TIBBIYOT
TA'LIMI & INNOVATSIYALARI

O'ZBEKISTON RESPUBLIKASI
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TOSHKENT TIBBIYOT AKADEMIYASI

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- 14.00.00 - Tibbiyot fanlari;
- 14.00.01 - Akusherlik va ginekologiya;
- 14.00.02 - Morfologiya;
- 14.00.03 - Endokrinologiya;
- 14.00.04 - Otorinolaringologiya;
- 14.00.05 - Ichki kasalliklar;
- 14.00.06 - Kardiologiya;
- 14.00.07 - Gigiena;
- 14.00.08 - Oftal'mologiya;
- 14.00.09 - Pediatriya;
- 14.00.10 - Yuqumli kasalliklar;
- 14.00.11 - Dermatologiya va venerologiya;
- 14.00.12 - Tibbiy reabilitologiya;
- 14.00.13 - Nevrologiya;
- 14.00.14 - Onkologiya;
- 14.00.15 - Patologik anatomiya;
- 14.00.16 - Normal va patologik fiziologiya;
- 14.00.17 - Farmakologiya va klinik farmakologiya;
- 14.00.18 - Psixiatriya va narkologiya;
- 14.00.19 - Klinik radiologiya;
- 14.00.20 - Tibbiy genetika;
- 14.00.21 - Stomatologiya;
- 14.00.22 - Travmatologiya va ortopediya;
- 14.00.23 - Hamshiralik ishini tashkil etish;
- 14.00.24 - Sud tibbiyoti;
- 14.00.27 - Xirurgiya;
- 14.00.28 - Neyroxirurgiya;
- 14.00.41 - Xalq tabobati;
- 14.00.35 - Bolalar xirurgiyasi;
- 14.00.34 - Yurak-qon tomir xirurgiyasi

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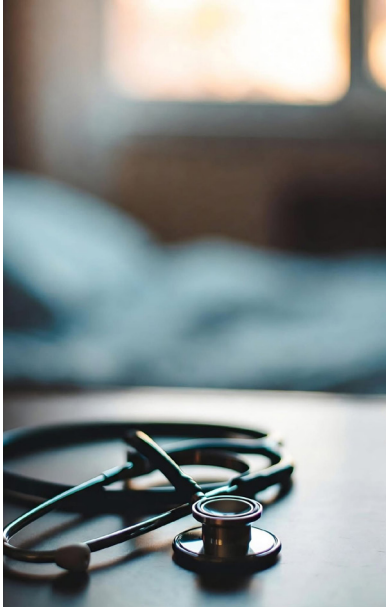
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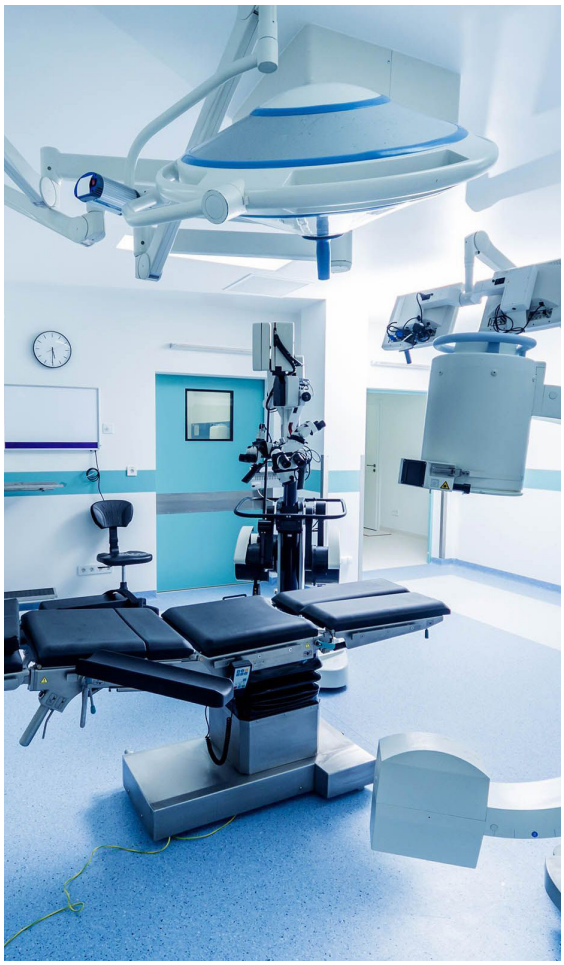
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MUNDARIJA

