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Review Article

The emerging role of glucagon-like peptide-1 receptor agonists in male infertility: mechanisms, experimental evidence, and future clinical perspectives

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ABSTRACT

Obesity and metabolic dysfunction are major modifiable risk factors for male infertility, affecting reproductive function via insulin resistance, oxidative stress, chronic inflammation, hypothalamic-pituitary-gonadal axis dysregulation and impaired steroidogenesis. Glucagon-like peptide-1 receptor agonists (GLP-1RAs) are commonly used to treat obesity and type 2 diabetes mellitus and have emerged as potential modulators of male reproductive health, due to their metabolic effects and potential direct effects on reproductive tissues. This narrative review aimed to assess the current evidence regarding the potential role of GLP-1RAs in obesity-related male infertility, including underlying mechanisms, experimental findings, clinical evidence, and future therapeutic implications. A narrative literature search was conducted in PubMed, Scopus, Google Scholar, and Web of Science for relevant publications from 2018 to 2025, including original research, clinical trials, observational studies, experimental studies, systematic reviews, and relevant guidelines on GLP-1RAs, male reproductive physiology, semen parameters, reproductive hormones, and obesity-associated infertility. Evidence suggests that GLP-1RAs may influence male reproductive function via both indirect and direct effects on reproductive tissues. Experimental studies have shown expression of the GLP-1 receptor in reproductive cells including Leydig cells, Sertoli cells and spermatozoa, suggesting potential roles in steroidogenesis, cellular metabolism, mitochondrial function and spermatogenesis. In addition, the relative contribution of direct pharmacological effects compared to metabolic improvement by weight loss remains unclear. Overall, GLP-1RAs appear to be a promising therapeutic approach for treating obesity-related male reproductive dysfunction by acting on both metabolic health and possibly restoring reproductive function. However, there is not enough evidence at present to justify the recommendation of GLP-1RAs as established fertility-enhancing agents. To determine their role in the evidence-based management of male infertility, large, adequately powered randomised controlled trials evaluating semen quality, sperm DNA integrity, reproductive hormone profiles, pregnancy, and long-term safety are required.

Keywords: Male infertility, GLP-1RAs, Obesity, Reproductive health, Spermatogenesis, Testosterone, Semen quality, Functional hypogonadism, Liraglutide, Semaglutide

INTRODUCTION

About 15% of couples who are of reproductive age globally experience infertility, which is still a serious public health issue with significant social consequences related to psychological and medical issues. It is extremely important to understand the reasons for reproductive disorders in men and find effective ways to treat them, as male factors cause about 20–30% of all cases of infertility and are associated with almost 50% of couples.[1,2] There are still few therapeutic options that address the underlying

reasons for male infertility, especially for men with obesity-related reproductive dysfunction, despite tremendous advancements in assisted reproductive technologies.

Obesity has been one of the most significant modifiable risk factors for reduced male fertility during the last few decades. Alongside the increased frequency of metabolic syndrome and type 2 diabetes mellitus, the prevalence of overweight and obesity is still rising alarmingly worldwide while 382 million individuals were affected globally in 2013, and by 2035, that number is predicted to rise to 592 million. Diabetes mellitus (DM) is a major cause of death, accounting for approximately 1.3 million deaths in 2013. New anti-hyperglycaemic medications have been created and put into clinical use to address this problem. These consist of glucagon-like peptide-1 receptor agonists (GLP1-RAs), sodium-glucose co-transporter-2 (SGLT2) inhibitors, and dipeptidyl peptidase-4 inhibitors (DDP4i).[3] Obesity adversely affects male reproductive health through a complex interplay of endocrine, metabolic, inflammatory, and vascular mechanisms. Excess adiposity promotes increased aromatization of testosterone into oestradiol, suppresses hypothalamic–pituitary–gonadal (HPG) axis function, induces insulin resistance, and contributes to chronic low-grade inflammation and oxidative stress. Collectively, these alterations impair Leydig and Sertoli cell function, compromise spermatogenesis, reduce semen quality, increase sperm DNA fragmentation, and predispose affected individuals to hypogonadism and erectile dysfunction. It is generally known that metabolic health and reproductive function interact, with diseases like diabetes and obesity being associated with lower sperm quality and male infertility.[4]

