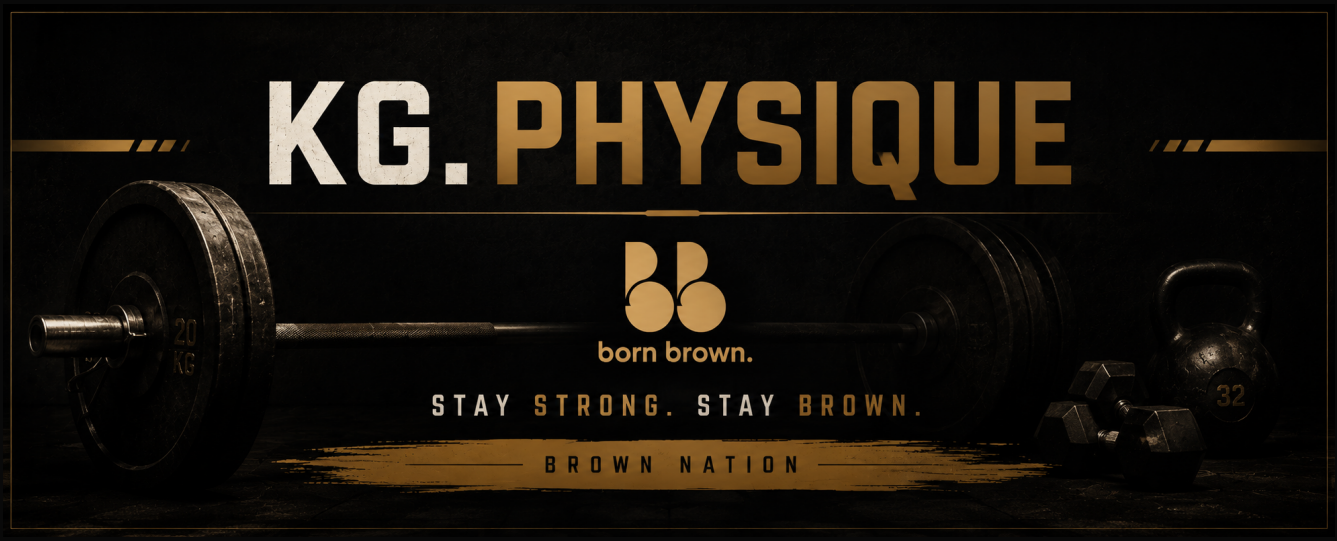


FAT BURNERS IN BODYBUILDING

From Clenbuterol to the Next Generation of Thermogenics

Research, bodybuilding experience and where the evidence actually stands.

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INTRODUCTION

Fat burners are discussed very differently depending on whether you are reading a clinical obesity paper or talking to competitive bodybuilders. Most medical research understandably asks whether a compound can help an overweight or obese person lose meaningful amounts of weight. That isn't the question I am interested in for this article.

I am talking about bodybuilders who are already lean, have their diet and cardio in place and are trying to move from lean into genuinely shredded contest condition - potentially around 5-6% body fat or lower. At that point, losing another kilogram of fat can make an enormous visual difference, and compounds that might appear relatively unimpressive in an obesity trial can potentially have a completely different application.

I also want to make a clear distinction throughout this article between published research and bodybuilding experience. There are compounds such as clenbuterol where bodybuilders have accumulated decades of practical experience and human research is increasingly confirming mechanisms and effects competitors have talked about for years. There are other compounds, such as SANA, BAM15 and ATX-304, where the science is developing ahead of bodybuilding experience.

Neither should be confused with the other.

This article is for educational purposes only. It is not medical advice, it does not recommend the use of any drug discussed, and it should not be interpreted as a dosing protocol. Individual responses can vary substantially depending on health, cardiovascular status, medications, drug exposure and numerous other factors.

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CHAPTER 1 - CLENBUTEROL: THE BODYBUILDING FAT BURNER THAT REFUSES TO DISAPPEAR

Clenbuterol was not developed as a bodybuilding drug. It was developed as a bronchodilator, with beta-2 adrenergic stimulation relaxing bronchial smooth muscle and helping open the airways. It has been used medically for respiratory disease in some countries, although it has never been approved for human use in others, including the United States. What eventually made clenbuterol far more interesting outside respiratory medicine was the discovery that beta-2 stimulation could influence energy expenditure, fat metabolism and skeletal muscle.