ТОШКЕНТ ХАЛҚАРО МОЛИЯ МАРКАЗИ ВА СОҒЛИҚНИ САҚЛАШ ТИЗИМИНИ РИВОЖЛАНТИРИШ: ЯНГИ ҚОНУННИНГ АҲАМИЯТИ 12
Шуҳрат Боймуротов

IMPROVEMENT OF THE SURGICAL APPROACH TO THE TREATMENT OF CRANIOVERTEBRAL JUNCTION ANOMALIES ASSOCIATED WITH SPINAL MALFORMATIONS (CLINICAL EXPERIENCE) 17
Arzikulov Sh.T., Ismailova R.O., Jabborov N.I.

ARTERIAL GIPERTENZIYA VA 2 TIP QANDLI DIABET: PATOGENETIK BOG‘LIQLIKLAR VA GENETIK PREDISPOZITSIYA 25
Ellamonov Suxrobjon No‘mon o‘g‘li

БОЛАЛАРДА ПСОРИАЗНИНГ КЛИНИК-БИОКИМЁВИЙ, ИММУНОПАТОГЕНЕТИК ХУСУСИЯТЛАРИНИ ҲИСОБГА ОЛГАН ҲОЛДА КОМПЛЕКС ДИАГНОСТИКА ВА ТЕРАПИЯСИНИ ТАКОМИЛЛАШТИРИШ 32
Хаитов Кахрамон Нажмитдинович, Абидов Хасанходжа Алишерович, Махкамова Мухлиса Фархадовна

GLUTEN BILAN BOG‘LIQ SALOMATLIK XAVFLARI VA OZIQ-OVQAT NAZORATINING ZAMONAVIY YONDASHUVLARI: TANQIDIY ADABIYOTLAR SHARHI 48
Abdullaeva Dilafruz G‘ayratovna, Razikov Farrux Ibragim o‘g‘li, Abdullaeva Dilafruz G‘ayratovna

SLEEP AND CIRCADIAN RHYTHMS IN RELATION TO ANDROGEN STATUS IN MIDDLE-AGED MEN: CURRENT EVIDENCE AND CLINICAL IMPLICATIONS 58
Feruza Lyutpillayevna Azizova, Abdukhalim Kholiddin ugli Abdurakhimov

BOLALARDAGI NEYROGEN DEFORMATSIYALARNING UMUMIY KLINIK TAVSIFI VA ULARNI KELTIRIB CHIQARUVCHI OMILLAR TAHLILI 66
Xalimov Ravshan Jurabayevich

KESARCHA KESISHDAN KEYINGI UZOQ MUDDATLI ASORATLAR VA REABILITATSIYA STRATEGIYALARI (ADABIYOTLAR SHARHI) 70
Atayeva Farzona Nuriddinovna

KATTALARDA MODIFIKATSIYALANGAN INTRAMEDULLYAR VINTLAR YORDAMIDA KAFT SUYAKLARI SINIQLARINI KAM INVAZIV OSTEOSINTEZ QILISH 75
Xudoyorov Farhod Rustamovich

УСОВЕРШЕНСТВОВАННАЯ МЕТОДИКА ДИНАМИЧЕСКОЙ АНГИОНЕФРОСЦИНТИГРАФИИ ДЛЯ РАСШИРЕННОЙ КОЛИЧЕСТВЕННОЙ ОЦЕНКИ ФУНКЦИИ ПОЧЕК 85
Хикматов Акром Азимджанович

SLEEP AND CIRCADIAN RHYTHMS IN RELATION TO ANDROGEN STATUS IN MIDDLE-AGED MEN: CURRENT EVIDENCE AND CLINICAL IMPLICATIONS

