PRIMOBOLAN

THE SCIENCE BEHIND BODYBUILDING'S

"MILD" STEROID



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INTRODUCTION

Primobolan is one of those anabolic steroids that has developed an almost unusual reputation in bodybuilding because, depending on who you speak to, it is either described as one of the cleanest and most desirable compounds available or dismissed as too mild, too expensive and too difficult to source to justify using at all. Somewhere between those two extremes sits the actual drug, methenolone, an anabolic androgenic steroid with a genuine pharmaceutical history stretching back more than sixty years.

What makes Primobolan particularly interesting is that its modern bodybuilding reputation is quite different from its original medical purpose. Methenolone emerged during the period when pharmaceutical companies were attempting to develop anabolic steroids that retained useful effects on protein metabolism while reducing some of the androgenic characteristics of testosterone. It subsequently appeared in both oral and injectable pharmaceutical forms and was investigated or prescribed in clinical settings involving postoperative recovery, catabolic illness, anaemia, liver disease and advanced cancer. Historical medical papers on Primobolan appeared by the early 1960s, including Fixson's 1962 report on methenolone enanthate in postoperative therapy and Heim et al.'s 1963 investigation of the mechanism of action of Primobolan acetate.

Over time, bodybuilding gave Primobolan a different identity. Rather than being used to help preserve tissue during illness, it became valued for adding anabolic activity without aromatisation, helping retain lean tissue during calorie restriction, and providing what many bodybuilders regard as a relatively manageable side effect profile compared with more aggressive anabolic steroids.

This article looks at Primobolan from the beginning, including what methenolone actually is, where it came from, how it was used medically, how it moved into bodybuilding, why it became known as a mild anabolic, what we actually know about its relationship with estrogen, how it compares with Masteron, what we can reasonably say about its health effects, and why Primobolan has become increasingly difficult to obtain in parts of the market.

CHAPTER 1

WHAT IS PRIMOBOLAN?

Primobolan is the trade name most closely associated with **methenolone**, also written as metenolone in European medical literature. Chemically, methenolone is a modified 5 alpha reduced anabolic androgenic steroid with a methyl group at carbon 1 and a double bond between carbon 1 and carbon 2. The German Sport University Cologne describes methenolone as a 5 alpha steroid with these structural modifications and dates its first synthesis to 1960.

Methenolone interacts with the androgen receptor rather than operating through some separate theoretical anabolic receptor. In receptor binding experiments published by Saartok et al. (1984), methenolone demonstrated substantial affinity for androgen receptors in skeletal muscle and prostate tissue. The study is particularly interesting historically because it helped support the concept that the anabolic effects of these steroids in skeletal muscle are mediated through the androgen receptor itself.

Primobolan has historically existed in two main pharmaceutical forms, **methenolone acetate** and **methenolone enanthate**. Methenolone acetate was the oral preparation historically sold as Primobolan or Primobolan S, while methenolone enanthate became the injectable preparation most bodybuilders recognise as Primobolan Depot.

One of methenolone's defining characteristics is that it does not aromatise into estradiol. This differentiates it from testosterone and is central to understanding why Primo eventually became so popular in bodybuilding.

Methenolone is also not a conventional 17 alpha alkylated anabolic steroid. This is particularly interesting with oral methenolone acetate because many familiar oral bodybuilding steroids rely on 17 alpha alkylation to survive hepatic metabolism. Methenolone acetate uses a different structural approach, which means it should not simply be placed in the same category as traditional 17 alpha alkylated oral steroids when discussing liver effects. Marquardt et al. (1964) specifically included methenolone in early research examining non 17 alpha alkylated anabolic steroids and liver function.

CHAPTER 2

THE HISTORY OF METHENOLONE

Methenolone emerged from the enormous pharmaceutical interest in anabolic steroids that followed the isolation and synthesis of testosterone during the 1930s. By the 1950s and early 1960s, researchers were developing numerous testosterone and DHT related molecules in an attempt to retain useful anabolic activity while reducing unwanted androgenic effects.

The German Sport University Cologne dates the first synthesis of methenolone to 1960 and records that Schering subsequently marketed methenolone acetate in Germany as Primobolan S and methenolone enanthate as Primobolan Depot. The former was supplied as a tablet, while the latter contained 100 mg of methenolone enanthate in 1 ml of injectable solution.

Clinical literature followed almost immediately. Fixson published on methenolone enanthate and Primobolan Depot in postoperative therapy in 1962, while Heim et al. examined the mechanism of action of Primobolan acetate in 1963. These publications are useful because they demonstrate that methenolone was already being actively investigated as a medical anabolic steroid at the beginning of the 1960s.

Schering eventually became part of Bayer, and the Primobolan name became one of the most recognisable pharmaceutical anabolic steroid brands in bodybuilding history.

