MASTERON

THE SCIENCE BEHIND BODYBUILDING'S

"HARDENING" STEROID



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INTRODUCTION

Drostanolone is better known in bodybuilding by one name, Masteron. Today, when most people hear that name they immediately think about contest preparation, getting harder and drier, reducing the amount of testosterone in a cycle, or using it alongside other compounds in the later stages of a cut. What often gets forgotten is that none of those things were the reason Drostanolone was originally developed.

Masteron began as a legitimate pharmaceutical during a period when researchers were modifying testosterone and dihydrotestosterone to create compounds with different therapeutic properties. Drostanolone eventually found a medical role in the treatment of advanced breast cancer in women, and for a period it was prescribed and studied as part of mainstream medicine before newer therapies gradually replaced it (Blackburn & Childs, 1959; Bennett et al., 1975).

That history is what makes Drostanolone interesting to me, because the way we talk about Masteron in bodybuilding today is almost completely removed from the reason the drug existed in the first place. In this article I want to look at that entire journey, from its origins and medical use to its adoption in bodybuilding, what it actually does, how it is used now, and which claims around it are supported by evidence rather than simply repeated because they have become part of bodybuilding folklore.

CHAPTER 1

WHAT IS DROSTANOLONE?

Drostanolone, historically also referred to as dromostanolone, is a synthetic anabolic androgenic steroid derived from dihydrotestosterone, or DHT. Chemically it is essentially 2 alpha methyl DHT, with a methyl group added at the 2 alpha position of the DHT molecule.

That small structural change is important because steroid chemistry is not simply a case of taking one hormone and making it stronger or weaker. Relatively small alterations to a steroid molecule can change the way it is metabolised and alter its anabolic, androgenic and other biological characteristics.

Like other anabolic androgenic steroids, Drostanolone produces much of its biological activity through the androgen receptor. Once an androgen binds to the receptor, the resulting complex influences gene transcription and contributes to the biological processes associated with androgen signalling.

The pharmaceutical form used historically was predominantly Drostanolone propionate, where a propionate ester was attached to the parent hormone to prolong its release following injection. Once the ester is removed, the active hormone is still Drostanolone. One of the characteristics that later became particularly important in bodybuilding is that Drostanolone is derived from the already 5 alpha reduced DHT structure and does not aromatise into estrogen. That is very different from testosterone, which can be converted into estradiol through the aromatase enzyme.

CHAPTER 2

THE HISTORY OF DROSTANOLONE

The history of Drostanolone begins in the 1950s, during a period when pharmaceutical companies and researchers were investigating a large number of modified androgens. Testosterone had already been isolated and synthesised during the 1930s, and the following decades saw considerable research into whether structural modifications could produce compounds with useful therapeutic effects.

Drostanolone emerged from that period of steroid research, and we know that it was already being investigated clinically by the late 1950s. In 1959, Blackburn and Childs published research examining 2 alpha methyl dihydrotestosterone in women with advanced breast cancer (Blackburn & Childs, 1959).

That date is important because it puts the history of Drostanolone into perspective. The compound was being investigated as a cancer treatment long before Masteron became associated with contest preparation, muscle hardness or the modern bodybuilding world.

Drostanolone propionate subsequently became available as a pharmaceutical and appeared internationally under several trade names, including Drolban and Masteril, with Masteron eventually becoming the name most familiar within bodybuilding. Drug records identify the early 1960s as the period in which Drostanolone propionate entered pharmaceutical use (NCATS Inxight Drugs, n.d.; NCI SEER*Rx, n.d.).

Historical medical literature from the following decades confirms that Drostanolone was not an obscure experimental compound that disappeared after its initial development. Researchers continued investigating it in women with advanced breast cancer throughout the 1960s and 1970s (Gaertner et al., 1968; Bennett et al., 1975; Chowdhury et al., 1976).

This is something worth remembering when discussing old anabolic steroids. Masteron did not begin as an underground bodybuilding product and it certainly was not designed to make somebody look harder before stepping on stage. It was a legitimate prescription pharmaceutical that was developed and investigated for a completely different purpose.