Research through the 1980s began producing results that immediately attracted attention. Animal studies demonstrated that clenbuterol could alter body composition, increase muscle protein deposition and, under some experimental conditions, reduce adiposity or increase thermogenesis. Research in livestock produced similar repartitioning effects, with beta-agonists increasing lean-tissue deposition relative to fat. By the 1990s, clenbuterol's reputation as both a fat-loss and anabolic compound was already firmly established within bodybuilding despite the lack of the controlled human trials we have today. [1-5]

This is where bodybuilding experience becomes important. Competitive bodybuilders did not wait thirty years for researchers to tell them clenbuterol increased energy expenditure. It became one of the most widely used contest-prep fat burners because competitors repeatedly observed that it helped push fat loss further when diet and cardio were already in place. The bodybuilding application was never primarily about taking an obese person and making them lose five kilograms. It was about helping somebody who was already lean become shredded.

What Clenbuterol Actually Does in Humans

Modern human research has now given us much better evidence for what clenbuterol actually does. Jessen and colleagues gave healthy young men 80 micrograms of clenbuterol and measured metabolic responses. Resting energy expenditure increased approximately 21%, while fat oxidation increased approximately 39%. Circulating fatty acids increased substantially, and signalling associated with skeletal-muscle growth, including mTOR phosphorylation, also increased. [6]

Those are significant acute effects. More recently, Hostrup and colleagues examined repeated clenbuterol exposure in healthy men. Two weeks at 80 micrograms per day increased lean mass by approximately 0.91 kg compared with placebo and increased muscle protein content, providing controlled human evidence that clenbuterol genuinely can exert anabolic effects. However, the same study showed a reduction in VO2 max and exercise capacity, reductions in markers of muscle oxidative capacity and attenuation of beta-2 signalling during repeated exposure. [7]

An even newer randomized trial published in 2026 examined eight weeks of resistance training with clenbuterol or placebo in healthy men and women. Clenbuterol augmented gains in lean mass by approximately 1.7 kg and increased improvements in bench press and leg press

strength compared with placebo. Interestingly, fat-mass reduction was not significantly greater than placebo in that particular resistance-training study. [8]

Is Clenbuterol Actually Anabolic?

Within bodybuilding, clenbuterol's anabolic properties have been discussed for decades. The argument was traditionally dismissed because much of the early evidence came from rodents and livestock, where the exposures were very different from normal human therapeutic use. That argument is becoming harder to make now.

We have controlled human evidence showing increased lean mass, increased muscle protein content and enhanced resistance-training adaptations. The 2025 Hostrup study demonstrated approximately 0.9 kg of additional lean mass after only two weeks, while the 2026 resistance-training study demonstrated approximately 1.7 kg greater lean-mass gains over eight weeks. [7,8]

That does not make clenbuterol comparable to anabolic-androgenic steroids. The mechanisms, magnitude of effect and overall pharmacology are completely different. But describing clenbuterol as having no anabolic effect in humans is no longer consistent with the available evidence. This also lines up with something bodybuilders have reported for years: at higher bodybuilding exposures, clenbuterol does not behave simply like caffeine with stronger fat-burning properties. There appears to be a genuine repartitioning and muscle-protein effect.

Where Clenbuterol Fits in Contest Prep

The bodybuilding application becomes much more relevant when somebody is already lean. A competitor deep into contest preparation may have spent months maintaining a calorie deficit, progressively increasing cardio and removing most of the easily lost body fat. At this point the objective may be moving from visibly lean into the type of condition where abdominal skin becomes extremely thin, lower-back fat disappears and separation through the glutes and hamstrings becomes visible.

