Androgen Society

November Newsletter

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

Letter from the President

Martin Miner, MD
TAS President

Dear Colleagues and Members of the Androgen Society,

As we transition into fall, I'm delighted to welcome you to the inaugural issue of *The Testosterone Times*, our new bimonthly newsletter designed to keep you connected to the vital work happening across our community.

The Androgen Society has always been built on a foundation of scientific excellence, collaborative learning, and a shared commitment to advancing patient care in recognition and management of testosterone deficiency. This newsletter represents our ongoing effort to strengthen these connections—bringing you the latest research insights, educational opportunities, and updates from across our society.

In this November issue, you'll find summaries of groundbreaking research that's shaping our field, updates on our educational initiatives, and important announcements about our 2026 Annual Meeting. We are grateful to our esteemed colleague and former President, Dr. Abdul Traish, for his continued dedication to advancing our mission. Abdul will be curating and summarizing 2-3 pivotal studies for each newsletter, offering our members his scholarly perspective on the latest breakthroughs and emerging trends in androgen science. His thoughtful analyses will serve as an essential resource for clinicians and researchers alike, bridging gaps between cutting edge research and practical application in our rapidly evolving field.

Congratulations to our colleagues on the publication of *Principles of Precision Hormone Therapy,* a new text edited by Dr. Franck Mauvais-Jarvis – a landmark contribution to our field that showcases the depth of expertise within our membership.

Many strategic initiatives are underway that will advance our society's mission. These include the GP Testosterone Consensus Meeting in Amsterdam, Princeton V (hopefully to broaden the use of T in men and women with functional hypogonadism), and the TRAVERSE Ad-Hoc committee. We're also excited to announce that a Learning Resources tab has been added to the website, which will feature evidence-based guidelines, clinical tools, and educational materials to support your practice.

We're also thrilled to introduce our Emerging Scholars Class of 2025-2026 and celebrate our recent award winners who exemplify the innovation and dedication that define our society.

I encourage you to take full advantage of your membership benefits. Update your profile to reflect your current interests and expertise and explore opportunities to get involved. We're actively seeking members to lead and join our social media committee—helping us amplify our society's impact and reach. If you missed last month's webinar, we've recorded it for you [link]. Your engagement makes our community stronger.

As we look ahead, I am energized by the momentum we're building together. The work we do—advancing precision hormone therapy, conducting rigorous research, and sharing knowledge—has real impact on the patients who depend on our expertise.

Thank you for your continued dedication to excellence in testosterone deficiency research and clinical practice. I look forward to seeing many of you at our upcoming webinars and at next year's annual meeting.

Warm regards,

Martin Miner, MD
President, the Androgen Society

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AbdulmagedM. Traish,
MBA, PhD

The Androgen Society Research Digest

Reviewed by Abdul Traish

Debate About the Free Testosterone Hypothesis: Useful Tool or Misguided Mirage?

Anawalt B. D. and Handelsman D.J. *JCEM*, 2025,00,1-5.

This article debates a central question in the clinical evaluation of hypogonadism. "Does free testosterone (FT) have better clinical value over that of total testosterone (TT) in diagnosis of hypogonadism?"

Before we delve into the debate itself, we should highlight few simple principles of biochemistry and physiology of testosterone circulation in blood so the reader can appreciate the big picture of this debate.

Plasma TT is comprised of albumin bound T, SHBG bound T and FT [Fig 1]. Indeed, we all learned in high school chemistry that lipophilic substances, such as steroids are portioned between the aqueous and lipophilic components of the solvent, in which "like dissolves like". Thus, a lipophilic substance such as testosterone will quickly partition between plasma proteins (e.g., SHBG and Albumin) and the aqueous environment of the serum.

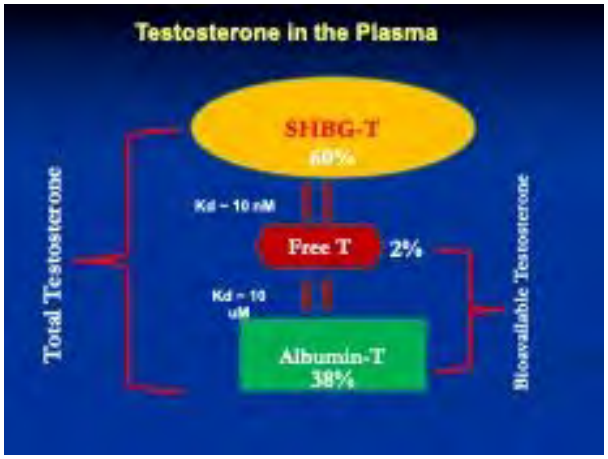

Figure 1. Distribution of protein bound and free T in the plasma

Note that the majority of T is bound to SHBG and serum albumin and only a small fraction remains soluble as free T and this free fraction is accessible to target cells. Target cells can only respond to the chemical messengers if these messengers reach their cognate receptors to initiate physiological signaling.