CHAPTER 3

DROSTANOLONE AS A MEDICINE

The principal medical use of Drostanolone propionate became the treatment of advanced breast cancer, particularly metastatic or inoperable disease in women. That sounds unusual from a modern perspective because we now associate androgens primarily with male hormone therapy and performance enhancement, but breast cancer treatment during the 1950s, 1960s and 1970s looked very different from the treatment options available today.

Modern endocrine therapies had not yet developed into what we have now, so physicians were investigating different ways of manipulating the hormonal environment that influenced hormone sensitive tumours. Androgens were one of those approaches, and Drostanolone attracted interest because researchers hoped it could produce useful antitumour activity while remaining clinically manageable.

The early work was followed by several clinical investigations. Bennett, Helman and Palmer examined Masteril in women with breast cancer between 1968 and 1972 and reported meaningful response rates across different menopausal groups (Bennett et al., 1975).

In the premenopausal group, 44 percent responded to Masteril compared with 22 percent undergoing oophorectomy. In the perimenopausal group, 34.5 percent responded to Masteril compared with 39 percent receiving nandrolone phenylpropionate, while in the postmenopausal group 38.5 percent responded to Masteril compared with 57 percent receiving estrogen therapy (Bennett et al., 1975).

Other researchers were investigating Drostanolone during the same period. Studies compared it with other hormonal treatments used for advanced breast cancer, including nandrolone and testolactone, showing that Drostanolone had genuine clinical activity rather than simply being a theoretical treatment (Rieche & Wolff, 1975; Chowdhury et al., 1976; Wolff & Rieche, 1978).

None of this means Drostanolone would be a sensible treatment for breast cancer today because oncology has moved on enormously. What it does establish is that Drostanolone was a genuine pharmaceutical hormone with measurable therapeutic activity in humans, and its original purpose had nothing to do with bodybuilding.

CHAPTER 4

FROM MEDICINE TO BODYBUILDING: THE 1970s AND 1980s

This part of the story is harder to document precisely because early bodybuilding drug use was rarely recorded in formal scientific literature. We know that anabolic steroids entered competitive sport during the middle of the twentieth century and that their use subsequently expanded through weightlifting, athletics and bodybuilding (Kanayama & Pope, 2018).

By the 1970s, anabolic steroids were firmly embedded within parts of competitive strength and physique sport, and by the 1980s their use was beginning to move beyond elite athletes into the broader gym population (Kanayama & Pope, 2018). Drostanolone was commercially available during this general period, which meant bodybuilders had potential access to pharmaceutical versions while the drug was still being used medically.

What I do not think we should do is pretend there is a reliable scientific record showing which famous 1970s bodybuilder used Masteron, exactly how much he used or what he combined it with, because that evidence simply does not exist. Much of early bodybuilding drug use was passed verbally from athlete to athlete, coach to athlete and gym to gym rather than being documented in medical journals.

What bodybuilders gradually learned through experience was that different steroids did not necessarily produce the same visual outcome, particularly when someone became extremely lean. Drostanolone had several characteristics that made it attractive because it provided anabolic and androgenic activity while not aromatising into estrogen.

As contest preparation became more sophisticated through the 1970s and particularly the 1980s, different compounds developed reputations for different purposes. Some became associated mainly with increasing bodyweight and strength, while Drostanolone increasingly became associated with contest preparation and the harder appearance bodybuilders wanted when they were already lean. That reputation eventually became far more famous than the original medical use of the drug.

CHAPTER 5

THE DECLINE OF PHARMACEUTICAL MASTERON

Drostanolone did not disappear from medicine because researchers suddenly discovered some unique catastrophic problem with the drug. The more straightforward explanation is that breast cancer treatment improved and newer endocrine therapies progressively replaced older androgen based approaches.

The androgenic nature of Drostanolone was always an obvious disadvantage when treating women because androgen exposure could produce virilising effects. As oncology developed more selective and effective options, there was progressively less reason to use strongly androgenic drugs for this purpose.