Importantly, however, Primobolan was never developed for bodybuilding. Its original purpose was pharmaceutical, with the objective of exploiting anabolic effects in clinical situations where maintaining or restoring tissue and protein metabolism was desirable.

CHAPTER 3

PRIMOBOLAN AS A MEDICINE

Historical medical use of methenolone was much broader than many modern bodybuilders realise. It appeared in research involving postoperative recovery, catabolic conditions, liver disease, anaemia and cancer.

Knöbel and Becker (1975), for example, studied methenolone enanthate in 15 patients with liver cirrhosis and examined its effects on albumin metabolism. They found that treatment increased albumin turnover, with the effect becoming more pronounced in patients whose turnover had been more impaired before treatment.

Lockner (1979) subsequently reported a clinical trial involving 19 patients with different forms of refractory anaemia. Responses occurred in several patient groups, although the study found no obvious prolongation of survival among responders. This is a good example of why the medical history of anabolic steroids should not be romanticised. Methenolone had genuine clinical applications, but it was not some universal treatment for catabolic or haematological disease.

Methenolone was also investigated in advanced breast cancer. Notter (1975) reported on 43 women with disseminated or inoperable breast carcinoma treated with methenolone enanthate. Objective remissions lasting longer than three months occurred in eight of 41 evaluable patients, and the author reported that methenolone produced less virilisation than testosterone propionate in that setting.

Perhaps most interestingly, pharmaceutical methenolone has not completely disappeared. Current Turkish pharmaceutical information still identifies Bayer's Rimobolan as a 100 mg per 1 ml methenolone enanthate product, while the Turkish Pharmacists' Association documents that Bayer's Primobolan Depot was officially renamed Rimobolan Depot in Turkey in 2014.

The significance of this history is that Primo's reputation for being a relatively manageable anabolic did not simply originate on bodybuilding forums. Methenolone was deliberately developed and clinically used as a pharmaceutical anabolic agent, although the populations and circumstances in those studies were obviously very different from modern bodybuilding.

CHAPTER 4

PRIMOBOLAN DEPOT VS ORAL PRIMOBOLAN

The two classic forms of Primobolan are often spoken about as though they are different steroids, but both ultimately deliver methenolone.

Primobolan Depot contains methenolone enanthate. Attaching the enanthate ester creates a depot preparation from which the compound is released gradually following intramuscular administration. Historical Schering Primobolan Depot contained 100 mg in 1 ml, and Bayer's Rimobolan continues to be listed as a 100 mg per 1 ml methenolone enanthate product in Turkey.

Oral Primobolan contained methenolone acetate. Unlike conventional 17 alpha alkylated oral steroids, methenolone acetate is orally active without that modification.

Goudreault and Massé (1990) directly investigated methenolone acetate metabolism in humans. Following oral administration to male volunteers, they identified twelve urinary metabolites and could still detect the parent steroid in urine up to 90 hours after administration. Their work confirmed that methenolone acetate is orally active and undergoes extensive human metabolism.

The practical distinction is therefore primarily one of delivery and pharmacokinetics. Methenolone enanthate provides a prolonged injectable depot, while methenolone acetate allows oral administration but is handled differently by the body. Neither should be confused with the other, but neither represents a completely different parent anabolic steroid.

CHAPTER 5

FROM MEDICINE TO BODYBUILDING: THE 1960s, 70s AND 80s

Primobolan entered medicine during exactly the same period in which anabolic steroids were spreading through strength and physique sports.

Kanayama and Pope (2018), reviewing the history of anabolic androgen use, describe how synthetic androgens became increasingly established among elite athletes and bodybuilders during the decades following the introduction of testosterone. By the 1980s, AAS use was beginning to extend beyond elite competitive sport into the broader gym population.

Primobolan fitted that environment particularly well because it was a legitimate pharmaceutical anabolic steroid with characteristics that were attractive to physique athletes. It did not aromatise, it did not directly produce estrogen mediated water retention, and it had been developed around the concept of producing useful anabolic effects while limiting some androgenic consequences.

Kopera's 1985 review of the history and clinical experience with anabolic steroids provides useful context for this pharmaceutical era, when anabolic agents had legitimate medical roles that were considerably broader than their current use.

As with the Masteron article, however, it is important not to rewrite bodybuilding folklore as scientific history. Primobolan was available during the Golden Era and became established in bodybuilding, but specific cycles attributed to famous bodybuilders decades later are generally much less reliably documented than the existence and use of the drug itself.

CHAPTER 6

WHAT PRIMOBOLAN ACTUALLY DOES

Methenolone is an androgen receptor agonist, and its anabolic effects are therefore best understood as part of androgen receptor signalling rather than as some unique muscle building pathway.

Saartok et al. (1984) compared several anabolic steroids and demonstrated that methenolone bound meaningfully to androgen receptors in skeletal muscle. Their receptor binding hierarchy placed methenolone ahead of testosterone in the experimental skeletal muscle system used in the study, although receptor binding affinity alone should never be interpreted as a direct ranking of how much muscle different drugs will build in humans.