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Abstract: Testosterone secretion in men exhibits pronounced temporal variation and is closely associated with the sleep–wake cycle. Short sleep duration, sleep fragmentation, obstructive sleep apnea, night-shift work, and circadian misalignment are common in modern populations and may interact with age, obesity, and metabolic dysfunction in shaping androgen status. To summarize current evidence on the associations of sleep duration, sleep architecture, circadian rhythms, shift work, and sleep-disordered breathing with circulating testosterone concentrations in men, with particular attention to middle age. A narrative review of relevant clinical, experimental, and epidemiological studies, systematic reviews, and meta-analyses was conducted. PubMed/MEDLINE-indexed publications were prioritized. Search concepts included combinations of the terms *testosterone*, *androgen status*, *sleep duration*, *sleep deprivation*, *sleep quality*, *slow-wave sleep*, *circadian rhythm*, *shift work*, *obstructive sleep apnea*, and *men*. Human studies with laboratory assessment of testosterone were given priority. Testosterone concentrations increase after sleep onset and generally reach higher levels during nocturnal sleep and in the early morning. Evidence regarding partial sleep restriction is heterogeneous. A meta-analysis of 18 studies involving 252 men found no statistically significant reduction in testosterone after short-term partial sleep deprivation, whereas total sleep deprivation of 24 hours or longer significantly reduced testosterone concentrations. Selective suppression of slow-wave sleep has also been associated with lower morning testosterone levels. Obstructive sleep apnea is consistently associated with lower testosterone levels in meta-analyses, especially in severe disease, although obesity and metabolic dysfunction contribute substantially to this relationship. Evidence regarding night-shift work remains inconsistent. Experimental data also suggest that molecular clock components, including CLOCK, BMAL1, PER, and CRY, may participate in the regulation of Leydig-cell steroidogenesis.

Sleep and circadian organization are biologically relevant to androgen homeostasis in men. However, current evidence does not support a simple causal model in which every reduction in sleep duration inevitably causes testosterone deficiency. Sleep architecture, obstructive sleep apnea, obesity, metabolic status, work schedule, and the timing of testosterone measurement should be considered jointly when evaluating androgen status in middle-aged men.

Key words: testosterone, androgen status, sleep, circadian rhythm, sleep deprivation, slow-wave sleep, obstructive sleep apnea, shift work, middle-aged men.

Annotatsiya: Erkaklarda testosteron sekretsiyasi yaqqol sutkalik o‘zgaruvchanlikka ega bo‘lib, uyqu–uyg‘oqlik sikli bilan chambarchas bog‘liq. Uyqu davomiyligining qisqarishi, uyquning fragmentatsiyasi, obstruktiv uyqu apnoesi, tungi smenali mehnat va sirkad desinxronizatsiya zamonaviy populyatsiyalarda keng tarqalgan bo‘lib, ular yosh, semizlik va metabolik buzilishlar bilan o‘zaro ta’sirlashib, androgen statusiga ta’sir ko‘rsatishi mumkin. Erkaklarda uyqu davomiyligi va

arxitekturasi, sirkad ritmlar, smenali mehnat hamda uyqu vaqtida nafas olish buzilishlarining testosteron konsentratsiyasi bilan bog‘liqligi haqidagi zamonaviy ilmiy dalillarni, ayniqsa o‘rta yoshdagi erkaklarga e’tibor qaratgan holda umumlashtirish. Klinik, eksperimental va epidemiologik tadqiqotlar, tizimli sharhlar hamda meta-tahlillar asosida narrativ adabiyotlar sharhi amalga oshirildi. PubMed/MEDLINE bazasida indekslangan nashrlarga ustuvorlik berildi. Qidiruvda *testosterone*, *androgen status*, *sleep duration*, *sleep deprivation*, *sleep quality*, *slow-wave sleep*, *circadian rhythm*, *shift work*, *obstructive sleep apnea* va *men* terminlarining kombinatsiyalaridan foydalanildi. Testosteron darajasi laboratoriya usulida aniqlangan inson tadqiqotlariga asosiy e’tibor qaratildi. Testosteron konsentratsiyasi uyqu boshlangandan keyin oshadi va odatda tungi uyqu vaqtida hamda erta tonggi soatlarda yuqoriroq darajaga yetadi. Qisman uyqu cheklanishining ta’siri bo‘yicha ma’lumotlar bir xil emas. 252 nafar erkakni qamrab olgan 18 ta tadqiqotning meta-tahlilida qisqa muddatli qisman uyqu deprivatsiyasidan keyin testosteron darajasining statistik jihatdan ahamiyatli pasayishi aniqlanmagan, biroq 24 soat va undan ortiq davom etgan to‘liq uyqu deprivatsiyasi testosteron konsentratsiyasining statistik jihatdan ahamiyatli pasayishi bilan kechgan. Sekin to‘lqinli uyquning selektiv ravishda bostirilishi ham ertalabki testosteron darajasining pasayishi bilan bog‘liq bo‘lgan. Obstruktiv uyqu apnoesi meta-tahlillarda, ayniqsa kasallikning og‘ir shakllarida, testosteronning past darajalari bilan izchil bog‘langan, biroq bu o‘zaro bog‘liqlikka semizlik va metabolik buzilishlar sezilarli hissa qo‘shadi. Tungi smenali mehnat bo‘yicha dalillar esa qarama-qarshi bo‘lib qolmoqda. Eksperimental ma’lumotlar CLOCK, BMAL1, PER va CRY kabi molekulyar sirkad soat komponentlari Leydig hujayralarida steroidogenezni boshqarishda ishtirok etishi mumkinligini ham ko‘rsatadi.

