

Testosterone Times



The Official Newsletter of the Androgen Society
Advancing Excellence in Testosterone Deficiency Research and Clinical Practice

- Letter from the President
- The Androgen Society Research Digest
- Annual Meeting Update
- Testosterone News
- Learning Resources
- Membership Matters
- Connect with the Androgen Society

Letter from the President



Martin Miner, MD
TAS President

Dear Colleagues and Members of the Androgen Society,

I want to welcome you to the second issue of *The Testosterone Times*, our bimonthly newsletter. The goal remains to keep you closely connected to the vital work across our community.

The Androgen Society rests on scientific excellence, collaborative learning, and a shared commitment to improving recognition and management of testosterone deficiency. This newsletter continues our effort to strengthen those connections by highlighting current research, educational opportunities, and key updates from across the society.

In this issue, you'll find concise summaries of landmark studies shaping our field, updates on our educational initiatives, and

important announcements about our 2026 Annual Meeting. We are especially grateful to our former President, Dr. Abdul Traish, who continues to curate and synthesize pivotal publications for each issue, offering expert perspective on emerging data and evolving trends in androgen science to guide both clinicians and researchers.

Dr. Franck Mauvais-Jarvis, our colleague from Tulane, is leading a multi-author effort urging the FDA to reconsider labeling for what has traditionally been termed secondary hypogonadism or adult-onset testosterone deficiency, following the FDA expert panel meeting on December 10, 2025. At that meeting, Drs. Morgentaler, Khera, and I emphasized that testosterone deficiency and its treatment should be central to a national men's health strategy, aiming to reduce disparities in care and longevity by making appropriate therapy more accessible.

As Dr. Dobbs noted in Heallo, regulators and medical societies have been overly focused on identifying a specific cause of testosterone deficiency before allowing treatment, even though many other conditions (such as hypertension) are treated despite often uncertain etiology. In addition, as Dr. Khera stressed, current FDA restrictions still reflect outdated concerns that testosterone therapy is primarily a lifestyle drug, a carcinogen, or a major cardiovascular risk—views that conflict with contemporary evidence and are addressed in our society position statement and upcoming webinar.

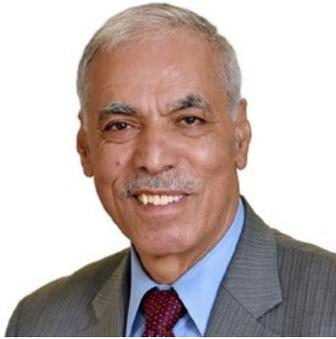
Several strategic initiatives continue to advance our mission, including the GP Testosterone Consensus Meeting in Amsterdam, the Princeton V initiative to expand appropriate use of testosterone in men and women with functional hypogonadism, and the TRAVERSE Ad-Hoc Committee focused on cardiovascular safety and regulatory implications. We are also pleased to introduce a new Learning Resources tab on our website, which will house evidence-based guidelines, clinical tools, and other materials to support your practice.

Please make full use of your membership benefits by updating your profile, exploring volunteer roles, and considering service on our social media committee to help extend the society's reach. If you were unable to attend the webinars, recordings are available online for on-demand viewing.

The momentum within our community is palpable, and the work we share—refining precision hormone therapy, generating high-quality data, and disseminating knowledge—directly improves the lives of patients who rely on our expertise. Thank you for your continued dedication to excellence in testosterone deficiency research and clinical care; I look forward to connecting with many of you at our upcoming webinars and at the Annual Meeting, which will once again precede the Endocrine Society's annual meeting in Chicago on June 11th and 12th.

Warm regards,

Martin Miner, MD
President, the Androgen Society



Abdulmaged M. Traish,
MBA, PhD

The Androgen Society Research Digest

Reviewed by Abdul Traish

Does Testosterone Therapy Increase Risk of Atrial Fibrillation (AF)? Appraisal of the Contemporary Scientific and Clinical Evidence.

Abdulmaged M Traish, Ph.D. Karim Sultan Haider, MD & Farid Saad, DVM, Ph.D.

Summary:

Here we review the evidence whether “testosterone” (T) therapy in hypogonadal men contributes to onset and increases the incidence of atrial fibrillation (AF). A MEDLINE review of the literature was performed, and relevant studies were analyzed and discussed. The effect of T therapy (Th) on the incidence of AF is a controversial clinical issue that remains hotly debated. Few studies have purported that TTh increases the incidence of AF. However, a closer look at these studies suggest that several confounding factors may have contributed to the authors conclusion that T increases the incidence of AF. On the contrary, a much larger number of studies have demonstrated that T deficiency contributes to incidence of AF and TTh does not increase the incidence of AF. Because this clinical issue is of critical importance in management of men with hypogonadism, it is critical that more clinical studies with AF as the primary outcome measure need to be considered. However, to date, none of the clinical trials have focused on AF as the primary outcome and in most of the reported studies AF was a secondary or tertiary outcome measure. Thus, lack of accurate clinical data on this issue will remain in the helm of debate, until new clinical trial data becomes available. We conclude that the findings of numerous studies suggest that low circulating levels (T deficiency) are associated with greater incidence of AF and higher endogenous T levels or TTh in hypogonadal men are associated with lower incidence of AF.