Reproductive results are less successful for people with higher body mass indices (BMIs). This group of patients seeking therapy for infertility is distinct; they are driven, obedient, knowledgeable, and ready to learn more. But merely advising them to eat less and exercise more to reduce weight is not enough to combat the obesity pandemic.[5]

The cornerstone of managing obesity is still lifestyle therapies, such as dietary changes and physical exercise, which have shown some benefits in reproductive function. However, sustained weight loss and long-term adherence are sometimes challenging to attain, which has led to an increase in interest in pharmaceutical treatments that can concurrently improve metabolic health and reproductive outcomes.[6]

Because of their powerful effects on appetite regulation, weight loss, glycaemic control, and cardiovascular risk reduction, GLP-1RAs have revolutionized the treatment of obesity and type 2 diabetes mellitus. While more recent dual incretin agonists have further increased therapy possibilities, agents including liraglutide, semaglutide, dulaglutide, and exenatide have shown significant success in improving metabolic markers.[7,8]

GLP-1 receptor agonists may have positive impacts on male reproductive health in addition to their known metabolic advantages, according to mounting experimental and clinical data. The hypothalamus, pituitary gland, testes, and reproductive tract have all been found to contain GLP-1 receptors, suggesting a potential physiological function in reproductive regulation.[9]

While GLP-1RA therapy has been shown to improve serum testosterone concentrations, erectile function, sperm concentration, and sperm motility, especially in obese men with metabolic dysfunction, experimental studies have shown that GLP-1 signalling may affect steroidogenesis, Sertoli cell metabolism, mitochondrial function, oxidative stress, and spermatogenesis.[9,10] Nevertheless, the available evidence remains heterogeneous, and it is still uncertain whether these reproductive benefits are mediated predominantly through weight loss and metabolic improvement or through direct pharmacological actions on reproductive tissues.

Understanding GLP-1 receptor agonists possible involvement in reproductive medicine has become more crucial given their fast-growing use in clinical practice and the rising incidence of male infertility linked to obesity. Strong data about fertility-specific outcomes, the ideal duration of therapy, and long-term reproductive safety is still lacking, despite a number of recent studies summarizing new findings. Thus, the purpose of this narrative review is to critically assess the available data on the effects of GLP-1 receptor agonists on male reproductive health, talk about the underlying biological mechanisms, summarize the preclinical and clinical research that is currently available, point out significant gaps in the literature, and suggest future research avenues that might make it easier to incorporate these agents into the treatment of male infertility linked to obesity.

GLP-1 PHYSIOLOGY AND PHARMACOLOGY IN MALE REPRODUCTION

GLP-1 is a peptide hormone generated through the enzymatic breakdown of proglucagon. It is synthesized in L-cells located in the intestinal mucosa, α-cells found in the pancreatic islet, and neurons residing in the nucleus of the solitary tract. GLP-1, an endocrine hormone, is secreted by enteroendocrine L-cells located in the distal jejunum, ileum, and colon in response to nutrient ingestion and neuroendocrine stimulation. It originates from the pre proglucagon precursor, which undergoes enzymatic processing within intestinal L-cells, ultimately giving rise to GLP1(1-37) and GLP-1(7-36) amide or GLP-1(7-37) peptide variants. GLP-1 is an incretin hormone that plays a pivotal role in the meticulous control of human blood glucose levels. Nevertheless, its duration of action is rather ephemeral, lasting a mere 1–2 min within the circulatory system under typical physiological conditions. Subsequently, GLP-1 undergoes enzymatic degradation facilitated by dipeptidyl peptidase IV (DPP-4), leading to the loss of its biological efficacy.[11]

Aside from being expressed in the gut and the pancreas, GLP-1 receptors are extensively expressed in other parts of the body, including the hypothalamus, brainstem, heart, kidneys, and male reproductive organs.[12] Functional GLP-1 receptors have been found on Leydig cells, Sertoli cells, and even mature spermatozoa. The presence of functional GLP-1 receptors on the aforementioned cells indicates that GLP-1 signalling could be involved directly in glucose homeostasis, steroidogenesis, and sperm energy metabolism, contrary to what is believed to be GLP-1's action through systemic weight loss and increased insulin sensitivity.[12] It is both the direct effect and the indirect effect of GLP-1 receptor signalling that give rise to the possibility of developing GLP-1 agonists for the treatment of infertility due to obesity.

