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Review Article

Menopause in Women, Andropause in Men: Myth, Reality, and Management

Helen Taylor MSc[1]* and Prof. Dr. Anabel Ternès von Hattburg [2]*

[1]*Managing Director Evexia Group Limited, Integrative Metabolic Health, UK*

[2]*Managing Director SRH Institute for Innovation and Sustainability Management, Germany*

***Corresponding author:** Helen Taylor MSC, Managing Director Evexia Group Limited, Integrative Metabolic Health, UK and Prof. Dr. Anabel Ternès von Hattburg, Managing Director SRH Institute for Innovation and Sustainability Management, Germany.

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Abstract

Menopause in women and the contested notion of "male menopause" raise overlapping biological, clinical and social questions about midlife endocrine change. This paper synthesises contemporary evidence on female reproductive senescence and male androgenic ageing (Late-Onset Hypogonadism, LOH), and proposes a clinical and research agenda to disentangle myth from biology. Drawing on epidemiology, endocrine physiology and clinical trials, we show that menopause is a well-defined, near-universal endocrine transition marked by ovarian failure and abrupt loss of oestrogen/progesterone, with predictable short- and long-term effects (vasomotor symptoms, genitourinary syndrome, bone and cardiovascular risk). By contrast, male ageing is characterised by a gradual, heterogeneous decline in testosterone and adrenal precursors, accompanied by rising SHBG, altered aromatisation and interactions with sleep, stress and adiposity; only a subset of men develops symptomatic LOH. We summarise the typical phenotype of LOH (sexual dysfunction, sarcopenia, fatigue, mood and metabolic disturbance), emphasise pitfalls in diagnosis (non-specific symptoms, reversible causes, single low measurements), and outline a stepped management approach prioritising lifestyle and sleep optimisation, targeted diagnostic testing, and guideline-based testosterone therapy when indicated. We highlight unresolved controversies diagnostic thresholds, long-term safety of testosterone replacement, and the need for biomarkers to predict treatment response and recommend integrated pathways combining lifestyle, endocrine and psychosocial care. Clinicians, workplaces and policymakers should recognise midlife endocrine change as a biopsychosocial transition: accurate diagnosis, personalised treatment and supportive social contexts can mitigate morbidity and preserve function for both women and men.

Scientific-Theoretical Agenda

Maria is 49. Once full of energy, she now wakes drenched in night sweats, her heart racing as if chased by an unseen predator. Meetings at work feel heavier, her focus slips, and she wonders if she is "losing herself." Doctors reassure her that it is "just menopause," yet the intensity of the transition feels anything but small.

Across the city, David, 52, notices a different erosion. His mornings feel slower, the gym no longer rewards him with the strength he once took for granted, and intimacy has become a struggle he does not voice aloud. He jokes about a "midlife crisis," but privately, he wonders whether something deeper is shifting.

These parallel stories capture a shared truth: midlife is not simply a chronological milestone but a biological and psychological turning point. For women, menopause is a well-defined transition marked by the abrupt cessation of ovarian function and a cascade of physiological consequences. For men, the picture is more ambiguous there is no sharp line, no universal signal, but rather a gradual decline in testosterone and vitality often referred to, controversially, as andropause or late-onset hypogonadism.

The question arises: is there truly a male menopause? If so, how does it compare to the female experience, and what are its medical, psychological, and societal implications? The answers matter not only for the individuals navigating these transitions but for the health systems, workplaces, and cultures that must respond.

This article sets out a theoretical and clinical agenda: to disen-

tangle myth from biology, explore the gendered nuances of midlife hormonal change, and assess what can be done to support men and women alike through these profound phases of life.

Biological Basis of Menopause

Menopause is defined as the permanent cessation of menstruation resulting from the natural decline in ovarian follicular activity and the subsequent loss of reproductive capacity. The World Health Organization [17] identifies the average age of menopause as around 50 years, although the range can vary significantly depending on genetic, environmental, and lifestyle factors [17,18]. This transition is not a sudden event, but rather the culmination of a gradual process that begins years earlier during perimenopause, when irregular cycles and fluctuating hormone levels first appear.

