International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
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ARTICLE TITLE	TESTOSTERONE AND PHYSICAL ACTIVITY: A REVIEW OF HORMONAL VARIABILITY IN SEDENTARY AND ACTIVE MEN
DOI	https://doi.org/10.31435/ijitss.3(47).2025.3645
RECEIVED	18 June 2025
ACCEPTED	09 September 2025
PUBLISHED	27 September 2025
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TESTOSTERONE AND PHYSICAL ACTIVITY: A REVIEW OF HORMONAL VARIABILITY IN SEDENTARY AND ACTIVE MEN

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ABSTRACT

Introduction and Purpose: Testosterone (T) is the main androgenic-anabolic hormone in men, playing a vital role in reproductive function, muscle and bone maintenance, mood regulation, and metabolic health. Its levels are dynamic and modulated by both intrinsic and extrinsic factors, notably physical activity. While sedentary behaviour is associated with age-related hormonal decline, various forms of exercise may induce either beneficial or adverse endocrine adaptations. The purpose of this review is to analyze how different types of physical activity—and inactivity—affect T levels in adult men, and to explore the physiological and behavioural modulators of these responses.
Materials and Methods: A thorough review of the literature available on PubMed and Google Scholar databases was conducted using the following keywords: "testosterone", "testosterone levels", "physical activity", "sedentary", "resistance training", "endurance training", "testosterone in athletes", "overtraining syndrome", and "exercise-hypogonadal male condition". Peer-reviewed studies published between 1987 and 2025 were included, with a focus on both clinical and physiological investigations examining acute and chronic T responses to various forms of physical activity in adult males. Special attention was given to studies addressing modulating factors such as age, energy availability, circadian rhythms, stress, and sleep quality.
Summary and Conclusions: Resistance training tends to acutely elevate and chronically sustain T concentrations. In contrast, prolonged endurance training may lead to suppressed baseline testosterone – EHMC. Factors like low energy availability, stress, and aging further influence hormonal patterns. Recognizing these variations is essential for clinicians, coaches, and researchers to properly interpret T data and avoid unnecessary medical interventions.

KEYWORDS

Testosterone, Testosterone Level, Physical Activity, Overtraining Syndrome, Exercise-Hypogonadal Male Condition, Circadian Rhythms

CITATION

Joanna Katarzyna Pergoł, Julia Sposób, Adam Borsuk, Martyna Narożniak, Zuzanna Krupa, Bartłomiej Czerwiec, Adrianna Ewa Pękacka, Julia Borkowska, Malwina Wojtas, Julia Skowrońska-Borsuk. (2025) Testosterone and Physical Activity: A Review of Hormonal Variability in Sedentary and Active Men. *International Journal of Innovative Technologies in Social Science*. 3(47). doi: 10.31435/ijitss.3(47).2025.3645

Introduction

Testosterone (T) is the main androgen-anabolic hormone in men, playing a critical role in reproductive function, the modulation of muscle mass, the maintenance of bone density, mood, and metabolic health. Circulating T levels are known to fluctuate throughout a man's life and are modulated by many internal and external factors, such as age, body composition, energy availability, stress, sleep, and especially physical activity. In common parlance, it is considered a typically"male" hormone, and for physically active individuals –particularly power sport athletes – it is often a symbol of good training, libido, and masculinity more generally. Unfortunately, as a result of a lack of knowledge concerning the physiology and regulation of the hypothalamic-pituitary-gonadal axis, determinations of T levels evaluations are commonly misused and misinterpreted, both by the general population lacking medical training and by specialists. This leads to false diagnoses, unnecessary anxiety and, in some cases, premature initiation of hormonal therapies with potentially harmful effects. On the other hand, T has remained an object of interest in medical and sports science for many years, including in the diagnosis of overtraining syndrome, monitoring the adaptation to training programs, or designing nutritional and recovery strategies. The relationship between physical activity and T levels is complex and is dependent on the type, intensity, and duration of exercise, as well as the subject's training status. Differences in hormonal responses to resistance exercise, sprinting, endurance exercise, or mixed modalities make linear comparisons impossible.

