WHY TESTOSTERONE IS BEST



powered by

born brown.

stay strong. stay brown.

INDEX

Chapters

1. Where Did Exogenous Testosterone Come From?
2. When Did Bodybuilders Start Using Testosterone?
3. Why Is Testosterone Used as a Base?
4. What Can Testosterone Alone Actually Do?
5. Why Is This Relevant for Indian Men?
6. Testosterone and Bloodwork
7. How Long Do People Stay on Testosterone?
8. What If You Want Children?
9. What Dose Do You Start With and How Do You Increase It?
10. TRT Versus a Bodybuilding Cycle
11. Want to Talk About Your Own Situation?
12. References

INTRODUCTION

Why Testosterone?

When people first start researching PEDs, they are immediately confronted with dozens of compounds, different esters, complicated cycles and people arguing online about what is "best." But almost every discussion eventually comes back to testosterone.

There is a reason for that. Testosterone is not some new bodybuilding drug that appeared in the last few decades. We have roughly 90 years of scientific and medical experience with exogenous testosterone, its pharmacology is extensively studied, and once an injectable testosterone ester is cleaved, the active hormone is testosterone itself, the principal androgen naturally produced by the male body.

So before talking about stacking multiple compounds, I think it makes sense to understand testosterone properly first.

1. Where Did Exogenous Testosterone Come From?

Testosterone's story goes back much further than bodybuilding. By the late nineteenth and early twentieth centuries, researchers already understood that the testes produced substances responsible for male sexual development and function, but the hormone itself had not yet been isolated.

The major breakthrough came in 1935. Ernst Laqueur and his team in Amsterdam isolated testosterone, while Adolf Butenandt and Leopold Ruzicka, working with their respective teams, succeeded in synthesising it during the same year. These discoveries established testosterone as a defined hormone that could be produced and developed for clinical use (Nieschlag & Nieschlag, 2019).

This was not bodybuilding research. Scientists and the emerging pharmaceutical industry were trying to identify the male sex hormone and develop effective treatments for androgen deficiency. Unmodified testosterone is rapidly inactivated when taken orally because of hepatic metabolism, which encouraged the development of injectable esters and other preparations capable of producing useful circulating testosterone concentrations (Nieschlag & Nieschlag, 2019).

That history matters. Testosterone has been available for clinical use since the 1930s, giving us a depth of medical and scientific experience that simply does not exist for many newer performance enhancing drugs.

2. When Did Bodybuilders Start Using Testosterone?

The transition from medicine into sport came later. Historical research indicates that anabolic androgenic steroids spread rapidly through elite athletics and bodybuilding from the 1950s through the 1970s, before widespread non athlete use became more prominent during the 1980s (Kanayama & Pope, 2018).

This distinction is worth making because a lot of early steroid history has become bodybuilding folklore. Stories about particular coaches, athletes and national teams are often repeated as established fact even when the historical documentation is less certain.

What we can say with confidence is that by the middle of the twentieth century athletes had recognised the powerful anabolic effects of androgens, and testosterone became part of the developing pharmacological culture of strength sports and bodybuilding. By the time bodybuilding exploded in popularity through the 1960s and 1970s, anabolic steroid use was already established within parts of the sport.

3. Why Is Testosterone Used as a Base?

This is probably the most important concept in the article. When you introduce suppressive anabolic steroids, your body does not simply continue producing its normal amount of testosterone alongside them.

Exogenous androgens suppress the hypothalamic pituitary gonadal axis. LH and FSH fall, and testicular testosterone production can become profoundly suppressed. Prospective research in anabolic steroid users has demonstrated this suppression during androgen use, along with marked suppression of spermatogenesis (Smit et al., 2021).

This is where the bodybuilding term "test base" comes from. Testosterone provides the fundamental androgen that the body would otherwise normally produce itself. It also gives rise to two physiologically important metabolites: estradiol through aromatisation and dihydrotestosterone, or DHT, through 5 alpha reduction.