This is where the combination of increased energy expenditure and increased fat oxidation becomes useful. Clenbuterol does not replace the calorie deficit. What it can potentially do is increase the expenditure side of that deficit while also increasing mobilisation and oxidation of fatty acids. There are no controlled trials taking competitive bodybuilders from 8% body fat to 5% using clenbuterol, so that specific application remains anecdotal. However, unlike some bodybuilding practices, the underlying effect is no longer speculative. Human research clearly demonstrates that clenbuterol can acutely increase energy expenditure and fat oxidation. [6]

The Limitation: Adaptation

Clenbuterol's effects do not necessarily remain identical indefinitely. Repeated beta-2 stimulation produces receptor and downstream signalling adaptation, and the 2025 human study demonstrated attenuation of beta-2 adrenergic signalling during only two weeks of exposure. [7] Bodybuilders have talked about clenbuterol becoming less noticeable after repeated use for decades. The research now gives us a physiological explanation for at least part of that observation.

What Can We Conclude About Clenbuterol?

Clenbuterol is probably the best example in this article of bodybuilding experience preceding modern human research. Bodybuilders used it for decades because they repeatedly observed that it assisted contest-prep fat loss and appeared to have muscle-preserving or anabolic characteristics. We now have controlled human evidence demonstrating a substantial acute increase in resting energy expenditure and fat oxidation, increased muscle-growth signalling, increased muscle protein content, increased lean mass and enhanced resistance-training adaptations. [6-8] That doesn't mean every claim made about clenbuterol in bodybuilding is automatically correct. It does mean that dismissing clenbuterol's fat-burning or anabolic properties because the early evidence came from animals is now outdated.

CHAPTER 2 - SALBUTAMOL/ALBUTEROL: THE SHORTER-ACTING BETA-2 ALTERNATIVE

Salbutamol, known as albuterol in the United States, belongs to the same broad beta-2 agonist family as clenbuterol, but its pharmacokinetics are very different. It is a legitimate and widely used asthma medication with a much shorter duration of action, which is one of the main reasons it has also attracted interest within bodybuilding.

From a contest-prep perspective, salbutamol is interesting because it potentially provides some of the same beta-2-mediated effects as clenbuterol without producing the same prolonged exposure. Bodybuilders who dislike feeling stimulated for most of the day or having clenbuterol interfere with sleep sometimes discuss salbutamol as a more controllable alternative.

There is also genuine human metabolic evidence behind it. Research examining beta-2 stimulation with salbutamol in lean men found an approximately 13% increase in energy expenditure during the experimental period, with fat oxidation increasing approximately 19%. When lipolysis was pharmacologically blocked, the increase in fat oxidation disappeared, demonstrating the relationship between beta-2 stimulation, mobilisation of stored fat and subsequent oxidation. [9]

Salbutamol and Muscle

Like clenbuterol, salbutamol also has human evidence suggesting effects on skeletal muscle. An older controlled study found that oral salbutamol increased quadriceps strength by approximately 12% after fourteen days, while dominant-leg hamstring strength increased approximately 22% after twenty-one days. The study did not demonstrate significant changes in lean body mass, but it established that beta-2 stimulation could influence human muscle function. [10]

More recent research has gone considerably further. A 2026 randomized controlled trial examining high-dose salbutamol during resistance training reported increases in lean mass alongside changes in skeletal-muscle oxidative characteristics and cardiac remodelling. [11] Again, this doesn't suddenly make salbutamol an anabolic steroid. It demonstrates that beta-2 agonism has meaningful effects on human skeletal muscle and that the old assumption that these compounds only affect the lungs and nervous system is far too simplistic.

Where Salbutamol Fits in Bodybuilding

Within bodybuilding, salbutamol has never developed quite the same reputation as clenbuterol, partly because clenbuterol's long duration of action makes it an extremely convenient thermogenic compound. Salbutamol's shorter duration can require more deliberate timing, but that same characteristic is exactly what some users prefer. For somebody already deep into a cut, the attraction is straightforward: increase beta-2-mediated thermogenesis and fat oxidation during selected parts of the day without necessarily carrying the same stimulatory effect into the evening.

The human evidence supports the underlying mechanism. What we don't have is the type of contest-prep evidence bodybuilders would actually like to see - trained competitors already at single-digit body fat being followed through the final stages of preparation.

CHAPTER 3 - MIRABEGRON: CAN BETA-3 ACTIVATION INCREASE HUMAN THERMOGENESIS?