The growing emphasis on optimizing male hormonal health in diverse populations - from elite athletes to those with sedentary lifestyles – makes the understanding of influences on T levels both clinically and physiologically relevant. The purpose of this review paper is to provide an overview of the current

understanding of the effects of various forms of physical activity, as well as inactivity, on T levels in adult men. Additionally, this paper will discuss, mediating factors such as age, diurnal rhythms, energy availability, and psychological stress will also be discussed, as well as the potential clinical implications of hypo- and hyperandrogenic states in both athletic and non-athletic settings.

Testosterone physiology

Testosterone, while present in small quantities in adrenal secretions, is primarily synthesized in the Leydig cells of the testes and is the most biologically potent of the endogenous androgens. In contrast, the adrenal cortex mainly produces weaker androgens such as dehydroepiandrosterone and androstenedione. Adrenocorticotropic hormone (ACTH) stimulates androgen secretion from the adrenal glands. T functions as the principal male sex hormone, and it is produced predominantly in the Leydig cells of the testes under the regulation of the hypothalamic-pituitary-gonadal (HPG) axis. Its secretion follows a circadian rhythm with a peak concentration in the morning and reduced concentrations in the evening and overnight (1, 2), regulated by higher brain centres, which modulate hypothalamic activity and promote the release of gonadotropin-releasing hormone (GnRH). GnRH, in turn, stimulates the anterior pituitary to secrete luteinizing hormone (LH), which acts on the Leydig cells of the testes to induce T synthesis that is repressed by a feedback inhibition (2, 3).

T, as well as androstenedione, can be converted to estradiol and estrone by the enzyme aromatase, which is found in various tissues, including the brain, liver, adipose tissue, and skin. A key factor in the development of hypogonadism in obese men is the activity of aromatase, an enzyme expressed in adipose tissue, which facilitates the conversion of free T into 17β-estradiol (2). Increased aromatase expression associated with excess adiposity results in a reduction of bioavailable T and enhances negative feedback on the HPG axis. This, in turn, suppresses gonadotropin secretion and further diminishes endogenous T production, contributing to the progression of hypogonadism (4).

The conversion reaction of cholesterol to pregnenolone, catalyzed by 20, 22-desmolase, is the main step in the biosynthesis of androgens, including T, in the adrenal glands. Androstenedione is a common final precursor in the synthesis of T. In tissues such as the prostate gland, seminal vesicles, epididymides, liver, skin, and others, there is an irreversible conversion of T to dihydrotestosterone due to the high activity of 5α-reductase, which catalyzes this reaction. Dihydrotestosterone exhibits greater androgenic potency and is considered the most potent endogenous androgen. It is further converted to 17-ketosteroids, which are excreted in the urine, the measurement of which allows the determination mainly of the androgenic activity of the adrenal cortex, and the testes only to a small extent. Androgens, also known as anabolic hormones, are responsible for numerous biological activities in the human body, particularly in males. Actions specifically related to reproduction are the regulation of spermatogenesis, the maintenance of secondary male sexual characteristics and the function of accessory genital organs, where the effect of androgens in males is to stimulate the development of organs in the urogenital sinus area, lower the sound of the voice, the formation of typical hair pattern with also the disappearance of hair on the head. In addition, T is directly responsible for libido levels and is integral in inhibiting the HPG hormonal axis. In addition, they stimulate cell proliferation, tissue maturation, and protein biosynthesis, which is a notable anabolic and anticatabolic effect on skeletal muscle (with its direct action by a cytoplasmic receptor) and bone density, as they stimulate body growth in length and increase muscle mass during puberty (2, 3, 5, 6).

The biologically active fraction, and at the same time a good marker of T concentration, is considered to be its free fraction, which accounts for about 0.5-2% of total serum T concentration. The remainder is bound to sex hormones (SHBG), in this form not available for uptake by most tissues, and to albumin, from which it rapidly dissociates, increasing its access to target tissues (7).

