrine profile by restoring central neuroendocrine signaling and reducing peripheral metabolic dysfunction [1-3,5]. Treatment leads to a significant increase in total testosterone (TT), driven primarily by the reduction of visceral fat mass—which decreases systemic aromatase activity—alongside the central restoration of luteinizing hormone (LH) pulse frequency [1,4,6,11-13]. Free testosterone (FT) demonstrates a stable to moderate rise, as total testosterone gains are counterbalanced by concurrent increases in sex hormone-binding globulin (SHBG) to preserve optimal bioavailable levels [6,12,13]. The significant increase in SHBG itself stems from the reversal of hepatic steatosis and hyperinsulinemia, which normalizes liver-derived SHBG gene expression and synthesis [7-9,11,20]. Central gonadotropins, LH and FSH, remain preserved or elevated due to the alleviation of hypothalamic suppression without triggering central negative feedback inhibition [1,5,6,12,13]. Concurrently, estradiol (E2) levels undergo a significant decrease, directly resulting from diminished adipose tissue mass limiting the peripheral aromatization of circulating androgen precursors [1,7,8,11,12] (Table 2).

- Sperm Concentration & Total Count: In a prospective study of obese men undergoing an 8-week low-calorie diet, sperm concentration increased approximately 1.71-fold and total sperm count approximately 1.41-fold; these improvements were associated with weight loss and were maintained at one year in men who sustained substantial weight reduction. Importantly, the subsequent liraglutide intervention did not produce additional semen-parameter improvements beyond the dietary intervention. Thus, these numerical changes should be interpreted primarily as evidence supporting the relationship between weight loss and semen quality rather than proof of a drug-specific effect. A separate 2025 retrospective propensity-matched TriNetX cohort study included 385 GLP-1RA-exposed men and 385 matched controls; sperm-count normalization occurred in 10/351 exposed men versus 0/358 controls, while motility did not differ significantly. This study was observational and did not establish causality.
- Sperm Motility & Energetics: Experimental and *in-vitro* studies have reported effects of GLP-1-related signaling on sperm mitochondrial function, ATP generation, and motility. These findings provide mechanistic support but should be distinguished from human clinical evidence, where consistent improvements in motility have not yet been demonstrated across studies.
- Sperm Morphology & DNA Integrity: Animal and *in-vitro* models have reported improvements in oxidative stress, sperm integrity, and testicular architecture following GLP-1RA exposure. Human data on sperm morphology and DNA fragmentation remain limited. These outcomes should therefore be presented as preclinical or exploratory rather than established clinical benefits.

Effects on Spermatogenesis and Semen Parameters

Clinical and preclinical studies suggest favorable effects on sperm concentration, total sperm count, and progressive motility, especially in metabolically compromised men [2,5,6,14,16]. Spermatogenesis is an energy-intensive process highly vulnerable to systemic inflammation, oxidative stress, and metabolic

Table 2: Systematic overview of male endocrine parameters under incretin therapy. Summary of observed clinical changes and biological mechanisms across key androgenic markers, transport proteins, gonadotropins, and estrogens.

Endocrine Marker	Observed Effect	Proposed Biological Mechanism
Total Testosterone (TT)	▲ Significant Increase	Reduction of visceral fat lowers systemic aromatase activity; central restoration of LH pulse frequency.
Free Testosterone (FT)	◄► / ▲ Stable to Moderate Rise	Increase in TT is balanced by concurrent rises in liver-derived SHBG, keeping bioavailable testosterone optimal.
SHBG	▲ Significant Increase	Reversal of hepatic steatosis and hyperinsulinemia normalizes hepatic SHBG gene expression and synthesis.
LH & FSH	◄► / ▲ Preserved or Elevated	Alleviation of hypothalamic suppression occurs without inducing central negative feedback inhibition.
Estradiol (E2)	▼ Significant Decrease	Marked reduction in adipose tissue mass curtails peripheral aromatization of circulating androgen precursors.

dysregulation [7,10,19].