Mirabegron is interesting because it moves us away from beta-2 receptors and toward beta-3 adrenergic receptors, which are heavily associated with adipose tissue and thermogenesis. Mirabegron was developed and approved for overactive bladder, not fat loss. However, beta-3 receptors are also expressed in human brown adipose tissue, which created an obvious research question: could pharmacologically activating beta-3 receptors increase human thermogenesis? The answer appears to be yes.

In a landmark 2015 study, Cypess and colleagues gave twelve healthy men a single 200 mg dose of mirabegron. Brown adipose tissue activity increased in every participant, while resting metabolic rate increased by approximately 203 calories per day, or 13%. [12] Further research confirmed that the effect was dose dependent. In another study comparing 50 mg with 200 mg, the higher exposure substantially increased brown-fat activity, circulating non-esterified fatty acids and resting energy expenditure, while the standard therapeutic exposure produced considerably smaller metabolic effects. [13]

Other research in lean South Asian and Europid men also demonstrated increases in resting energy expenditure following 200 mg mirabegron, although effects on lipid oxidation varied between groups. [14]

What About Repeated Use?

O'Mara and colleagues treated healthy women with 100 mg mirabegron daily for four weeks. Chronic treatment increased brown adipose tissue metabolic activity and produced favourable changes in insulin sensitivity and other metabolic markers. [15] This demonstrated that beta-3 activation was not simply an acute laboratory curiosity. However, the bodybuilding question remains different. Increasing brown-fat activity and resting energy expenditure does not automatically mean mirabegron will produce dramatic visible fat loss in somebody already at 6% body fat. We simply do not have that study.

Why Mirabegron Became Interesting to Bodybuilders

Clenbuterol pushes beta-2 signalling hard throughout the body. Mirabegron offered the possibility of targeting a receptor more closely associated with adipose-tissue thermogenesis. The complication is that receptor selectivity is not absolute, particularly as exposure increases. The stronger metabolic effects in human studies have generally occurred at exposures above the standard doses used clinically for overactive bladder, and cardiovascular effects including increases in heart rate and blood pressure become increasingly relevant at those levels. [12,13,16]

Mirabegron therefore shouldn't be viewed as a perfectly selective fat-burning switch with none of the systemic consequences associated with adrenergic stimulation. Nevertheless, the human evidence establishes an important principle: pharmacological beta-3 activation can increase

human brown-fat thermogenesis and resting energy expenditure.

CHAPTER 4 - YOHIMBINE: TARGETING STUBBORN FAT WHEN YOU ARE ALREADY LEAN

Yohimbine is different from the compounds discussed so far because it isn't primarily interesting for producing a massive increase in resting metabolic rate. Its bodybuilding appeal comes from interfering with alpha-2 adrenergic signalling, which normally acts as a brake on catecholamine-driven lipolysis.

Different areas of human adipose tissue do not respond identically to adrenaline and noradrenaline. Beta-adrenergic receptors generally stimulate lipolysis, while alpha-2 receptors inhibit it. Yohimbine is an alpha-2 adrenergic antagonist, meaning that rather than simply pushing harder on the accelerator, it partially removes the brake. This becomes particularly relevant when somebody is already extremely lean. A competitive bodybuilder may have visible abdominal separation, vascularity and striations through much of the physique while still carrying disproportionately more subcutaneous fat around the lower abdomen, lower back, glutes or thighs.

The Human Evidence

Galitzky and colleagues demonstrated the effect directly in humans. Oral yohimbine at 0.2 mg/kg increased plasma glycerol and non-esterified fatty acids in fasting healthy subjects, demonstrating increased lipid mobilisation. Exercise strengthened the effect. When yohimbine was administered after a meal, however, the lipid-mobilising effect was completely suppressed. [17]

That experiment explains much of the bodybuilding practice surrounding yohimbine and fasted cardio. Insulin is strongly anti-lipolytic, so attempting to remove the alpha-2 brake on fat mobilisation makes considerably more sense when insulin is relatively low. Exercise also matters because mobilising fatty acids does not automatically mean those fatty acids disappear. They still need to be oxidised, and exercise creates an energetic demand capable of using some of the fatty acids being released.