Once androgen receptor signalling is activated, downstream changes can influence protein synthesis, nitrogen balance and the maintenance of lean tissue, which is consistent with the reasons anabolic steroids were originally investigated medically.

In bodybuilding, Primo is generally not regarded as a compound that produces enormous changes in scale weight over a short period. Its reputation is instead built around gradual lean tissue accumulation or retention, particularly when calorie intake is controlled.

This distinction is important because scale weight is not the same thing as new muscle tissue. A compound that produces changes in glycogen, water or estrogen related fluid can create a rapid increase in bodyweight, while another anabolic may produce a less dramatic change on the scales.

Because methenolone does not aromatise, any increase in estrogen mediated fluid retention during a Primo based cycle is not coming from the methenolone itself. That helps explain why bodybuilders frequently describe Primo gains as relatively clean or gradual rather than dramatic.

CHAPTER 7

WHY PRIMOBOLAN BECAME KNOWN AS A “MILD” STEROID

Calling Primobolan mild is understandable, but the word needs context.

Its reputation has a genuine historical basis. Methenolone was developed during an era when pharmaceutical researchers were actively attempting to improve the relationship between anabolic and androgenic effects, and it became clinically useful partly because it could provide anabolic activity without some of the more pronounced androgenic characteristics of other available drugs.

It also does not aromatise, so methenolone itself does not directly create estrogenic water retention or gynecomastia.

Injectable methenolone enanthate is not 17 alpha alkylated, and oral methenolone acetate is also not a conventional 17 alpha alkylated anabolic. Marquardt et al. (1964) found that the non 17 alpha alkylated anabolic steroids they studied, which included methenolone, did not produce the abnormal liver function pattern associated with the more hepatotoxic 17 alpha alkylated steroids.

All of that makes the mild reputation understandable. Where the word becomes misleading is when mild gets translated into does nothing to health markers.

Methenolone is still an anabolic androgenic steroid. It still activates androgen receptors, contributes to suppression of endogenous reproductive function, can produce androgenic effects in susceptible individuals and may influence cardiovascular risk markers. The better description is therefore **comparatively mild in certain respects**, rather than harmless.

CHAPTER 8

WHY BODYBUILDERS USE PRIMOBOLAN TODAY

Modern bodybuilding primarily values Primobolan because it allows additional anabolic androgenic activity without adding another aromatising compound.

That makes it particularly attractive during calorie restriction, when preserving lean tissue becomes increasingly important as body fat falls.

Primo should not automatically be called a cutting steroid, though, because the anabolic activity does not disappear when calories increase. It can also be used in gaining phases where the objective is accumulating lean tissue without relying entirely on increasing testosterone exposure.

The association with cutting largely comes from the characteristics of the drug. Methenolone itself does not aromatise, so it does not directly increase estrogen related water retention, and its effects on bodyweight can therefore appear less dramatic than those of compounds that alter both tissue and fluid.

Another reason people like Primo is that it can make the hormonal structure of a cycle conceptually simpler. Testosterone can provide an aromatising component, while methenolone adds androgen receptor stimulation without becoming another direct source of estradiol.

The disadvantages are practical. Genuine Primo has historically been expensive, pharmaceutical preparations are relatively low concentration, and recent raw material supply issues have made reliable methenolone considerably more difficult to obtain in some markets.

CHAPTER 9

PRIMOBOLAN AND TESTOSTERONE: UNDERSTANDING THE RELATIONSHIP

Primobolan and testosterone both activate androgen receptors, but they are not interchangeable hormones.

Testosterone can be converted to estradiol through aromatase, whereas methenolone cannot. Testosterone therefore contributes to both androgenic and estrogenic physiology in a way methenolone does not.

This distinction matters because estradiol is not simply an unwanted bodybuilding hormone. In men it has physiological roles in sexual function, bone health and other biological systems, meaning the objective should not automatically be to drive estrogen as low as possible.

This is why bodybuilding discussions about universal testosterone to Primo ratios can become overly simplistic. A ratio that produces an acceptable hormonal environment in one person may produce a very different result in somebody else because aromatase activity, body fat, genetics, total testosterone exposure, SHBG, other medications and individual metabolism vary.

The useful principle is therefore not finding one supposedly perfect ratio. It is understanding what each compound contributes and then observing what actually happens in the individual.

CHAPTER 10

PRIMOBOLAN AND ESTROGEN: DOES PRIMO REALLY LOWER E2?

What bodybuilders actually report

One of the most interesting debates surrounding Primobolan is its relationship with estradiol. Spend enough time speaking to experienced bodybuilders and you will repeatedly hear some variation of the same story: they added Primo to testosterone, their estradiol dropped, they required less aromatase inhibitor than they normally would, or in some cases they drove their E2 considerably lower than they intended.