By the 1990s, Drostanolone had effectively disappeared from mainstream clinical practice. FDA records show that the approval for Drolban Injection was formally withdrawn in 1994 after the manufacturer indicated that the product was no longer being marketed and requested withdrawal of the application (U.S. Food and Drug Administration, 1994).

This is an important distinction because older PEDs are sometimes described online as though they were suddenly banned after medicine discovered they were extraordinarily dangerous. The reality is often much less dramatic. Drugs can disappear because better treatments become available, commercial demand disappears, or manufacturers simply stop producing them. In Drostanolone's case, medicine moved on, while bodybuilding did not.

CHAPTER 6

WHAT DROSTANOLONE ACTUALLY DOES

Drostanolone is an androgen receptor agonist, which means it binds to and activates the androgen receptor. Through androgen receptor signalling, anabolic androgenic steroids can influence protein metabolism and other processes involved in maintaining or increasing lean tissue.

The straightforward pharmacology, however, is only part of the reason Masteron became popular because bodybuilders commonly describe something much more visual when they talk about it. The words you constantly hear are harder, drier, denser and more detailed.

There are no high quality controlled trials where competitive bodybuilders were given Drostanolone and researchers objectively measured changes in muscle hardness or visual dryness, so I think it is important to be clear that those descriptions come largely from practical bodybuilding experience rather than controlled clinical research.

There is still a pharmacological reason why part of that experience makes sense. Drostanolone does not aromatise and therefore does not directly create additional estradiol or estrogen mediated water retention in the way an aromatising androgen can. That does not mean Masteron somehow removes water from underneath the skin, and it certainly cannot reveal conditioning that is covered by body fat.

What it does mean is that increasing androgen exposure with Drostanolone is hormonally different from achieving the same increase simply by adding more testosterone. This is why the visual effect associated with Masteron tends to become much more relevant when somebody is already very lean, because relatively small changes in fullness, separation, vascularity and water balance become considerably easier to see.

CHAPTER 7

MASTERON PROPIONATE VS MASTERON ENANTHATE

The original pharmaceutical form of Drostanolone was Drostanolone propionate, and this is the ester that appears throughout the historical breast cancer literature (Bennett et al., 1975; Chowdhury et al., 1976). Modern bodybuilding also commonly uses Drostanolone enanthate, which is a longer ester version of the same parent hormone.

The important point is that the active hormone is still Drostanolone. The ester primarily changes how quickly the compound is released following injection and therefore influences the practical injection schedule rather than changing Drostanolone into an entirely different anabolic steroid. Propionate produces a shorter acting depot, while enanthate produces a longer release profile.

Drostanolone enanthate does not have the historical therapeutic evidence base that Drostanolone propionate has, so when we talk about the Drostanolone used medically in the twentieth century we are principally talking about Drostanolone propionate.

I also think it is worth being careful with the exact half life figures constantly quoted online because they are often repeated with far more confidence than the available human pharmacokinetic research justifies. The useful practical distinction is simply that propionate is the shorter ester and enanthate is the longer ester.

CHAPTER 8

WHY BODYBUILDERS USE MASTERON TODAY

Modern bodybuilding uses Masteron for a completely different purpose from the one for which Drostanolone was originally prescribed. The interest now is almost entirely physique related, and the compound has become most strongly associated with cutting phases and contest preparation rather than simply trying to add as much bodyweight as possible.

There are several reasons for this. Drostanolone does not aromatise, so adding it does not create another direct source of estrogen, while at the same time it provides additional androgenic and anabolic signalling without every additional milligram of androgen exposure needing to come from testosterone.

During a prolonged calorie deficit, maintaining muscle becomes increasingly difficult as body fat drops and recovery begins to suffer. Preserving anabolic signalling therefore becomes important, but a competitive bodybuilder is also concerned with much more than simply maintaining muscle because the final appearance on stage depends on fullness, separation, vascularity, hardness and overall presentation.