From a biological perspective, menopause is characterized by a sharp reduction in ovarian production of oestrogen and progesterone, the two central hormones regulating the menstrual cycle. This hormonal decline underpins the cluster of physiological changes women may experience. Among the most common are vasomotor symptoms, such as hot flushes and night sweats, which affect up to 75% of women in Western populations [12]. Other immediate consequences include disturbances in sleep, changes in mood, and difficulties with concentration, all of which can impact daily functioning and quality of life. A further clinical hallmark of menopause is the Genitourinary Syndrome of Menopause (GSM), which encompasses vaginal dryness, irritation, urinary urgency, and increased risk of infections. These symptoms are closely linked to declining oestrogen levels and can significantly affect sexual health and intimate relationships. Importantly, while vasomotor symptoms may resolve over time, GSM often persists or worsens if untreated, underscoring the chronic dimension of menopause-related changes.

Beyond the acute symptoms, menopause has long-term health implications due to the protective role of oestrogen in multiple systems. Reduced oestrogen accelerates bone resorption, heightening the risk of osteoporosis and fracture, especially in the postmenopausal years. Cardiovascular health is also impacted, as oestrogen decline alters lipid metabolism and vascular function, contributing to an increased risk of heart disease in postmenopausal women [12]. These associations illustrate that menopause is not only a reproductive milestone but also a systemic biological transition with wide-ranging consequences.

At the same time, menopause cannot be understood as purely a biological phenomenon. Increasingly, it is framed as a biopsychosocial transition, where they lived experience is shaped as much by cultural narratives, social context, and personal coping strategies as by hormone levels. Studies show that women in societies where menopause is seen as a natural phase of life often report fewer symptoms than those where it is pathologised or stigmatised *El Khoudary, et al.,* (2019) [4]. Workplace culture plays a critical role as well: access to flexible working arrangements, open communication, and supportive leadership can substantially buffer the negative impact of menopausal symptoms on professional life. On the individual level, factors such as physical activity, mindfulness, and social support networks are associated with more positive experiences during this transition. Taken together, menopause represents a multifaceted turning point in women's lives simultaneously biological, psychological, and social. While its biological underpinnings are well-established, the diversity of women's experiences highlights the importance of broad, interdisciplinary perspectives when studying and addressing menopause.

Male "Menopause"– Andropause or Myth?

In contrast to the female menopause, which is marked by a clear and universal biological event- the cessation of ovarian follicular activity men does not experience a sharply defined reproductive endpoint. Instead, male reproductive ageing is characterised by a gradual and variable decline in circulating testosterone levels, typically beginning around the ages of 30 to 40 years, at an average rate of approximately 1% per year [6]. This slow and heterogeneous process stands in stark contrast to the abrupt hormonal transition in women and forms the basis of ongoing debates around whether there can truly be said to be a male "menopause."

Some men, particularly in later life, do develop clusters of symptoms often attributed to what is termed Late-Onset Hypogonadism (LOH). These include reduced libido, erectile dysfunction, low energy, fatigue, mood disturbances, irritability, and decreased motivation. Physiological changes may also occur, such as loss of muscle mass, increased central fat accumulation, and reductions in bone mineral density. In certain cases, cognitive complaints such as memory difficulties and reduced vitality are also reported *Wu, et al.,* (2008) [18]. These symptoms resemble some aspects of female menopause, fuelling the popularisation of the term "andropause" in both medical and media contexts. However, the universality of this phenomenon is contested. Unlike menopause, which is inevitable for all women, not all men experience clinically significant declines in testosterone or associated symptoms. Indeed, many men retain testosterone concentrations within the normal reference range well into advanced age, without any notable impairment of sexual function, mood, or vitality [14]. This variability underscores the difficulty of equating male ageing with a single, discrete transition comparable to menopause.