Various GLP-1 receptor agonists are currently available for medical application, and they vary in terms of their structural makeup, selectivity for the GLP-1 receptor, and longevity of pharmacological effect. Short-acting compounds like exenatide and lixisenatide provide pulsatile activation of the receptor, while long-acting drugs like liraglutide, dulaglutide, and semaglutide allow continuous binding and produce more pronounced weight loss effects. In turn, a new dual agonist – tirzepatide – which acts on both the glucose-dependent insulinotropic polypeptide (GIP) receptor and the GLP-1 receptor – was proven to produce unprecedented reductions in glycated haemoglobin and body weight in the course of clinical trials of the SURPASS and SURMOUNT programmes, showing even better results than GLP-1-selective agonists.[13] The GIP receptor is activated by tirzepatide with relatively higher potency than the GLP-1 receptor, and it may be this ratio that contributes to the compound's better metabolic properties, while the impact of GIP signal transduction specifically on reproductive function in males has never been studied.[14] The rising popularity of the dual agonist, along with developing triple agonists, makes reproductive research especially urgent.

MECHANISTIC LINKS BETWEEN OBESITY, METABOLIC DYSFUNCTION, AND MALE REPRODUCTIVE IMPAIRMENT

The burden at hand in this case is quite heavy, as male factors are responsible for up to fifty percent of infertility cases, while the prevalence of obesity has doubled over the past three decades and currently affects over a billion people worldwide.[20,25] The impact of obesity on male reproduction is caused by numerous interconnected processes. Adipose tissue, especially visceral fat, produces large amounts of aromatase that converts testosterone into estradiol and causes hyperestrogenism with subsequent negative feedback inhibition of the HPG-axis, suppression of gonadotropin-releasing hormone and gonadotropin production, and decrease in intratesticular and serum testosterone.[15] At the same time, hyperinsulinemia due to insulin resistance decreases production of sex hormone-binding globulin, reducing available testosterone concentration, and chronic low-level inflammation induced by adipocytokines including leptin and pro-inflammatory cytokines inhibits steroidogenesis in Leydig cells.[15] Additionally, reactive oxygen species produced as a result of excessive adiposity destroy sperm membrane and genetic material, causing DNA fragmentation and reducing motility, while the presence of scrotal adipose tissue increases the temperature in the testes and negatively impacts spermatogenesis.[15] Overall, all of these processes form a vicious cycle in which obesity and hypogonadism reinforce one another, sometimes termed male obesity-associated secondary hypogonadism (Figure 1). Because GLP-1 receptor agonists simultaneously reduce adiposity, improve insulin sensitivity, and dampen systemic inflammation, they are mechanistically well positioned to interrupt this cycle, independent of any direct testicular action.

GLP-1 RECEPTOR EXPRESSION AND DIRECT TESTICULAR ACTIONS

Functional GLP-1 receptors have been demonstrated on Leydig cells, Sertoli cells, and spermatozoa using immunohistochemistry and receptor-binding assays. In Leydig cells of both humans and rodents, Caltabiano et al identified GLP-1 receptors by immunohistochemistry and suggested a possible function in steroidogenesis and oncosuppression in Leydig cell tumors.[26] On the other hand, Martins et al in an experiment using human Sertoli cells *in vitro*, reported that low physiological levels of GLP-1 enhance the conversion of glucose into lactate which is crucial for the maturation of germ cells, while supra-physiological levels decreased the mitochondrial membrane potential and increased oxidative stress.[30] In isolated human spermatozoa, Rago and colleagues demonstrated functional GLP-1 receptor expression by western blot and immunofluorescence, with receptor activation increasing progressive motility and cholesterol efflux through kinase-dependent pathways, supporting a role for GLP-1 in regulating sperm capacitation-related processes.[27] These findings, summarized in a comprehensive physiological review of GLP-1 in reproduction, indicate that GLP-1 receptor agonists may influence male fertility through mechanisms that extend beyond systemic metabolic improvement, although the clinical significance of direct testicular signalling relative to weight-loss-mediated effects remains unresolved (Figure 2).[28]