This is one reason testosterone occupies a unique position compared with compounds such as Masteron, Primobolan, nandrolone or trenbolone. Those compounds can activate androgen receptors, but they do not simply reproduce the full physiological role of testosterone and its metabolites. This is why testosterone commonly remains the hormonal foundation when other anabolic compounds are introduced.

4. What Can Testosterone Alone Actually Do?

Quite a lot. One of the clearest controlled demonstrations comes from Shalender Bhasin and colleagues. In a 20 week study, 61 healthy men aged 18 to 35 had endogenous testosterone secretion suppressed with a GnRH agonist and were then assigned to receive 25, 50, 125, 300 or 600 milligrams of testosterone enanthate per week (Bhasin et al., 2001).

The results demonstrated a clear dose response relationship. Fat free mass increased by approximately 3.4 kilograms in the 125 milligram group, 5.2 kilograms in the 300 milligram group and 7.9 kilograms in the 600 milligram group. Leg strength, power, thigh and quadriceps muscle volume, haemoglobin and IGF 1 also increased in relation to testosterone exposure, while HDL cholesterol moved in the opposite direction as exposure increased (Bhasin et al., 2001).

A related analysis of the same experimental programme showed that testosterone induced increases in muscle size were associated with hypertrophy of both type I and type II muscle fibres (Sinha-Hikim et al., 2002).

These studies do not mean every bodybuilder using testosterone will gain those exact amounts. The subjects were studied under controlled conditions and their endogenous testosterone had been deliberately suppressed. But the research gives us something much stronger than gym anecdotes: direct evidence that testosterone itself has a substantial and dose dependent anabolic effect.

For bodybuilding, the injectable esters most commonly encountered include testosterone enanthate, testosterone cypionate and testosterone propionate. The ester primarily changes the release characteristics and duration of the preparation. Once the ester is cleaved, the active hormone is testosterone. Enanthate and cypionate are longer acting preparations, while propionate is shorter acting.

The practical point is simple: you do not necessarily need three or four anabolic compounds to produce substantial changes in muscle mass and strength. Testosterone itself is a very effective anabolic agent.

5. Why Is This Relevant for Indian Men?

This is something I specifically wanted to include because a large proportion of the people reading my content are Indian.

There is research examining testosterone deficiency in Indian men, although the available data are not strong enough to claim that Indian men inherently have lower testosterone simply because they are Indian.

A pilot study from King George's Medical University in Lucknow examined 157 working Indian men aged 40 to 60. Of the 106 men who screened positive for symptoms associated with testosterone deficiency, 41 had low free testosterone and 32 had low total testosterone. The authors reported symptomatic biochemical hypogonadism in 26.1% of the study population. This was a relatively small occupational study rather than a nationally representative survey, so that figure should not be treated as the prevalence of testosterone deficiency across all Indian men (Goel et al., 2009).

The more interesting question is what has been happening to testosterone levels across male populations more broadly.

Are Men's Testosterone Levels Actually Declining?

There is evidence from several populations suggesting that age matched men studied in more recent periods have sometimes had lower testosterone concentrations than men of the same age studied previously.

The Massachusetts Male Aging Study found a substantial age independent population level decline in testosterone across measurements taken from the late 1980s through 2004. The investigators reported that the decline was not explained by the measured changes in obesity, smoking and other health and lifestyle characteristics (Travison et al., 2007).

Danish population research involving 5,350 male serum samples also found lower total testosterone in more recently born or studied age matched men. However, this is where the detail matters: after adjustment for the concurrent increase in BMI, the cohort and period related changes in total testosterone were no longer significant, and there was no significant age independent trend in calculated free testosterone (Andersson et al., 2007).

Finnish data provide another important piece of the picture. Researchers analysed 3,271 men from population surveys conducted in 1972, 1977 and 2002 and found lower total and free testosterone in more recently born age matched men. Among men aged 60 to

69, mean total testosterone fell from 21.9 nmol/L in the earlier birth cohort to 13.8 nmol/L in the more recent cohort, and the cohort effect remained significant after adjustment for BMI (Perheentupa et al., 2013).