The diagnostic challenge lies in differentiating pathological hypogonadism from the normal consequences of ageing. Factors such as obesity, chronic illness, stress, poor sleep, and medications can suppress testosterone, confound the picture and complicate clinical decisions. Furthermore, reliance on symptoms alone can be misleading, as many of the complaints attributed to "andropause" such as fatigue or low mood are non-specific and may reflect psychosocial stressors or comorbid conditions rather than androgen deficiency [15,16].

The medicalisation of normal ageing is therefore a central concern. Overzealous diagnosis risks exposing men to unnecessary Testosterone Replacement Therapy (TRT), which carries its own risks, including cardiovascular events and prostate health concerns, though data remain contested. Conversely, under-recognition of genuine hypogonadism can leave affected men without

effective treatment. This delicate balance continues to drive controversy in endocrinology, with some experts rejecting the notion of "male menopause" altogether, while others argue for a refined clinical framework under the terms "late-onset hypogonadism" or "testosterone deficiency syndrome."

In sum, the notion of a male "menopause" is best viewed as a metaphorical analogy rather than a biological equivalence. While women experience a sudden, universal reproductive transition, men undergo a slow, highly variable trajectory of endocrine ageing. For some, this leads to a symptomatic syndrome with meaningful health implications; for others, testosterone levels remain robust, and no equivalent "pause" is experienced. The controversy over andropause reflects both biology and culture, highlighting the need for nuanced research and careful clinical judgment.

Mental Health and Midlife Transitions

Hormonal change in midlife is not only a biological process but also a profound psychosocial and emotional transition. Importantly, the ways in which men and women experience these changes differ substantially, reflecting both physiological contrasts and cultural narratives. For women, the perimenopausal period is associated with a rapid decline in ovarian function, leading to a sudden and dramatic reduction in oestrogen and progesterone levels. This "hormonal collapse" is biologically abrupt and has been consistently linked to an elevated risk of depression and anxiety, particularly when compounded by factors such as sleep disturbance, hot flushes, or psychosocial stressors [5]. Women in this stage are also more likely to experience cognitive complaints, mood variability, and increased vulnerability to burnout, especially in contexts where caregiving responsibilities and professional demands intersect.

In men, by contrast, hormonal ageing takes the form of a gradual decline in Hypothalamic-Pituitary-Gonadal (HPG) axis function, with testosterone levels typically decreasing at around 1% per year from midlife onwards. Unlike female menopause, this process is highly variable, influenced by genetics, lifestyle, obesity, and comorbidities, and does not result in a universal hormonal endpoint. Some men present with symptoms suggestive of late-onset hypogonadism, including fatigue, irritability, loss of libido, erectile dysfunction, and depressive symptoms. However, the causal role of testosterone deficiency in mental health outcomes remains debated, as many of these symptoms are non-specific and can also result from psychosocial stressors, chronic illness, or ageing itself [21].

This divergence between sudden collapse in women and gradual decline in men has implications for physical, mental, and sexual health. In both sexes, hormonal change intersects with bone density, cardiovascular function, and muscle integrity, shaping long-term risks such as osteoporosis, frailty, or cardiovascular disease. On the mental health side, depression, anxiety, and burnout are common midlife concerns, though their underlying mechanisms may differ by sex. Sexual health is also frequently affected: women report genitourinary syndrome of menopause with discomfort and reduced satisfaction, while men may struggle with erectile dysfunction and diminished sexual wellbeing linked to testosterone decline. Beyond biology, stigma and cultural framing play a central role in shaping lived experiences. Women often face dismissive or reductive attitudes in healthcare and workplaces, with menopausal symptoms trivialised as "just part of ageing." This lack of recognition can exacerbate feelings of isolation and negatively affect mental health. Men, conversely, are often constrained by norms of masculinity that make it difficult to acknowledge vulnerability related to sexuality, vitality, or ageing. Many are reluctant to seek help, leaving symptoms underreported and untreated [8].