Evidence in Already-Lean Athletes

Ostojic studied twenty elite male soccer players receiving yohimbine or placebo for twenty-one days. Bodyweight and muscle mass did not significantly change, but measured body-fat percentage in the yohimbine group decreased from approximately 9.3% to 7.1%, while the placebo group did not experience the same reduction. Exercise performance was not significantly impaired. [18]

It was a small study and body-fat measurements at these levels have limitations, but these weren't sedentary obese individuals. They were trained athletes already starting at roughly single-digit body fat. That makes the study considerably more relevant to what we are discussing here.

Stubborn Fat and Bodybuilding Experience

Bodybuilders have used yohimbine for years during the later stages of contest preparation, particularly when most body fat has already disappeared but specific areas remain behind. The research does not prove that yohimbine magically selects someone's lower abdominal fat and burns it away. What it does demonstrate is that alpha-2 receptors inhibit lipolysis, yohimbine can increase lipid mobilisation in fasting humans, exercise strengthens that response and feeding suppresses it. [17-20]

For somebody carrying substantial excess body fat, worrying about alpha-2 receptors in their lower back is missing the bigger picture. For somebody approaching 5-6% body fat or lower, relatively small regional differences in subcutaneous fat can completely change how the physique looks. That is where yohimbine becomes much more interesting.

CHAPTER 5 - SANA/MVD1: CREATINE-DEPENDENT THERMOGENESIS

SANA takes us away from traditional adrenergic fat burners altogether. Its chemical name is 5-(2-nitroethenyl)salicylic acid, and during clinical development it has been referred to as MVD1. Its attraction is a different mechanism involving creatine-dependent thermogenesis within adipose tissue.

When bodybuilders hear creatine, they normally think about creatine monohydrate, phosphocreatine, ATP regeneration and strength. That isn't what we are talking about here. Creatine also participates in metabolic cycling within adipose tissue, where repeated phosphorylation and dephosphorylation can consume energy without producing an equivalent amount of useful external work. The easiest way to think about this is as deliberately making part of metabolism less fuel efficient. More energy has to be consumed, with some of that energy dissipated as heat. Research by Kazak and colleagues had already demonstrated that a creatine-driven substrate cycle could increase energy expenditure and thermogenesis in beige adipose tissue. [21]

What SANA Did

Cal, Leyva and colleagues found that SANA increased mitochondrial respiration and stimulated non-shivering thermogenesis in both brown and white adipose tissue. Interestingly, the effect did not depend on UCP1, the classic protein associated with brown-fat thermogenesis, and it did not depend on AMPK activation. When researchers depleted creatine, however, much of SANA's thermogenic effect disappeared. Mice lacking mitochondrial creatine kinase also showed impaired responses to SANA, providing strong evidence that creatine metabolism was actually involved in producing the effect rather than simply changing alongside it. [22]

In diet-induced obese mice, SANA reduced adiposity and improved liver fat, insulin sensitivity and glucose regulation. The effects also persisted under thermoneutral conditions. [22]

Then SANA Was Tested in Humans

SANA subsequently progressed into a randomized, double-blind, placebo-controlled Phase 1A/B trial. The single-dose component examined 200-800 mg in healthy lean volunteers, while participants with overweight or obesity received repeated exposures up to 400 mg per day for fifteen days.

The study was primarily designed to assess safety and pharmacokinetics rather than weight loss. Nevertheless, at the highest repeated exposure participants lost approximately 3% of bodyweight in fifteen days, while placebo did not produce the same change. Improvements were also reported in glucose, insulin and HOMA-IR. [22] The study was tiny, with only six treated participants in the highest repeated-dose cohort, so the result should not be interpreted like a large obesity trial.

Why I Think SANA Is a Compound to Watch, Not Use

This is where I would draw a clear distinction between being interested in a compound and believing bodybuilders should start experimenting with it. SANA has human data, but the human exposure is still extremely limited. We don't know what happens after six, eight or twelve weeks, whether the thermogenic response diminishes over time, or how the compound behaves in somebody already at extremely low body fat.