PRECLINICAL EVIDENCE

Rodent-based research is the most reliable evidence of the positive impact of GLP-1 receptor agonists on male reproductive characteristics. Exenatide, liraglutide, or semaglutide administration in obese and diabetic rodents results in the restoration of body weight, improvement of serum testosterone levels, as well as increased sperm concentration, motility, and integrity of sperm DNA, by mechanisms that include the cAMP/PKA and PI3K/Akt signalling pathways responsible for supporting Sertoli cells and reducing testicular inflammation and oxidative

stress.[16] However, the preclinical literature is not uniformly favourable: there is evidence from some of the high fat diet rodent trials showing that the use of GLP-1 receptor agonists does not manage to restore semen volume, sperm concentration, or motility despite the improvement in metabolic indices, whereas some researchers have shown that GLP-1 signalling, under specific circumstances, can reduce testosterone levels.[4] Such variability could be attributable to factors such as different experimental models, dose regimes, periods of treatments and extent of metabolic dysregulation, indicating that laboratory results cannot be directly applied in the clinic.

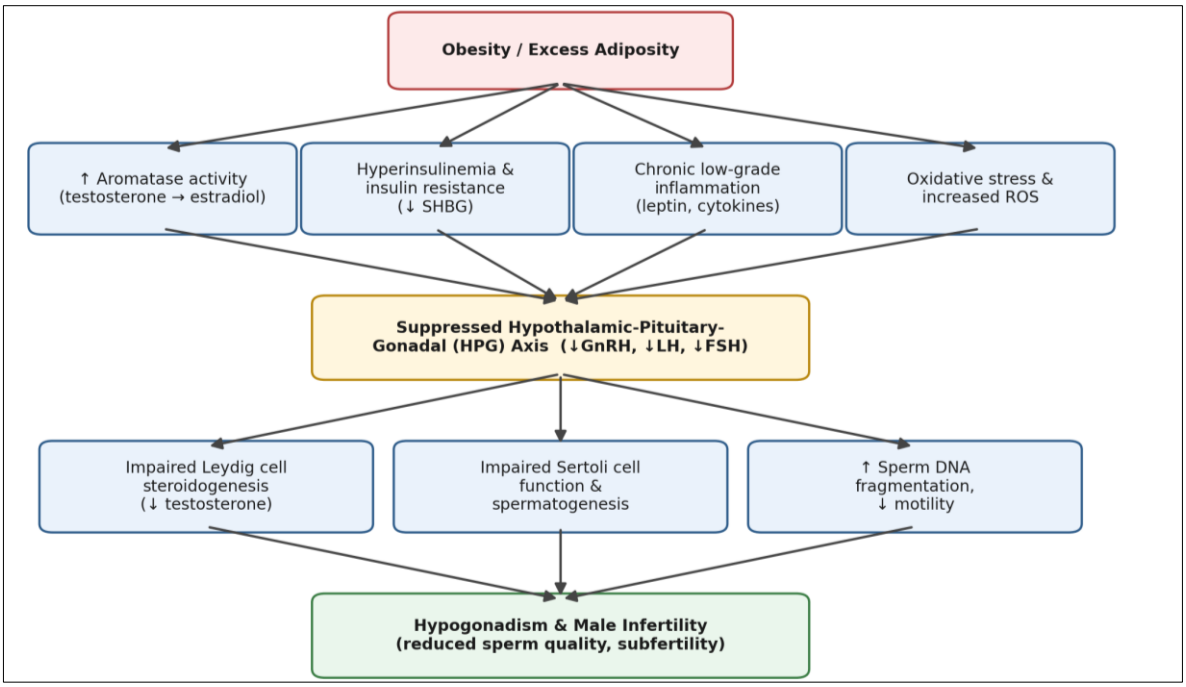

Figure 1: Mechanistic pathways linking obesity to male reproductive dysfunction.[15,17,20,22]