A critical question, therefore, is how much of midlife wellbeing is determined by hormonal trajectories versus lifestyle and social factors. Evidence suggests that while biological decline is real, its impact is strongly modulated by exercise, nutrition, sleep, stress management, and supportive environments. In this sense, midlife transitions represent not only a biological challenge but also a social opportunity to redesign healthcare, workplace policies, and cultural narratives in ways that reduce stigma and promote resilience.

Discussion/Review

The phrase "male menopause" is commonly used in public discourse but is scientifically imprecise. Clinically, the phenomenon it attempts to describe is better termed Late-Onset Hypogonadism (LOH) or Partial Androgen Deficiency of the Ageing Male (PADAM): an age-associated, progressive decline in androgen production and action that, in a subset of men, crosses a threshold producing persistent symptoms and impaired function [13,7]. The difference with female menopause is fundamental: menopause is a relatively abrupt, universal endocrine event (permanent cessation of ovarian follicular activity) occurring in midlife that produces near-complete loss of ovarian sex steroids; male androgenic ageing is gradual, heterogeneous and not universal in its clinical manifestation [10]. For many men the decline in Testosterone (T) is clinically silent; for others, biochemical fall plus symptom burden constitute a treatable endocrine syndrome. This paper synthesises the evidence for the biological reality of andropause, characterises its typical course and mechanisms, summarises the clinical phenotype and differential diagnosis, outlines health implications, and discusses contemporary management principles, including risks and evidence gaps.

Evidence for the Existence of Andropause

Age-related androgen decline is demonstrable across populations and study designs. Longitudinal cohorts and cross-sectional surveys show that serum testosterone falls on average by approximately 1%-2% per year after the third decade of life; estimates of 1.3%-1.6% per annum have been reported in men over 40 [1,20]. By late old age many men have lost a substantial fraction often cited as roughly 20%-30% by age 75 compared with young adult levels though individual trajectories vary widely [3,1]. The decline in total testosterone is compounded by increases in Sex Hormone-Binding Globulin (SHBG) such that free (bioavailable) testosterone may fall disproportionately [7].

Prevalence estimates of clinically relevant LOH depend heavily on case definitions. When strict criteria are applied persistent low T on repeated morning measures plus a defined cluster of symptoms prevalence estimates in community cohorts are modest (single digits overall but rising markedly with age). For example, studies combining biochemical thresholds with sexual symptom requirements report prevalences near 2% in some European cohorts, rising to double digits in older age groups when less stringent symptom criteria are applied [19]. Broader symptom surveys (which capture non-specific ageing complaints) yield higher figures [10]. Importantly, prevalence and symptom patterns have been described in diverse geographic settings (India, Europe, Middle East), indicating that androgenic ageing is not restricted to a single population or ethnicity [13,10]. Taken together, the data indicate a universal biological process (age-related androgen decline) with a variable clinical expression (LOH in a subset).

Typical Onset and Course (a Staged Continuum)

Unlike the defined stages of female reproductive ageing (perimenopause, menopause, postmenopause), male androgen declines lack universally accepted staging. A pragmatic clinical framework recognises a continuum:

Early Androgen Decline (Mid-Life)

Testosterone begins to fall in many men from the mid-30s onward; in the 40s-50s many men show reductions that remain within or near the low-normal range. Symptoms at this stage tend to be mild or attributed to stress, work, family responsibilities, or poor sleep. Some authors have used terms such as “partial andropause” or colloquially “penopause” to denote this phase [7].

Progressive Decline and Symptomatic LOH

In later decades (60s-70s and beyond) some men experience further reductions in total and free testosterone that cross symptom-provoking thresholds. The prevalence of symptomatic LOH rises with age for example, one large study reported LOH prevalence of ~5.6% across men aged 30-79 but higher proportions among older subsets, with some analyses showing ~18% prevalence in men over 70, depending on definitions used [10].

