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Andropause: Myths and Management

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Over the past two decades, prescription rates for testosterone have risen sharply, due to direct-to-consumer advertising and the spread of online misinformation. As a result, the topic of andropause, or aging-associated testosterone deficiency, is increasingly encountered by Canadian primary care physicians. Despite its growing visibility, up to 25% of healthcare practitioners report challenges in accurately diagnosing and managing andropause. In this article, we clarify the common myths around andropause to better inform primary care physicians and their patients. We discuss the Canadian guidelines on diagnosis and management of testosterone deficiency. Finally, we provide insight into the safety of testosterone-replacement therapy. By implementing structured monitoring and shared decision making, primary care physicians can ensure safe, effective, and evidence-based care for aging Canadians.

Introduction

Andropause is a colloquial term used to describe age-associated testosterone deficiency. It is characterized by deficient production of testosterone, leading to three major categories of symptoms: **1)** sexual symptoms including reduced libido, erectile dysfunction, and ejaculatory dysfunction; **2)** cognitive symptoms including fatigue, depression, and insomnia; and **3)** physical symptoms including decreased energy levels, increased visceral fat, and alterations in bone and muscle density.[1]

Testosterone deficiency has been receiving considerable attention in recent years, with a growing proportion of men receiving exogenous testosterone despite the absence of a conclusive diagnosis of hypogonadism.[2,3] Due to considerable misinformation about andropause and the benefits of testosterone-replacement therapy (TRT), coupled with a push for its direct-to-consumer advertising,[4,5] primary care physicians are increasingly facing patient-initiated requests for testosterone testing and treatment. Further, up to 25% of Canadian healthcare practitioners report difficulty with the diagnosis and treatment of testosterone deficiency.[1] Demystifying the diagnosis and management of andropause will thus benefit Canadian physicians and patients alike.

In this article, we will examine some of the common myths surrounding aging-related testosterone decline and discuss its management.

Common Myths

Myth 1: Andropause is simply the male equivalent of menopause.

Reality: Andropause is highly variable and not universally experienced by men.

Unlike menopause, which is universally experienced by women typically around age 50, there is great variability in the clinical and biochemical manifestations of andropause. The onset of andropause is insidious, with a gradual decline in testosterone beginning as early as age 30, with serum testosterone levels decreasing at a rate of 1–2% per year[6] The decline in testosterone levels is influenced by many factors beyond chronological age, including smoking status, glycemic control (HbA1c), body mass index, and other comorbidities.[7]

While menopause is a well-characterized phenomenon resulting in complete loss of fertility, the criteria for andropause is subject to multiple guideline variations and does not follow a definitive path.[8] Indeed, the term andropause is somewhat misleading, as the true 'pausing' of male androgen production can only occur from the

complete loss of testicular function, such as from medical castration. This ambiguity is reflected in the diverse names given to andropause, including hypogonadism or late-onset hypogonadism (LOH) by the Endocrine Society and European Association of Urology, testosterone deficiency by the Canadian and American Urological Associations, or androgen deficiency in the aging male (ADAM).

Myth 2: Andropause is a "silver bullet" diagnosis for the symptoms of aging.

Reality: Andropause is often a symptom of aging rather than a cause.

Aging is associated with functional decline across multiple physiological systems, leading to a broad range of non-specific symptoms. Many of these symptoms—erectile dysfunction, reduced libido, fatigue, reduced muscle and bone mass, cognitive complaints, and weight gain—are loosely associated with reduced testosterone levels. Because of the inverse relationship between testosterone and biological age, patients may be misled to believe that testosterone reduction causes aging.

In practice, these symptoms are multifactorial, and their underlying causes are often difficult to determine. Erectile dysfunction, for example, is often more strongly associated with vascular health and psychosocial factors than with reduced testosterone levels. Comorbid conditions such as obesity and metabolic syndrome, diabetes, and cardiovascular diseases can also exert an influence on sexual function, energy levels, and cognition, independent of androgen status. Indeed, many of the classic signs of low testosterone, including the sexual symptoms of androgen deficiency, show only weak associations with low testosterone in older men.[9] Consequently, a large proportion of men with testosterone deficiency confirmed with bloodwork remain asymptomatic.[10]

Attributing aging-related symptoms to testosterone deficiency risks oversimplification that could lead to inappropriate treatment with TRT, which shows limited benefit in men without confirmed testosterone deficiency.[4] Despite this, testosterone deficiency should remain part of the differential diagnosis, and biochemical evaluation may be appropriate in patients presenting with multiple relevant comorbidities.