So I would not repeat the common internet claim that men today automatically have half the testosterone their grandfathers had. The evidence is more complicated than that. Different populations have produced different findings, and factors such as obesity clearly explain part of the change in some datasets.

Modern lifestyle factors are still relevant. A 2026 review examined sleep deprivation and ultra processed food consumption in relation to testosterone and male reproductive health and concluded that both are plausible contributors to impaired hormonal health, while also making clear that the underlying evidence varies in strength and that these relationships are complex (Schimenes et al., 2026).

For Indian men, the message is not that being Indian means you need testosterone. It is that metabolic health, obesity, sleep, diet and physical activity matter, and the only way to know your own testosterone status is to measure it properly rather than making assumptions from age or symptoms.

6. Testosterone and Bloodwork

This ties directly into my previous article because I do not believe anyone should start using PEDs without establishing proper baseline bloodwork first.

Before testosterone, I want to know what the body looks like naturally. That means establishing total testosterone, free or calculated free testosterone, SHBG, estradiol, LH, FSH and prolactin, alongside a full blood count, haemoglobin, haematocrit, total cholesterol, LDL, HDL, triglycerides, ApoB, liver markers, kidney markers including Cystatin C and urine albumin to creatinine ratio, fasting glucose and HbA1c. Blood pressure should also already be established.

Once testosterone is introduced, several of those numbers can change. Haemoglobin and haematocrit can rise. Estradiol can increase through aromatisation. HDL can fall as androgen exposure rises. LH and FSH are expected to become suppressed.

The Endocrine Society's clinical guideline recognises erythrocytosis as an important effect of testosterone therapy and recommends monitoring testosterone and haematocrit during treatment, alongside appropriate clinical monitoring (Bhasin et al., 2018).

For PED users, the same basic monitoring principle is useful even though bodybuilding use is not the same as medically prescribed TRT: establish the comprehensive baseline, repeat the relevant testing once testosterone has been in the system long enough to assess your response, and continue monitoring if use continues. Do not wait until something feels wrong.

7. How Long Do People Stay on Testosterone?

This is where medical testosterone replacement and bodybuilding use become very different conversations.

Someone with clinically diagnosed hypogonadism who receives TRT may remain on testosterone long term because the objective is ongoing replacement of testosterone that the body is not producing adequately. TRT is therefore not designed as a short bodybuilding cycle followed by recovery of natural production (Bhasin et al., 2018).

Bodybuilding culture developed a different pattern. Cycling means using anabolic steroids for a period and then discontinuing them, while continuous use or what bodybuilders often call "blast and cruise" keeps exogenous androgens in the system rather than allowing the hypothalamic pituitary gonadal axis to recover.

Real world research confirms that there is no single standard cycle length. In the prospective HAARLEM study of 100 male amateur athletes, the median AAS cycle lasted 13 weeks, but the range was enormous at 2 to 52 weeks (Smit et al., 2020). Research in gym based AAS users has also documented both cycling and continuous patterns of use (de Zeeuw et al., 2023).

That is important because there is no scientifically established bodybuilding rule saying everyone should run testosterone for exactly 10, 12, 16 or 20 weeks. Those numbers largely come from bodybuilding practice rather than clinical trials identifying an optimal cycle duration.

If someone drops from a higher bodybuilding exposure to a lower ongoing testosterone exposure, they may call that a cruise or a break, but physiologically they have not come off testosterone. Exogenous testosterone is still present and suppression of natural gonadotropin production continues.

Recovery after discontinuation also varies. In the HAARLEM cohort, testosterone concentrations normalised within three months after stopping androgen abuse in most subjects, while recovery of spermatogenesis took considerably longer, approximately one year on average (Smit et al., 2021). Those findings describe a particular cohort and should not be treated as a guaranteed recovery timetable for every user.