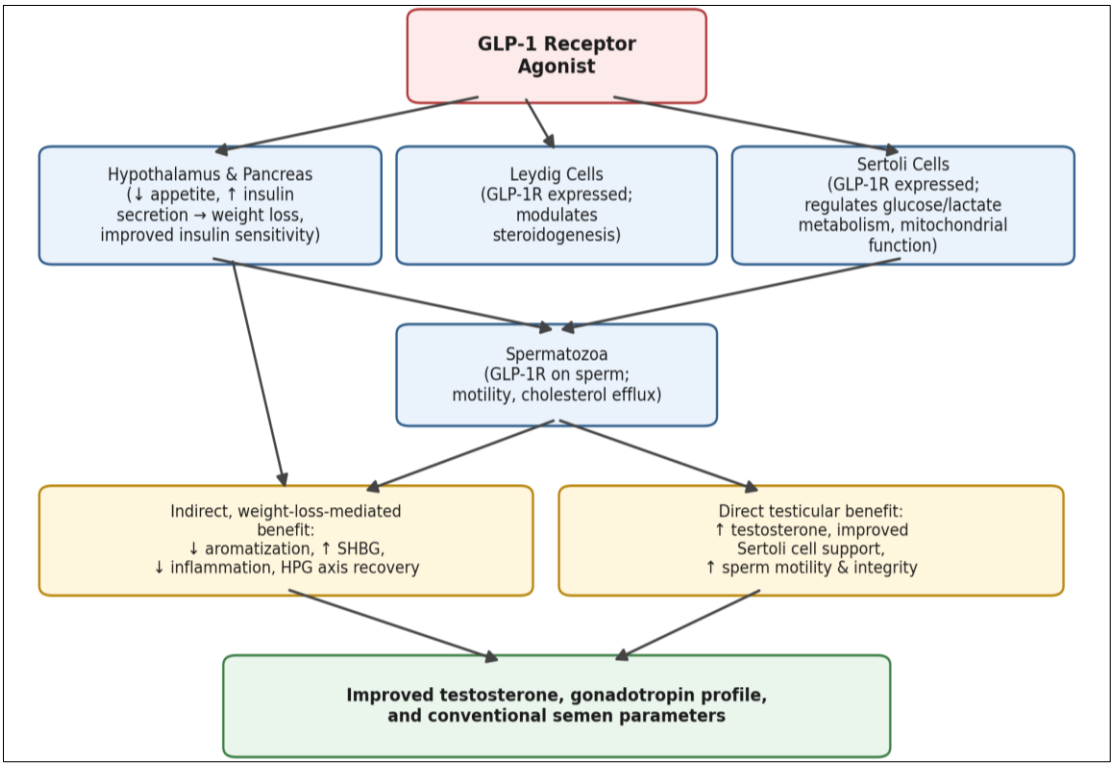

Figure 2: Proposed sites of GLP-1 receptor agonist action across the male reproductive axis, including indirect (weight-loss-mediated) and direct testicular effects.[4,12,26,27,30]

CLINICAL EVIDENCE IN MEN

Liraglutide

By far, the most convincing evidence from humans is from liraglutide studies. Liraglutide at 3.0 mg per day was compared in a prospective trial of 110 young men with obesity-induced functional hypogonadism to gonadotropins and transdermal testosterone administration over four months, where those receiving liraglutide exhibited improved sperm parameters in terms of concentration, motility, and morphology in addition to erectile function scores superior to those on either comparative therapy, with many men restored to the eugonadal status.[17] In another study by Giagulli et al the combination of liraglutide with testosterone and metformin therapy in obese men with hypogonadism and type 2 diabetes resulted in improved erectile function and normalization of testosterone levels.[18]

In a randomized comparison of liraglutide to testosterone replacement therapy in men with obesity-induced functional hypogonadism, Jensterle et al discovered similar total testosterone increases in liraglutide treatment without any reductions in luteinizing hormone and follicle-stimulating hormone production in contrast to exogenous testosterone which suppressed these hormones and thus indicated the restoration of HPG axis functioning instead of its suppression, an especially important point for reproductive-age men.[19] In all the above-mentioned studies, testosterone improvement corresponded to the amount of weight loss.