Myth 3: Andropause is an inevitable consequence for aging men, and all men should consider TRT.

Reality: Not all men experience symptoms of andropause, and TRT shows limited benefit without confirmed testosterone deficiency.

Despite an annual decline of testosterone levels, most men will maintain normal testosterone levels, and fewer than 25% meet biochemical criteria for testosterone deficiency.[10] Further, only approximately 2% of men are estimated to experience symptoms of andropause,[9] demonstrating the complex interactions between genetics, lifestyle, comorbidities, and medications that play a role in the manifestation of andropause. Rather than an inevitable consequence of aging, it is possible that andropause only impacts a small subset of men.

The goal of TRT is to restore physiological levels of testosterone in men with confirmed testosterone deficiency to alleviate the symptoms of andropause.[11] Clinical trials have shown that TRT can improve sexual, mood, and osteoporotic symptoms in men with testosterone deficiency.[12,13] However, TRT confers limited benefit in men without pathological hypogonadism,[4] and even among men with confirmed testosterone deficiency, improvements in vitality and cognition are modest.[12,13]

Testosterone replacement is not without risk, with commonly reported side effects including polycythemia, acne, fluid retention, gynecomastia, and worsening of sleep apnea.[11] TRT also leads to infertility and is thus contraindicated in patients wishing to remain fertile (see **Box 1**).[13] Despite these risks, off-label prescribing of TRT has substantially increased over the past two decades, largely driven by direct-to-consumer advertising and misinformation about its benefits in aging men without testosterone deficiency.[4,5] Although short trials of TRT (3–6 months) may be warranted for symptomatic men when alternative causes of the symptoms have been excluded, TRT should be used as a tool for the treatment of symptoms of low testosterone, rather than a 'fountain of youth' for aging men: We treat the patient, not the blood-test.

Contraindications for TRT
Allergy or hypersensitivity to testosterone
Male breast cancer (known or suspected)
Desire to preserve fertility
Metastatic or high-risk prostate cancer requiring androgen deprivation therapy
Unstable cardiovascular disease or recent cardiac events within the previous 3–6 months

Box 1. Contraindications for TRT; *courtesy of Eviatar Fields, PhD, Naeem Bhojani, MD, Bilal Chughtai, MD and Dean Elterman, MD*.

Clinical Pearls
Prioritize clinical history: Testosterone testing should not be performed routinely in all men but reserved for those with a positive history and persistent symptoms.
The equivocal range: In men with total testosterone between 8–12nmol/L, always calculate free testosterone because elevated sex hormone-binding globulin (SHBG) (elevated in ageing and liver disease) can mask a functional androgen deficiency.
Circadian testosterone: Always ensure that blood draws for testosterone are obtained between 7:00 and 11:00 AM. Testing outside this window increases the risk of false-positive results due to natural circadian decline.
TRT is not a "Fountain of Youth": Although TRT can help symptomatic patients with confirmed testosterone deficiency, clinical trials demonstrate minimal benefits for vitality, or for asymptomatic men with numerous contraindications, side effects and risks, and should be avoided in asymptomatic men regardless of testosterone levels.

Box 2. Clinical Pearls; *courtesy of Eviatar Fields, PhD, Naeem Bhojani, MD, Bilal Chughtai, MD and Dean Elterman, MD*.

Myth 4: Total testosterone is the definitive metric for diagnosing andropause.

Reality: Although total testosterone is useful as an initial test, it requires clinical correlation, as well as supplementary testing in equivocal cases.