The bodybuilding community therefore commonly describes Primo as having an **AI like effect**, and there are plenty of users who are convinced from their own bloodwork that the effect is real. The important distinction is between observing that **estradiol decreased** and proving that **methenolone is an aromatase inhibitor**. They are not necessarily the same thing.

Anecdotally, responses appear to vary enormously. Some bodybuilders report being able to combine testosterone and Primo without using a conventional aromatase inhibitor while maintaining estradiol within a range that works well for them. Others report that relatively modest amounts of Primo significantly reduce their E2, while some notice very little change at all.

This variation is also reflected in bodybuilding forums, where users frequently discuss adjusting their testosterone to Primo relationship according to bloodwork rather than following one universal ratio. Reports range from Primo having very little apparent effect on estradiol to E2 being pushed into very low territory.

These reports are not controlled clinical trials and should not be treated as such. Underground product accuracy, differences in laboratory assays, body fat, testosterone exposure, genetics, other drugs and individual metabolism can all influence the result. However, repeatedly seeing lower estradiol on bloodwork after introducing methenolone is still a legitimate observation. The mistake would be either treating those anecdotes as definitive proof of aromatase inhibition or dismissing them simply because a controlled bodybuilding trial does not exist.

What the science actually tells us

Methenolone does not aromatise, which means it cannot itself be converted into estradiol. What has **not** been established in humans is that methenolone directly inhibits the aromatase enzyme in the same manner as conventional pharmaceutical aromatase inhibitors such as anastrozole or exemestane.

Patt et al. (2020) investigated several anabolic androgenic steroids, including methenolone, for their effects on adrenal steroidogenesis. In human H295R adrenocortical cells, methenolone altered steroid hormone production, with increased mineralocorticoid production and reduced adrenal androgen production. The pattern suggested interference with CYP17A1 related steroidogenesis, although subsequent cell free testing did not demonstrate direct CYP17A1 inhibition.

That study shows that methenolone may influence steroidogenesis in ways that are more complicated than simply saying it does not aromatise. However, it **does not demonstrate that methenolone inhibits aromatase**, and it does not establish Primo as a pharmaceutical AI.

Then why might E2 fall?

There are several possibilities, and at this stage we should be careful about pretending that one has been definitively proven. The simplest explanation in some cases is cycle composition. If somebody reduces testosterone and replaces part of that androgen exposure with non aromatising methenolone, there is less testosterone available to be converted into estradiol. In that situation E2 can fall without Primo directly inhibiting anything.

But that explanation does **not account for every bodybuilding report**. There are bodybuilders who report lower estradiol after adding Primo while maintaining the same testosterone exposure, and there are also reports of testosterone being increased while E2 nevertheless remains lower than expected. These observations are one reason the idea of an estrogen lowering effect from Primo has persisted for so long.

It is possible that methenolone influences steroidogenesis through a mechanism that has not yet been adequately characterised in humans. It is also possible that several mechanisms are involved. At present, we simply do not have enough controlled human data to say exactly what is happening.

Lower E2 is not necessarily better

For years, estrogen was often treated as something that simply needed to be controlled or suppressed during testosterone use. We now have a much better appreciation that estradiol performs important physiological functions in men. Driving E2 excessively low can be associated with poor libido, erectile problems, mood changes and joint discomfort, while estradiol also contributes to bone and broader physiological health.

This becomes particularly relevant with Primo because someone who already responds strongly to methenolone's apparent estrogen lowering effect could potentially compound the problem by automatically adding a pharmaceutical AI on top. The assumption that every testosterone based cycle requires an AI therefore makes little sense.

There is probably no universal Testosterone to Primo ratio

Bodybuilding discussions often try to solve the issue by creating ratios such as 1:1, 2:1 or 1:2 testosterone to Primo. Those ratios may describe what works for an individual, but they should not be treated as biological rules.

Two bodybuilders running exactly the same amounts of testosterone and methenolone can produce very different estradiol results because aromatase activity, body fat, genetics, SHBG, metabolism and other drugs can all influence the hormonal environment. The bodybuilding community itself demonstrates this variability. Some users report that equal testosterone and Primo works extremely well, while others find that the same relationship drives E2 too low. That is why bloodwork becomes much more useful than copying somebody else's ratio.

So is Primobolan an AI?

Based on the evidence we currently have, **I would not call Primobolan an aromatase inhibitor**. There is no convincing human evidence demonstrating that methenolone directly inhibits aromatase in the way established pharmaceutical AIs do. At the same time, I would also not say that **Primo cannot lower estradiol**.

There is a substantial amount of bodybuilding anecdote, including reports supported by before and after bloodwork, suggesting that methenolone can reduce measured estradiol in at least some individuals. The size of that effect appears to vary considerably, and its precise mechanism has not been established.

The most accurate position therefore sits somewhere between the two extremes: **Primobolan is not a proven aromatase inhibitor, but it appears capable of producing an estrogen lowering effect in**

some users, and we currently do not fully understand why.