Semaglutide, dulaglutide, and weight-loss-mediated effects

Fewer studies have focused on the effects of semaglutide and dulaglutide in particular on male fertility, but much of the data comes from weight reduction trials in which semen analyses were done as part of the secondary endpoints. In a sub-study within the S-LiTE trial, Andersen et al randomly assigned obese men to receive liraglutide, exercise, both or neither after low-calorie diets initially and showed that sperm concentration and total sperm count were increased significantly at eight weeks following weight loss by the diets and were sustained for one year only in patients who achieved 12 kg of weight loss, independently of any treatment groups.[10]

In a placebo-controlled cross-over study, Lengsfeld et al studied the effects of dulaglutide on healthy men without obesity and showed no clinically significant negative effects on sexual functions, which was a preliminary reassuring safety data on this agent in reproductive-aged men.[29] This is a consistent finding in the literature that the degree of sustained weight loss and not the GLP-1 receptor agonist molecule seems to play the key role in improving semen parameters, though the possible contribution of direct testicular effects remains uncertain.[16]

Tirzepatide and emerging dual and triple agonists

Despite its clinical success, tirzepatide has not yet undergone assessment in clinical trials aimed at evaluating the drug's impact on the male reproductive system or fertility. As has been seen in the pivotal SURMOUNT-1 trial, the drug demonstrated a mean reduction of 20.9% in body weight at the highest dose level over 72 weeks, which was significantly higher compared with the 15% body weight reduction obtained by semaglutide 2.4 mg in the STEP-1 trial.[23,21] The higher efficacy of the drug in terms of body weight reduction may indicate that the drug could provide even greater benefits for male reproduction through metabolism optimization; however, the added action on the GIP receptor adds an extra variable in the equation because the role of this receptor in male reproduction has never been studied.[13,14]

GAPS IN THE LITERATURE AND FUTURE RESEARCH DIRECTIONS

Despite some promising signs in terms of preclinical studies and preliminary clinical results, there are still several drawbacks that make it impossible to apply GLP-1 agonists clinically for the treatment of male infertility. First, most clinical trials involve too few patients, are non-randomised, or take semen parameters as the secondary outcome measure, and do not last more than four to six months – less time than required for a full spermatogenic cycle (approximately three months).[4,16] There is no evidence comparing different GLP-1 agonists in relation to their effects on reproduction. No one studied the effects of tirzepatide or triple agonists on fertility-seeking men. It should be noted, however, that the dangers of the administration of exogenous testosterone on the process of spermatogenesis via negative feedback inhibition of the HPG axis in hypogonadal men of reproductive age have been confirmed. Therefore, GLP-1 agonists and similar drugs with positive influence on gonadotropin secretion may become quite promising for future studies.[22] The effectiveness of surgical means, such as bariatric surgery for the treatment of obesity, has been proved. However, in small prospective studies the impact of this procedure on semen parameters was inconsistent.[24] Of critical importance, pregnancy and live birth rates — the most relevant outcomes for infertile couples — are rarely reported.[12] Future studies must focus on adequately powered RCTs of obese men with male infertility using semen quality, sperm DNA fragmentation, and pregnancy rates as the main end points; studies that separate the direct effect of GLP-1 receptor activation within the testis from any secondary effect mediated by weight loss; studies of dose and duration of GLP-1R agonism that take into consideration the spermatogenic cycle; and long-term safety data in young males, with attention to the epigenetics of sperm. Such information is needed before GLP-1R agonists, such as tirzepatide, or even dual or triple GLP-1R agonists, can become a part of treatment algorithms in obese male infertility.

CONCLUSION

Obesity is one of the key factors contributing to infertility in males, operating via HPG axis impairment, insulin resistance, inflammation, and oxidative stress. The ability of GLP-1 receptor agonists to cause weight loss and metabolic changes, along with the recently discovered GLP-1 receptor signalling in Leydig cells, Sertoli cells, and sperm, makes these agents promising candidates for the treatment of infertility for obesity-associated male reproductive dysfunction. Recent studies, mainly involving liraglutide, indicate a favourable impact on levels of testosterone and gonadotropins and semen parameters in general, without spermatogenic side effects that might occur due to the administration of exogenous testosterone. Nevertheless, the available evidence is diverse and is mainly based on small-sized trials of limited duration; there is virtually no information on tirzepatide or other new dual or triple agonists. Further research with appropriate trial design and fertility-oriented endpoints is required before recommendations for GLP-1 receptor agonists as a treatment modality in obese males with infertility.

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