Introduction:

Atrial fibrillation (AF) is a major public-health concern associated with increased Atrial fibrillation (AF) is a major public-health concern associated

with increased mortality and morbidity including stroke, heart failure (HF), and cognitive decline. AF is a multifactorial and heterogeneous disease. Among the many factors contributing to development of AF are hypertension, heart failure, valve disease, coronary artery disease, obesity and congenital heart disease. AF may be the result of multiple structural, electrophysiologic, ionic, and molecular changes related both to aging itself and to aging-related comorbidities and cardiovascular risk factors, such as hyperlipidemia, hypertension, testosterone (T) deficiency, metabolic syndrome, diabetes, sarcopenia and frailty. The underlying molecular mechanisms of AF onset and persistence in hypogonadal men are poorly understood.

Does Testosterone (T) Therapy Contribute to Higher Incidence of AF?

A. Studies suggesting that T Therapy or High Circulating T Levels Increase the Risk of AF.

A recent study by Bonnet et al., [2025] [1] using data from the TriNetX Research Network compared 117,908 cis men with hypogonadism treated with T with 1:1 propensity-score matched cis men not treated with T. After 5 years of follow-up, cis men treated with TTh had a lower risk of myocardial infarction with no differences for stroke or mortality. However, these men exhibited higher risks of AF and acute pulmonary embolism/deep vein thrombosis. The authors suggested that TTh increases the onset of AF.

Close examination of the findings of this study, however, raised several concerns and suggested that confounding factors may have contributed to their findings. For example, the study did not account for variables that may contribute to AF such as co-morbidities including diabetes, hypertension, chronic obstructive pulmonary disease, obstructive sleep apnea, periodontal disease, congestive heart failure, peripheral vascular disease, coronary artery disease, hyperthyroidism, rheumatic valve disease, nonrheumatic valve disease, structural heart disease, and cardiomyopathy; baseline body mass index; low-density lipoprotein; and use of aspirin, β -blockers, and statins. Since many of these comorbidities contribute to incidence of AF, this study did not provide critical information regarding the role of TTh on the incidence of AF. In addition, as acknowledged by the authors “We cannot determine how the diagnosis of hypogonadism was done, and we did not have the dose of testosterone, the way of administration nor the adherence to testosterone therapy by each participant over the follow-up” [Bonnet et al., 2025][1]. Therefore, the findings of this study do not unequivocally conclude that TTh contributes to higher incidence of AF.

Furthermore, this study did not provide laboratory T values and no data on patient’s adherence to TTh or if the TTh normalized physiological T levels as discussed by Sharma et al., [2017] [2]. This study lacks critical information necessary to ascertain the relationship between TTh and incidence of AF. In addition, the authors concluded that TTh in transmen did not increase the risk of AF and went further to state that transmen treated with TTh had

a lower rate of AF than untreated cis-males and lower acute pulmonary embolism/deep vein thrombosis (APE/DVT) rate than cis-females using/oral contraceptive. It should be noted that transmen are biologically women (female-to-male transsexuals); thus, unless the pathophysiology of AF is different in women and men, this conclusion is considered baseless and at best unwarranted. Interestingly, however, a recent study by the same investigators [Bonnet et al 2025] [3] reported that TTh in diabetic men was associated with reduced risk of atrial fibrillation (HR: 0.91 [95% CI 0.85–0.98], $p = 0.01$) instead of increased risk of AF as they reported previously. Thus, it is difficult to reconcile such findings by the same group of investigators.

Data from the recent TRAVERSE trial [Lincoff et al 2023] [6] had also indicated that TTh may increase the risk of AF. Although the incidence of AF in the T arm that TTh may increase the risk of AF. Although the incidence of AF in the T arm was 91 (3.5%) compared to 63 patients (2.4%) in the placebo group, this increased risk, albeit small, was attributed to TTh. It should be noted that the Traverse Trial, in men with or without Covid showed no significant differences between T treated and the placebo group. In men with Covid AF was noted in 3/178(1.8%) in the T arm and 3/180 (2.1%) in the placebo group. Similarly, in men who did not get Covid, but received T treatment there were 32 cases of AF out of 2406 (1.4%). In the placebo group in men without Covid, there were 26 cases of AF out of 2410 (1.1%). This shows only 6 more cases of AF in the T treated group when compared with the placebo group [Pencina et al., 2025] [8]. It is unclear why the values provided in these two publications by the same group of authors differed significantly from one another. These discrepancies do not align with original findings of Lincoff et al., 2023 [6] suggest that such cases may be due to chance alone. In addition, it should be noted that in this trial, no precise information was provided on patients' adherence to TTh or achieving normal physiological levels of T over the entire course of treatment. The fact that a total of 61.4% of the patients in the T group discontinued TTh, and 61.7% of the patients in the placebo group discontinued placebo, suggests poor but normal compliance for a study of this sort. Furthermore, since all patients in this trial had either vascular disease or a history of vascular disease, it is not surprising that some of these patients are likely to have AF over the course of follow-up period, since many of the vascular disease risk factors contribute to onset of AF [Laredo et al., 2018][9]. with placebo group.