Uyqu va sirkad tashkil topish erkaklarda androgen gomeostazini boshqarishning biologik jihatdan muhim komponentlari hisoblanadi. Biroq mavjud dalillar uyqu davomiyligining har qanday qisqarishi muqarrar ravishda testosteron yetishmovchiligiga olib keladi degan soddalashtirilgan sababiy modelni qo‘llab-quvvatlamaydi. O‘rta yoshdagi erkaklarda androgen statusini baholashda uyqu arxitekturasi, obstruktiv uyqu apnoesi, semizlik, metabolik holat, ish rejimi va testosteron darajasini aniqlash uchun qon olish vaqtini kompleks ravishda hisobga olish zarur.

Kalit so‘zlar: testosteron, androgen statusi, uyqu, sirkad ritm, uyqu deprivatsiyasi, sekin to‘lqinli uyqu, obstruktiv uyqu apnoesi, smenali mehnat, o‘rta yoshdagi erkaklar.

Аннотация: Секреция тестостерона у мужчин характеризуется выраженной суточной вариабельностью и тесно связана с циклом «сон–бодрствование». Сокращение продолжительности сна, его фрагментация, обструктивное апноэ сна, ночная сменная работа и циркадная десинхронизация широко распространены в современных популяциях и могут взаимодействовать с возрастом, ожирением и метаболическими нарушениями, влияя на андрогенный статус. Обобщить современные научные данные о взаимосвязи продолжительности и архитектуры сна, циркадных ритмов, сменной работы и нарушений дыхания во сне с концентрацией тестостерона у мужчин, с особым акцентом на мужчин среднего возраста. Проведён нарративный обзор клинических, экспериментальных и эпидемиологических исследований, систематических обзоров и метаанализов. Приоритет отдавался публикациям, индексированным в PubMed/MEDLINE. При поиске использовались сочетания терминов *testosterone*, *androgen status*, *sleep duration*, *sleep deprivation*, *sleep quality*, *slow-wave sleep*, *circadian rhythm*, *shift work*, *obstructive sleep apnea* и *men*. Основное внимание уделялось исследованиям на людях с лабораторным определением уровня тестостерона. Концентрация тестостерона повышается после начала сна и обычно достигает более высоких значений во время ночного сна и в ранние утренние часы. Данные о влиянии частичного ограничения сна неоднородны. Метаанализ 18 исследований с участием 252 мужчин не выявил статистически значимого снижения уровня тестостерона при кратковременной частичной депривации сна, тогда как полная депривация сна продолжительностью 24 часа и более приводила к статистически значимому снижению концентрации тестостерона. Селективное подавление медленноволнового сна также ассоциировалось со снижением утреннего уровня тестостерона. Обструктивное апноэ сна в метаанализах устойчиво связано с более низкими уровнями тестостерона, особенно при тяжёлом течении заболевания, хотя ожирение и метаболические нарушения вносят существенный вклад в эту взаимосвязь. Данные относительно ночной сменной работы остаются противоречивыми. Экспериментальные исследования также указывают на возможное участие компонентов молекулярных циркадных часов, включая CLOCK, BMAL1, PER и CRY, в регуляции стероидогенеза в клетках Лейдига.

Сон и циркадная организация являются биологически значимыми компонентами регуляции андрогенного гомеостаза у мужчин. Вместе с тем имеющиеся данные не подтверждают упрощённую причинно-следственную модель, согласно которой любое сокращение продолжительности сна неизбежно приводит к дефициту тестостерона. При оценке андрогенного статуса у мужчин среднего возраста следует комплексно учитывать архитектуру сна, наличие обструктивного апноэ сна, ожирение, метаболический статус, режим работы и время забора крови для определения уровня тестостерона.

Ключевые слова: тестостерон, андрогенный статус, сон, циркадные ритмы, депривация сна, медленноволновой сон, обструктивное апноэ сна, сменная работа, мужчины среднего возраста.