- Sperm Concentration & Total Count: Long-term liraglutide 3.0 mg clinical studies confirm that GLP-1 RA therapy maintains or expands weight-loss-associated gains in sperm concentration (1.49-fold increase) and total sperm count (1.41-fold increase) [6,12,14-16]. Furthermore, real-world retrospective database analyses (such as TriNetX cohort evaluations) demonstrate significantly higher rates of sperm count normalization in GLP-1-treated men compared to non-treated obese controls [14,15].
- Sperm Motility & Energetics: GLP-1 RAs enhance progressive sperm motility through improved mitochondrial membrane potential and Sertoli-cell lactate secretion [1,5,19]. Direct *in vitro* incubation studies confirm heightened intracellular ATP yield and boosted flagellar beat frequency upon receptor activation [1,13].
- Sperm Morphology & DNA Integrity: Preclinical models show marked reductions in sperm DNA fragmentation index (DFI) and structural restoration of seminiferous tubule architecture under GLP-1 RA administration, driven by reductions in testicular reactive oxygen species (ROS) [1,4,7,13]. Human data concerning morphology and DNA integrity remain an active area of ongoing confirmation [1,10,17] (Figure 3).

Direct Testicular and Cellular Mechanisms

Potential direct testicular actions are biologically important because Leydig cells regulate testosterone biosynthesis and Sertoli cells provide metabolic and structural support for developing germ cells [1,6,13]. At the cellular level, GLP-1-related signaling in Sertoli cells upregulates glucose transport via GLUT8 transporters, boosts mitochondrial bioenergetics, and increases lactate secretion—the principal energy substrate essential for germ cell maturation—while suppressing apoptotic cascades [1,3,7,19,13]. In mature spermatozoa, direct receptor pathways regulate mitochondrial membrane potential, ATP yield, flagellar motility, capacitation-related processes, and acrosome reaction readiness [1,11,13,19]. The overall clinical significance of these direct cellular pathways continues to be defined alongside systemic weight loss benefits [1,5,17].

Vascular Health, Erectile, and Sexual Function

Metabolic syndrome, visceral obesity, and endothelial dysfunction are primary pathophysiological drivers of erectile dysfunction (ED) [9,11,20]. GLP-1-based therapy improves erectile and sexual function through three integrated physiological pathways [1,3,5,9,20]:

1. Endothelial Nitric Oxide Synthase (eNOS) Activation: GLP-1 signaling in cavernous endothelial and smooth muscle tissues upregulates eNOS phosphorylation, restoring nitric oxide (NO) bioavailability and promoting corpus cavernosum relaxation [1-3].
2. Mitigation of Glycemic & Oxidative Damage: Reductions in advanced glycation end-products (AGEs) and vascular cell adhesion molecules attenuate microvascular damage and neural degeneration within penile tissue [1,3,19].
3. Psychosocial and Body Image Gains: Substantial weight loss correlates directly with improved International Index of Erectile Function (IIEF-5) scores, boosted sexual desire, increased confidence, and enhanced psychological well-being [1,12,20].

Dual GLP-1/GIP Agonists and Emerging Therapies

Multi-target incretin therapies, such as the dual GIP/GLP-1 receptor agonist tirzepatide, represent a major emerging area of interest [3,18,15]. GIP receptors are expressed in testicular capillary endothelium and central neural networks [1,3]. Preliminary clinical observations suggest that tirzepatide's superior weight loss and glycemic control yield proportionate or greater