Tran et al., [2024] [7] analyzed findings from the ASPREE study with 4570 participants with mean follow-up of 4.4 years and suggested that higher circulating T levels are independently associated with an increased risk of AF in healthy older men. Similarly, data from the prospective Atherosclerosis Risk in Communities (ARIC) study [Berger et al., 2019][5] identified incident AF among 9,282 participants who had plasma total T measured by liquid chromatography tandem mass spectrometry at Visit 4. After a mean of 13.7 years of follow-up, 1664 incident cases of AF were identified. There was a positive association between plasma total T and incident AF in men. It was concluded that a higher plasma total T concentration was associated with a modestly greater incidence rate of AF in men.

O'Neal et al., 2017 [4] analyzed 4,883 participants from the Multi-Ethnic Study of Atherosclerosis. Over a median follow-up of 10.9 years, higher bioavailable testosterone levels were associated with increased AF risk in men. The authors concluded that higher levels of endogenous bioavailable T contribute to AF development in men. Based on the aforementioned studies it appears that higher T levels in circulation or TTh in men with T deficiency may increase the incidence of AF.

B. Studies Suggesting That T Deficiency and Not Higher T Levels Increases the Risk of AF.

In contrast to the studies discussed above, Magnani et al., [2014][10]. identified a significant interaction between age and T, in a cohort of 1251 men of whom 275 developed incident AF and therefore stratified men into age 55–69 (n=786), 70–79 (n=351), and ≥80 (n=114). In men 55–69 each 1-standard deviation (SD) decrease in T was associated with hazard ratio (HR) 1.30 (95% confidence interval [CI], 1.07 to 1.59) for incident AF [Table 2]. The association between T and 10-year incident AF in men 70–79 did not reach statistical significance. However, in men ≥80 years a 1-SD decrease in T was associated with HR 3.53 (95% CI, 1.96 to 6.37) for AF risk. It was concluded that T is associated with incident AF in a cohort of older men. In study assessing the link of sex hormones with AF [Costa et al., 2021][11], it was suggested that testosterone deficiency is associated with increased atrial fibrosis, impaired connexin-mediated gap junctional communication, and heightened autonomic imbalance, all of which promote conduction heterogeneity and re-entry circuits that facilitate AF initiation and maintenance.

Sharma et al 2017 [2] investigated a national cohort of 76,639 veterans with low TT levels who were divided into 3 groups. Group 1 had TTh resulting in normalization of TT levels (normalized T), group 2 had TTh without normalization of TT levels (nonnormalized T), and group 3 did not receive T (no T). Normalization of T levels after TTh was associated with a significant decrease in the incidence of AF. This is contrary to the studies cited above and suggests caution should be exercised prior to assigning a false premise that TTh contributes to increased AF risk.

Zeller et al., [2018][12] reported on a population cohort of 7892 subjects (3876), aged 25–74 years, with median follow-up of 13.8 years, indicating that increasing levels of T were associated with lower risk of AF in men and noted that low T levels are associated with increased risk of AF in men. In a study of 1019 men with median follow-up of 9.5 years in which plasma DHT levels were measured, Rosenberg et al., [2018][13] reported that 304 (30%) men developed AF. A nonlinear association with risk of incident AF was noted with both free and total DHT, in which subjects with the lowest levels of DHT had a higher risk of incident AF. The authors concluded that among older men, low free DHT is associated with an increased risk of incident AF. More recently, Cho et al., [2024][14] reported on data from the UK Biobank, with a population-based prospective cohort, focusing on 179,988 men who

had complete serum TT level data and no AF history. During the median follow-up of 11.8 years the authors noted 13,847 incidents of AF (7.7%). Participants with normal TT levels (≥ 300 ng/dL) showed a 15% reduced risk of AF compared to those with low TT (< 300 ng/dL). Furthermore, each 1-standard deviation increase in TT levels was associated with a reduced AF risk. These findings suggest that low TT levels are associated with reduced LA function and increased risk of AF.

In a retrospective observational study [Blackwell et al., 2024][15] in which 133,584 with hypogonadism were included after propensity matching who were prescribed TTh versus no T treatment. The authors reported a decreased risk of AF (3.6% vs 4.0%; RR 0.900; $P < .001$) for patients treated with testosterone. In a systematic review and meta-analysis, Corona et al., [2024][16] reported that out of 8126 patients with hypogonadism treated with TTh and 7310 patients who remained untreated (placebo), the incidence of non-fatal arrhythmias and AF was increased only in one study. In addition, the authors pointed out that “both nonfatal arrhythmia and AF were not trial endpoints of the TRAVERSE study but only investigator-reported adverse events, and hence they should be interpreted with caution.” This, however, was not confirmed when all other studies were considered. Furthermore, no relationship between endogenous T levels and AF incidence was observed after the adjustment for confounders.