8. What If You Want Children?

This is something that should be considered before starting testosterone rather than after someone has already been suppressed for years and suddenly decides they want children.

Before testosterone, establish your natural LH and FSH. If future fertility genuinely matters to you, a baseline semen analysis gives you information that LH and FSH alone cannot provide because it directly assesses sperm concentration and other semen parameters.

Exogenous testosterone suppresses LH and FSH. This reduces intratesticular testosterone, which normally exists at concentrations far higher than serum testosterone and is important for normal spermatogenesis.

An integrated analysis of 1,549 men from 30 hormonal male contraception studies found that the median time for sperm concentration to recover to 20 million per millilitre was approximately 3.4 months after treatment stopped. The estimated probability of recovery was 67% by six months, 90% by 12 months and 96% by 16 months (Liu et al., 2006). These were controlled contraceptive studies, not long term bodybuilders using multiple anabolic steroids, so the numbers should not be treated as a guaranteed recovery timeline for PED users.

What About HCG?

HCG is relevant because it acts at the LH receptor in the testes. Coviello and colleagues studied healthy men whose gonadotropins were suppressed using testosterone enanthate. In the testosterone plus placebo group, intratesticular testosterone fell by 94%. Adding low dose HCG maintained intratesticular testosterone in a dose dependent manner despite suppression of LH and FSH (Coviello et al., 2005).

That is an important physiological finding and helps explain why HCG is discussed so often in relation to testicular function during testosterone exposure. But maintaining intratesticular testosterone is not the same thing as proving that fertility will be fully preserved in every individual.

If children are part of your future plans, baseline LH, FSH and semen analysis give you valuable information before natural production is suppressed. Sperm banking is another option to consider before prolonged androgen exposure if preserving fertility is particularly important.

9. What Dose Do You Start With and How Do You Increase It?

This is where medical evidence and bodybuilding convention need to be separated. There is no medically established "beginner bodybuilding testosterone dose." Clinical testosterone guidelines are designed to treat genuine hypogonadism and restore testosterone to an appropriate physiological range, not to prescribe supraphysiological testosterone for muscle growth (Bhasin et al., 2018).

What we do have is useful dose response research. In the Bhasin study, increasing weekly testosterone enanthate exposure from 25 through to 600 milligrams produced progressively greater increases in fat free mass, muscle size and strength, but also progressively greater changes in some physiological markers, including haemoglobin and HDL cholesterol (Bhasin et al., 2001).

That gives us an important principle: more testosterone can produce more anabolic effect, but more also changes more things physiologically. Rather than changing several variables at once, understanding how your own body responds to testosterone exposure and bloodwork changes is far more useful than blindly chasing a number.

What Should You Watch For?

Estradiol: Testosterone aromatises into estradiol. As testosterone exposure increases, estradiol can increase as well. Estradiol is not simply a bad hormone that men should automatically try to eliminate because it has important physiological roles. Bloodwork and symptoms need to be interpreted together rather than reacting blindly to a single number.

DHT and hair loss: Testosterone can be converted into DHT through 5 alpha reductase. Men who are genetically susceptible to androgenic alopecia may notice accelerated hair loss as androgen exposure increases.

Skin: Greater androgen activity can increase sebaceous gland activity and contribute to oilier skin and acne in susceptible individuals.

Haemoglobin and haematocrit: Testosterone stimulates erythropoiesis. Controlled dose response research has shown haemoglobin increasing in relation to testosterone exposure (Bhasin et al., 2001).

Lipids: Higher androgen exposure can adversely affect HDL and other cardiovascular risk markers, which is why I would never assess testosterone use simply by looking at a testosterone number on a pathology report.

Blood pressure: Monitor it regularly rather than assuming it is fine because you feel normal.

This is also why introducing several compounds simultaneously makes troubleshooting much harder. If testosterone changes and three other compounds are introduced at the same time, working out which variable caused a new symptom or bloodwork change becomes considerably more difficult.

10. TRT Versus a Bodybuilding Cycle

These two things are constantly confused.