Protein bound T does not trespass the plasma membrane. Only the free T fraction can trespass the plasma membrane of target cells. Thus, how would it be possible that TT reflects the biological signaling if a major fraction had never entered the target cell.

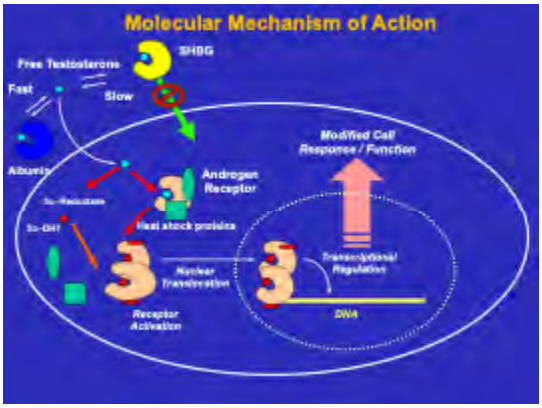

Figure 2. Androgen Receptor Signaling in Target Cells

A clinical study of more than one thousand patients had demonstrated that there is a wide variability of SHBG concentrations, and this may affect the FT levels with minimal effect on TT levels [Krakowsky Y *et al..* 2017].

Back to the debate: Simply put, Dr. Anawalt addressed this question in a crystal-clear manner with sufficient biochemical, physiological and clinical evidence. Dr. Anawalt cites one key clinical study from the European Male Aging Study which demonstrated unequivocally that hypogonadal symptoms are correlated more closely with the free T than total T [Antonio L., J. 2016].

Dr. Anawalt cites a laboratory study in which transgenic male mice were transfected with gene overexpressing human SHBG. These animals demonstrated elevated serum total testosterone, TG-SHBG TT ~10000 ng/dL compared to the wild type with TT ~120 ng/dL. FT was markedly reduced in the SHBG transgenic animals and concomitant decreased weight of the most androgen-sensitive organs (seminal vesicles and levator ani/bulbocavernosal muscles) [Laurent MR, *et al.,* 2016].

The relationship between age and total T, free T, LH & SHBG was clearly presented in the study by Wu et al., indicating that with age, SHBG levels rise, and FT levels decline, albeit the total T may not be reduced significantly. These findings make it clearer that FT is the fraction that should be used clinically to diagnose hypogonadism [Wu FCW et al., [2008]. Also, the work of Bhasin Group in which increased levels of SHBG dramatically reduces the FT is of critical relevance to this discussion [Zakharove *et al.,* 2015].

The Endocrine Society Guidelines Committee [Bhasin et al., 2018] has recommended that FT should be measured in men with conditions of Obesity, Diabetes, use of glucocorticoids, progestins and androgenic steroids. Also, the committee recommends use of FT in conditions of nephrotic syndrome, hypothyroidism, acromegaly and in polymorphism of the SHBG gene. It also recommends use of FT in aging, HIV disease, Cirrhosis and hepatitis, hyperthyroidism, use of estrogens and use of anticonvulsants.

Dr. Handelsman made several points questioning the usefulness of FT measurement. One is that FT is more susceptible to cellular degradation than bound T. While true, it is of no biological importance since target cells have a capture system for FT via the androgen receptor. Once bound to its receptors, FT will transduce the biochemical signaling pathway it is designed to elicit.

Another argument is that it is difficult to measure free T, and there are conflicting views on how to best perform a calculated FT. These are old arguments that have been shown to be incorrect. In a blinded study in which clinical blood samples were split between two labs- one that performed direct measurement of FT and another that performed equilibrium dialysis- the correlation between the results was excellent. Also, the correlation between calculated FT and equilibrium dialysis was excellent. [Kacker et al., 2013]. In sum, Dr. Handelsman arguments about the ease of measuring total T vs free T holds no water, in my view.

To this end, I believe that Dr. Anawalt had done a marvelous job clarifying this concept and leaving no shadow of doubt in the minds of those who have a clear understanding of biochemistry and physiology.

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Testosterone and Insulin Resistance in Men: Evidence For a Complex Bi-Directional Relationship

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A considerable body of evidence exists suggesting a link among reduced testosterone (T) plasma levels, increased insulin resistance (IR) and type 2 diabetes mellitus (T2DM) [Traish et al., 2009]. Low T precedes elevated fasting insulin, glucose, and hemoglobin A1c (HbA1c) values and may even predict the onset of diabetes (T2DM) [Figures 1 and 2].