Xu B et al., [2024] [17] investigated a cohort of 173, 498 men (aged 37-73 years, followed for 11 years) from the UK Biobank. Among these men 11, 368 had incident AF. Compared with the third quartiles, the lowest category of serum T was associated with increased risks of AF. Similar associations were found between cFT values and AF events. Interestingly however, higher levels of cFT were associated with increased risks of AF. In addition, higher SHBG concentrations were associated with increased risks of AF. Therefore, lower T concentrations were an independent risk predictor for incident AF in men.

A recent study utilizing data from the TriNetX Research Network compare hypogonadal men who had a prescription for topical T therapy with men who did not [Greenberg et al., 2024] [21]. There were 2134 men included in each cohort after propensity score matching. The findings of this study suggested that TTh was not associated with a significantly increased risk of new-onset AF. Finally, a meta-analysis of randomized clinical trials by Sood et al., [2024] [22] demonstrated that TTh was not associated with a statistically increased or decreased risk of AF. It should be noted that data from several other observational studies [Lai et al., 2009] [18] ; Rosenberg et al 2018] [13] and meta-analyses [Hu et al., 2022] [20] and Sood et al., 2024 [22] ; Cho et al., 2024[14] also suggested that low T is a contributor to incidence of AF in older men.

C. Findings from Animal Studies Suggesting That T Deficiency Increases the Risk of AF.

Findings from animal studies by Tsuneda et al., [2009] [23] had demonstrated that repetitive atrial responses induced by electrical stimuli significantly increased in castrated rats without changes in other electrophysiological properties and were abolished by administration of T. In men, medical castration prolongs the QT/QTc intervals in most patients with prostate cancer treated with androgen deprivation therapy and it could cause TdP/ VFs even in patients with no risk of QT prolongation before the therapy [Hasegawa et al., 2021] [19]. Thus, it appears that physiological T levels do not increase the incidence of AF while low T contribute to the pathophysiology of AF in animals and humans. Similar studies by Tsai et al., [2013] [24] have suggested that T deficiency plays a role in the pathophysiology of AF. Androgen regulates cardiac electrophysiology and calcium (Ca²⁺) homeostasis, and findings from androgen receptor knock out (ARKO) animal models demonstrated that loss of the androgen receptor alters atrial electrophysiology with increased atrial arrhythmogenesis. Furthermore, Zhang et al., [2017] [25] demonstrated that castration in male mice increased the incidence of AF. The number of AF episodes in castrated mice increased by 13.5-fold when compared with sham mice. The authors demonstrated that androgen treatment alleviated the occurrence of AF. The authors concluded that TD contributed to the increased late sodium current, prolonged action potential repolarization, and increased susceptibility to AF.

Summary and Conclusions:

The relationship between circulating T levels or TTh and increased incidence of AF remains controversial and hotly debated. It is important to ask the key question: Do normal physiological or higher T levels represent a risk for AF? If this were the case, then we expect younger men between ages of 18 years old and 60 years old to be at the highest risk of AF than older men, since they have normal or high physiological T levels compared to older men. However, the incidence of AF is higher in older men where T levels are often low compared to younger men. Therefore, in absence of real scientific and clinical evidence for the etiology and the pathophysiological mechanisms of T action on the onset of AF, these claims remain unjustified and at, best, anecdotal. It must be noted that almost all studies suggesting that TTh increases the risk of AF do not provide key information on T levels or patients' adherence to TTh and other known comorbidities that contribute to onset of AF. Hence, it is our opinion that adequate TTh (achieving physiological T levels with good patient compliance) either has no effect or a beneficial effect in reducing incidence of AF.

Based on the findings of the contemporary clinical literature, T deficiency is a real contributor to increased risk of AF in older men and normalization of T levels with TTh may decrease the rates of AF [Sharma et al 2017] [2]. However, the impact of TTh on the onset or incidence of AF remains equivocal and requires further studies. The studies described in Table 1

advocating that higher circulating T or TTh increase the risk of AF remind us of the purported studies on the adverse effects of TTh on increasing the risk of cardiovascular disease [Basaria et al., 2010; [26] Vigen et al., 2013[27]; Finkle et al., 2014[28] Xu et al 2013[29] which were proven false by findings from the T-Trials [Snyder et al., 2016] [30] T4DM trial [Wittert et al., 2021[31]] and the TRAVERSE Trial [Lincoff et al., 2023] [6]. The findings of the studies described here suggest that T deficiency and not TTh may be associated with cardiometabolic co-morbidities and in turn may contribute to the increased incidence of AF in older men.