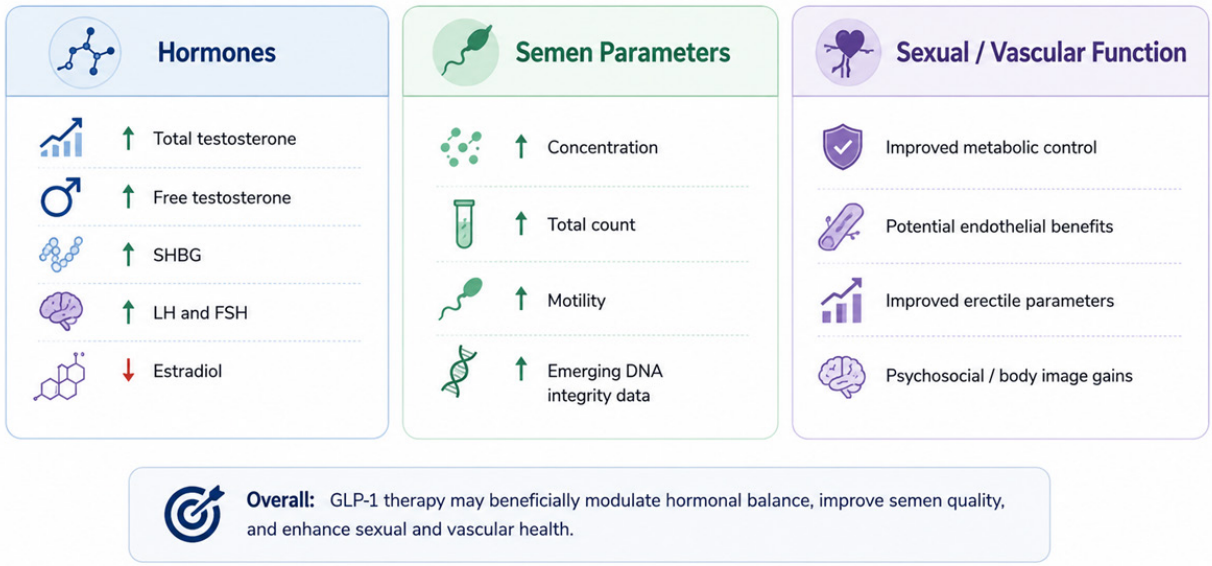

Figure 3: Major hormonal, semen, and sexual-function domains reported as potentially modified by GLP-1-based therapy.

improvements in endogenous testosterone restoration compared to single-agent GLP-1 RAs [1,14,15,18]. Dedicated long-term trials are required to establish whether direct GIP signaling provides additive reproductive benefits independent of enhanced weight loss [1,3,18].

Paternal Epigenetics and Intergenerational Considerations

Paternal obesity impairs sperm microRNA expression, histone modifications, and DNA methylation patterns, transmitting metabolic disease and cardiovascular risk to offspring [7,10]. The possibility that GLP-1-mediated metabolic clearance and reduced systemic inflammation could favorably remodel the paternal sperm epigenome prior to conception is scientifically important [1,10,18]. While an intriguing hypothesis for mitigating intergenerational metabolic disease transmission, human longitudinal studies are required to confirm offspring outcomes [1,10,18].

Clinical Considerations, Safety, and Fertility Timing

While clinical evidence supports metabolic and reproductive improvements, several practical clinical nuances warrant attention [1,16-18]:

- Semen Volume Dynamics: While sperm concentration and total count generally improve, real-world comparative observations have noted stable or slightly reduced ejaculatory volume in some GLP-1 users relative to controls [1,14,17]. This effect may relate to transient shifts in seminal vesicle fluid dynamics during active rapid weight loss, underscoring the need to evaluate the complete semen profile rather than single isolated parameters [5,14,17].
- Micronutrient Co-Factors: Rapid weight loss and active caloric restriction can lead to micronutrient deficiencies in key co-factors required for optimal spermatogenesis, including zinc, folate, selenium, and L-carnitine [7,16,18]. Clinicians should ensure nutritional monitoring and targeted supplementation during active weight-loss phases [18,16].
- Assisted Reproductive Technology (ART) Timing: For men undergoing ART or active fertility treatments, weight stabilization should ideally be achieved prior to final sperm collection to avoid transient fluctuations in semen parameters caused by active caloric restriction [16,18].