Smith et al., [2001] demonstrated that gonadotropin-releasing hormone (GnRH) agonist treatment in men with prostatae cancer for 12 weeks elevated HbA1c and decreased insulin sensitivity index and HOMA-IR measurements. Similarly, use of androgen deprivation therapy (ADT) in men with prostate cancer significantly increases the risk of T2DM with a hazard ratio (HR) of 1.4 [Keating et al. 2006].

On the other hand, T therapy of hypogonadal men improves insulin sensitivity (IS), fasting glucose, and HbA1c levels. Clearly T deficiency is associated with increased IR, reduced IS and T2DM, increased onset of metabolic syndrome (MetS) [Traish et al., 2009].

Interplay Between Testosterone Deficiency and Insulin Sensitivity

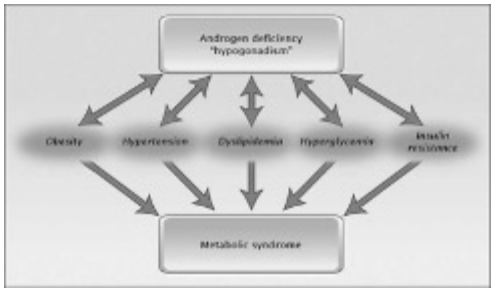

Figure 1. Bidirectional relationship between T deficiency and Metabolic Syndrome [Adopted from Traish et al., 2009].

Yialamas et al., [2007] demonstrated that acute sex steroid withdrawal for 2 weeks reduced insulin sensitivity in young healthy men with idiopathic hypogonadotropic hypogonadism, suggesting that T modulates insulin sensitivity directly and further suggesting that this pathway is not mediated by changes in body composition. On the other hand, Chen et al., [2006] argued that androgen deficiency is a consequence of, and is not a cause of, poor metabolic status. Thus, it is possible that such interplay is bidirectional. As shown in the diagram above important links exist between androgen deficiency and key components of MetS, especially obesity, hypertension, and dyslipidemia, together with glucose abnormalities (hyperglycemia) and insulin resistance [Mulligan et al., 2006].

Bidirectional Relationship Between Hypogonadism, T2DM, and IR.

- *Testosterone Deficiency, Insulin Resistance, and Type 2 Diabetes*

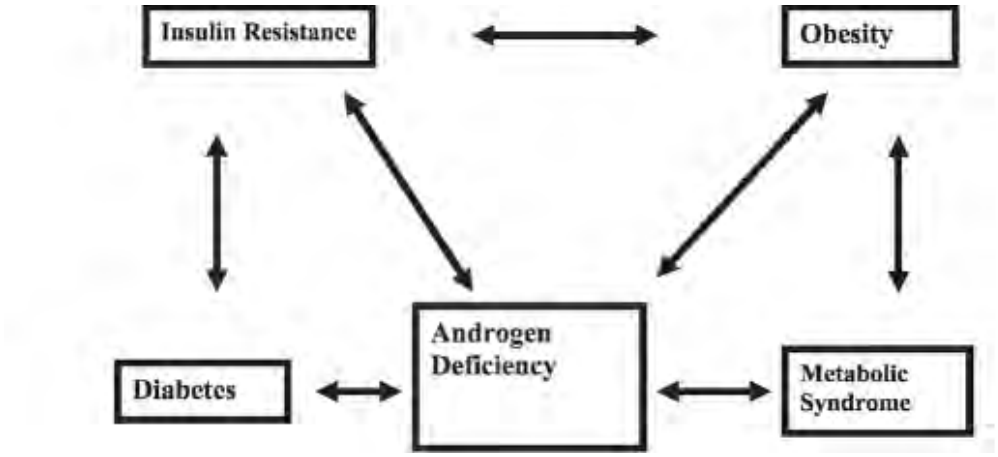

Figure 2. Putative Bidirectional relationship between T deficiency and Metabolic Syndrome, Obesity, Insulin resistance and Diabetes [Adopted from Traish et al., 2009].

Hypogonadism and T2DM are often diagnosed together in the same patient (Dhindsa et al., 2004; Kapoor et al., 2007). Hypogonadism was more prevalent in diabetic patients with increasing BMI, or in those who were severely obese. Dhindsa et al. (2004)

Cellular Mechanisms Linking Testosterone Deficiency to IR, Glycemic Control, and T2DM

T biosynthesis is regulated primarily by pulsatile secretion of luteinizing hormone (LH), and compelling evidence exists that Leydig cell steroidogenesis is further modulated locally by circulating hormones, growth factors, and cytokines (Saez 1994). Serum T levels reflect the integrity of the hypothalamic-pituitary-gonadal (HPG) axis, and low T levels noted in cases of IR may indicate a defect at one or more functional levels of the HPG axis.