This is where Masteron's reputation comes from. Experienced competitors frequently report that the compound produces a harder and more detailed appearance when they are sufficiently lean, but I think expectations need to remain realistic because somebody carrying significant body fat is not going to add Masteron and suddenly look dry and stage ready.

Once body fat becomes extremely low and the skin is already thin, relatively small changes become much easier to see. That is why Masteron makes considerably more sense as a contest preparation or finishing compound than as something somebody adds at a much higher body fat percentage expecting a dramatic cosmetic transformation.

CHAPTER 9

MASTERON AND TESTOSTERONE: UNDERSTANDING THE RELATIONSHIP

One of the most misunderstood areas of modern Masteron use is the relationship between Drostanolone and testosterone. Both compounds activate the androgen receptor, but that does not make them interchangeable hormones because testosterone has physiological roles that extend beyond androgen receptor activation.

The biggest difference in this context is that testosterone can be converted by the aromatase enzyme into estradiol, whereas Drostanolone cannot aromatise. Estradiol is not simply a hormone that men should try to eliminate because it contributes to normal sexual function, bone physiology and several other biological processes.

For that reason, describing Masteron as a direct replacement for testosterone is too simplistic. What bodybuilders sometimes do instead is alter the relative contribution of aromatising and non aromatising androgens within the overall cycle. If testosterone exposure is reduced while Drostanolone exposure is increased, there may be less testosterone available for aromatisation even though total androgen exposure remains substantial.

That is completely different from saying that Masteron kills estrogen or somehow neutralises testosterone. The better way to understand it is that changing the relative amount of testosterone and Drostanolone changes the hormonal environment, which can influence estradiol exposure, side effects and ultimately the way an individual looks and feels.

CHAPTER 10

DOES MASTERON REALLY CONTROL ESTROGEN?

One of the most repeated claims about Masteron is that it acts like an aromatase inhibitor, but I do not think the evidence supports presenting that as fact. Drostanolone does not aromatise, which means Drostanolone itself cannot be converted into estradiol. That is completely different from directly inhibiting the aromatase enzyme.

Aromatase inhibitors work by interfering with the enzyme responsible for converting aromatisable androgens into estrogens. There is no good human evidence demonstrating that Drostanolone acts as a clinically meaningful aromatase inhibitor in men using testosterone.

The historical breast cancer research probably contributed heavily to Masteron's anti estrogen reputation, because if a drug was once used against breast cancer it is easy to assume that it must somehow directly block estrogen. The actual research is more complicated.

Trams examined Drostanolone propionate and its interaction with estradiol and DHT binding in normal and malignant tissues in 1977. The study found competition at androgen binding sites rather than estrogen receptors, and it does not provide evidence that Drostanolone functions as a conventional systemic aromatase inhibitor (Trams, 1977).

The most accurate way I would put it is that Masteron does not produce estrogen, but that does not mean we should automatically assume it reliably lowers serum estradiol being produced from testosterone. If you actually want to know what your estradiol is doing, measure it.

CHAPTER 11

MASTERON IN CONTEST PREPARATION

Contest preparation is where Drostanolone has built most of its modern identity because the priorities of a bodybuilder change dramatically once the objective becomes stepping on stage rather than simply gaining more muscle. Calories are restricted for an extended period, body fat becomes extremely low, cardio generally increases, training still needs to remain productive and recovery becomes progressively harder as the preparation continues.

At that point the objective is not simply to build more tissue. The goal becomes retaining as much muscle as possible while producing the best visual appearance on stage, which is where the characteristics of a non aromatising androgen such as Masteron become attractive.

Drostanolone can provide anabolic and androgenic activity without adding another direct source of estrogen, and bodybuilders have long associated it with the harder and more detailed appearance desired when body fat is already very low.

What I would not do is treat Masteron as a substitute for conditioning because no steroid can compensate for body fat covering muscle separation. The so called Masteron look is better understood as the interaction between very low body fat, developed muscle, glycogen, diet, water balance and androgen exposure. Masteron can contribute to that final appearance, but it is one part of the equation rather than the thing that creates conditioning by itself.

