RESEARCH ARTICLE

JENER Journal of Empirical and Non-Empirical Research
Volume 2, Issue 3, Page 193-196 | Article No. 27
DOI: 10.4898/jener.v2i3.a27
ISSN: 7895-9216
Journal homepage: https://jenerjournal.com
Journal email: editor@jenerjournal.com

JENER Journal
JENER Journal of Empirical and Non-Empirical Research

Testosterone Use and Preventable Male Infertility: A Public Health Perspective

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ARTICLE INFORMATION

Article history:
Published: March 2026

Keywords:
Testosterone therapy
Male infertility
Public health
Spermatogenesis
Reproductive health

ABSTRACT

Exogenous testosterone has been on the rise in the United States, even in men of reproductive age. Testosterone can be used in the treatment of fatigue, low libido, and decreased vitality. It has a known suppressive impact on sperm production. Exogenous testosterone impairs the hypothalamic pituitary gonadal axis and decreases the intratesticular testosterone that is needed to induce spermatogenesis. A lot of men enter therapy without a definite counseling of fertility hazard. This facilitates a means of male infertility against which one can act. In the context of the health of the population, infertility caused by testosterone raises the need to use assisted reproductive technologies, as well as leads to the imbalance of access to care. The paper discusses biological processes and clinical findings about recovery and unmet needs in patient education. It also presents prevention measures which concentrate on training of the providers, counselling standards, and policy alignment. By resolving this problem, reproductive autonomy is facilitated, and the unnecessary healthcare burden is minimized.

1. Introduction

Male infertility is one of the health issues that raise worldwide concern where it has contributed to close to half of cases of infertility among couples [1]. Although it is highly prevalent, prevention and early intervention measures are not given the due emphasis both in clinical practice and in public health policy [2]. More focus is normally placed to assisted reproductive technologies instead of focusing on the modifiable risk factors including obesity, smoking, exposure to environmental toxins, metabolic disorders, and late medical cheque-ups [3]. This has led to the obvious loss of useful fertility preservation and early management opportunities.

Meanwhile, testosterone treatment has grown beyond the well-defined medical conditions [4]. Even though testosterone replacement therapy is suitable in men who have been clinically diagnosed with hypogonadism, it is being increasingly used in the treatment of younger men who are being treated with nonspecific symptoms like fatigue, low libido, or decreased sexual performance [5]. Nonetheless, exogenous testosterone suppresses the hypothalamic–pituitary–gonadal axis and reduces intratesticular testosterone and disrupts spermatogenesis [6]. This suppression in men wishing to be fertile in the future may cause temporary or permanent infertility. As a result, there is a developing new at-risk group of men who are facing the possible avoidance of infertility because of hormones. These trends emphasize the necessity to implement more and more stringent prescribing policies, better fertility counselling before the testosterone therapy is provided, and more powerful preventive policies of public health.

1.1 Scope of the Problem

There has been an increase in the prescription rates of testosterone within the last ten years [5]. Another significant segment of users is comprised of men who are below the age of 45. Treatment is initiated in most instances without the laboratory evidence of hypogonadism being confirmed [4]. There is also an increased access to direct-to-consumer advertising and online clinics, which is a cause of concern in terms of inappropriate use and lack of control [2].

1.2 Public Health Relevance

There are profound clinical, psychological, and monetary repercussions of infertility to the affected couples [6]. Long periods of indecision, frequent visits to doctors, and high cost of treatment may lead to emotional pressure and strain the family pocket and are often experienced by individuals. Where infertility is caused by unchangeable exposures or medical procedures, it becomes a preventable health issue in the population. The example of the male infertility linked with exogenous testosterone intake is one of them [2][3]. Early education, proper prescribing practice and fertility counselling play a pivotal role in eliminating preventable reproductive harm in such situations. Individual wellbeing is enhanced by tackling the causes of infertility that may be prevented instead of depending on the expensive assisted reproductive technologies, which further points to its significance in the wider scope of the population health planning.