Heterogeneity in Timing and Severity

Importantly, trajectories are heterogeneous: many men in their 70s maintain adequate androgenicity and remain asymptomatic, while others in their 50s can already manifest frank LOH. Comorbidities (obesity, diabetes, chronic illness), medications, lifestyle, sleep and stress all modulate the pace and clinical impact of decline. Clinicians often conceptualise andropause pragmatically as mild, moderate and severe reflecting symptom severity and biochemical reduction rather than as strict endocrine stages. This approach aligns management with functional impairment rather than with arbitrary age cut-offs.

Hormonal Changes Underlying Andropause

The endocrine substrate of andropause is complex and multi-factorial:

Testosterone and Gonadotropins

Testosterone production declines because of combined testicular (Leydig cell) ageing and alterations in hypothalamic-pituitary signalling. Unlike female menopause, which produces huge rises in gonadotropins, men typically retain partial gonadal function: LH may rise modestly to compensate, but in many ageing men the pituitary response is blunted or insufficient to normalise testosterone, producing a mixed picture of partial primary gonadal failure and central (secondary) hypogonadism related to illness or medications [13,5].

SHBG and Bioavailable Androgen

With advancing age SHBG concentrations generally increase; because SHBG binds testosterone, the rise causes a sharper decline in free (bioavailable) testosterone than in total testosterone. Consequently, older men with apparently “borderline” total T may experience clinically meaningful low free T an important diagnostic caveat [7].

Adrenopause: DHEA and Pregnenolone

Adrenal androgen precursors fall markedly with age DHEA and DHEA-S typically decline by several percent per year from the third decade onward and are substantially lower in elderly men compared with young adults [11]. Pregnenolone, a neurosteroid precursor, also decreases with age. Loss of these adrenal steroids may contribute to subjective declines in vitality, mood and some cognitive complaints, though therapeutic data are mixed [11,1].

Testosterone → Estrogen Balance

Estrogens in men arise chiefly via aromatisation of androgens in peripheral adipose tissue. Two opposing effects occur with age: falling testosterone might lower estradiol, but increasing visceral adiposity and aromatase activity often increase conversion of testosterone to estradiol, producing relative hyperestrogenism in obese men. Elevated peripheral aromatisation can suppress the HPG axis further and exacerbate androgen deficiency, while very low estradiol (in lean hypogonadal men) may contribute to bone loss [7].

Cortisol, Stress and Circadian Rhythms

The adrenal stress hormone cortisol often remains stable or increases relative to declining anabolic steroids; the resulting anabolic/catabolic imbalance may promote sarcopenia, visceral fat and insulin resistance. Sleep and circadian disruption common in modern life depress nocturnal testosterone pulses and blunt morning peaks. Empiric sleep restriction protocols in young men have produced declines in circulating testosterone comparable to years of ageing, underlining the physiological link between sleep and an-

drogen status [14]. Melatonin declines with age as well and may indirectly influence testosterone secretion through sleep regulation.

Collectively, these hormonal shifts reduced gonadal steroidogenesis, rising SHBG, falling adrenal precursors, altered aromatisation, and perturbations in stress and circadian physiology interlock to produce the endocrine substrate for symptomatic LOH.

Signs and Symptoms of Male "Menopause"

LOH produces a wide array of symptoms spanning sexual, physical, psychological and metabolic domains. Two points are important: (1) Individual symptom specificity varies considerably reduced libido and certain sexual complaints are relatively androgen-specific, while fatigue, mood changes and cognitive complaints are common to many conditions and (2) The syndrome often develops gradually, so patients may normalise slow declines until symptoms become marked.

Sexual and Reproductive

Diminished libido is the cardinal androgen-related symptom and correlates best with serum testosterone [19]. Loss of spontaneous and morning erections, decreased masturbation/sexual thoughts and reduced sexual satisfaction are commonly reported. Erectile Dysfunction (ED) co-occurs frequently but is often multifactorial (vascular disease, medications) and therefore less specific solely to LOH. Spermatogenesis and fertility decline with ageing and chronic hypogonadism, though men can remain fertile into older ages.