That distinction matters because bodybuilding experience can sometimes identify a phenomenon before research has adequately explained the mechanism. It does not mean the anecdote automatically becomes scientific fact, but it also does not make sense to ignore consistent bloodwork simply because the specific mechanism has not yet been established.

With Primo, the sensible approach is therefore not to assume what it will do to E2 based on somebody else's testosterone to Primo ratio. Establish the baseline, introduce the compounds, repeat the bloodwork and see how **that individual** actually responds.

CHAPTER 11

PRIMOBOLAN VS MASTERON

Primobolan and Masteron are regularly compared because both are modified DHT related anabolic steroids, neither aromatises, and both are commonly used when bodybuilders want additional androgenic anabolic activity without adding another aromatising drug.

Their reputations in bodybuilding are nevertheless quite different.

Masteron, or drostanolone, became particularly associated with cosmetic conditioning and contest preparation. Bodybuilders commonly describe increased hardness and visual detail when already very lean, although these cosmetic observations are not clinical endpoints that have been quantified in controlled trials.

Primobolan's reputation is less about producing an immediate cosmetic effect and more about providing steady anabolic activity with relatively little estrogenic complexity.

Neither aromatises. Neither should automatically be called an aromatase inhibitor. Both can produce androgenic effects. Both should be considered within the context of the entire cycle rather than being treated as isolated drugs.

If I had to simplify the bodybuilding distinction, I would describe Masteron as being more closely associated with **cosmetic androgenic effects and contest finishing**, while Primobolan is more closely associated with **steady lean tissue retention or accumulation without adding another aromatising anabolic**.

CHAPTER 12

SIDE EFFECTS AND HEALTH CONSIDERATIONS: PUTTING THE RISKS INTO CONTEXT

Primobolan is often described as one of the milder anabolic steroids, but discussing its health effects properly requires acknowledging a major limitation in the evidence. We do not have good long term controlled trials in which healthy competitive male bodybuilders use pharmaceutical methenolone alongside a standardised amount of testosterone while researchers control their training, diet, alcohol consumption, recreational drug use, other PEDs, cardiovascular fitness and bodyweight.

That matters because much of the methenolone specific clinical literature comes from populations that look nothing like a modern male bodybuilder, including patients with cancer, anaemia, cirrhosis and other serious illnesses. Those studies remain scientifically useful because they tell us what biological effects methenolone is capable of producing, but it is poor interpretation to assume that the magnitude of an effect observed in an ill clinical population will automatically occur in a healthy male bodybuilder.

The opposite argument is equally weak. The absence of a perfect bodybuilding trial does not prove that Primo has no cardiovascular, hormonal or androgenic consequences. The evidence simply needs to be interpreted in context.

Primo is rarely used by itself

One of the biggest problems with assigning a specific side effect to Primobolan is that bodybuilders generally use multiple compounds. Smit et al. (2020) documented this clearly in the HAARLEM study of 100 male amateur athletes using anabolic steroids. Participants used highly variable combinations of AAS and other performance enhancing substances, while chemical analysis also showed that products obtained through the illicit market did not always contain what their labels claimed.

That creates an obvious problem when somebody develops an abnormal health marker. If HDL falls during a cycle containing testosterone, methenolone and additional compounds, we cannot automatically calculate what percentage of the change came specifically from Primo. If blood pressure rises while somebody is simultaneously using stimulants, gaining significant bodyweight and sleeping poorly, assigning the entire change to methenolone makes little scientific sense.

This is why statements such as Primo destroys cholesterol or, at the opposite extreme, Primo has no cardiovascular effects are both more confident than the evidence warrants.

Lifestyle matters

PED use does not happen inside a laboratory, and bodybuilding lifestyle factors can influence many of the same health outcomes attributed to anabolic steroids. Diet quality, bodyweight, cardiovascular fitness, sleep, smoking, alcohol, recreational drugs, stimulant use and genetic cardiovascular risk all matter.

Schwingel, Zoppi and Cotrim (2014) examined 145 male recreational bodybuilders and specifically investigated the combined influence of anabolic steroids, alcohol, tobacco and cocaine on lipid profiles. Among the participants, 118 reported alcohol intake, 27 reported smoking and 33 reported cocaine use. The study is useful precisely because it demonstrates how messy real world bodybuilding exposure can be when researchers try to attribute a cardiovascular outcome to one variable.

This is particularly relevant when looking retrospectively at professional bodybuilders from previous generations. We often know that they used anabolic steroids, but we may know considerably less about cumulative doses, recreational drugs, alcohol, smoking, stimulants, blood pressure, sleep apnoea, diet, family history and decades of very high bodyweight. That does not mean steroids can simply be excluded from the discussion. It means blaming one specific anabolic for a multifactorial health outcome is often impossible.