T levels can be modulated by a variety of endogenous and exogenous factors, including the light–dark cycle, nonphotic entrainment cues, social interactions, lifestyle habits, occupational schedules, and dietary patterns. In particular, nutritional factors such as adherence to a low-fat diet and the use of dietary supplements, including caffeine, have been shown to influence circulating T concentrations (3). Several physiological factors are known to contribute to reduced serum T concentrations, including advancing age, increased body weight, suboptimal nutritional status, psychological or physical stress, insufficient sleep, and excessive alcohol consumption (8, 9). Furthermore, the age-related decline in T levels has been implicated in the increased incidence and mortality associated with cardiovascular diseases. T deficiency is commonly observed in men with type 2 diabetes mellitus and is associated with impaired insulin sensitivity, elevated body fat percentage, central (truncal) obesity, dyslipidemia, hypertension, end-stage renal disease, human immunodeficiency virus, chronic obstructive pulmonary disease, and some genetic conditions. Other causes include castration, trauma,

radiation or chemotherapy, acute illness, and pituitary tumors (4, 10, 11). Controversy exists regarding whether the decline in T with increasing age is a normal physiologic process or whether it is a result of chronic comorbidities and lifestyle choices (10). T replacement therapy, while gradually gaining recognition for its therapeutic potential (12, 13), remains a partially limited and inconsistent intervention (10). Consequently, physiologically based strategies for restoring normal T levels are strongly recommended, not only due to their ability to enhance endogenous hormonal regulation but also because they may address and reverse underlying contributors to metabolic disease risk factors.

Numerous studies have demonstrated that exercise influences T levels, but the effects depend on several variables, including training type, duration, intensity, and the individual's fitness level (14, 15). Athletes, particularly those engaged in endurance sports, may experience suppressed resting T (16), while sedentary individuals often exhibit age-related T decline (17). The complexity of these hormonal responses has led to the recognition of adaptive and maladaptive patterns of endocrine regulation in different athletic populations (18).

Testosterone in Sedentary vs. Active Men

Sedentary behaviour, particularly in the context of aging, is closely associated with a progressive decline in serum T concentrations (17). This decline has been linked to unfavourable health outcomes, including sarcopenia, reduced sexual function, impaired cognitive performance, and elevated risk of metabolic disorders such as insulin resistance and visceral obesity (9, 11). The underlying mechanisms likely involve both central dysregulation of the HPG axis and peripheral factors such as increased aromatase activity in adipose tissue and diminished testicular responsiveness (14).

Conversely, regular physical activity—particularly resistance exercise and high-intensity interval training (HIIT)—is associated with increased circulating concentrations of both total and free T, especially in individuals who were previously sedentary (15, 17). These hormonal improvements are thought to stem from enhanced Leydig cell steroidogenesis, improved pituitary LH secretion patterns, and reduced fat mass, which collectively mitigate aromatization of T to estradiol (19). A recent meta-analysis further confirmed that acute bouts of physical exercise can significantly elevate T levels, with resistance and high-intensity exercise producing the most robust responses. In the analyzed studies, mean increases ranged from 15% to 25% for total T and up to 30% for free T within 30 minutes post-exercise (20).

Comparative data consistently show that physically active men maintain higher baseline T levels than sedentary individuals, independent of age and body composition (17). This observation was supported by findings from Cinar et al. (21), in which both athletes and sedentary men underwent exhaustive exercise. Athletes exhibited significantly higher resting total T (23.3 ± 1.2 vs. 15.8 ± 0.9 nmol/L) and free T levels (0.39 ± 0.02 vs. 0.27 ± 0.01 nmol/L) compared to sedentary subjects, both at rest and after exercise, suggesting that regular training enhances the hormonal response to physical stress. In this study, participants also received magnesium supplementation (10 mg/kg/day), which was associated with further increases in T levels—highlighting the modulatory role of micronutrient status alongside physical training (21).

In addition, long-term moderate physical activity has been highlighted as a protective factor against age-related T decline. A systematic review by Zouhal et al. (22) in men and women over 40 years of age demonstrated that diverse exercise interventions—including resistance, HIIT, and endurance training—consistently increased basal concentrations of total T (effect sizes d = 0.19–3.37), along with SHBG, IGF-1, and growth hormone. These results support the concept that regular exercise promotes maintenance of eugonadal status and may attenuate metabolic and cardiovascular risks associated with sedentary aging.

Active men also demonstrate more favourable anabolic–catabolic (A-C) hormone ratios and exhibit better hormonal resilience throughout the day. Importantly, differences in exercise modality, intensity, and training status may contribute to variability in T responsiveness, which should be accounted for in interpretation of results (14, 23).