According to Canadian clinical guidelines, the clinical diagnosis of testosterone deficiency is based on several factors (see **Figure 1** and **Box 2**).[1,3] First, as mentioned, an initial diagnosis requires a total testosterone level below 8 nmol/L (230 ng/dL), in conjunction with a positive clinical history and physical examination. However, because total testosterone measures both protein-bound and unbound hormone, patients whose testosterone falls within the borderline range of 8–12 nmol/L are considered equivocal. These patients warrant further biochemical workup to determine whether symptoms are driven by a lack of active, unbound hormone.

Up to 60% of plasma testosterone is bound to sex hormone-binding globulin (SHBG), rendering it biologically inactive, with only approximately 2% of total testosterone being unbound (i.e., free testosterone) and thus bioactive.[14] Variations in SHBG levels will skew the proportion of bound to unbound hormone for a given total testosterone value. Several factors are known to increase SHBG levels, including aging, hyperthyroidism, and liver disease.[14] As a result, many aging patients with testosterone levels within this equivocal range have a 'normal' appearing total testosterone level, despite having reduced free testosterone due to elevated SHBG, which could explain their symptoms. Calculating free testosterone in older men with total testosterone within the equivocal range can prevent clinicians from missing biochemical hypogonadism.

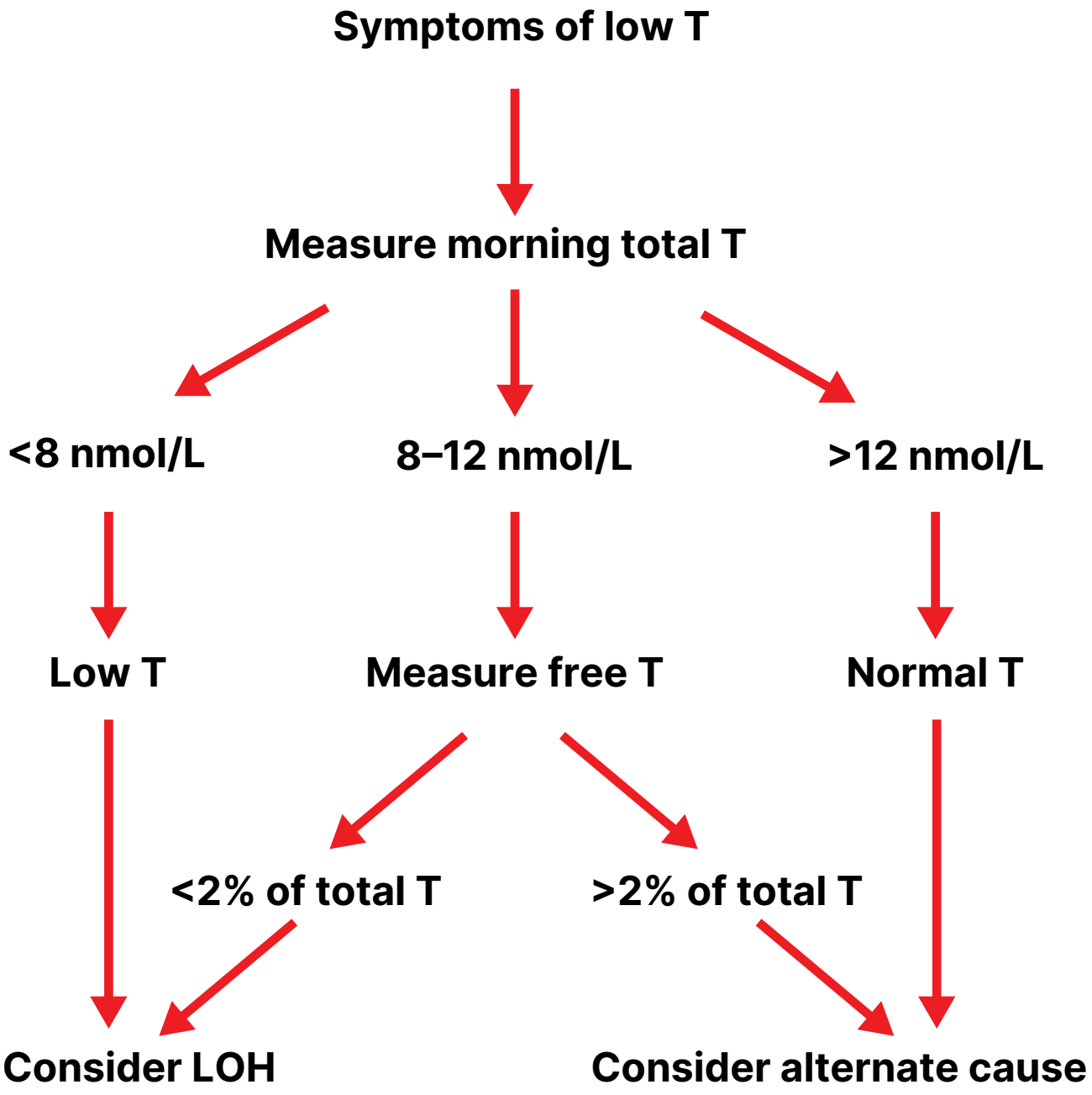

Figure 1. Diagnostic algorithm for men experiencing the symptoms of low testosterone.[1] Men with symptoms of low testosterone (T) in the absence of a cause should undergo measurement of morning total T. If total T falls within the equivocal range (8–12 nmol/L), free T should be calculated. Late-onset hypogonadism (LOH) can be considered in patients with low total T (<8 nmol/L) or with free T comprising less than 2% of total T; *courtesy of Eviatar Fields, PhD, Naeem Bhojani, MD, Bilal Chughtai, MD and Dean Elterman, MD.*

Diagnosis of Andropause