In the insulin-resistance state Leydig cell function, particularly steroidogenesis, may be impaired by changes in the production of hormones and cytokines locally in the target tissue and in adipose tissue. Although several studies suggest that increasing IR may be attributed to a decrease in T secretion in men, it is not fully clear how the HPG axis mediates the interplay between T and insulin levels.

Pitteloud et al., [2005a, b, 2008] explored the effect of suppression of endogenous reproductive hormones, followed by sequential stimulation of the pituitary and the testes with GnRH and human chorionic gonadotropin (hCG), respectively. hCG-stimulated T levels at 48 hours were positively correlated with insulin sensitivity as well as with baseline serum T levels. The authors suggested that alteration in Leydig cell function may account in part for the mechanisms by which a decrease in T leads to IR.

Several observations have suggested that a relationship exists between insulin/glucose and LH/follicle-stimulating hormone (FSH) levels. Diabetic men often have reduced serum levels of FSH, LH, prolactin, and growth hormone, consistent with secondary hypogonadism [Hutson et al., 1983; Benitez and Perez Diaz, 1985].

In this current review, Dr. Hayes indicated that a positive correlation exists between serum T levels and IS in men across the full spectrum of glucose tolerance [Pitteloud et al. 2005a,b]. This is not surprising since T modulates glucose uptake, utilization and disposal [Roa et al., 2013; Kelly and Jones, 2013; Tishova Y et al., 2024].

T regulates the expression of GLUT4, insulin receptor, insulin receptor substrate 1 and 2 and increases the expression of AKT and PKC and phosphofructokinase. T also increased the expression of Hexokinase and ubiquitin Cytochrome C reductase binding protein (UQRCB) and increases glycogen synthase and glucose 6 phosphate dehydrogenase and reduces the expression of glycogen phosphorylase [Kelly and Jones, 2013]. T increases cellular responsiveness to insulin and modulating GLUT4 expression and membrane transport resulting in increased glucose uptake and utilization [Dhindsa S, et al., 2016, Roa et al., 2013].

Dr. Hayes' group dissected the relationship between T and IR in men by performing a detailed hormonal and metabolic evaluation in 60 men with a spectrum of glucose tolerance. IS was measured using a hyperinsulinemic-euglycemic clamp. Mitochondrial function was assessed by measuring maximal aerobic capacity (VO2 max) and expression of oxidative phosphorylation genes in skeletal muscle.

Dr. Hayes' group demonstrated a positive correlation between serum T levels and IS in men across the full spectrum of glucose tolerance. Men with T deficiency were twice as insulin resistant as eugonadal controls. The study also showed a positive correlation between physiological markers of mitochondrial function such as maximal aerobic capacity (VO2 max;) as well as genes involved in oxidative phosphorylation (UQCRB,). These data indicate that low serum T levels are associated with an adverse metabolic profile and suggest a novel unifying mechanism for the previously independent observations that low T levels and impaired mitochondrial function promote IR in men. Dr. Hayes' group and others have shown that T levels are significantly reduced in men with impaired glucose tolerance and T2DM than in normoglycemic controls (Pitteloud et al., 2005a,b; 2008).

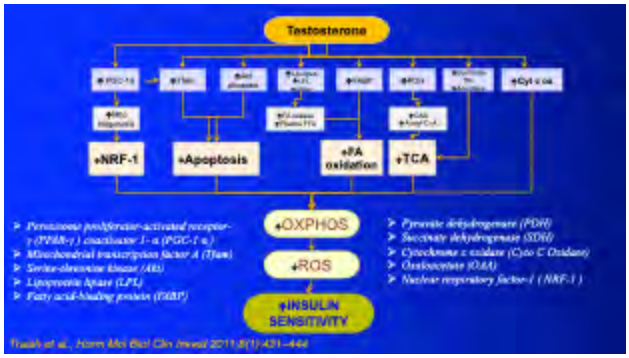

Figure 3. Testosterone modulates mitochondrial metabolism and regulates sugar and fat metabolism [Traish et al., 2011].

In this review, Dr. Hayes shed light on a very critical scientific and clinical question pertaining to the pathophysiology of insulin resistance (IR) in relation to reduced circulating testosterone (T) levels. Dr. Hayes acknowledges that "low T levels predict type 2 diabetes mellitus (T2DM) in men." Dr. Hayes sought to dissect the bidirectional physiological relationship between T and insulin sensitivity (IS) in men.