There is also an important safety signal. At the highest 800 mg single exposure, two participants developed reversible renal tubular abnormalities reflected by proteinuria and glucosuria. Repeated exposure at lower levels for fifteen days was considerably better tolerated, but fifteen days is nowhere near enough information to establish longer-term safety. [22]

SANA therefore remains very interesting scientifically, but I think it is far too early for bodybuilders to be experimenting with it. Let the clinical research progress, let larger numbers of people be exposed under controlled conditions and let us see what longer-term safety data actually show. There is no benefit in being one of the first people experimenting with a compound while researchers are still establishing its basic human safety profile.

CHAPTER 6 - BAM15: MITOCHONDRIAL UNCOUPLING WITHOUT DNP?

BAM15 moves the discussion into mitochondrial uncoupling. Normally, mitochondria use the proton gradient generated by nutrient oxidation to produce ATP efficiently. A mitochondrial protonophore partially uncouples this process, forcing the body to oxidise more fuel to maintain its energy requirements. Anyone familiar with bodybuilding pharmacology will immediately think of DNP.

DNP demonstrated that mitochondrial uncoupling can produce substantial fat loss in humans, but it also demonstrated how dangerous uncontrolled uncoupling can become. BAM15 was developed as a structurally different mitochondrial protonophore with the objective of producing metabolic uncoupling across a wider experimental window.

What Happened in the BAM15 Research?

The 2020 Nature Communications study produced striking results. BAM15 was orally bioavailable and increased oxygen consumption and nutrient oxidation in mice. In obese mice continuing to consume the same Western diet, the BAM15 group eventually weighed approximately 15% less than untreated controls, with the difference largely attributable to lower fat mass while lean mass was preserved. [23] Food intake did not decrease.

BAM15 also improved insulin sensitivity, reduced liver fat and improved several metabolic abnormalities associated with diet-induced obesity. Researchers did not observe a significant increase in body temperature at the tested exposures, and ATP levels remained maintained despite the increased mitochondrial energy expenditure. [23] From a bodybuilding perspective, increased nutrient oxidation, substantial reductions in fat mass and preservation of lean tissue obviously make the mechanism extremely interesting.

BAM15 Is Not 'Safe DNP'

This is where the conversation around BAM15 needs to become considerably more cautious. It is very easy to look at the animal research and describe BAM15 as a safer version of DNP. We do not have the human evidence required to make that statement.

The impressive body-composition results are primarily preclinical. We do not have an established human bodybuilding exposure, a meaningful long-term human safety database or enough information to understand how the therapeutic window translates from animals into humans. That matters particularly with a mitochondrial uncoupler because we are interfering directly with cellular energy production. A compound producing a wider experimental window than DNP in mice does not automatically mean that window will remain wide in humans.

There have also been concerns and discussion around inflammatory markers such as hsCRP, but without adequate controlled human evidence I would not state that BAM15 is proven to increase hsCRP in humans. This is precisely the problem: there is still too much we do not know.

Why I Wouldn't Experiment With BAM15 Currently

Of all the newer compounds covered in this article, BAM15 is the one I would be most cautious about experimenting with at this stage. The mechanism is fascinating and the animal results are impressive, but that isn't enough. Until we have proper human trials establishing pharmacokinetics, effective exposures, inflammatory responses, cardiovascular effects, organ toxicity and longer-term safety, I don't think the potential fat-loss benefit justifies experimenting with it.

The comparison with DNP actually strengthens that argument rather than weakening it. Mitochondrial uncoupling can clearly be extremely powerful, but history has already shown us why potency in this particular pathway deserves enormous respect. BAM15 belongs in this article because it may represent an important future direction in metabolic pharmacology. It does not belong in the category of compounds where I think the current evidence justifies bodybuilders being early adopters.

For now, my position is simple: interesting research compound, but not worth trying until considerably more human safety data exist.

CHAPTER 7 - ATX-304: AMPK, MITOCHONDRIA AND INCREASING ENERGY EXPENDITURE

ATX-304 is another compound that potentially moves fat-loss pharmacology away from simply suppressing appetite or stimulating adrenergic receptors. It was previously developed under the name O304 and has been investigated as an activator of the cellular energy-sensing network involving AMP-activated protein kinase, or AMPK, alongside effects on mitochondrial metabolism.