Canadian guidelines emphasize that biochemical evaluation be undertaken in conjunction with a thorough clinical history and physical examination. Thus, routine screening of men with non-specific symptoms such as fatigue, stress, or new-onset sexual dysfunction, is not recommended. Testosterone testing should be considered in men presenting with persistent sexual symptoms, including reduced libido or impaired quality of erections, especially after an inadequate response to phosphodiesterase-5 inhibitors. Other indications include unexplained sarcopenia and obesity in the absence of other metabolic syndrome components.[3]

The diagnostic criteria for andropause are not straightforward; however, the *Canadian Medical Association Journal* (CMAJ) provides official guidelines (**Figure 1**).[3,8] In men with a positive history and physical examination, the initial workup should include measurement of total testosterone levels. This blood sampling must be performed in the morning, ideally between 7:00 and 11:00 AM, to account for the circadian pattern of testosterone secretion and to avoid false-positive results. While Canadian guidelines do not specify a formal diagnostic threshold value for total testosterone, other international guidelines suggest that levels below 8 nmol/L are indicative of testosterone deficiency.[8] Investigation into reversible causes of hypogonadism is strongly recommended. Conversely, investigation for hypogonadism is suggested for men with unexplained anemia or sarcopenia of undetermined origin.[3]

Management of Andropause

Non-pharmacological Management

Non-pharmacological management of andropause should constitute first-line therapy for andropause and revolves around education and lifestyle modification aimed at managing comorbidities. Weight management is among the most impactful modifications that men could make, as adipose tissue forms a bidirectional relationship with testosterone. First, adipose tissue expresses high levels of aromatase, which converts testosterone to estradiol; elevated estradiol, in turn, inhibits hypothalamic gonadotropin release, which reduces testosterone production.[15] Conversely, low testosterone levels also trigger deposition of visceral adipose tissue, forming a vicious cycle that further reduces testosterone levels.[15] Thus, significant loss of visceral fat can increase serum total testosterone levels.