Men with T deficiency exhibit greater insulin resistant (IR) compared to men with normal T levels. Using functional and genetic studies, Dr. Hayes group have provided evidence that T deficiency may induce IR by eliciting mitochondrial dysfunction. As shown in Figure 3, a host of metabolic mitochondrial enzymes are regulated by androgens. It is not surprising that sugar metabolism and insulin sensitivity are regulated by T levels [Traish et al., 2011]

Interestingly however, in a prospective population study of a random population sample of 1400 men, aged 30–74, Ottarsdottir et al., 2018 investigated whether a bidirectional association between T concentrations and IR exists. Low concentrations of total T at baseline were significantly associated with high log HOMA-IR. Similar results were observed for bioavailable T. Men within the lowest quartile of total T at baseline had significantly higher log HOMA-IR. Multivariable analysis of the impact of IR at baseline on T levels at follow-up revealed no significant associations regarding T concentrations Low T concentrations at baseline predicted higher IR at follow-up, but high IR at baseline did not predict low T at follow-up. The authors noted a robust association between low T concentrations and IR, both cross-sectionally and longitudinally. However, in this study the relationship did not appear to be bidirectional. Indeed, low T independently predicted IR, however, no significant impact of IR on T levels was recorded during the follow-up time.

It should be noted that the T4DM trial had suggested that T therapy may prevent progression from pre-diabetes to diabetes and may result in remission of diabetes in some patients [Wittert et al., 2021]. These findings are supported by data from registry studies [Haider et al., 2020 and Yassin et al., 2019]

Indeed, weight loss contributes to increased T levels and reverse T deficiency in over 50% of obese men with impaired glucose tolerance and increased body fat mass and body weight results in reduced T levels [Reddy KC et al., 2021]. Dr. Hayes used this observation to conclude that the relationship between T and IR in men is bidirectional. It is my view that this relationship is more complex and merits thorough investigation.

Instead of providing more specific physiological and biochemical pathways to illustrate the nature of this relationship, Dr. Hayes pivoted to discuss direct-to-consumer advertising, development of more patient-friendly T formulations, and an expansion of the clinical indications for T replacement as well as the expansion of the T market to 1.6 billion Dollars.

Also, the discussion pertaining to the Endocrine Society guidelines is irrelevant to the bidirectionality of the relationship between T and IR and provides no real information that may explain this relationship.

None of these issues add any relevant information or shed any light on the physiological bidirectional relationship between T levels and IR. It would have been more appropriate to provide scientific bases for this relationship than addressing increased T prescriptions, patient preference to various formulations or market size.

Dr. Hayes provided what she deemed as evidence from 4 of the largest T trials and concluded that T does have positive effects [Lincoff et al., 2023; Snyder et al., 2016, Wittert et al., 2021; Basaria et al., 2015]. However, her understanding of the findings on T and prostate cancer seems to be overshadowed by other biases. For Dr. Hayes to state that the studies on prostate cancer were not adequately powered in the Traverse Trials and/or of insufficient duration to rule out an effect, is at best naïve if not born by an old belief that T is dangerous for the prostate.

If one examines the data in the four largest clinical trials and express the number of prostate cancer incidence in this large population of patients, you will quickly realize that the incidences of prostate cancer are negligible in all 4 trials. As to the duration effect, if her premise is true, then men in their 50s and 60s should be afflicted with prostate cancer since they have been exposed to high levels of T in their 20s, 30s, 40s and 50s, however, this is not the case. Also, in the last two decades T therapy has increased by several fold in the USA in the meantime there is no increase in prostate cancer incidence in the USA. Therefore, this argument is ill founded at best

In sum, the complex bidirectional relationship between T and IR may be true as suggested by Dr. Hayes. However, more pertinent physiological and biochemical information are needed to substantiate this relationship.

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Fink J, Bentzen K, and Horie S.

This narrative review focused specifically on highlighting the relationship between endogenous, exogenous testosterone (T) and the novel methods which may be used to treat hypogonadism while minimizing side effects related to increased hematocrit (HCT)levels. Increased hematocrit levels in response to T therapy remains a controversial and hotly debated topic which merits further discussion and debate. While several studies advocated non-inferior effects of T as compared to placebo other studies, however, point to an adverse side effect on cardiovascular health possibly mediated through HCT elevation with TTh. Indeed, the jury are still out on this topic, simply the contemporary clinical literature offered no conclusive evidence that increases in hematocrit produced by T therapy indeed result in major cardiovascular events (MACE).

Below is a brief synapse of each of the key issues addressed.