Here we review the evidence whether “testosterone” (T) therapy in hypogonadal men contributes to onset and increases the incidence of atrial fibrillation (AF). A MEDLINE review of the literature was performed, and relevant studies were analyzed and discussed. The effect of T therapy (Th) on the incidence of AF is a controversial clinical issue that remains hotly debated. Few studies have purported that TTh increases the incidence of AF. However, a closer look at these studies suggest that several confounding factors may have contributed to the authors conclusion that T increases the incidence of AF. On the contrary, a much larger number of studies have demonstrated that T deficiency contributes to incidence of AF and TTh does not increase the incidence of AF. Because this clinical issue is of critical importance in management of men with hypogonadism, it is critical that more clinical studies with AF as the primary outcome measure need to be considered. However, to date, none of the clinical trials have focused on AF as the primary outcome and in most of the reported studies AF was a secondary or tertiary outcome measure. Thus, lack of accurate clinical data on this issue will remain in the helm of debate, until new clinical trial data becomes available. We conclude that the findings of numerous studies suggest that low circulating levels (T deficiency) are associated with greater incidence of AF and higher endogenous T levels or TTh in hypogonadal men are associated with lower incidence of AF.

References:

- [1]. Bonnet F, Vaduva P, Balkau B, Genet T, de Freminville JB, Ducluzeau PH, Fauchier L. Testosterone therapy and the risk of atrial fibrillation, venous thromboembolism and cardiovascular events in cis men with hypogonadism and trans men. *Eur J Endocrinol.* 2025 Aug 29;193(3):374-382.
- [2]. Sharma R, Oni OA, Gupta K, Sharma M, Sharma R, Singh V, Parashara D, Kamalakar S, Dawn B, Chen G, Ambrose JA, Barua RS. Normalization of Testosterone Levels After Testosterone Replacement Therapy Is Associated with Decreased Incidence of Atrial Fibrillation. *J Am Heart Assoc.* 2017 May 9;6(5):e004880
- [3]. Bonnet F, Vaduva P, Halimi JM, Dosda A, Ducluzeau PH, Koppe L, Fauchier L. Testosterone therapy is associated with reduced risk of acute kidney injury, kidney failure with renal replacement therapy, and cardiovascular events in men with diabetes and hypogonadism. *Cardiovasc Diabetol.* 2025 Oct 1;24(1):378. doi: 10.1186/s12933-025-02930-2. PMID: 41035033; PMCID: PMC12487499.

- [4]. O'Neal WT, Nazarian S, Alonso A, Heckbert SR, Vaccarino V, Soliman EZ. Sex hormones and the risk of atrial fibrillation: The Multi-Ethnic Study of Atherosclerosis (MESA). *Endocrine*. 2017 Oct;58(1):91-96.
- [5]. Berger D, Folsom AR, Schreiner PJ, Chen LY, Michos ED, O'Neal WT, Soliman EZ, Alonso A. Plasma total testosterone and risk of incident atrial fibrillation: The Atherosclerosis Risk in Communities (ARIC) study. *Maturitas*. 2019 Jul; 125:5-10.
- [6]. Lincoff AM, Bhasin S, Flevaris P, Mitchell LM, Basaria S, Boden WE, Cunningham GR, Granger CB, Khera M, Thompson IM Jr, Wang Q, Wolski K, Davey D, Kalahasti V, Khan N, Miller MG, Snabes MC, Chan A, Dubcenco E, Li X, Yi T, Huang B, Pencina KM, Trivison TG, Nissen SE; TRAVERSE Study Investigators. Cardiovascular Safety of Testosterone-Replacement Therapy. *N Engl J Med*. 2023 Jul 13;389(2):107-117.
- [7]. Tran C, Yeap BB, Ball J, Clayton-Chubb D, Hussain SM, Brodtmann A, Tonkin AM, Neumann JT, Schneider HG, Fitzgerald S, Woods RL, McNeil JJ. Testosterone and the risk of incident atrial fibrillation in older men: further analysis of the ASPREE study. *EClinicalMedicine* 2024;72:102611.
- [8]. Pencina KM, Lincoff AM, Klein EA, Nissen SE, Shang YV, Khan N, Li X, Chan A, Miller MG, Bhasin S. Testosterone Replacement Therapy and Risk of COVID-19 and Effect of COVID-19 on Testosterone's Treatment Effect. *Journal of the Endocrine Society*. 2025 Mar;9(3):bvaf002
- [9]. Laredo M, Waldmann V, Khairy P, Nattel S. Age as a Critical Determinant of Atrial Fibrillation: A Two-sided Relationship. *Can J Cardiol*. 2018 Nov;34(11):1396-1406.
- [10]. Magnani JW, Moser CB, Murabito JM, Sullivan LM, Wang N, Ellinor PT, Vasan RS, Benjamin EJ, Coviello AD. Association of sex hormones, aging, and atrial fibrillation in men: the Framingham Heart Study. *Circ Arrhythm Electrophysiol*. 2014 Apr;7(2):307-12.
- [11]. Costa S, Saguner AM, Gasperetti A, Akdis D, Brunckhorst C, Duru F. The Link Between Sex Hormones and Susceptibility to Cardiac Arrhythmias: From Molecular Basis to Clinical Implications. *Front Cardiovasc Med*. 2021 Feb 17; 8:644279. doi: 10.3389/fcvm.2021.644279. PMID: 33681311; PMCID: PMC7925388.
- [12]. Zeller T, Schnabel RB, Appelbaum S, Ojeda F, Berisha F, Schulte-Steinberg B, Brueckmann BE, Kuulasmaa K, Jousilahti P, Blankenberg S, Palosaari T, Salomaa V, Karakas M. Low testosterone levels are predictive for incident atrial fibrillation and ischaemic stroke in men, but protective in women - results from the FINRISK study. *Eur J Prev Cardiol*. 2018 Jul;25(11):1133-1139.
- [13]. Rosenberg MA, Shores MM, Matsumoto AM, Bůžková P, Lange LA, Kronmal RA, Heckbert SR, Mukamal KJ. Serum androgens and risk of atrial fibrillation in older men: The Cardiovascular Health Study. *Clin Cardiol*. 2018 Jun;41(6):830-836.