Similarly, exercise has been shown to increase testosterone levels and improve androgen receptor sensitivity.[16] Resistance training provides the best benefit for testosterone production and release,[17] while the added benefit of increased lean mass and management of comorbidities such as obesity and diabetes further improves testosterone levels. Sleep hygiene should also be optimized, as testosterone production follows a circadian pattern, with peak plasma testosterone levels occurring in the morning; unsurprisingly, sleep restriction is known to reduce testosterone levels.[18] Lifestyle modifications targeting other reversible causes of testosterone deficiency—including medications, nutrition, extreme aerobic exercise, obstructive sleep apnea, and acute illness—should also be considered.[1]

Pharmaceutical Options

The current Canadian guidelines for testosterone deficiency strongly recommend TRT in men with biochemical and clinical evidence of testosterone deficiency, provided no contraindications are present.[1,3] The goals of therapy are to relieve symptoms of testosterone deficiency through restoration of the patient's testosterone levels to physiological levels (14–17.5 nmol/L);[8] the guidelines acknowledge biological variability.[3]

Various formulations of TRT are available in Canada, including short-acting injectables, oral formulations, and transdermal delivery systems such as patches, gels, and solutions (see Table 3 in [1]). Selection of the appropriate formulation should be made through shared decision making between the physician and patient, based on the patient's goals and lifestyle. Given that exogenous testosterone suppresses gonadotropin production leading to infertility, alternative therapies, such as human chorionic gonadotropin, aromatase inhibitors, and selective estrogen receptor modulators are indicated in patients wishing to remain fertile.[3,13]

Monitoring and Safety

The safety profile of TRT remains an active area of study, and the risks of treatment should be balanced with the goals of the patient. Current evidence suggests that testosterone treatment does not increase the risk of developing prostate

cancer,[12] although patients with active prostate cancer should consult a urologist prior to initiation of TRT.[1] Similarly, there is no evidence suggesting that TRT worsens symptoms of benign prostatic hyperplasia, and some patients may experience improvements in lower urinary tract symptoms.[1] Furthermore, while earlier studies raised concerns regarding cardiovascular risk, the TRAVERSE trial demonstrated that TRT does not increase the incidence of cardiac events in patients who are at risk of- or have preexisting- cardiovascular disease risk.[19] TRT can thus be provided to men with stable cardiovascular disease if appropriately monitored.[1,3]

Ongoing monitoring for adverse effects is critical to ensure the success and safety of TRT (see Table 4 in [1]). A baseline assessment is recommended prior to the initiation of treatment, followed by follow-up at 3, 6, and 12 months to evaluate symptoms, side effects, prostate specific antigen and serum testosterone levels, with or without assessment of other gonadotropins.[3] Hematocrit levels are important to evaluate at baseline, as well as at 3 and 12 months to monitor for polycythemia. A baseline digital-rectal exam is recommended, with annually assessments thereafter. A review of medications should be conducted in patients who undergo TRT, as testosterone can interact with numerous medications and increase the risk of hepatotoxicity, bleeding, and cardiac or thrombolytic events.[20]

Conclusions

Andropause is a heterogenous and often over-diagnosed condition, largely due to inconsistent guidelines and the non-specific nature of its symptoms. Diagnostic criteria require both biochemical confirmation in conjunction with clinical symptoms. While lifestyle modification remains first-line therapy, selected men with confirmed testosterone deficiency can benefit from TRT, provided that potential risks of testosterone therapy are considered. Ultimately, primary care physicians play a central role in the diagnosis and treatment of andropause.