CHAPTER 12

SIDE EFFECTS AND HEALTH CONSIDERATIONS

The fact that Masteron does not aromatise does not mean it is free of side effects, it simply means the side effect profile differs from that of an aromatising compound. When I look at Masteron or any other AAS, I think it makes more sense to consider the entire androgen exposure and the overall cycle rather than pretending every change can be attributed to one compound in isolation.

Cardiovascular health and lipids

Cardiovascular health is one of the areas that deserves the most attention with prolonged AAS use. Research on AAS users has repeatedly identified changes including reduced HDL cholesterol, increased LDL cholesterol, higher blood pressure and adverse cardiovascular effects associated with long term exposure (Kanayama et al., 2010).

We do not have long term controlled trials that allow us to assign an exact cardiovascular risk percentage specifically to Drostanolone in competitive bodybuilders, so I would not pretend we can put an exact number on it. What we do know is that it belongs to a drug class where cardiovascular risk and deterioration in important cardiovascular markers are well documented.

Testosterone production and fertility

Exogenous anabolic steroids suppress the hypothalamic pituitary gonadal axis, which can reduce LH and FSH, lower intratesticular testosterone and impair sperm production. These reproductive consequences of AAS exposure are well established in the medical literature (Nieschlag et al., 2015). Being DHT derived does not make Masteron exempt from suppression, which becomes particularly important for somebody who intends to discontinue AAS completely or is concerned about fertility.

Acne and skin

The androgenic activity of Drostanolone can increase sebaceous gland activity and contribute to oily skin and acne in susceptible people. How strongly somebody experiences this varies considerably because genetics, total androgen exposure and the rest of the cycle all influence the outcome.

Liver

Masteron is not a 17 alpha alkylated oral anabolic steroid, so it should not automatically be placed in the same category as the classic hepatotoxicity associated with many oral steroids. That does not mean liver markers should be ignored, particularly when somebody is using several PEDs simultaneously, but directly equating injectable Drostanolone with a 17 alpha alkylated oral is pharmacologically inaccurate.

CHAPTER 13

MASTERON AND HAIR LOSS: IT IS NOT JUST ABOUT GENETICS

Hair loss gets oversimplified far too often in bodybuilding, particularly when someone starts shedding during a cycle and immediately assumes the entire problem is DHT or genetics. Genetics and androgen sensitivity are obviously important, especially with a DHT derived compound like Drostanolone, but they are only part of the picture because hair loss can be influenced by several different processes occurring at the same time.

The first thing to understand is that there are different types of hair loss. Androgenetic alopecia is the familiar male pattern recession and thinning around the temples, hairline and crown, whereas telogen effluvium generally presents as more diffuse shedding and can be triggered by physiological stress, illness, surgery, medications, rapid weight loss and nutritional deficiencies. A bodybuilder can potentially have both processes occurring at the same time, which is why every episode of shedding should not automatically be blamed on DHT alone (Durusu Turkoglu et al., 2024).

Androgens and genetic susceptibility

Drostanolone is a DHT derived androgen, so androgen signalling remains an obvious consideration when discussing Masteron and hair loss. Androgenetic alopecia is strongly influenced by genetic susceptibility and androgen receptor signalling within scalp follicles, with progressive follicular miniaturisation producing the typical pattern of recession and thinning (Trüeb, 2002).

This is also why finasteride needs to be understood properly. Finasteride reduces conversion of testosterone into DHT by inhibiting 5 alpha reductase, but Drostanolone is already based on a reduced DHT structure and does not require that conversion step before it can exert androgenic activity. Blocking 5 alpha reductase therefore does not simply stop Drostanolone from acting at susceptible scalp follicles.

Oxidative stress and inflammation

Hair follicle biology is more complicated than simply saying that DHT is high and therefore hair falls out. Oxidative stress and inflammatory signalling have also been investigated as contributors to follicular ageing and androgenetic alopecia, which means the local scalp environment and general health can influence what is happening alongside androgen exposure.