- [14]. Cho SH, Yang PS, Jang ES, Kim DH, Yu HT, Kim TH, Uhm JS, Pak HN, Lee MH, Joung BY. Association of serum testosterone levels with left atrial size, function, and risk of atrial fibrillation in adult men: observational and mendelian randomization analyses. *European Heart Journal*. 2024 Oct;45(Supplement_1):ehae666-2747.
- [15]. Blackwell KM, Buckingham H, Paul KK, Uddin H, Jehle DVK, Blackwell TA. Benefits of Testosterone Replacement Therapy in Hypogonadal Males. *J Am Board Fam Med*. 2024 Sep-Oct;37(5):816-825. doi: 10.3122/jabfm.2024.240025R1. PMID: 39978846.
- [16]. Corona G, Rastrelli G, Sparano C, Carinci V, Casella G, Vignozzi L, Sforza A, Maggi M. Cardiovascular safety of testosterone replacement therapy in men: an updated systematic review and meta-analysis. *Expert Opinion on Drug Safety*. 2024 May 3;23(5):565-79.
- [17]. Xu B, Mo W, Tan X, Zhang P, Huang J, Huang C, Guo D, Wei X, Liu Y, Lei X, Dou W, Lin J, Liu D, Yang L, Huang Y, Zhang H, Liao Y. Associations of Serum Testosterone and Sex Hormone-binding Globulin With Incident Arrhythmias in Men From UK Biobank. *J Clin Endocrinol Metab*. 2024 Jan 18;109(2):e745-e756.
- [18]. Lai J, Zhou D, Xia S, et al. Reduced testosterone levels in males with lone atrial fibrillation. *Clin Cardiol* 2009;32:43-6.
- [19]. Hasegawa K, Ito H, Kaseno K, Miyazaki S, Shiomi Y, Tama N, Ikeda H, Ishida K, Uzui H, Ohno S, Horie M. Impact of medical castration on malignant arrhythmias in patients with prostate cancer. *Journal of the American Heart Association*. 2021 Mar 2;10(5):e017267.
- [20]. Hu P, Huang J, Lu Y, Zheng M, Li H, Duan X, Deng H, Zhao W, Liu X. Circulating sex hormones and risk of atrial fibrillation: A systematic review and meta-analysis. *Frontiers in Cardiovascular Medicine*. 2022 Aug 22; 9:952430.
- [21]. Greenberg DR, Kohn TP, Asanad K, Brannigan RE, Halpern JA. Association of testosterone replacement therapy with atrial fibrillation and acute kidney injury. *J Sex Med*. 2024 Dec 1;21(12):1201-1203.
- [22]. Sood A, Hosseinpour A, Sood A, Avula S, Durrani J, Bhatia V, Gupta R. Cardiovascular Outcomes of Hypogonadal Men Receiving Testosterone Replacement Therapy: A Meta-analysis of Randomized Controlled Trials. *Endocr Pract*. 2024 Jan;30(1):2-10.
- [23]. Tsuneda T, Yamashita T, Kato T, et al. Deficiency of testosterone associates with the substrate of atrial fibrillation in the rat model. *J Cardiovasc Electrophysiol* 2009;20:1055-60.
- [24]. Tsai W-C, Yang L-Y, Chen Y-C, et al. Ablation of the androgen receptor gene modulates atrial electrophysiology and arrhythmogenesis with calcium protein dysregulation. *Endocrinology* 2013; 154:2833-42.