2. Literature Review

There are established findings on the dependence of exogenous testosterone and sperm production suppression [5]. Endocrinology and urology studies prove to be consistent [3].

Increased lecithin depletion in the brain is known to cause neurochemical changes, which affect mental functions and cognition.

2.1 Hormonal Control and Repression

The male reproduction is hormonally controlled by an accurate regulation of the hypothalamic-pituitary-gonadal (HPG) axis [1]. This axis is involved in signalling of the endocrine systems required to support normal spermatogenesis and general reproductive wellbeing. The hypothalamus secretes Gonadotropin-releasing hormone (GnRH) which prompts the secretion of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) by the anterior pituitary [2]. LH stimulates Leydig cell in the testes to stimulate the production of testosterone which is critical in sustaining intratesticular testosterone levels that are necessary in the formation of sperm [3]. At the same time, FSH has a direct effect on Sertoli cells to stimulate the process of spermatogenesis and sperm maturation [4].

Neurochemical balance is another factor that helps in regulation of hormonal activity. After having changes in brain chemistry, such as that related to lecithin deprivation, has been reported to affect cognition and neuroendocrine regulation, which may in turn change the hormonal control processes [5].

This well-regulation system however is disturbed in case exogenous testosterone is introduced. High amounts of testosterone in the blood inhibit GnRH secretion by negative feedbacks and decrease the secretions of LH and FSH by the pituitary gland [2]. This causes a significant decrease in intratesticular levels of testosterone in the presence of normal or high levels of serum testosterone. This decrease negatively affects spermatogenesis and could result in reduced sperm production or result in azoospermia. Hence, external testosterone treatment may destroy the fertility by disrupting the natural hormonal balance.

2.2 Clinical Evidence on Fertility Effect

It is always shown by clinical evidence that the tests of sperm counts will reduce in a few months after the commencement of the testosterone replacement therapy (TRT) [3]. Suppressive effect is attained because exogenous testosterone suppresses the release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) which are vital to the maintenance of intratesticular testosterone and normal spermatogenesis. Consequently, the concentration of sperm in most men gets considerably lowered, and some of them develop azoospermia, which refers to the total lack of sperm in the ejaculate [3]. This inhibitory ability is so potent that testosterone has even been proposed as a possible male hormonal contraceptive since it can be used with great confidence to inhibit the development of sperm [3].

Clinical practise thus discourages the use of testosterone in males who wish to remain fertile now or in the future due to its already known suppressive impact on the production of sperm [8]. Nevertheless, recent studies released in 2025 and 2026 also show that the prescription of testosterone in younger men still has been on the increase [8]. The crux upon these studies is that there is a lack of records on fertility counselling preceding the therapy, which implies that many patients are poorly informed of the possible reproductive effects [3]. The issue with this lack of congruence between evidence-based guidelines and prescribing is that it is possible to avoid infertility and better clinical supervision should be provided.

2.3 Gaps in Clinical Practice

In spite of evident facts, there are still shortcomings in clinical care. Most providers concentrate on symptom management without talking about reproductive long-term objectives [2]. Lack of time and standardized protocols are some of the factors that contribute to missed counselling [6]. Besides, the care can be fragmented. One provider may administer testosterone to patients and another care provider administer fertility care to patients, which puts off the identification of the issue [7].

3. Methodology

The paper uses a narrative review design by combining evidence on the effects of testosterone therapy on male fertility focusing on biological, clinical, and public health. The narrative approach enables the integration of various forms of evidence, including mechanistic data and population-wide implications to find patterns, gaps and possible spheres of intervention. It is not concerned with producing new primary data, but with assessing the outcomes of hormone, reproductive and systemic effects together, hence the study would be appropriate in informing clinical practice as well as the policy on health of the populace.