AMPK is one of the body's major cellular fuel sensors. When cellular energy availability falls, including during exercise and calorie restriction, AMPK signalling shifts metabolism toward producing and conserving usable energy. This includes increasing glucose uptake and fatty-acid oxidation while suppressing some energy-consuming synthetic pathways.

O304 was originally investigated primarily for diabetes rather than obesity. A 2018 human study demonstrated improvements in fasting glucose, insulin resistance and microvascular perfusion in people with type 2 diabetes, providing early evidence that the mechanism translated beyond animal experiments. [24] Later research also demonstrated that O304 could increase glucose utilisation through skeletal muscle and heart while creating additional metabolic demand through mitochondrial mechanisms. [25]

The 2026 Human Data

At the 2026 American Diabetes Association Scientific Sessions, investigators reported results from a randomized Phase 1b study involving 23 participants with obesity and prediabetes. Participants received 400 mg ATX-304 once daily or placebo for eight weeks, with an optional extension.

The reported findings included significant increases in plasma adiponectin, reductions in liver and visceral fat and an increase in resting metabolic rate. The conference abstract reported increases in resting metabolic rate reaching up to 33% compared with baseline. [26] That is an interesting metabolic signal, but it needs to be interpreted correctly. This was a small Phase 1b study and the results were conference data rather than a large peer-reviewed weight-loss trial.

Why ATX-304 Could Eventually Matter to Bodybuilding

Someone deep into contest preparation may already be eating exactly what their plan permits regardless of hunger. What becomes more useful is increasing how much energy the body is expending without simply adding another stimulant. That is what makes ATX-304 interesting.

If future trials confirm a meaningful and sustained increase in resting metabolic rate with an acceptable safety profile, it could eventually become relevant to physique athletes because the objective would be increasing calories out rather than continually forcing calories in lower. But once again, I don't think this is something bodybuilders need to rush into.

ATX-304 remains in early clinical development. The human sample sizes are small, longer-term safety data are limited and we don't know what happens when the compound is used in extremely lean athletes or alongside the pharmacology frequently encountered during contest preparation. My position on ATX-304 is therefore similar to SANA: watch the research and wait. If larger and longer trials confirm the metabolic effects while establishing a reasonable safety profile, then the bodybuilding conversation becomes considerably more interesting. Right now, there simply isn't enough evidence to justify being an early adopter.

CHAPTER 8 - FAT-BURNER STACKS REPORTED DURING CONTEST PREPARATION

Before We Discuss Stacking

Before discussing fat-burner stacks, this section needs to be understood for what it is. This is not medical advice, it is not a recommended protocol, and it is not an instruction to use any of these compounds. It is a discussion of combinations repeatedly reported within bodybuilding forums, contest-prep discussions and anecdotal accounts from competitors and coaches.

Every individual is different, and factors such as bodyweight, body-fat level, cardiovascular health, blood pressure, medications, previous drug exposure and individual tolerance can completely change the response. The fact that a combination is commonly discussed or used within bodybuilding does not establish that it is safe. The purpose of this section is simply to document what is commonly heard and reported within competitive bodybuilding during cutting phases and contest preparation.

Clenbuterol + Yohimbine

Probably one of the most recognisable combinations discussed in bodybuilding is clenbuterol and yohimbine, particularly as the athlete becomes progressively leaner. The reasoning comes directly from their different adrenergic mechanisms. Clenbuterol stimulates beta-2 receptors and can increase energy expenditure, thermogenesis and fat oxidation, whereas yohimbine blocks alpha-2 receptors that normally inhibit lipolysis.

This combination has become particularly associated with the later stages of contest preparation because yohimbine's reputation within bodybuilding is strongest when the athlete is already lean and attempting to remove resistant fat from areas such as the lower abdomen and lower back. There is substantial anecdotal bodybuilding experience behind the combination, although there is no controlled clinical trial taking stage-lean bodybuilders and comparing clenbuterol alone with clenbuterol plus yohimbine during contest preparation.

Salbutamol + Yohimbine

A similar combination reported within bodybuilding substitutes salbutamol for clenbuterol. The reasoning remains largely the same because salbutamol is also a beta-2 agonist while yohimbine blocks alpha-2-mediated inhibition of lipolysis. The major practical difference is pharmacokinetics. Salbutamol is substantially shorter acting than clenbuterol, which is precisely why some bodybuilders report preferring it during cutting phases.