This does not mean taking antioxidants will reverse genetically driven hair loss, but it does mean that androgen signalling occurs within a broader biological environment involving inflammation, cellular stress and nutritional status. For bodybuilders this becomes relevant because hard training, prolonged dieting, poor sleep and the physiological stress of contest preparation can all exist simultaneously.

Rapid weight loss and aggressive dieting

Significant calorie restriction, rapid weight loss and nutritional deficiencies are recognised triggers for telogen effluvium. This matters in bodybuilding because shedding may become noticeable

weeks or months after the original physiological stress, so somebody can blame the compound they most recently added even though the hair cycle may have been disrupted earlier in the prep (Durusu Turkoglu et al., 2024).

Iron and ferritin

Iron status is one of the nutritional areas most commonly investigated in diffuse hair loss. Low ferritin can be associated with telogen effluvium and other forms of shedding, although the literature does not support one universal ferritin target for every person. The important point is to investigate iron status rather than automatically supplementing it, because excess iron is not harmless.

Vitamin D

Vitamin D repeatedly appears in the hair loss literature. A 2024 systematic review of micronutrients and androgenetic alopecia identified vitamin D among the nutrients potentially involved in hair growth and maintenance, while also showing that the evidence is not completely consistent across studies (Wang et al., 2024).

Vitamin B12 and folate

Vitamin B12 and folate are involved in DNA synthesis and normal cellular division, making them biologically relevant to rapidly dividing tissues such as the hair follicle. A large retrospective study of 3,028 patients with telogen effluvium found vitamin D deficiency in 72.2 percent of those screened, vitamin B12 deficiency in 30.7 percent, folate deficiency in 4.4 percent and thyroid dysfunction in 4.6 percent (Öner & Akdeniz, 2021). These figures do not prove that each deficiency caused the hair loss, but they show why nutritional and endocrine factors are worth investigating in diffuse shedding.

Zinc

Zinc is involved in cellular division, protein metabolism and normal follicular biology. A 2024 study of telogen effluvium reported lower zinc concentrations in affected patients than in controls, while the broader systematic review of androgenetic alopecia also identified zinc as one of the micronutrients potentially relevant to hair growth and maintenance (Durusu Turkoglu et al., 2024; Wang et al., 2024).

Selenium and copper

Selenium and copper are involved in normal cellular and antioxidant processes, but this is an area where balance matters more than simply increasing supplementation. In the 2024 telogen effluvium study, selenium was actually higher in affected patients while copper was not significantly different from controls, which is a useful reminder that blindly supplementing every micronutrient associated with hair is not sensible (Durusu Turkoglu et al., 2024).

Biotin

Biotin is probably one of the most heavily marketed hair supplements, yet genuine deficiency is uncommon. The same 2024 telogen effluvium study did not find a significant difference in serum or urinary biotin between affected patients and controls, so taking large doses without evidence of deficiency should not be treated as a universal solution (Durusu Turkoglu et al., 2024).

Vitamin A

Vitamin A is another good example of why more is not always better. Adequate vitamin A is necessary for normal physiology, but excessive vitamin A or retinoid exposure can itself contribute to hair shedding. This becomes relevant when somebody takes multiple supplements without checking the combined intake across all of them.

Vitamin K

Vitamin K is important for normal human physiology, but there is currently not strong clinical evidence showing that vitamin K deficiency is a common direct cause of hair loss. The major 2024 systematic review examining micronutrients and androgenetic alopecia looked at vitamin B, vitamin D, vitamin A, vitamin C, iron, selenium, zinc, manganese and copper rather than identifying vitamin K deficiency as an established driver (Wang et al., 2024). I therefore would not put vitamin K in the same category as iron, vitamin D, zinc or some B vitamins when investigating unexplained shedding.