3.1 Data Sources

Peer-reviewed journals, clinical guidelines, and public health reports were used to obtain data. It focused on the literature on the testosterone therapy and its effects on the male reproductive outcome [1]. It has used sources such as experimental studies, observational studies, and consensus reports by experts, which assure the consideration of both the mechanistic and applied evidence.

3.2 Analytical Approach

The reviews were conducted in a systematic fashion to determine common trends in terms of biological processes, clinical outcomes, and overall public health [2][3]. The levels of evidence based on the combinations of the hormonal pathways impacted by the exogenous testosterone, clinical results rated on the basis of the reproductive outcomes, analyzing the public health data on the basis of the systemic and societal implications were all evaluated. It was an integrated method which allowed the holistic interpretation of the evidence.

3.3 Inclusion Criteria

Only relevant studies, focusing on the use of testosterone and male fertility, were included, whereby the inclusion was limited to recent studies by 2010 or so [5]. Both experimental and observational studies which gave quantifiable or mechanistic evidence were taken into consideration.

3.4 Limitations

Primary data collection is not used in the study, but the literature available is used instead; this literature may have different quality and methodology [3]. Nevertheless, the evidence-based support of conclusions is due to the consistent results of numerous studies [6], which makes it possible to draw evidence-based conclusions about the reproductive impacts of testosterone.

4. Findings

The pattern of hormonal suppression makes treated cases of testosterone treatment that has direct influence on the production of sperm [1]. Exogenous testosterone increases the level of blood androgen, and this provokes a negative feedback inhibition of the hypothalamic-pituitary-gonadal axis. This decreases the release of gonadotropin-releasing hormone (GnRH), luteinizing hormone (LH) and follicle-stimulating hormone (FSH) which are needed to sustain intratesticular testosterone and normal spermatogenesis. With lowered intratesticular testosterone, there is a sharp reduction of sperm production and this may result in oligospermia or azoospermia. These results indicate that there is an obvious biological association between testosterone therapy and compromised male fertility results.

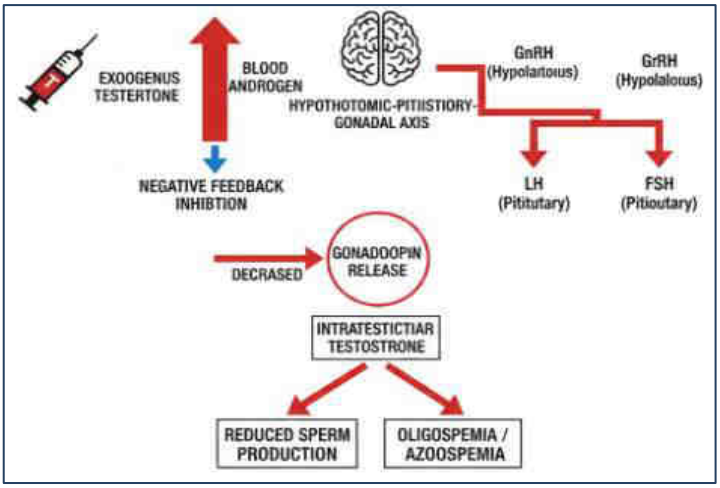

Figure-1: Hormonal Mechanisms of Testosterone Therapy and Male Infertility; SOURCE: [2,3]

4.1 Biological and Clinical Results

The use of exogenous testosterone alters physiological regulation of the hypothalamic-pituitary-gonadal axis by negative feedback inhibition [2]. Increased circulating testosterone inhibits the release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH), which are needed to sustain intratesticular testosterone and also to ensure spermatogenesis [3]. With a reduction in the intratesticular level of testosterone, sperm production will reduce very fast and some men can develop severe oligospermia, or even azoospermia. This suppression is possible clinically in a matter of months when the therapy is initiated. The post-discontinuation recovery is unpredictable and inconsistent. Clearly, some men recover normal levels of sperm count in a few months, but others are able to recover after a period of more than a year, partially or completely. In severe instances, some medical treatments like the use of gonadotropin are needed to induce spermatogenesis and regain fertility potential [5].