In conclusion, structured and consistent physical activity supports the preservation of endogenous T production and may serve as an effective, non-pharmacological intervention for maintaining endocrine and metabolic health in men across different age groups.

Acute vs. Chronic Exercise Effects on Testosterone

Acute bouts of physical exercise — whether resistance or endurance-based — typically induce a transient elevation in circulating T levels. The magnitude and duration of this response are influenced by exercise intensity, muscle mass involved, rest intervals, and circadian factors (3, 24, 25). Hormonal elevations generally occur within minutes post-exercise, peaking around 15–30 minutes and returning to baseline within

approximately 60 minutes (22, 24). This pattern is consistent for both total and free T, and is influenced by several factors including time of day, nutritional status, training history, and rest intervals (24).

In contrast, chronic adaptations to training exert more complex and heterogeneous effects. Regular resistance training has been shown to maintain or modestly increase basal T concentrations in men of various age groups, especially when paired with adequate recovery and progressive overload (3, 26). These adaptations are partially attributed to improved HPG axis sensitivity and reductions in adipose tissue, which decreases peripheral aromatization of T to estradiol.

However, endurance training presents a distinct endocrine profile. Multiple studies have documented a consistent reduction in resting T levels among endurance-trained males, often accompanied by disruptions in LH pulsatility and hypothalamic signalling—hallmarks of the EHMC (16, 27). These findings suggest a downregulation of the HPG axis as a physiological adaptation to chronic endurance stress. For example, males preparing for endurance events such as marathons exhibit significantly lower basal T concentrations after prolonged training, despite increased physical fitness (28, 29).

Overall, acute and chronic exercise affect T regulation in divergent but complementary ways — where acute bouts temporarily boost circulating hormone levels, chronic resistance training supports basal endocrine function, while long-term endurance training may suppress T as part of an adaptive energy-conserving strategy.

Sport-Specific Testosterone Profiles

Different types of sports impose unique endocrine demands, which can modulate T levels differently:

- **Endurance Sports (e.g., marathon running, cycling):** Often associated with chronically reduced resting T. Studies by Hackney and colleagues (1989, 1996, 2001, 2005) (16, 27, 30, 31) have consistently demonstrated that endurance-trained men may exhibit suppressed gonadal function due to sustained energy deficiency, reduced LH pulsatility, and hypothalamic downregulation (16, 27). During the regeneration phase, decreased levels at rest were observed for hours to days, for example after triathlon competitions, but also after repeated anaerobic exercise (32). Maïmoun et al. (29) observed significantly lower T levels in male endurance athletes training for marathons, despite improved aerobic fitness. Similarly, Lucia et al. (33) documented hormonal disturbances in older professional cyclists during the Tour de France, with T suppression attributed to prolonged endurance load. The EROS-Profile study (34) further confirmed that post-triathlon or repeated anaerobic effort, T levels can remain reduced for up to 72 hours.
- **Resistance Sports (e.g., weightlifting, bodybuilding):** Resistance exercise leads to robust acute surges in T, particularly in response to high-load, multi-joint movements with short rest intervals. Kraemer et al. (24, 35, 36) and Häkkinen et al. (37) reported elevated T immediately post-exercise and in some cases chronically with appropriate training protocols. However, baseline levels may remain stable, and chronic adaptations depend on training load and recovery. A-C balance, as measured by the T/C ratio, does not always increase proportionally with strength gains. In untrained individuals, no significant changes in T have been observed despite improvements in muscular performance (38, 39).
- **Mixed/Intermittent Sports (e.g., soccer, basketball, rugby):** Hormonal responses in these disciplines are heterogeneous. Competition-induced stress, training load, and match frequency influence T and cortisol dynamics (40, 41). For instance, it was noted that soccer players experience lower in-season T and elevated cortisol, likely due to accumulated fatigue and dense competition schedules (41). Similar findings have been reported in basketball athletes during high-intensity competitive periods (42).
- **Combat Sports (e.g., boxing, MMA):** Combat sports induce significant acute hormonal responses. Slimani et al. (43) conducted a systematic review and meta-analysis revealing that T rises sharply during competition or simulated combat stress. However, chronic effects are highly dependent on training load, weight management practices, and frequency of matches. In MMA and boxing, hormonal fluctuations are particularly marked before and after weigh-ins and fights.