- [25]. Zhang Y, Wang HM, Wang YZ, Zhang YY, Jin XX, Zhao Y, Wang J, Sun YL, Xue GL, Li PH, Huang QH, Yang BF, Pan ZW. Increment of late sodium currents in the left atrial myocytes and its potential contribution to increased susceptibility of atrial fibrillation in castrated male mice. *Heart Rhythm*. 2017 Jul;14(7):1073-1080.
- [26]. Basaria S, Coviello AD, Travison TG, Storer TW, Farwell WR, Jette AM, Eder R, Tennstedt S, Ulloor J, Zhang A, Choong K, Lakshman KM, Mazer NA, Miciek R, Krasnoff J, Elmi A, Knapp PE, Brooks B, Appleman E, Aggarwal S, Bhasin G, Hede-Brierley L, Bhatia A, Collins L, LeBrasseur N, Fiore LD, Bhasin S. Adverse events associated with testosterone administration. *N Engl J Med*. 2010 Jul 8;363(2):109-22.
- [27.] Vigen R, O'Donnell CI, Barón AE, Grunwald GK, Maddox TM, Bradley SM, Barqawi A, Woning G, Wierman ME, Plomondon ME, Rumsfeld JS, Ho PM. Association of testosterone therapy with mortality, myocardial infarction, and stroke in men with low testosterone levels. *JAMA*. 2013 Nov 6;310(17):1829-36
- [28]. Finkle WD, Greenland S, Ridgeway GK, Adams JL, Frasco MA, Cook MB, Fraumeni JF Jr, Hoover RN. Increased risk of non-fatal myocardial infarction following testosterone therapy prescription in men. *PLoS One*. 2014 Jan 29;9(1):e85805.
- [29]. Xu L, Freeman G, Cowling BJ, Schooling CM. Testosterone therapy and cardiovascular events among men: a systematic review and meta-analysis of placebo-controlled randomized trials. *BMC Med*. 2013 Apr 18; 11:108.
- [30]. Snyder PJ, Bhasin S, Cunningham GR, Matsumoto AM, Stephens-Shields AJ, Cauley JA, Gill TM, Barrett-Connor E, Swerdloff RS, Wang C, Ensrud KE, Lewis CE, Farrar JT, Cella D, Rosen RC, Pahor M, Crandall JP, Molitch ME, Cifelli D, Dougar D, Fluharty L, Resnick SM, Storer TW, Anton S, Basaria S, Diem SJ, Hou X, Mohler ER 3rd, Parsons JK, Wenger NK, Zeldow B, Landis JR, Ellenberg SS; Testosterone Trials Investigators. Effects of Testosterone Treatment in Older Men. *N Engl J Med*. 2016 Feb 18;374(7):611-24
- [31]. Wittert G, Bracken K, Robledo KP, Grossmann M, Yeap BB, Handelsman DJ, Stuckey B, Conway A, Inder W, McLachlan R, Allan C, Jesudason D, Fui MNT, Hague W, Jenkins A, Daniel M, Gebiski V, Keech A. Testosterone treatment to prevent or revert type 2 diabetes in men enrolled in a lifestyle programme (T4DM): a randomised, double-blind, placebo-controlled, 2-year, phase 3b trial. *Lancet Diabetes Endocrinol*. 2021 Jan;9(1):32-45.

Annual Meeting Information

8th Annual
Meeting of the
Androgen
Society

Androgen Society

Registration is Open

June 11-12, 2026

Chicago, Illinois

8th Annual Meeting of The Androgen Society

Registration Pricing

Androgen Society Active Members – \$550

Androgen Society Associate Members – \$300

Androgen Society Trainee Members – \$275

Non-Member Physicians – \$600

Non-Member Non-Physicians – \$350

Non-Member Trainees – \$275

Register today for the 8th Annual Meeting of the Androgen Society — join us for two days of engaging and innovative programming at the University of Chicago – David Rubenstein Forum.

This meeting will once again serve as the premier forum for research and education regarding testosterone deficiency and its treatment, bringing together healthcare professionals from around the world to share the latest advances in androgen therapy.



Please sign into the site to ensure that you receive Androgen Society Member pricing for your registration.

Not an Androgen Society member? [Join today!](#)

Register Today!

Don't miss our Early Bird pricing!

Save \$50 on your meeting registration by signing up before April 10, 2026!



Hotel Accomodations

A room block has been reserved at The Study at University of Chicago for meeting attendees. Conveniently located near the venue!

To book your room:

Use the room block link for the Androgen Society or Call 773-643-1600, press "1" for reservations.

Please note: the cut-off to receive room block pricing is May 19, 2026.

[Reserve Your Room](#)

2026 Annual Meeting Draft Agenda

Watch for updates to the 2026 Agenda - click on link for current information.



[Draft Agenda](#)

Abstract Submission Deadline Extended!