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References

1. Grober ED, Krakowsky Y, Khera M, Holmes DT, Lee JC, Grantmyre JE, et al. Canadian Urological Association guideline on testosterone deficiency in men: evidence-based Q&A. Can Urol Assoc J. 2021;15(5):E234-E243. doi:10.5489/cuaj.7252
2. Jasuja GK, Bhasin S, Reisman JI, Berlowitz DR, Rose AJ. Ascertainment of testosterone prescribing practices in the VA. Med Care. 2015;53(9):746-752. doi:10.1097/MLR.0000000000000398.
3. Morales A, Bebb RA, Manjoo P, Assimakopoulos P, Axler J, Collier C, et al. Diagnosis and management of testosterone deficiency syndrome in men: clinical practice guideline. CMAJ. 2015;187(18):1369-1377. doi:10.1503/cmaj.150033
4. Mok SF, Fennell C, Savkovic S, Turner L, Jayadev V, Conway A, et al. Testosterone for androgen deficiency-like symptoms in men without pathologic hypogonadism: a randomized, placebo-controlled cross-over with masked choice extension clinical trial. J Gerontol A Biol Sci Med Sci. 2020;75(9):1723-1731. doi:10.1093/gerona/glz195
5. Bandari J, Ayyash OM, Emery SL, Wessel CB, Davies BJ. Marketing and testosterone treatment in the USA: a systematic review. Eur Urol Focus. 2017;3(4-5):395-402. doi:10.1016/j.euf.2017.10.016
6. Harman SM, Metter EJ, Tobin JD, Pearson J, Blackman MR. Longitudinal effects of aging on serum total and free testosterone levels in healthy men. J Clin Endocrinol Metab. 2001;86(2):724-731. doi:10.1210/jcem.86.2.7219
7. Kanabar R, Mazur A, Plum A, Schmied J. Correlates of testosterone change as men age. Aging Male. 2022;25(1):29-40. doi:10.1080/13685538.2021.2023493
8. Kwong JCC, Krakowsky Y, Grober E. Testosterone deficiency: a review and comparison of current guidelines. J Sex Med. 2019;16(6):812-820. doi:10.1016/j.jsxm.2019.03.262
9. Wu FCW, Tajar A, Beynon JM, Pye SR, Silman AJ, Finn JD, et al. Identification of late-onset hypogonadism in middle-aged and elderly men. N Engl J Med. 2010;363(2):123-135. doi:10.1056/NEJMoa0911101

10. Araujo AB, Esche GR, Kupelian V, O'Donnell AB, Travison TG, Williams RE, et al. Prevalence of symptomatic androgen deficiency in men. J Clin Endocrinol Metab. 2007;92(11):4241-4247. doi:10.1210/jc.2007-1245
11. Miner M, Barkin J, Rosenberg MT. Testosterone deficiency: myth, facts, and controversy. Can J Urol. 2014;21 Suppl 2:39-54.
12. Snyder PJ, Bhasin S, Cunningham GR, Matsumoto AM, Stephens-Shields AJ, Cauley JA, et al. Effects of testosterone treatment in older men. N Engl J Med. 2016;374(7):611-624. doi:10.1056/NEJMoa1506119
13. Mian AH, Yang DY, Kohler TS. Current management and controversies surrounding andropause. Urol Clin North Am. 2022;49(4):583-592. doi:10.1016/j.ucl.2022.07.003
14. Krakowsky Y, Conners W, Morgentaler A. Serum concentrations of sex hormone–binding globulin vary widely in younger and older men: clinical data from a men's health practice. Eur Urol Focus. 2019;5(2):273-279. doi:10.1016/j.euf.2017.05.007
15. Carrageta DF, Oliveira PF, Alves MG, Monteiro MP. Obesity and male hypogonadism: tales of a vicious cycle. Obes Rev. 2019;20(8):1148-1158. doi:10.1111/obr.12863
16. Vingren JL, Kraemer WJ, Ratamess NA, Anderson JM, Volek JS, Maresh CM. Testosterone physiology in resistance exercise and training. Sports Med. 2010;40(12):1037-1053. doi:10.2165/11536910-000000000-00000
17. Cheng H, Zhang X, Li Y, Cao D, Luo C, Zhang Q, et al. Age-related testosterone decline: mechanisms and intervention strategies. Reprod Biol Endocrinol. 2024;22(1):144. doi:10.1186/s12958-024-01316-5
18. Liu PY, Reddy RT. Sleep, testosterone and cortisol balance, and ageing men. Rev Endocr Metab Disord. 2022;23(6):1323-1339. doi:10.1007/s11154-022-09755-4
19. Lincoff AM, Bhasin S, Flevaris P, Mitchell LM, Basaria S, Boden WE, et al. Cardiovascular safety of testosterone-replacement therapy. N Engl J Med. 2023;389(2):107-117. doi:10.1056/NEJMoa2215025
20. Luther PM, Spillers NJ, Talbot NC, Sinnathamby ES, Ellison D, Kelkar RA, et al. Testosterone replacement therapy: clinical considerations. Expert Opin Pharmacother. 2024;25(1):25-35. doi:10.1080/14656566.2024.2306832