Thyroid function

Both hypothyroidism and hyperthyroidism can disturb the normal hair cycle. In the study of more than 3,000 patients with telogen effluvium, thyroid dysfunction was identified in a proportion of patients, which is why thyroid testing can be worth considering when diffuse shedding does not have an obvious explanation (Öner & Akdeniz, 2021).

Stress, illness, scalp disease and medications

Telogen effluvium can follow major physiological stress, illness, surgery, inflammatory conditions and certain medications. Scalp conditions including seborrhoeic dermatitis, psoriasis, inflammatory disorders and fungal disease can also contribute to shedding or scalp irritation, while autoimmune conditions such as alopecia areata produce a different pattern of loss altogether.

How I would look at hair loss during a cycle

If someone starts noticing hair loss while using Masteron, I would not automatically blame one factor. I would first look at the pattern of hair loss because progressive recession around the temples and crown points more towards androgenetic alopecia, while sudden diffuse shedding across the scalp makes me think more about telogen effluvium and other systemic triggers.

From there I would look at total androgen exposure, family history, how aggressive the diet has been, recent weight loss, stress, sleep, recent illness, thyroid function and nutritional status, particularly iron and ferritin, vitamin D, vitamin B12, folate and zinc. Depending on the situation, copper and selenium may also be worth assessing.

The important point is that Masteron can accelerate androgen related hair loss in somebody who is susceptible, but not every case of shedding that occurs during a Masteron cycle is caused solely by Drostanolone or DHT. A bodybuilder can simultaneously have genetic susceptibility, high androgen exposure, aggressive dieting, poor recovery and a nutritional deficiency, which makes the real cause much more complicated than blaming one molecule.

CHAPTER 14

BLOODWORK: BEFORE, DURING AND AFTER A DROSTANOLONE CYCLE

One of the biggest mistakes I see with PED use is people waiting until something feels wrong before checking bloodwork. Bloodwork becomes considerably more useful when you know exactly where you started because a proper baseline allows you to compare how your individual markers change during the cycle rather than simply looking at one abnormal result and wondering whether it was already like that beforehand.

Clinical guidance written specifically for people using anabolic androgenic steroids supports monitoring areas including full blood count, lipids, liver function, renal markers, estradiol and blood pressure, with additional testing depending on the drugs being used and the individual's circumstances (Gibbons et al., 2024).

For a typical 12 to 14 week period, I would look at bloodwork at three main points. The first is before starting so you have a genuine baseline, the second is around week 6 so you can see what is changing while the cycle is underway, and the third is around week 12 to 14 so you can assess the cumulative effect.

Before starting: establish your baseline

Before introducing Drostanolone or making a major change to an existing PED protocol, I would want a baseline covering the major blood count, cardiovascular, liver, kidney, hormonal and metabolic markers that are likely to matter during the cycle.

Full Blood Count
Haemoglobin Haematocrit Red blood cell count White blood cell count Platelets

Lipid and Cardiovascular Markers
Total cholesterol LDL C HDL C Triglycerides Non HDL cholesterol ApoB ApoA1

Liver Markers
ALT AST GGT ALP

Bilirubin Albumin

Kidney Markers
Creatinine eGFR Urea Electrolytes

Hormonal Markers
Total testosterone SHBG Calculated or measured free testosterone Estradiol LH FSH Prolactin

Metabolic Markers
Fasting glucose HbA1c

Additional Markers
PSA where appropriate based on age, individual risk and clinical circumstances.

I would also include blood pressure as part of the baseline rather than treating it as something completely separate. Ideally, use several resting readings taken under similar conditions because one measurement after coffee, training, rushing to an appointment or sitting in traffic can look very different from normal resting blood pressure.

Around week 6: see how your body is responding

The six week blood test is where the baseline starts becoming genuinely useful because now you can compare what has changed rather than simply asking whether every number remains inside the laboratory reference range. At approximately week 6, I would repeat the main markers most likely to change during AAS exposure, particularly Full Blood Count with attention to haemoglobin and haematocrit, HDL C, LDL C, non HDL cholesterol, ApoB, ApoA1, ALT, AST, GGT, creatinine, eGFR, estradiol and blood pressure.