Somatic and Body Composition

Typical physical changes include reduced energy and exercise tolerance, loss of muscle mass and strength (sarcopenia), increased fat mass especially central adiposity and decreased bone mineral density. Hair thinning on the beard and body (not scalp androgenic alopecia) can occur with longstanding androgen deficiency. Hot flushes and sweating are less common than in women but occur in some hypogonadal men, particularly when testosterone loss is abrupt (e.g., after androgen-ablative therapy) [7].

Psychological and Cognitive

Men with LOH frequently report low mood, irritability, reduced motivation, fatigue and cognitive complaints such as poor concentration and memory lapses. These symptoms are non-specific and often overlap with depression, sleep deprivation and chronic disease; nevertheless, systematic reviews and some randomized trials indicate that testosterone replacement in confirmed hypogonadal men can yield modest mood improvements and, in selected cognitive domains, small gains [11,1].

Metabolic and Cardiovascular

Low testosterone is associated with adverse metabolic parameters higher visceral fat, insulin resistance, dyslipidaemia and increased prevalence of metabolic syndrome and type 2 diabetes. Observational data link low T with endothelial dysfunction, increased inflammatory markers and higher rates of cardiovascular events; whether low testosterone is a causal driver or a marker of underlying poor health remains partially unresolved [2,5].

Syndrome Prevalence of Specific Symptoms

When symptomatic LOH is identified (i.e., low T + required symptom cluster), sexual complaints and fatigue predominate. Large cohort summaries report high frequencies of low libido and lack of energy among men meeting LOH criteria, with other complaints (sleepiness after meals, memory issues, hair loss, mood symptoms) common but variably present depending on sample selection [19,10].

Comparison to Female Menopause and "Stages"

Several features distinguish male androgenic ageing from female menopause:

Universality

Men will all experience some decline in testosterone with age, but not all will develop clinically meaningful LOH. In contrast, menopause is essentially universal for women [13].

Onset and Abruptness

Female menopause is a relatively focused transition (often in late 40s to early 50s) with marked endocrine changes (gonadotropin surge; near-zero ovarian steroids). Male androgen decline is gradual, usually spanning decades with variable symptom onset and magnitude; there is no single biologic event analogous to cessation of menses [10].

Hormonal Profiles

Women enter a state of profound estrogen/progesterone deficiency accompanied by high gonadotropins. Aging men typically retain partial gonadal function and rarely experience the dramatic gonadotropin surges seen in women; many older men show only modest LH elevations and continuing (though diminished) testosterone production [5].

Symptoms Overlap but Differ in Scale

Some menopausal-type symptoms (hot flushes, mood lability, sleep disturbance) can occur in men with very low T, but the prevalence and intensity tend to be lower and more heterogenous [7]. Given these differences, the clinical approach to male androgenic ageing focuses on the presence and impact of symptoms together with biochemistry rather than on age alone or on fixed "stages."

Misdiagnosis and Differential Diagnosis

Andropause is commonly missed or misattributed because its cardinal complaints (fatigue, low libido, mood disturbance) overlap with many other conditions. Clinicians should rule out and address alternative and co-existing causes before attributing symptoms to LOH.

Key Conditions to Exclude

Major Depressive Disorder and Other Psychiatric Illnesses: Low mood, anhedonia and psychomotor slowing can mirror LOH. Sometimes psychiatric disorder is primary; conversely, hypogonadism can cause depressive symptoms. A careful psychiatric assessment is essential [19].

Thyroid Dysfunction: Hypothyroidism may present with fatigue, weight gain and low libido and should be excluded via TSH/T4 testing [7].

Chronic Systemic Disease: Diabetes, chronic liver or renal disease, HIV, inflammatory illnesses and malignancy can reduce testosterone and cause constitutional symptoms. Treating the underlying illness can partially reverse functional hypogonadism.