Limitations of Current Evidence and Future Directions

The current literature remains heterogeneous with respect to study populations, treatment duration, comparator groups, and reproductive outcomes [1,5,17,18]. Most published clinical studies evaluate surrogate markers (such as testosterone levels, sperm concentration, and motility) rather than definitive fertility endpoints such as natural pregnancy rates, ART success, or live birth [1,5,17,18] (Figure 4).

Critical Research Priorities

1. Head-to-Head Comparative Trials: Prospective randomized controlled trials (RCTs) directly comparing semaglutide or tirzepatide against selective estrogen receptor modulators (e.g., clomiphene citrate), exogenous gonadotropins, or lifestyle modification alone [1,17,18].
2. Sperm DNA Integrity & Epigenetics: Standardized human studies assessing sperm DNA fragmentation index (DFI) and longitudinal sperm epigenomic remodeling during GLP-1 therapy [1,10].
3. Definitive Fertility Outcomes: Multicenter studies evaluating natural pregnancy rates, time-to-pregnancy, and live birth outcomes in couples where the male partner receives GLP-1 therapy [1,5,18].

Conclusions

GLP-1 receptor agonists represent an important emerging

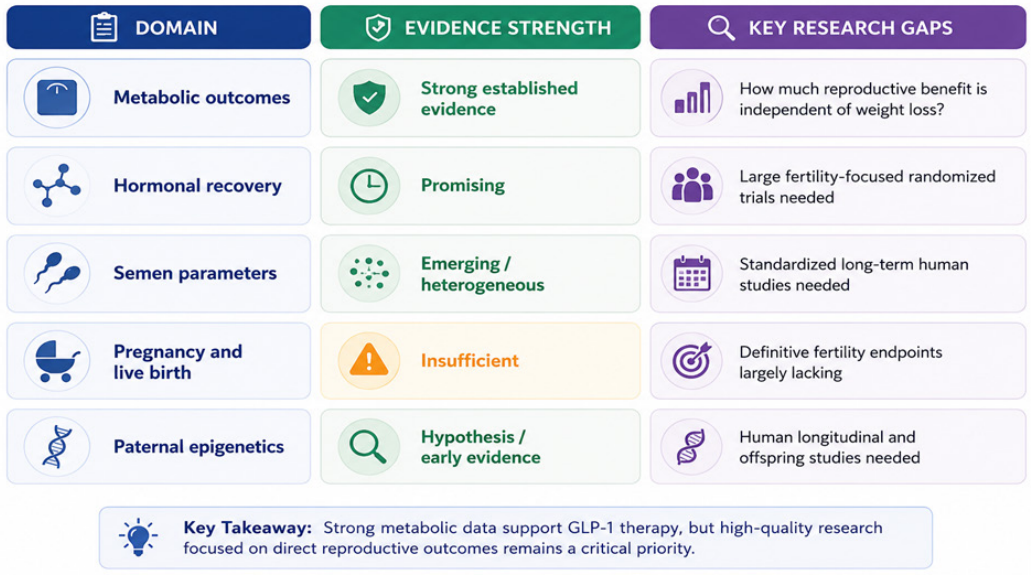

Figure 4: Current evidence map and major research gaps in GLP-1 therapy and male reproductive health.

therapeutic class in male reproductive medicine. The reviewed evidence supports a biologically plausible relationship between metabolic improvement, endocrine recovery, and selected improvements in male reproductive parameters, particularly in men with obesity or metabolic dysfunction [1,6,12-15]. Potential direct effects on central reproductive pathways, Leydig cells, Sertoli cells, and mature spermatozoa provide an additional mechanistic framework, operating in synergy with systemic metabolic restoration [1,6,13,19]. At present, improvements in surrogate reproductive markers should not be equated with established improvements in natural pregnancy or live-birth outcomes [1,5,17,18]. Large, fertility-focused prospective trials remain a critical priority to fully define the clinical role of GLP-1-based therapies in male reproductive healthcare [1,5,18].

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