INTRODUCTION

Testosterone is the principal circulating androgen in men and contributes to reproductive and sexual function, the maintenance of skeletal muscle and bone mass, erythropoiesis, body composition, and several metabolic processes [1]. Circulating testosterone concentrations are not constant throughout the day. In healthy men, they generally reach higher concentrations during nocturnal sleep and the early morning and decline toward the afternoon and evening [2–4]. This temporal pattern has direct clinical implications. Current endocrine guidelines recommend measurement of fasting morning total testosterone when evaluating suspected male hypogonadism, with confirmation of low concentrations on a separate occasion [1]. However, clock time alone does not fully explain testosterone variability. Experimental physiology suggests that the nocturnal increase in testosterone is strongly related to sleep itself, its timing, and its architecture [2–5].

This distinction is particularly relevant to middle-aged men. Aging is associated with changes in both androgen physiology and sleep architecture, while obesity, metabolic disorders, psychological stress, physical inactivity, obstructive sleep apnea (OSA), and occupational shift work become increasingly common potential confounders [6–9]. Several of these factors may affect sleep and testosterone simultaneously. The public health relevance of the issue has increased as short and irregular sleep schedules have become common features of modern lifestyles. Nevertheless, the frequently stated assumption that insufficient sleep inevitably lowers testosterone is an oversimplification. Experimental studies have yielded heterogeneous findings, while cross-sectional associations cannot establish temporal or causal relationships. The aim of this narrative review was therefore to critically summarize current evidence on the relationships between sleep duration, sleep architecture, circadian regulation, night-shift work, and sleep-disordered breathing and androgen status in men, with particular attention to factors relevant to middle age.

RESEARCH METHODOLOGY

A narrative review methodology was used. Relevant literature was identified primarily through PubMed/MEDLINE using combinations of the following search terms: testosterone, androgen, male hypogonadism, sleep, sleep duration, sleep restriction, sleep deprivation, slow-wave sleep, sleep fragmentation, circadian rhythm, circadian clock genes, shift work, night work, obstructive sleep apnea, and men. Original human studies, controlled experimental studies, population-based investigations, systematic reviews, and meta-analyses examining testosterone concentrations in relation to sleep or circadian characteristics were prioritized. Mechanistic reviews and experimental evidence were included when required to interpret biological pathways. Particular attention was given to study design, sample size, age distribution, timing of blood sampling, and potential confounding by obesity, metabolic disorders, and OSA. Because this was a narrative rather than a systematic review, no quantitative pooling of results was performed.

ANALYSIS AND RESULTS

Sleep-dependent and diurnal variation in testosterone secretion. The daily pattern of testosterone secretion represents an interaction between hypothalamic–pituitary–gonadal regulation, sleep, and circadian organization. Early physiological studies demonstrated that testosterone concentrations begin to increase after sleep onset and remain elevated during the night, with peak concentrations generally occurring during sleep or around awakening [2,3]. Luboshitzky et al. demonstrated that, in healthy young men, testosterone began to rise with sleep onset and reached high concentrations around the first rapid-eye-movement period [2]. Subsequent studies showed that fragmentation of normal sleep could disrupt this nocturnal testosterone rhythm [3]. Accordingly, the apparent “morning peak” in testosterone should not be considered exclusively a clock-driven phenomenon. Sleep timing and continuity contribute substantially to the temporal profile of testosterone secretion [4,5]. The amplitude of diurnal testosterone variation also changes with age. These effects are clinically relevant because a blood sample obtained at an inappropriate time may result in substantially different testosterone concentrations in the same individual.

Sleep duration and partial sleep restriction. One of the most frequently cited experimental studies was conducted by Leproult and Van Cauter [10]. Ten healthy young men underwent one week of sleep restriction to approximately five hours per night. Daytime testosterone concentrations were approximately 10–15% lower after sleep restriction than under the rested condition. Although influential, this study involved only ten young men and therefore should not be interpreted as definitive evidence that habitual short sleep universally causes testosterone deficiency. Subsequent randomized studies produced less consistent results. Smith et al. studied acute and repeated sleep restriction under both inpatient and outpatient conditions and did not demonstrate a consistent, significant reduction in circulating testosterone [11].