Medications and Substances: Opioids, long-term glucocorticoids, some antidepressants, cimetidine, spironolactone, ketoconazole and alcohol misuse can suppress T; medication review is a routine step [1].

Sleep Disorders: Obstructive Sleep Apnoea (OSA) reduces nocturnal testosterone secretion and causes daytime fatigue and low libido; treating OSA can improve and sometimes normalise androgen levels [14].

Pituitary or Testicular Pathologies: Macroprolactinoma, haemochromatosis, orchitis or testicular injury may cause hypogonadism requiring specific management. Measurement of LH/FSH and prolactin helps distinguish primary gonadal from central causes.

Practical Diagnostic Precautions

a) Do not diagnose LOH on the basis of a single low testosterone measurement obtained during acute illness or under the influence of interfering medications. Repeat morning testing on two separate occasions and ensure sampling conditions are appropriate [20].

b) Beware of over-reliance on symptom checklists alone (e.g., ADAM, AMS) because of low specificity; use them for screening but confirm with biochemistry.

c) Consider the whole patient: if a man presents with cognitive decline, weight loss or frank systemic features, a broader geriatric and endocrine assessment is indicated rather than reflexive initiation of TRT.

Health Implications and Associated Conditions

LOH has important clinical implications beyond symptomatic distress. Several health domains are consistently associated with low testosterone.

Metabolic and Cardiovascular Disease

Low testosterone is linked to insulin resistance, central obesity, dyslipidaemia and increased prevalence of metabolic syndrome and type 2 diabetes. Observational data also link low T with endothelial dysfunction, markers of oxidative stress and inflammation, and higher rates of cardiovascular events and mortality [2,5]. However, disentangling causality is challenging because chronic illness, obesity and inflammation both decrease T and increase cardiovascular risk. Contemporary randomized evidence including large trials suggests that, when used appropriately in symptomatic hypogonadal men and monitored according to guidelines, TRT does not necessarily increase cardiovascular events [9]. Nevertheless, cardiovascular risk assessment should inform decisions about initiating hormone therapy.

Bone Health and Frailty

Androgen deficiency contributes to bone loss; men with sustained low testosterone are at increased risk of osteopenia and osteoporosis. TRT has been shown to improve bone mineral density in hypogonadal men, supporting its role in fracture-risk management when clinically indicated [10]. Loss of muscle mass and strength associated with LOH contributes to frailty, falls and loss of independence in older men.

Mood, Cognition and Quality of Life

Although trials report heterogeneous cognitive outcomes, testosterone therapy in confirmed hypogonadal men can improve energy, mood and certain cognitive domains; even when objective cognitive change is modest, subjective improvements in concentration, motivation and quality of life are commonly reported *Samara, et al.,* (2013); *Ajayi, et al.,* (2024) [11,1].

Longevity and Mortality Associations

Observational cohort studies have linked low testosterone to higher all-cause mortality over long follow-up, but these associations are attenuated after adjustment for comorbidity and frailty markers, leaving open whether low T is a causal driver or an integrated biomarker of poor health and biological ageing [4,1]. The pragmatic clinical implication is that very low testosterone warrants assessment for underlying systemic disease and interventions to mitigate frailty and cardiometabolic risk.

Management Principles

Approach LOH with a staged, patient-centred strategy.

Confirm The Diagnosis and Identify Reversible Factors

Obtain two morning testosterone measurements, assess symptom burden with validated instruments (while recognising limits of specificity), and screen for reversible contributors (thyroid disease, medications, alcohol, OSA, systemic illness). Measure SHBG and calculate free testosterone when indicated (e.g., obesity, liver disease) [20].

Lifestyle Optimisation as First-Line

Lifestyle interventions are powerful: weight loss (particularly reduction of visceral fat), resistance exercise to rebuild lean mass, optimisation of sleep (treating OSA, improving sleep hygiene), moderation of alcohol and reduction of chronic stress can raise endogenous testosterone and improve symptoms in many men, especially those with borderline biochemical findings [11,14].