Cardiovascular health and lipids

There is legitimate evidence that AAS exposure as a class can adversely affect cardiovascular risk factors, and the prospective HAARLEM study is considerably more relevant to modern steroid users than many older clinical reports because researchers followed actual male AAS users through their cycles.

Smit et al. (2021) documented both positive and negative physiological effects during AAS exposure in 100 men. However, participants used different combinations and amounts of steroids, meaning the study tells us much more confidently about the effects of supraphysiological androgen exposure than it does about methenolone in isolation.

There is also methenolone specific evidence. Garbrecht et al. (1981) studied methenolone enanthate in 28 menopausal women with metastatic breast cancer and observed hyperlipoproteinaemia in a number of patients, with the abnormalities regressing after methenolone was discontinued.

That is evidence that methenolone **can influence lipid metabolism**. It is not evidence that a healthy male bodybuilder using Primo alongside testosterone will experience exactly the same effect, because the population, health status and treatment circumstances were completely different.

The common sense conclusion is therefore to measure the individual response. If LDL, ApoB or non HDL cholesterol rises substantially while HDL falls after a cycle is introduced, that information matters regardless of whether somebody online describes Primo as mild.

Blood pressure

Primobolan does not aromatise and therefore does not directly create estrogen mediated fluid retention in the same way that increasing testosterone exposure can. That does not guarantee normal blood pressure because blood pressure is influenced by far more than estradiol.

Bodyweight, sodium intake, genetics, kidney function, cardiovascular conditioning, stimulants, sleep apnoea, stress and other drugs can all contribute. Patt et al. (2020) found that methenolone altered

mineralocorticoid production in human adrenal cells, which provides an interesting mechanistic reason to continue studying its possible relationship with blood pressure, but an in vitro adrenal cell experiment should not be presented as proof that Primo causes hypertension in bodybuilders.

The practical answer is much simpler. Measure blood pressure consistently and judge the individual's response.

Haemoglobin and haematocrit

Androgen exposure can stimulate erythropoiesis, making Full Blood Count relevant during AAS use. However, Primo is generally paired with testosterone, and testosterone itself is well established as capable of increasing haemoglobin and haematocrit. A rise during a testosterone and Primo cycle therefore cannot automatically be assigned to methenolone.

Hydration, altitude, smoking and obstructive sleep apnoea can also affect these measurements. Again, baseline matters because without knowing where someone started, attributing a later result to one compound becomes guesswork.

Testosterone production and fertility

There is considerably less ambiguity around suppression. Methenolone is an exogenous anabolic androgenic steroid and contributes to negative feedback on the hypothalamic pituitary gonadal axis. When Primo is being used alongside exogenous testosterone, LH and FSH are generally already suppressed by the overall androgen exposure.

The more useful question is therefore not how much suppression belongs to each individual steroid, but whether fertility and restoration of endogenous testosterone production are relevant goals for that individual.

Liver health

Primobolan is also frequently discussed incorrectly in relation to the liver. Methenolone enanthate is not a 17 alpha alkylated anabolic steroid, and it should not automatically be placed in the same hepatotoxic category as classic 17 alpha alkylated oral steroids.

Marquardt et al. (1964) specifically examined non 17 alpha alkylated anabolic steroids, including methenolone, and found that they failed to produce the abnormal liver function tests associated with the alkylated compounds studied historically.

That does not make liver testing irrelevant. A bodybuilder may be using other PEDs, oral steroids, medications, supplements or alcohol simultaneously, and intense resistance training itself can influence AST and ALT. The broader picture, including GGT, bilirubin, ALP, training history and other exposures, is therefore more useful than looking at one isolated enzyme and immediately blaming Primo.

The dose and total exposure matter

Another major weakness in discussions about steroids is treating a drug's name as though it determines the outcome regardless of exposure. The total amount of androgen being used, duration of exposure, number of compounds and cumulative years of PED use all matter.

A short period of exposure to one anabolic in an otherwise well managed individual is not biologically equivalent to decades of high dose polypharmacy, even though both people can technically say they have used Primobolan. This is also why looking at the death or disease of a professional bodybuilder and attributing it to one specific steroid without knowing the complete medical and drug history is rarely scientifically defensible.

Where the evidence actually leaves us

I do not think the evidence supports describing Primobolan as harmless simply because it has a relatively favourable reputation. I also do not think the evidence supports taking every negative finding from the general AAS literature or from seriously ill historical patients and presenting it as proof that methenolone itself caused the same outcome in a healthy male bodybuilder.

We know enough to say that methenolone is biologically active, that it can affect physiology and that supraphysiological androgen exposure as a whole carries genuine health consequences. What we do not have is the perfect study that isolates pharmaceutical Primobolan in modern competitive bodybuilders while controlling testosterone, other PEDs, diet, alcohol, recreational drugs, training and lifestyle.