More informative evidence comes from a 2021 systematic review and meta-analysis by Su et al. [12]. Eighteen studies involving 252 men were included. Short-term partial sleep deprivation did not significantly reduce serum testosterone:

SMD −0.22; 95% CI −0.50 to 0.06; p=0.13.

In contrast, total sleep deprivation produced a significant reduction:

SMD −0.64; 95% CI −0.87 to −0.42; p<0.001.

The reduction was evident after 24 hours of total sleep deprivation and also following 40–48 hours of deprivation [12]. These findings demonstrate an important dose- and context-dependent relationship. Acute complete loss of sleep appears capable of suppressing testosterone, whereas the endocrine consequences of more typical partial sleep restriction remain less certain.

Sleep architecture: quantity is not the whole story. Total sleep duration may not adequately describe the biological interaction between sleep and testosterone. Sleep architecture, particularly slow-wave sleep (SWS), may also be relevant. In a randomized experimental study, Ukraintseva et al. selectively suppressed SWS in healthy men while largely preserving total sleep duration and sleep efficiency [13]. SWS was reduced by approximately 54%, and morning testosterone concentrations were significantly lower following SWS suppression than during the control condition. This experiment suggests that the endocrine consequences of disturbed sleep may depend on sleep quality and architecture rather than merely on the total number of hours spent asleep.

Observational studies support this possibility. In older men, objective sleep characteristics have been associated with morning testosterone concentrations [14]. Barrett-Connor et al. also found that men with lower testosterone demonstrated lower sleep efficiency, more nocturnal awakenings, less SWS, and more sleep-disordered breathing [15]. Importantly, several of these relationships were attenuated after adjustment for body mass index or waist circumference [15]. This observation highlights obesity as an important shared determinant of disturbed sleep and lower testosterone.

Population-based evidence. Large observational datasets permit evaluation of sleep and testosterone under real-world conditions but introduce greater potential for residual confounding. Patel et al. analyzed NHANES 2011–2012 data from 2,295 men with available testosterone measurements [16]. The median age was 46 years, and the median testosterone concentration was 377 ng/dL. After multivariable adjustment, serum testosterone was lower with increasing age, increasing BMI, and decreasing sleep duration. Each hour of sleep loss was associated with approximately 5.85 ng/dL lower testosterone, whereas each one-unit increase in BMI was associated with approximately 6.18 ng/dL lower testosterone [16]. These results are highly relevant to middle-aged populations because they demonstrate that sleep and adiposity may contribute simultaneously to variation in testosterone. However, the cross-sectional design prevents determination of directionality. Short sleep could contribute to metabolic abnormalities and altered testosterone; low testosterone could influence sleep-related physiology; or common factors such as obesity could affect both. Recent population studies similarly indicate complex, potentially nonlinear relationships between sleep characteristics and circulating sex hormones [17]. Consequently, sleep duration should not be interpreted in isolation.

Obstructive sleep apnea. OSA provides one of the strongest clinical links between disturbed sleep and androgen status. The biological plausibility is considerable. Recurrent upper-airway obstruction produces intermittent hypoxemia, sympathetic activation, and repeated arousals, resulting in fragmentation of normal sleep architecture. OSA is also strongly associated with obesity and insulin resistance, which themselves are related to lower testosterone levels. A systematic review and meta-analysis by Su et al. found significantly lower serum testosterone concentrations in men with OSA compared with controls [18]. Importantly, the association persisted in analyses accounting for age and BMI and appeared stronger with increasing OSA severity. A subsequent systematic review and meta-analysis by Wang et al. similarly concluded that OSA is significantly associated with reduced total testosterone levels in men [19]. Nevertheless, the relationship is not straightforward. OSA and functional hypogonadism frequently share obesity as an upstream determinant, making complete separation of their independent effects difficult.

Furthermore, evidence that treatment of OSA reliably restores testosterone levels is weak. A systematic review and meta-analysis evaluating continuous positive airway pressure (CPAP) found no consistent substantial increase in testosterone following CPAP treatment [20]. This distinction is clinically important. An association between OSA and low testosterone does not necessarily demonstrate that sleep apnea is the sole cause of hypogonadism.

Testosterone treatment and sleep-disordered breathing. The relationship between testosterone and sleep is potentially bidirectional. While disturbed sleep may be associated with lower testosterone levels, exogenous testosterone can influence ventilatory physiology. Experimental administration of high-dose testosterone to older men has been reported to transiently worsen sleep-disordered breathing [21]. Therefore, symptoms of OSA should be considered in men being assessed for testosterone treatment, particularly in

individuals with obesity or other risk factors. The interaction should not be overstated, however. Available evidence differs according to testosterone formulation, dose, treatment duration, and baseline OSA status.