Testosterone and SHBG can also be useful depending on what else is being used and what question you are trying to answer. This comparison is much more useful than simply looking for red numbers because somebody can technically remain within the laboratory reference interval while their HDL has fallen substantially, ApoB has increased and haematocrit has climbed compared with baseline.

Week 12 to 14: assess the cumulative effect

By week 12 to 14, the purpose changes slightly because you are no longer looking at the initial response. You are looking at what sustained exposure has done over the entire period. At this point

I would again repeat the major panels so the results can be compared directly with both baseline and week 6, including Full Blood Count, haemoglobin, haematocrit, lipid profile, ApoB, ApoA1, ALT, AST, GGT, ALP, bilirubin, creatinine, eGFR, urea, electrolytes, fasting glucose, HbA1c, estradiol, testosterone, SHBG and blood pressure.

PSA can also be reassessed where clinically appropriate. If somebody intends to discontinue all suppressive AAS and assess recovery of natural testosterone production, LH, FSH and testosterone become more useful once enough time has passed for the exogenous hormones and their esters to clear rather than being interpreted immediately after the final injection.

The main thing I want people to take from this chapter is that bloodwork should be viewed as a trend across time. Baseline, week 6 and week 12 to 14 tell a story that one isolated blood test cannot. I want to know whether HDL is progressively falling, whether LDL or ApoB is increasing, whether haematocrit is climbing, whether blood pressure has changed, whether kidney or liver markers are moving in the wrong direction and what has happened to estradiol. That tells you considerably more about how your own body is responding than somebody else's bloodwork or cycle posted on social media.

CHAPTER 15

FINAL THOUGHTS: WHERE DROSTANOLONE FITS

Masteron is a good example of why I think understanding the history of a PED matters. If your only exposure to Drostanolone comes from bodybuilding forums, social media or gym conversations, it would be easy to believe that the compound was somehow developed specifically to make bodybuilders look harder and drier before a show, when in reality it came out of pharmaceutical steroid research and was being investigated as a treatment for advanced breast cancer by 1959 (Blackburn & Childs, 1959).

Clinical research continued investigating Drostanolone in breast cancer during the following decades, but as oncology progressed and more appropriate treatments became available, its medical usefulness gradually disappeared (Bennett et al., 1975; Chowdhury et al., 1976). Bodybuilding then gave the drug a completely different purpose, which is how Masteron survived long after it stopped being relevant to modern medicine.

The appeal within bodybuilding is not difficult to understand because Drostanolone is a DHT derived anabolic androgenic steroid that activates the androgen receptor, does not aromatise and can provide additional androgenic and anabolic activity without creating another direct source of estrogen. Experienced bodybuilders have also long associated it with the harder and more detailed appearance desired during contest preparation, although that visual reputation comes primarily from practical bodybuilding experience rather than controlled trials in competitive athletes.

Where I think people get carried away is when that practical experience becomes exaggerated into claims that Masteron is a fat burner or that it automatically controls estrogen like an aromatase inhibitor. It does neither. Getting lean is what reveals conditioning, and managing estradiol is considerably more complicated than simply adding Drostanolone.

Understanding the difference between what a drug actually does and what bodybuilding culture has come to say about it is one of the main reasons I wanted to write this article in the first place.

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CHAPTER 16

WANT TO TALK ABOUT YOUR OWN SITUATION?

PED use should not be approached as simply choosing compounds from a list because your goals, age, training history, previous PED exposure, cardiovascular risk, blood pressure, bloodwork, fertility considerations and individual response all matter. Two people can use the same compounds and see very different changes in bloodwork, side effects and overall results, which is why I prefer looking at the full picture rather than discussing a cycle in isolation.

KG Physique offers individual consultations covering PED bloodwork review, cycle review and discussion, interpretation of health markers relevant to PED use, and contest preparation considerations.

For consultation information, visit KG Physique.

Instagram: @kg.physique YouTube: @kg.physique

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CHAPTER 17

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