TRT means testosterone replacement therapy. Its medical purpose is to treat men with genuine testosterone deficiency by restoring testosterone to an appropriate physiological range and improving symptoms associated with hypogonadism. The Endocrine Society recommends diagnosing hypogonadism in men who have relevant symptoms together with consistently and unequivocally low testosterone measurements and recommends aiming for testosterone concentrations in the mid normal range during treatment (Bhasin et al., 2018).

A bodybuilding cycle has a different objective. The purpose is not simply to correct a deficiency. It is to deliberately create androgen exposure above normal physiological production to increase muscle mass, strength, recovery or physique outcomes.

The Bhasin dose response study illustrates the distinction. Mean nadir testosterone concentrations were approximately 542 ng/dL in the 125 milligram group, 1,345 ng/dL in the 300 milligram group and 2,370 ng/dL in the 600 milligram group (Bhasin et al., 2001). Those figures do not establish universal TRT or bodybuilding doses because individual pharmacokinetics vary, but they demonstrate how progressively greater testosterone exposure can move circulating concentrations well beyond normal physiological replacement.

Replacement and enhancement are not the same thing. Calling a supraphysiological bodybuilding protocol "TRT" does not make it TRT.

11. Want to Talk About Your Own Situation?

If you are considering testosterone or other PEDs and want to understand your bloodwork, what you should be checking, how testosterone fits into a broader PED approach or what you should be considering before making that decision, reach out to me through the KG Physique website to book a consultation.

You can also follow me on Instagram, where I share more of my own bodybuilding experience, PED discussions, bloodwork and contest prep.

@kg.physique

@kg.physique

stay strong. stay brown.

© KG Physique. This article is proprietary content owned by KG Physique and may not be copied, reproduced, distributed, republished or used in whole or in part without prior written permission.

12. References

Each PubMed label below is clickable.

Andersson, A. M., Jensen, T. K., Juul, A., Petersen, J. H., Jørgensen, T., & Skakkebaek, N. E. (2007). Secular decline in male testosterone and sex hormone binding globulin serum levels in Danish population surveys. The Journal of Clinical Endocrinology & Metabolism, 92(12), 4696-4705. https://doi.org/10.1210/jc.2006-2633 PubMed

Bhasin, S., Brito, J. P., Cunningham, G. R., Hayes, F. J., Hodis, H. N., Matsumoto, A. M., Snyder, P. J., Swerdloff, R. S., Wu, F. C., & Yialamas, M. A. (2018). Testosterone therapy in men with hypogonadism: An Endocrine Society clinical practice guideline. The Journal of Clinical Endocrinology & Metabolism, 103(5), 1715-1744. https://doi.org/10.1210/jc.2018-00229 PubMed

Bhasin, S., Woodhouse, L., Casaburi, R., Singh, A. B., Bhasin, D., Berman, N., Chen, X., Yarasheski, K. E., Magliano, L., Dzekov, C., Dzekov, J., Bross, R., Phillips, J., Sinha-Hikim, I., Shen, R., & Storer, T. W. (2001). Testosterone dose-response relationships in healthy young men. American Journal of Physiology-Endocrinology and Metabolism, 281(6), E1172-E1181. https://doi.org/10.1152/ajpendo.2001.281.6.E1172 PubMed

Coviello, A. D., Matsumoto, A. M., Bremner, W. J., Herbst, K. L., Amory, J. K., Anawalt, B. D., Sutton, P. R., Wright, W. W., Brown, T. R., Yan, X., Zirkin, B. R., & Jarow, J. P. (2005). Low-dose human chorionic gonadotropin maintains intratesticular testosterone in normal men with testosterone-induced gonadotropin suppression. The Journal of Clinical Endocrinology & Metabolism, 90(5), 2595-2602. https://doi.org/10.1210/jc.2004-0802 PubMed

de Zeeuw, T. I., Brunt, T. M., van Amsterdam, J., & van den Brink, W. (2023). Anabolic androgenic steroid use patterns and steroid use disorders in a sample of male gym visitors. European Addiction Research, 29(2), 99-108. PubMed