Molecular circadian regulation of testosterone synthesis. Recent research has expanded from physiological circadian rhythms to molecular clock mechanisms. The core molecular circadian machinery consists of transcriptional feedback loops involving CLOCK, BMAL1, PER, and CRY genes. These systems regulate rhythmic gene expression in numerous peripheral tissues in addition to the central circadian pacemaker. Recent evidence reviewed by Ning et al. indicates that circadian-clock components may participate in the regulation of Leydig-cell steroidogenesis and testosterone production [22]. Experimental alterations in clock-gene activity can influence steroidogenic pathways, including the expression of enzymes and regulatory proteins necessary for androgen synthesis. Potential interactions have been described between circadian signaling and steroidogenic acute regulatory protein (StAR), cholesterol transport, and steroidogenic enzyme expression [22]. Nevertheless, an important limitation is that a substantial proportion of molecular evidence originates from cellular and animal models. The clinical importance of individual clock-gene alterations for testosterone deficiency in middle-aged men remains insufficiently established. Thus, molecular circadian biology provides a plausible mechanistic framework rather than definitive clinical evidence.

Shift work and circadian misalignment. Night-shift work represents a natural model of chronic circadian disruption. Shift workers may experience misalignment between endogenous circadian timing and behavioral cycles of sleep, activity, food intake, and light exposure. Human investigations of testosterone in shift workers have produced heterogeneous results. Differences between studies may reflect sampling time, duration and type of shift work, chronotype, sleep duration, age, occupational stress, and metabolic characteristics. Kelly et al. demonstrated that shift work can modify temporal patterns of steroidogenesis and emphasized that interpretation depends strongly on biological rather than conventional clock time [23]. Bracci et al. subsequently reported lower pregnenolone and total testosterone concentrations among male shift workers compared with daytime workers when sampling was standardized at the beginning of the morning shift [24].

A 2025 systematic review and meta-analysis by Viramgami et al. evaluated night-shift work and male reproductive outcomes [25]. Overall, the evidence suggested adverse associations with aspects of male reproductive health, although evidence specific to testosterone was less consistent than that for some reproductive outcomes. Consequently, night-shift work should currently be considered a plausible modifier of androgen physiology rather than an established independent cause of testosterone deficiency.

Table 1. Key human evidence linking sleep-related factors with testosterone in men

Study	Design/population	Exposure	Main finding
Luboshitzky et al., 1999 [2]	Physiological study, healthy men	Normal sleep architecture	Testosterone increased after sleep onset and reached high levels during nocturnal sleep
Penev, 2007 [14]	Older men, objective sleep assessment	Sleep quantity/quality	Better sleep characteristics associated with higher morning testosterone
Barrett-Connor et al., 2008 [15]	Community-dwelling older men	Sleep efficiency/SWS/ OSA	Lower testosterone associated with poorer sleep; associations attenuated by adiposity
Leproult & Van Cauter, 2011 [10]	Experimental, n=10	~5 h/night for one week	Daytime testosterone approximately 10–15% lower
Ukraintseva et al., 2018 [13]	Randomized experimental study	Selective SWS suppression	Reduced SWS associated with lower morning testosterone
Patel et al., 2019 [16]	NHANES, n=2,295 men	Self-reported sleep duration	Each hour less sleep associated with ~5.85 ng/dL lower testosterone
Smith et al., 2019 [11]	Randomized controlled studies	Acute/repeated partial restriction	No consistent significant testosterone reduction
Su et al., 2021 [12]	Meta-analysis, 18 studies, 252 men	Partial/total deprivation	Partial deprivation NS; total deprivation significantly reduced testosterone
Su et al., 2022 [18]	Systematic review/ meta-analysis	OSA	OSA associated with lower testosterone, especially severe OSA

Study	Design/population	Exposure	Main finding
Bracci et al., 2023 [24]	Occupational study	Shift vs daytime work	Lower pregnenolone and testosterone reported in shift workers
Wang et al., 2023 [19]	Systematic review/ meta-analysis	OSA	OSA associated with lower total testosterone
Viramgami et al., 2025 [25]	Systematic review/ meta-analysis	Night-shift work	Evidence of adverse male reproductive effects; testosterone evidence heterogeneous