A. Endogenous T levels and hematocrit

Shin et al 2016, reported on 1200 patients with varying total T levels. Those with low total T levels (<235ng/dL) were significantly correlated with low hemoglobin and hematocrit levels (P<0.001). However, no statistical differences were noted in patients with normal total T (235-996 ng/dL) or high total T (>996ng/dL) levels and hemoglobin and hematocrit levels [Shin et al., 2016].

Interestingly however, Rich JM et al., 2022 reported on findings from 3673 men. Those men who had T levels > 800ng/dL (146; 4%), tended to have elevated hematocrit levels when compared with those men having normal T levels. However, there was a correlation between endogenous T and hematocrit, with a mean difference of 1.28%.

In study of 1273 patients, Paller CJ et al., showed that those men with low free T levels had lower hematocrit levels as compared with patients with normal free T levels. Endogenous total T levels did not increase hematocrit levels above normal range. The observation that free T levels correlated better with hematocrit levels when compared with total T levels was also reported by Bhatia V et al., 2006.

B. Exogenous T levels and hematocrit and hemoglobin

In a study of more than 10,000 patients Kohn T P et al., 2024 noted that that rise in hematocrit from base line are correlated with increased risk of MACE, as compared with patients having a stable hematocrit level while being treated with T.

Similarly, Ory et al., 2022 suggested that patients who developed polycythemia had increased risk of MACE/VTE as compared with patients with normal hematocrit levels. These findings suggest that T therapy is an independent risk factor for MACE and VTE during the first year of therapy. It should be noted that the increases in hemoglobin and hematocrit are significantly greater in older than younger men, and the increase is mostly in the first 48 months of

younger men. and the increase is mostly in the first 48 months of the intervention [Caviello AD et al., 2008; Strange RC et al., 2021]

C. Differences Among T Preparations in Raising HCT

Depending on the T formulations and route of administration T therapy may lead to significant increases in hematocrit and potential cardiovascular incidents. On the other hand, for hypogonadal patients with anemia, T therapy is beneficial not only for restoring healthy T levels but also red blood cells. A recent study [Diaz et al., 2022] showed that T cypionate leads to polycythemia (HCT>52%) after 4 months of treatment in 5.5% of the participants. Hematocrit levels increased by 5.1%.25.

It appears that subcutaneous administration of T cypionate leads to a reduced rate of erythrocytosis as compared with intramuscular injection therapy [Williamson et al., 2022]. In a study comparing hematocrit changes between 12 weeks of intra-muscular T (100mg/week) and subcutaneous injections of T enanthate (100mg/week), the subcutaneous injection group showed lower post-therapy hematocrit levels as compared to the intramuscular injection group [Choi et al 2022]

A retrospective cross-sectional study [Nolan et al., 2021] included data from 180 individuals who were on T therapy (intramuscular (TU n=125), intramuscular T enanthate (n =31), or transdermal T (n=24). One in four patients on intramuscular T enanthate and one in six on TU developed polycythemia during the intervention, whereas none of the patients on transdermal T showed signs of polycythemia. In a study of 95 hypogonadal men who received 225 mg of oral TU (Tlando) twice daily for 24 days the mean increase in HCT was 0.9±3.00% [DelConte A et al., 2022]. Another oral preparation Jatenzo appears to be will tolerated regarding HCT increases [Engel et al., 2023]. Smaller changes in HCT were noted in studies with T gel and higher changes in HCT were noted in patients receiving T pellets [Pastuszak AW et al., 2015, Grober ED et al., 2008].

D. Risk Factors for T Therapy-Induced Hematocrit Increases

One recent study evaluated the data of 566 patients who were treated ≥3-month with T therapy showed that the occurrence of T therapy induced hematocrit elevation was higher in patients with higher levels of hemoglobin and hematocrit pretreatment [Ryu et al., 2022]. Hematocrit levels ≥52 % were recorded in 33 patients (5.83%), occurring 16.7 months of T therapy. Patients in this group displayed statistically significantly higher pretreatment hemoglobin and hematocrit levels as compared with patients <52% (hemoglobin:14.48 vs.15.83, P<0.001; hematocrit:42.25 vs. 46.5, P<0.001).40 Pre-T Therapy hemoglobin and hematocrit levels contribute to hematocrit elevations, therefore it is very important to monitor these values during T therapy in patients with high pre-T therapy hemoglobin and hematocrit levels.