Please note that all presenters must plan to attend the conference in person. When preparing your submission, please include:

- Abstract title
- Complete abstract text
- All authors and affiliations
- Confirmation of author approval

For questions regarding abstract submission, please contact androgensociety@affinity-strategies.com

Submission Deadline: April 15
(Extended!)
Notification Date: May 1

[Submit Your Abstract](#)



Dan Kelly, MD
Program Committee Chair

“I’m both delighted and deeply honoured to lead the Scientific Programme for next year’s Annual Meeting of the Androgen Society. Building on last year’s exceptional scientific line-up, we are committed to raising the bar even further. Our goal is to gather the global community of testosterone researchers and clinicians – bringing the world’s foremost experts under one roof – and curate an exhilarating, intellectually vibrant programme that reflects the very latest in testosterone science.

The agenda will balance cutting-edge breakthroughs with practical insights: from foundational mechanisms and translational discoveries to real-world patient care strategies. Expect lively, interactive debates that take on long-standing clinical controversies, insightful case-study discussions, and the chance to network with peers and leaders in an open, dynamic setting.

This meeting is designed to expand your expertise on all things testosterone. Whether you’re a seasoned investigator or a clinician seeking fresh perspectives, I can confidently promise a stimulating and fulfilling two-day experience that will both inform and inspire.

I am eager to welcome you to what promises to be a memorable and transformative gathering. Let’s push the boundaries of what’s possible in testosterone research and care – together.”

~ Dan Kelly, Program Committee Chair

**THANK
YOU
TO OUR
SPONSORS**

Androgen Society



Groundbreaking FDA Presentation Marks Historic Moment for Testosterone Therapy

The Androgen Society made history as three of the field's most distinguished leaders — Dr. Mohit Khera, Dr. Marty Miner, and Dr. Abe Morgentaler— participated in a groundbreaking FDA panel that could fundamentally transform testosterone therapy access and patient care.

This pivotal panel addressed critical barriers that have long hindered optimal patient treatment, with key recommendations presented to the FDA:

Panel Recommendations:

- **Eliminate the “age-related” restriction** – Many medical conditions are “agerelated” in that they become more prevalent with aging, including hypertension, vheart disease, and most adult cancers. These conditions are treated to improve health. The same should be true for testosterone deficiency
- **Remove testosterone from the list of controlled substances** – No other hormone is controlled. This step will reduce barriers to testosterone therapy access

- **Raise treatment threshold to 350 ng/dL** – Allow therapy initiation at this level when accompanied by clinical signs and symptoms
- **Remove prostate cancer warnings from labeling** – Reflecting current evidence and eliminating outdated concerns that prevent appropriate care

This event represents a watershed moment in our field's efforts to advance evidence-based care and remove regulatory obstacles that have prevented countless men from accessing appropriate treatment.

The Androgen Society remains committed to advocating for policies that prioritize patient health and reflect the latest scientific evidence in men's health.

Expert Panel on Testosterone Replacement Therapy for Men



Mohit Khera



Martin Miner



Abraham
Morgentaler



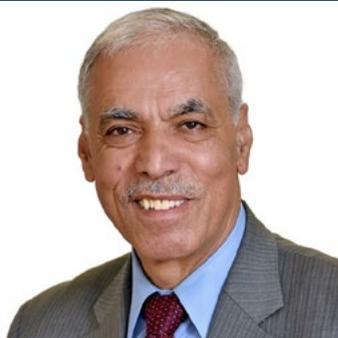
Learning Resources

We're excited to announce the launch of our new [Learning Resources tab](#) on the Androgen Society website!

As part of our mission to promote excellence in research, education, and clinical practice regarding testosterone deficiency and its treatment, we've created a centralized hub where members can access valuable educational materials, research updates, and clinical resources—all in one convenient location.

Have articles or resources to share? We welcome contributions from our members! If you have relevant publications, research findings, or educational materials you'd like to share with the Androgen Society community, please send them to androgensociety@affinitystrategies.com.

Membership Matters



Abdulmaged M. Traish,
MBA, PhD
Membership Co-Chair



Abraham Morgentaler,
MD, FACS
Membership Co-Chair

Get More Involved - Update Your Profile

- Log into your membership profile
 - Update contact information
 - Add your specialty and areas of interest
-

Social Media Team – Join Us!

Calling 1-3 Passionate Members Help amplify our society's impact through social media:

- We'll provide the graphics and content support
 - Flexible time commitment
 - Make a difference in our community outreach
 - Creative input welcome
-

Mentor/Mentee Program

Share Knowledge • Build Connections • Grow Together

- **Experienced Members:** Share your expertise with emerging professionals
- **Early Career Members:** Gain valuable guidance and insights

Interested? Please contact:
androgensociety@affinitystrategies.com

Connect with The Androgen Society

WE WANT TO HEAR FROM YOU!
Help Us Improve Society Communications

Your feedback drives our improvements:

- What communication methods work best for you?
- What topics would you like to see covered?
- How can we better serve your professional needs?
- What resources are missing from your toolkit?

Share your thoughts at AndrogenSociety@affinity-strategies.com.



Androgen Society