The available evidence supports a biologically meaningful relationship between sleep, circadian organization, and male androgen physiology. However, three distinctions are necessary for appropriate interpretation. First, sleep duration, sleep quality, and sleep architecture are not equivalent exposures. Complete sleep deprivation produces a clearer reduction in testosterone than short-term partial sleep restriction [12], while selective suppression of SWS may affect morning androgen concentrations despite relatively preserved total sleep time [13]. Second, association is not equivalent to causation. Population-based studies show that shorter or poorer sleep is associated with lower testosterone, but adiposity represents a particularly important confounder. Obesity can promote OSA and sleep fragmentation while simultaneously lowering total testosterone through metabolic and hypothalamic-pituitary-gonadal mechanisms. Third, testosterone itself may affect sleep physiology, particularly sleep-disordered breathing. The relationship is therefore potentially bidirectional. These issues are especially important in middle-aged men, in whom multiple modifiable factors frequently coexist. Increasing visceral adiposity, reduced physical activity, psychosocial stress, irregular work schedules, metabolic dysfunction, and sleep disturbances may form an interrelated network rather than independent exposures. From a methodological perspective, future studies should avoid interpreting a single testosterone measurement without considering blood-sampling time and preceding sleep-wake behavior. Standardized morning fasting sampling is particularly important when comparing individuals [1].

Studies of shift workers deserve additional attention because “08:00” does not have the same biological meaning for a worker who has just completed a night shift and for a person who has slept conventionally during the night. For preventive medicine, the current evidence supports incorporating sleep assessment into the broader evaluation of men with suspected androgen deficiency. This should include habitual sleep duration, sleep quality, shift-work status, and symptoms suggesting OSA. These variables should be assessed jointly with BMI, waist circumference, physical activity, and metabolic comorbidities rather than interpreted independently.

At the same time, current evidence does not justify promising that sleep extension alone will normalize testosterone in men with established hypogonadism. Intervention trials demonstrating a clinically meaningful, sustained increase in testosterone after correction of habitual short sleep remain limited.

Implications for research in middle-aged men. A particularly important evidence gap concerns population-based studies specifically targeting middle-aged men. Many experimental sleep studies have recruited small samples of young, healthy volunteers, whereas studies of OSA often include older men or clinical populations. Therefore, extrapolation to community-dwelling men in the transitional middle-age period is uncertain.

Future studies should simultaneously quantify: objective and/or standardized measures of sleep; total testosterone measured under standardized morning conditions; adiposity and central obesity; physical activity and functional status; shift-work exposure; psychological stress; OSA risk; metabolic comorbidities.

Multivariable modeling could then determine whether sleep-related characteristics remain independently associated with testosterone after adjustment for major anthropometric, functional, and lifestyle factors. Most population-based evidence is observational and therefore susceptible to reverse causality and residual confounding. Experimental studies are generally small and predominantly involve young, healthy men. Definitions of “short sleep” vary substantially between studies. Sleep is assessed using self-report in some populations, whereas others use actigraphy or polysomnography. The timing of testosterone sampling is also heterogeneous. This is a critical limitation because testosterone concentrations vary across the sleep-wake cycle. OSA, obesity, and metabolic dysfunction frequently coexist, making their individual contributions difficult to distinguish. Finally, evidence regarding molecular circadian-clock regulation in humans remains less developed than experimental evidence from animal and cellular systems.

CONCLUSION

Sleep and circadian organization are important components of male androgen physiology. Testosterone secretion is closely linked to the sleep–wake cycle, and disruption of normal sleep architecture may alter its physiological temporal pattern. The strongest experimental evidence indicates that prolonged total sleep deprivation can reduce circulating testosterone, whereas the effect of short-term partial sleep restriction is inconsistent. Disturbance of slow-wave sleep may also reduce morning testosterone levels. Obstructive sleep apnea (OSA) is consistently associated with lower testosterone levels in systematic reviews and meta-analyses, although obesity and metabolic dysfunction contribute substantially to this relationship. Evidence regarding night-shift work and testosterone remains heterogeneous. Therefore, androgen status in middle-aged men should be evaluated within a multidimensional framework incorporating sleep, circadian behavior, adiposity, metabolic health, and lifestyle characteristics.

Prospective population-based studies are required to determine whether sleep and circadian characteristics independently predict testosterone decline and whether correction of these factors can prevent or reverse clinically relevant androgen deficiency.

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