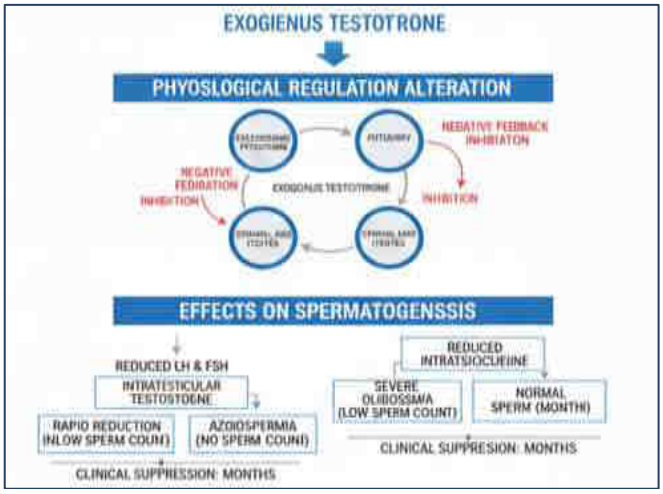

Figure-2: Exogenous Testosterone: Biological & Clinical Results; SOURCE:[3]

4.2 Public Health Impact

The lasting effects of infertility caused by testosterone is not only limited to the individual patients but is a general population health issue. Demand In the growing infertility rates in males, the use of assisted reproductive technologies (ART) such as in vitro fertilisation and intracytoplasmic sperm injection become more popular [6]. Such interventions are expensive, resource consuming and in most cases can not be fully paid by the insurance systems. Consequently, most couples find themselves in a big financial strain in their bid to undergo fertility treatment. Increasing dependency on ART further puts more pressure on health facilities infrastructure in terms of special clinics, laboratory facility, and skilled personnel. Moreover, avoidable infertility associated with improperly prescribed testosterone shows the gaps in preventive health education and clinical practise, which underline the necessity to implement policy-level interventions that would minimise preventable reproductive harm [6].

4.3 Health Equity Implications

Access to fertility assessment and therapy is still unevenly distributed within the socioeconomic groups. Patients with greater levels of income will experience increased access to specialised consultations, diagnostic tests, and sophisticated technology of reproductive technologies [2]. Conversely, poor populations tend to have late diagnosis and low chances of getting treatment. Infertility caused by testosterone can also increase the difference, since men under therapy who are not counselled appropriately can subsequently seek costly fertility treatment [8]. Since this kind of infertility can be prevented significantly, the lack of such control and patient education can contribute to the further reduction of inequities in reproductive healthcare. To avoid further proliferation of disparities in reproductive health, preventive measures, especially those that relate to prescribing, need to be reinforced; equitable access to fertility counselling needs to be insured [6].

5. Conclusion and Recommendations

5.1 Conclusion

Infertility caused by testosterone can be prevented to a great extent. The unnecessary exposure of men to risk is caused by current prescribing practises and the absence of fertility counselling, makes the problem even worse. This problem needs to be addressed by concerted efforts, such as more strict clinical recommendations, education of patients, and community health community programmes. By carrying out both healthcare and community-based measures, one will be able to decrease preventable infertility, safeguard the health of reproductive organs, and educate men who may opt to use testosterone therapy to make informed choices.

5.2 Recommendations

Before embarking on testosterone therapy, providers need to evaluate reproductive objectives. Fertility risks must be delivered in an unambiguous manner. Men that would like to have children should be offered alternative treatments. Guidelines to be taken into consideration in the clinic should be observed more.

5.3 Policy Implications

Medical systems are expected to insist on recording of fertility counselling prior to the prescribing of testosterone. Training in medicine needs to have more emphasis on male reproductive health. The common myths about testosterone and fertility should be corrected through public health campaigns.

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