Overtraining syndrome

Overtraining syndrome (OTS), considered as a"vegetative dysregulation", is generally characterized by a long-term (weeks to years) decline in sport-specific performance despite the maintenance or reduction of training intensity, in opposition to other short-term overtraining states: functional overreaching and nonfunctional overreaching (32, 44-47). This condition is also associated with increased fatigability and a range of autonomic and psychological symptoms, including altered mental excitability, sleep disturbances, emotional instability, and somatic complaints that are not attributable to identifiable organic pathology (32). A definitive diagnosis of OTS requires the exclusion of underlying organic or medical conditions that could

account for the observed symptoms (45). Assessment of overtraining necessitates a highly individualized approach, as the physiological response to training stress varies significantly among individuals. Exercise testing at high intensities of the individual's anaerobic threshold has been proposed as a valuable method for evaluating exercise-induced alterations in hormonal concentrations associated with overtraining (32). OTS has been associated with reduced T levels, a finding previously reported in the literature. This hormonal suppression may contribute to impaired muscle recovery, decreased muscle mass, a lowered basal metabolic rate, and diminished fat oxidation capacity (34, 48). Monitoring changes in T and cortisol concentrations, and in particular their reciprocal ratio (e.g., free testosterone/cortisol (T/C) >30%), which varies with the intensity and duration of exercise, has been proposed as a marker of A-C state that may be useful in the diagnosis of OTS. However, numerous studies have reported inconsistent findings, largely depending on the type of physical activity examined (45) and may be partly explained by methodological variability across studies. For instance, in research involving Finnish weightlifters, a positive correlation between the T/C ratio and strength performance was observed (37). In contrast, similar associations were not consistently observed in studies focusing on endurance athletes often failed to confirm such associations (47). These discrepancies may stem from differences in the type of exercise protocols used (e.g., resistance vs. aerobic), timing of hormone sampling (e.g., morning vs. post-exercise), duration of training cycles, baseline fitness levels of participants, and even the methods of hormone analysis (e.g., total vs. free T, serum vs. salivary measurements). Moreover, the dynamic nature of the T/C ratio, influenced by acute versus chronic stress responses, may render it a context-dependent marker that is more suitable for monitoring short-term training load rather than serving as a universal indicator of performance or overtraining risk. Novel studies polemize with that hypothesis by applying more accurate methodology and exploring the potential of other hormones in comparison to T. In the EROS BASAL study by Cadegiani et al. (44), the T/C ratio did not differ between overtrained and healthy athlete subjects, which strongly undermined its usefulness in diagnosing OTC. Moreover, taking into account rather anabolic effect of chronic hypercortisolism and less probable simultaneous cortisone and T regulation (due to different hypothalamus-pituitary axes), the T/C ratio was considered inappropriate for evaluating the chronic A-C state. The same study demonstrated that OTS specifically reduces the testosterone-to-estradiol (T/E2) ratio, without significantly altering total T or other individual hormone levels. This finding suggests that the T/E2 ratio may serve as a more sensitive marker of the A–C balance, as the conversion of T to estradiol via aromatase involves a single enzymatic step, allowing for a rapid and direct hormonal response to physiological demands (44, 46). Furthermore, OTS can be independently triggered by eating patterns, regardless of training patterns, which may better explain underperformance, which is the key characteristic of OTS, and has to be taken into account in future analysis (46).

Exercise-Hypogonadal Male Condition

The EHMC refers to a state observed in some endurance-trained men, which is described by chronically lowered resting T levels despite normal or even enhanced physical fitness. T plays a central role, with its chronic decrease being a reflection of a broader endocrine adaptation to chronic physical stress. First fully systematically described by Hackney et al. (16), chronic endurance training can lead to a functional downregulation of the HPG axis, with the inhibition of T being the key endocrine feature of EHMC. This condition is also described by alterations in LH pulsatility, impaired testicular steroidogenesis, and a reduction in circulating bioavailable T.