Until we have that evidence, the most sensible way to evaluate Primo is through the research we do have, interpreted in the correct population and context, combined with bloodwork, blood pressure and the individual's actual response.

CHAPTER 13

PRIMOBOLAN AND HAIR LOSS: MORE THAN JUST DHT

Hair loss during Primobolan use is often immediately blamed on the fact that methenolone is DHT related, but hair biology is considerably more complicated than that.

Androgenetic alopecia is strongly influenced by genetic susceptibility and androgen receptor signalling within scalp follicles, meaning increased androgen exposure can accelerate hair loss in susceptible individuals. Methenolone binds to the androgen receptor, as demonstrated by Saartok et al. (1984), so there is a plausible androgenic pathway through which Primo can contribute to scalp hair loss.

That does not mean every episode of shedding during a Primo cycle is caused by methenolone. Bodybuilders frequently use Primo during contest preparation, which can simultaneously involve rapid weight loss, prolonged calorie restriction, low dietary fat, high training volume, large amounts of cardio, psychological stress and restricted food variety. Those factors can contribute to **telogen effluvium**, which usually presents as more diffuse shedding rather than the classic temple and crown pattern of androgenetic alopecia.

Yorulmaz et al. (2022) reviewed 3,028 patients with telogen effluvium and found abnormalities including iron deficiency anaemia, thyroid dysfunction and deficiencies of vitamin D, vitamin B12, folate and zinc among subsets of patients. These findings are associations and should not be interpreted as proof that every deficiency caused the hair loss, but they demonstrate why unexplained diffuse shedding deserves a broader investigation.

Wang et al. (2024), in a systematic review of 49 studies examining micronutrients and androgenetic alopecia, found evidence implicating imbalances involving B vitamins, vitamin D, iron, selenium and zinc, although they also emphasised that the literature is inconsistent.

Oxidative stress and inflammation may also contribute to follicular ageing and androgenetic alopecia, but this does not mean taking large doses of antioxidants will reverse genetically driven hair loss. Vitamin A deserves particular attention because excessive intake can itself promote shedding, while zinc and selenium are also good examples of nutrients where more is not automatically better.

Vitamin K is sometimes mentioned in online discussions about hair loss, but the current evidence is nowhere near strong enough for me to present vitamin K deficiency as an established common cause of alopecia.

The practical distinction is the pattern. Progressive recession at the temples, hairline and crown points more strongly towards androgenetic alopecia, whereas sudden diffuse shedding across the scalp makes nutritional, thyroid, illness, medication and physiological stress factors more important to investigate. Primo may still be contributing, but blaming the compound automatically can mean

missing another problem that may actually be correctable.

CHAPTER 14

BLOODWORK: BEFORE, DURING AND AFTER A PRIMOBOLAN CYCLE

Bloodwork becomes most useful when it is treated as a comparison over time rather than a pass or fail test. Gibbons et al. (2024) published a practical clinical guideline specifically addressing blood testing in people using anabolic androgenic steroids. Their recommendations include Full Blood Count, lipids, liver function, renal markers, estradiol and blood pressure, with additional testing depending on the drugs being used and whether the individual is currently on or off AAS.

Before starting: establish the baseline

Without a baseline, it becomes very difficult to know whether a later abnormality actually developed 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
Free testosterone
Estradiol
LH
FSH
Prolactin

Metabolic Markers

Fasting glucose
HbA1c

Additional Markers

PSA where appropriate based on age and individual circumstances.

Blood pressure should also be measured using several properly rested readings rather than relying on one measurement after caffeine, training or rushing into an appointment.

Around week 6: assess the response

At around week 6, the objective is to identify the direction in which markers are moving rather than simply asking whether they remain inside a laboratory reference interval. I would repeat Full Blood Count, lipid markers including ApoB and ApoA1, liver markers, renal markers, estradiol and relevant hormone measurements, together with blood pressure.

The trend is what matters. If HDL has fallen substantially from baseline while ApoB and LDL have increased, that deserves attention even if the laboratory has not yet marked the result as critically abnormal.

Week 12 to 14: assess cumulative change

At the end of the monitoring period, the major panels should be repeated so baseline, week 6 and week 12 to 14 can be compared directly.

Gibbons et al. (2024) also make an important point about interpreting creatinine and eGFR in muscular individuals because high muscle mass can affect creatinine based renal estimates. Likewise, resistance training can influence AST and ALT, which means liver enzymes should be interpreted alongside GGT, bilirubin, ALP, training history and the broader clinical picture.

LH and FSH are generally much more useful before exposure or when assessing recovery than while somebody is actively using suppressive exogenous androgens. Bloodwork cannot make PED use risk free, but it can replace assumptions with information about how the individual is actually responding.

CHAPTER 15

THE PRIMOBOLAN SHORTAGE: WHY HAS PRIMO BECOME SO HARD TO FIND?