The Traverse Trial, a multicenter, randomized, double-blind, placebo-controlled, non-inferiority trial, enrolled over 5000 hypogonadal men ages 45 to 80 with preexisting or high risk of cardiovascular disease. The primary cardiovascular safety endpoint (first occurrence of any component of a composite of death from cardiovascular causes, nonfatal myocardial infarction, or nonfatal stroke) took place in 182 patients (7.0%) in the testosterone group and in 190 patients (7.3%) in the placebo group. This means that in men with hypogonadism and preexisting or a high risk of cardiovascular disease, T therapy is non-inferior as compared with placebo regarding cardiovascular events [Lincoff AM et al., 2023]

In Summary, T therapy may elicit varying levels of HCT depending on the T formulation and its route of administration. Whether the

increases in HCT in response to T therapy are correlated with potential cardiovascular incidences remain to be established. It should be noted that patients with anemia of known or unknow causes seem to benefit from T therapy. This contradiction in HCT levels in response to T therapy will need to be evaluated further.

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The Definitive Guide to Precision Hormone Therapy

"Principles of Precision Hormone Therapy: Healthy Aging and Prevention of Chronic Disease" has just been published, representing a collaborative masterpiece from **(9) of our distinguished ANDROGEN SOCIETY members**.

This isn't just another book on hormones – it's the resource our field has been waiting for.

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Current hormone therapy resources fall into two problematic categories: outdated academic textbooks that haven't kept pace with modern science, or popular books written by non-experts that lack scientific foundation. Our ANDROGEN SOCIETY contributors recognized this critical gap and took action.

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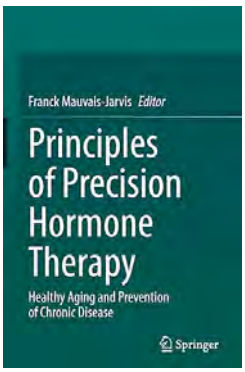

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We're incredibly proud of our ANDROGEN SOCIETY members who contributed their expertise to create this essential resource. Their collaborative effort represents the best of our community's commitment to advancing the science of healthy aging.

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Online Education

Did you miss last month's webinar – **Debate: Should Functional Hypogonadism Be Treated?**

We've recorded it for you – go to link to watch!

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Annual Meeting Information

8th Annual Meeting of The Androgen Society

Mark your calendars!

Abstract submissions for the 8th Annual Meeting of The Androgen Society will open on **November 16, 2025**.

Save the Date: June 11-12, 2026 | Chicago, Illinois, USA

The 8th Annual 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.

Important Dates

Submission Opens: November 16, 2025
Submission Deadline: February 16, 2026
Notification Date: March 16, 2026

We encourage you to start preparing your abstracts now for submission when the portal opens next month. All presenters must plan to attend the conference in person.

Watch for the full Call for Abstracts announcement in mid-November with complete submission guidelines and the abstract submission portal link.

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

We look forward to receiving your submissions and welcoming you to Chicago in June 2026!

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

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Abdulmaged M. Traish, MBA, PhD

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Membership Co-Chair

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MEMBER SPOTLIGHT

Congratulations to Our Award Winners from the 2025 Annual Meeting

Dedication to Education Award: Dr. Adrian Dobs

Presented by Sandeep Dhindsa

The Androgen Society is pleased to announce Dr. Adrian Dobs as the recipient of the Dedication to Education Award. Dr. Dobs is a professor of medicine and oncology at Johns Hopkins University School of Medicine and serves as director of the Johns Hopkins Clinical Research Network.

Dr. Dobs's journey began in Albany, New York, followed by an Internal Medicine residency at Albert Einstein at Montefiore Hospital and an Endocrine Fellowship at Johns Hopkins. With over 300 peer-reviewed publications, 40 book chapters, and numerous grants from the NIH, industry, and foundations, Dr. Dobs is a leader in medical education and research.

Her accomplishments include being the first to identify and report on male hypogonadism in HIV disease, leading the development of new testosterone formulations, and directing a major clinical research partnership in the Mid-Atlantic States. Dr. Dobs is a fixture at medical education events worldwide, consistently sharing her expertise and advancing the field.

Please join us in congratulating Dr. Adrian Dobs on receiving the Dedication to Education Award for her outstanding commitment to research, education, and clinical excellence.

Dr. Adrian Dobs

Lifetime Achievement Award: Dr. Irwin Goldstein

Presented by Abdul Traish

We are delighted to present Dr. Irwin Goldstein with the Lifetime Achievement Award, recognizing his extraordinary impact on sexual medicine and erectile physiology. Dr. Goldstein is a creative and innovative clinician scientist who consistently pushes the boundaries of research and clinical practice.

Dr. Goldstein's career includes professorships at Boston University and the University of California, San Diego, as well as directorships at the Institute of Sexual Medicine at both institutions. He has served as Editor-in-Chief of the Journal of Sexual Medicine and Sexual Medicine Reviews and has led both the North American Society of Sexual Medicine and the International Society for the Study of Women's Sexual Health.