The T decline in EHMC is not due to testicular or pituitary pathology but is a centrally mediated suppression, likely triggered by a hypothalamic response to prolonged energy imbalance and physical overload (18, 49). Low T in this context may represent a physiological attempt to conserve energy by downregulating reproductive function—a theory supported by the overlap of EHMC and syndromes such as Relative Energy Deficiency in Sport (50).

Several mechanisms are involved in the inhibition of T in athletes with EHMC. Elevated cortisol levels, which occur due to chronic stimulation of the HPA axis, have been shown to have inhibitory action on the release of GnRH and consequently LH release. Simultaneously, reduced concentrations of leptin and elevated prolactin levels further attenuate hypothalamic signalling. All together, these endocrine changes cumulatively result in reduced testicular T production (18, 51). In addition, increased central opioid activity and systemic inflammatory cytokines, which occur due to prolonged endurance training, can act as inhibitory signals at the hypothalamic level. Clinically, decreased T in EHMC may be expressed as fatigue, decreased libido, mood changes, and slowed muscle recovery. Chronic hypogonadism can also impair bone mineral density and lean

body mass. Notably, T suppression in EHMC can also occur separately from OTS and can be evident even in well-performing athletes without overt symptoms of fatigue or psychological distress (18).

While TRT is not generally recommended in EHMC owing to its functional and reversible nature, therapeutic interventions must aim to restore the hormonal balance by non-pharmacological means. These include a reduction in either the volume or intensity of training, optimization of caloric and micronutrient intake, and maximization of recovery periods. These interventions are intended to promote endogenous T production by reducing hypothalamic suppression and optimizing energy availability (52).

In conclusion, T suppression is a hallmark of EHMC and an important biomarker of maladaptive endocrine adaptation to chronic endurance exercise. Elucidation of the mechanisms and clinical sequelae of low T in this setting is critical for early detection and management of affected athletes, and for the discrimination of EHMC from pathological hypogonadism or overtraining syndromes.

Modulating Factors

Several factors modulate testosterone responses to exercise:

- **Age:** Older males hormonally gain from starting exercise, although increases in T can be less than in younger men. Habitual exercise can reduce the normal age-associated reduction in T, particularly when resistance exercise is included (17, 19).
- **Training Status:** Novice or untrained individuals tend to have more pronounced acute hormonal responses to exercise compared to trained athletes. This is partly because of neuromuscular novelty and the larger relative physiological stress placed upon identical workloads. Chronic training, nevertheless, can dampen this responsiveness (14, 24).
- **Energy Availability and Nutrition:** Low energy availability, caused by an insufficient caloric intake compared to the energy expenditure, can suppress the HPG axis and T production. This process, a key component of relative energy deficiency in sport, leads to functional hypogonadism, especially in endurance athletes or those with disordered eating behaviors (50, 53).
- **Psychological Stress and Sleep:** Chronic psychological stress and poor sleep increase cortisol, which in itself can suppress T production. Such lifestyle issues can exacerbate training-related stress and lead to endocrine imbalance (10, 54).
- **Circadian Rhythms:** T has a very distinct diurnal rhythm, typically peaking in the early morning hours. Morning exercise may have different hormonal effects than afternoon or evening exercise, and timing must be taken into account in research as well as training contexts (3).

Conclusions

Testosterone plays a key role in male physiology, and its levels are subject to dynamic changes influenced by lifestyle, particularly physical activity. A review of the available literature indicates that regular physical activity can support the maintenance of a eugonadal hormonal profile, but the effect of exercise on T levels depends on a number of variables, such as age, training status, energy availability, diurnal rhythm, and mental workload.

Resistance exercise, especially intense and multi-joint exercise, leads to transient increases in T levels and potentially beneficial adaptations with long-term use. Chronic endurance training, on the other hand, can result in a functional decrease in T levels, leading in extreme cases to the development of EHMC. This condition, while potentially reversible, requires early recognition and appropriate non-pharmacological intervention.

Disclosure

Author's contribution:

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All authors have read and agreed with published version of the manuscript.

Funding Statement – Not applicable.

Institutional Review Board Statement – Not applicable.

Informed Consent Statement – Not applicable.

Data Availability Statement – The authors confirm that the data supporting this study are available in the article's references.

Conflict of Interest – The authors declare no conflict of interest.

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