Goel, A., Kumar, S., Natu, S. M., Dalela, D., Sinha, R. J., & Awasthi, S. (2009). A cross-sectional pilot study to determine the prevalence of testosterone deficiency syndrome in working population of Indian men. Indian Journal of Urology, 25(2), 190-194. https://doi.org/10.4103/0970-1591.52911 PubMed

Kanayama, G., & Pope, H. G., Jr. (2018). History and epidemiology of anabolic androgens in athletes and non-athletes. Molecular and Cellular Endocrinology, 464, 4-13. https://doi.org/10.1016/j.mce.2017.02.039 PubMed

Liu, P. Y., Swerdloff, R. S., Christenson, P. D., Handelsman, D. J., Wang, C., & Hormonal Male Contraception Summit Group. (2006). Rate, extent, and modifiers of spermatogenic recovery after hormonal male contraception: An integrated analysis. The Lancet, 367(9520), 1412-1420. https://doi.org/10.1016/S0140-6736(06)68614-5 PubMed

Nieschlag, E., & Nieschlag, S. (2019). Endocrine history: The history of discovery, synthesis and development of testosterone for clinical use. European Journal of Endocrinology, 180(6), R201-R212. https://doi.org/10.1530/EJE-19-0071 PubMed

Perheentupa, A., Mäkinen, J., Laatikainen, T., Vierula, M., Skakkebaek, N. E., Andersson, A. M., & Toppari, J. (2013). A cohort effect on serum testosterone levels in Finnish men. European Journal of Endocrinology, 168(2), 227-233. https://doi.org/10.1530/EJE-12-0288 PubMed

Schimenes, B. C., Alvarenga, T. A., Toricelli, M., Moyses-Oliveira, M., Vasco, M. B., Tufik, S., & Andersen, M. L. (2026). The 24/7 lifestyle and male hormonal health: How sleep deprivation and ultra-processed foods impact testosterone and reproduction. Reviews in Endocrine and Metabolic Disorders, 27(4), 731-746. https://doi.org/10.1007/s11154-026-10030-z PubMed

Sinha-Hikim, I., Artaza, J., Woodhouse, L., Gonzalez-Cadavid, N., Singh, A. B., Lee, M. I., Storer, T. W., Casaburi, R., Shen, R., & Bhasin, S. (2002). Testosterone-induced increase in muscle size in healthy young men is associated with muscle fiber hypertrophy. American Journal of Physiology-Endocrinology and Metabolism, 283(1), E154-E164. https://doi.org/10.1152/ajpendo.00502.2001 PubMed

Smit, D. L., de Hon, O., Venhuis, B. J., den Heijer, M., & de Ronde, W. (2020). Baseline characteristics of the HAARLEM study: 100 male amateur athletes using anabolic androgenic steroids. Scandinavian Journal of Medicine & Science in Sports, 30(3), 531-539. https://doi.org/10.1111/sms.13592 PubMed

Smit, D. L., Buijs, M. M., de Hon, O., den Heijer, M., & de Ronde, W. (2021). Positive and negative side effects of androgen abuse. The HAARLEM study: A one-year prospective cohort study in 100 men. Scandinavian Journal of Medicine & Science in Sports, 31(2), 427-438. https://doi.org/10.1111/sms.13843 PubMed

Smit, D. L., Buijs, M. M., de Hon, O., den Heijer, M., & de Ronde, W. (2021). Disruption and recovery of testicular function during and after androgen abuse: The HAARLEM study. Human Reproduction, 36(4), 880-890. PubMed

Travison, T. G., Araujo, A. B., O'Donnell, A. B., Kupelian, V., & McKinlay, J. B. (2007). A population-level decline in serum testosterone levels in American men. The Journal of Clinical Endocrinology & Metabolism, 92(1), 196-202. https://doi.org/10.1210/jc.2006-1375 PubMed