A pioneer in the field, Dr. Goldstein was the lead author of the seminal Viagra paper in the New England Journal of Medicine, revolutionizing sexual medicine. He has a huge commitment to mentorship, welcoming trainees from around the world and guiding many to successful careers. Dr. Goldstein's dedication extends beyond science—he is a devoted father and husband, always making time for his family.

Having known Dr. Goldstein for over five decades, I can attest to his tireless pursuit of knowledge and his generosity as a mentor. He has lectured globally, delivering insights to audiences in Latin America, Australia, Asia, Europe, and the US. For his unparalleled contributions to clinical and sexual medicine, we are honored to recognize Dr. Irwin Goldstein with the Lifetime Achievement Award.

Dr. Irwin Goldstein

Lifetime Achievement Award: Dr. Basin

Presented by Abe Morgentaler, MD

The Androgen Society is proud to announce Dr. Shalender Bhasin as the recipient of our 2025 Lifetime Achievement Award. Dr. Bhasin's contributions to the field of testosterone research are unparalleled; so much of our discussions on the benefits and risks of testosterone are based on randomized clinical trials directed by Dr. Bhasin.

Born in Yamuna Nagar, India, Dr. Bhasin earned his medical degree from the All India Institute of Medical Sciences, and he completed his fellowship in endocrinology and metabolism at UCLA under Dr. Ron Swerdloff, a previous Lifetime Achievement Award winner. Today, Dr. Bhasin serves as Professor of Medicine at Brigham and Women's Hospital and Harvard Medical School.

With over 500 publications and an astonishing h-index of 101, Dr. Bhasin's influence spans both basic science and clinical trials. He is widely recognized for the clinical trials that he's generated and participated in and his leadership as the lead author of the Endocrine Society Guidelines since 2006. Among his many accomplishments, Dr. Bhasin is most proud of his large randomized controlled trials establishing the safety and efficacy of testosterone therapy, his research into the mechanisms by which testosterone increases muscle mass and erythropoiesis, and his work elucidating the binding of testosterone and estradiol to SHBG and albumin.

On a personal note, Dr. Bhasin is known for his graciousness, generosity, and humility. His voluminous body of work has contributed enormously to the field of testosterone therapy being on an equally solid scientific foundation as any other clinical entity. For these reasons, we honor him as the Androgen Society's 2025 Lifetime Achievement Award recipient.

Dr. Shalender Basin

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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.

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Industry News

Besins Healthcare is Ready for Movember

Did you know that Movember began in 2003 with a few friends enjoying a drink near Melbourne?

Since then, it's become a global movement supporting men's health: from prostate and testicular cancer to mental health and suicide prevention.

But there's another important health topic many don't talk about – low testosterone.

Low testosterone can't be seen, but it can be felt through low energy, mood changes or decreased libido.[1] It's something many men experience, especially as they age.[2]

If you or someone close to you has noticed these symptoms, low testosterone could be a factor.

Drop your thoughts or questions below so that we can all show our support during Movember!

#BesinsHealthcare #Movember #MensHealth #LowTestosterone #MensWellbeing

1. TraishAM, Miner MM, Morgentaler A, et al. Testosterone Deficiency. Am J Med. 2011;124(7):578-87.
2. TsametisCP, IsidoriAM. Testosterone replacement therapy: For whom, when and how? Male Reproductive Endocrinology. 2018;86:69-78.

Grünenthal

Grünenthal has recently updated **Nebido.com**, a platform which brings together essential knowledge, powerful digital tools and the latest research to support clinical decision-making and patient education. Whether you need quick access to Nebido dosing guidance or in-depth clinical data, **Nebido.com** delivers clarity, confidence, and convenience – all in one place. The link to the platform can be found **here**.

Marius Pharmaceuticals

Marius Pharmaceuticals is conducting a clinical study led by Dr. Mohit Khera evaluating the impact of their FDA-approved oral testosterone undecanoate capsule on fertility outcomes. This study explores the maintenance of the hypothalamic-pituitary-gonadal (HPG) axis, a key factor in preserving sperm production and testicular function among men receiving testosterone therapy.

- **Marius Pharmaceuticals Website**
- **Marius Pharma Announces Pilot Study with Dr. Mohit Khera**

Please contact Marius Pharmaceuticals with any questions at **info@mariuspharma.com**.

TackleTD.com is a website designed by Grünenthal for patients and the public to raise awareness of testosterone deficiency and aims to provide men who may have the condition with the information and resources they require to seek the help they need. The link to the platform can be found **here**.

Verity

If you're interested in learning more about oral testosterone therapy, please visit the following webpage **to review a White Paper.**

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WE WANT TO HEAR FROM YOU!

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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.

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