Consider Testosterone Replacement Therapy (TRT) for Symptomatic, Biochemically Proven LOH

When persistent, functionally limiting symptoms coexist with confirmed low testosterone despite optimisation of reversible factors, guideline-based TRT is an evidence-based option. Expected benefits include improved libido, lean mass, bone density and aspects of mood and energy [20]. Decisions should be individualised with shared decision-making, taking into account patient preferences and comorbidities.

Choice of Formulation and Monitoring

Multiple preparations exist (topical gels, transdermal patches, intramuscular injections including long-acting depot formulations and oral preparations where available). Monitoring should include haemoglobin/haematocrit (to detect polycythaemia), Prostate Surveillance (PSA and digital rectal examination consistent with local guidance), assessment of symptom response and routine review of cardiovascular risk factors [20]. TRT may worsen sleep apnoea or provoke erythrocytosis; prompt review and dose adjustment or cessation is indicated if complications arise.

Contraindications and Caution

Active prostate cancer remains a usual contraindication; poorly controlled heart failure and very unstable cardiovascular disease warrant caution. For men with significant polycythaemia, sleep apnoea or high baseline PSA, thoughtful risk-benefit discussion and close follow-up are mandatory.

Integrated Care and Follow-Up

Management often benefits from a multidisciplinary approach: primary care, endocrinology, urology, sleep medicine and mental health input as needed. Longitudinal follow-up is essential both to optimise outcomes and to collect real-world data on safety and benefit.

Contested Areas and Research Priorities

Several issues remain unresolved and merit further research:

Precise Biochemical Thresholds and Biomarkers

There is no universally accepted single cut-off for testosterone treatment; better biomarkers (including tissue-level indices or predictive markers of treatment responsiveness) would aid targeting.

Long-Term Safety and Benefit

Although randomized data reduce earlier safety concerns, longer follow-up studies are needed to fully clarify cardiovascular, prostate and cognitive long-term outcomes across diverse patient groups.

Predictors of Treatment Response

Identifying which men will derive meaningful symptomatic and functional improvement from TRT would improve cost-effectiveness and safety.

Non-Hormonal Strategies and Multimodal Care

Trials that combine lifestyle, sleep and metabolic interventions with selective TRT versus multimodal non-hormonal strategies could clarify optimal care pathways.

Population Health Implications

With ageing populations worldwide, robust evidence on when and how to screen for LOH in primary care settings and how best to deliver integrated lifestyle and medical care is needed.

Conclusion

Male androgenic ageing colloquially “male menopause” or andropause is a real, biologically grounded phenomenon. It differs fundamentally from female menopause in its gradualism, heterogeneity and incomplete universality. The core endocrine picture is one of progressive decline in testosterone and adrenal androgens, rising SHBG and disturbed interaction with metabolic, stress and circadian axes. Clinically relevant LOH is diagnosed by the concurrence of persistent, functionally impairing symptoms and reproducible low testosterone, after exclusion and management of reversible causes. From a clinical perspective the priorities are clear: (1) Use careful, guideline-based diagnostic practices (repeat morning measurements, assessment for reversible causes), (2) Prioritise lifestyle and sleep optimisation as first-line therapy in borderline cases, (3) Offer TRT to men with persistent, symptomatic, biochemically confirmed LOH using informed, shared decision-making and appropriate monitoring, and (4) Avoid both under-recognition of treatable hypogonadism and over-prescription of testosterone on the basis of non-specific ageing complaints or a single laboratory value.

Finally, the field needs better predictive biomarkers and longer-term safety data; until these are available, the balanced approach above combining lifestyle medicine with selective, monitored hormone therapy is the prudent path to restoring function and quality of life for men who truly have late-onset hypogonadism [20,9].

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Conflict of Interest

None.

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