The availability of Primobolan has become a major topic among bodybuilders over roughly the last two years, but describing the situation accurately requires separating the **legitimate pharmaceutical market** from the **underground bodybuilding market**. They are not the same supply chain.

Pharmaceutical methenolone has become geographically limited

Germany is historically central to the Primobolan story because Schering marketed both Primobolan S and Primobolan Depot there. The German Sport University Cologne now states that methenolone is no longer commercially available in Germany.

That does not mean pharmaceutical methenolone disappeared everywhere. In Turkey, Bayer's product was officially renamed from Primobolan Depot to Rimobolan Depot in 2014, according to documentation from the Turkish Pharmacists' Association. Current Turkish medicine databases continue to identify Rimobolan as a Bayer 100 mg per 1 ml methenolone enanthate preparation.

This tells us something important about modern Primo availability. Pharmaceutical methenolone still exists, but its legitimate market is much smaller and more geographically restricted than it once was.

The underground supply problem is different

The more obvious recent shortage discussed in bodybuilding concerns underground methenolone and the raw material used to manufacture it. This is much harder to document scientifically because underground steroid manufacturers do not publish audited production figures and Chinese raw material suppliers do not provide transparent global methenolone production statistics.

One supplier operating in this market, Integra Pharmaceuticals, has publicly reported severe shortages of methenolone and drostanolone raw material and attributes those problems to tighter regulatory restrictions and enforcement affecting Chinese raw material manufacturers. It specifically describes methenolone and drostanolone as specialised compounds that are not currently being manufactured at scale.

That report is useful as **industry evidence of a supply problem**, but it is not an independent government or peer reviewed source, so I would not use it to claim that one particular Chinese regulatory action definitively explains every Primo shortage worldwide.

Why Primo is particularly vulnerable

Methenolone is a niche pharmaceutical compound compared with testosterone. Testosterone has enormous legitimate medical demand and numerous manufacturers around the world. Methenolone does not have that same pharmaceutical manufacturing base.

When the availability of raw steroid material contracts, a lower volume and relatively expensive compound such as methenolone is therefore potentially more vulnerable than testosterone.

The economics also matter because Primo has traditionally been one of the more expensive anabolic steroids to manufacture in the underground market. When raw material becomes scarcer and more expensive, manufacturers have greater incentive to reduce production, increase prices, underdose products or substitute cheaper compounds. That is one reason authenticity has always mattered particularly with Primo.

So is there really a global Primo shortage?

I would phrase the answer carefully. There is credible evidence that pharmaceutical methenolone availability has become geographically limited, and there are consistent reports from within the bodybuilding supply chain of substantial methenolone raw material pressure during the recent period.

What I cannot substantiate is the much stronger claim that Bayer has globally stopped producing methenolone or that one single government action caused every Primobolan shortage around the world. The evidence supports **restricted pharmaceutical availability combined with disruption in the underground raw material market**, rather than one simple worldwide explanation.

CHAPTER 16

FINAL THOUGHTS: WHERE PRIMOBOLAN FITS

Primobolan is one of the better examples of how bodybuilding can take a genuine pharmaceutical anabolic steroid and gradually build an entirely different identity around it.

Methenolone was developed during an era when researchers were attempting to produce useful anabolic effects while limiting excessive androgenic activity. It subsequently found legitimate medical applications in postoperative recovery, catabolic disease, anaemia, liver disease and advanced cancer, as demonstrated across the historical literature from Fixson, Knöbel and Becker, Lockner and Notter.

Bodybuilding eventually valued it for different reasons. Primo does not aromatise, it provides androgen receptor stimulation without adding another direct source of estradiol, and it does not have the classic 17 alpha alkylated structure responsible for much of the hepatotoxic reputation of traditional oral anabolic steroids.

Those are genuine characteristics. Where things become exaggerated is when those characteristics are translated into claims that Primo is harmless, that it cannot affect cardiovascular markers, or that it functions as a pharmaceutical aromatase inhibitor.

The evidence does not support those conclusions. At the opposite extreme, taking every health problem observed among polypharmacy using bodybuilders or seriously ill historical patients and attributing it directly to methenolone is equally poor science.

Primo should be judged for what it actually is: a non aromatising anabolic androgenic steroid with a long pharmaceutical history, a relatively favourable reputation in certain areas, genuine physiological effects, significant individual variability and some major gaps in the evidence surrounding the way modern bodybuilders actually use it.

That middle ground may not make for the most dramatic social media headline, but it is a much more accurate way of understanding Primobolan.

stay strong. stay brown.

CHAPTER 17

WANT TO TALK ABOUT YOUR OWN SITUATION?

PED use should not be approached simply as choosing compounds from a list because 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 experience completely different changes in bloodwork, side effects and results, which is why I prefer looking at the full picture rather than discussing an isolated drug or cycle without context.

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.

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

CHAPTER 18

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https://integra-pharm.com/index.php/news-updates/raw-materials-batch-updates/global-raw-material-supply-update

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