Clenbuterol + Caffeine

Another extremely common combination encountered during cutting phases is clenbuterol with caffeine, although in many cases this may happen almost by default rather than because somebody deliberately designed it as a pharmacological stack. Caffeine is already deeply

embedded in bodybuilding through coffee, pre-workouts and energy drinks. During calorie-restricted contest preparation, caffeine can also become increasingly attractive because of its effects on alertness, perceived fatigue and training drive.

The important practical consideration is that the subjective increase in stimulation shouldn't automatically be interpreted as a proportional increase in fat loss. There is eventually a point where adding more stimulation may contribute considerably more to how stimulated somebody feels than to the amount of additional energy actually being expended.

Clenbuterol + Yohimbine + Caffeine

Once clenbuterol, yohimbine and caffeine are combined, we arrive at one of the more aggressive combinations repeatedly discussed within bodybuilding contest preparation. The attraction is straightforward. Clenbuterol provides the primary beta-2 component, yohimbine blocks alpha-2-mediated inhibition of lipolysis, and caffeine provides additional stimulation while helping some competitors manage the fatigue associated with low calories and high cardio volumes.

This combination is generally discussed later in a cut rather than as something necessary from the beginning of preparation. Anecdotally, competitors using combinations like this are usually already very lean and attempting to push conditioning further rather than relying on the stack to create the majority of their fat loss.

Salbutamol + Caffeine

Salbutamol and caffeine also appear together in cutting discussions for many of the same reasons caffeine is combined with clenbuterol. The beta-2 agonist provides the primary pharmacological component while caffeine contributes additional stimulation. Again, the major distinction between this combination and clenbuterol-caffeine is duration. Salbutamol's shorter action is one reason some users report finding it easier to manage around their training schedule and sleep.

Yohimbine + Caffeine

Yohimbine and caffeine are another frequently reported combination, particularly around fasted cardio. Unlike the previous combinations, there is no direct beta-2 agonist involved. Yohimbine blocks alpha-2 receptors and increases noradrenergic activity, while caffeine provides additional stimulation and can improve alertness and perceived energy during cardio.

The popularity of this combination around fasted cardio also fits reasonably well with the human yohimbine research. Yohimbine's lipid-mobilising effect was demonstrated in fasting subjects, exercise strengthened the response and feeding suppressed it.

Mirabegron Combinations

Mirabegron occasionally appears in bodybuilding discussions alongside more traditional fat burners because it offers a somewhat different approach to increasing thermogenesis. Rather than being another conventional beta-2 agonist, mirabegron was developed as a beta-3 agonist and has human evidence demonstrating activation of thermogenic adipose tissue and increased

resting energy expenditure. This has led to discussion of combining mirabegron with compounds such as clenbuterol or yohimbine, although the bodybuilding evidence here is considerably thinner.

The Traditional ECA Stack

The ephedrine-caffeine-aspirin stack, usually abbreviated ECA, deserves inclusion because it is one of the most famous cutting stacks in bodybuilding history and predates much of the modern discussion surrounding clenbuterol-based combinations. Ephedrine increases adrenergic activity while caffeine complements its stimulant and thermogenic effects. Aspirin was historically added under the theory that inhibiting prostaglandin production could help prolong the thermogenic response.

ECA became particularly prominent during the 1980s and 1990s and, unlike many bodybuilding fat burners, ephedrine and caffeine have a substantial amount of controlled human research demonstrating effects on thermogenesis and weight loss. The aspirin component has become considerably more questionable. Its contribution to fat loss is much less convincing than that of ephedrine and caffeine, which is one reason modern bodybuilding discussions frequently refer simply to ephedrine and caffeine rather than treating aspirin as an essential part of the combination.

What About SANA, BAM15 and ATX-304?

Mechanistically, the three newer compounds could eventually create interesting stacking possibilities because they are not simply variations on conventional adrenergic stimulation. But I would not currently consider them established stacking compounds. SANA and ATX-304 need considerably more human safety and longer-duration data before we know where they genuinely fit. BAM15 is further behind in terms of demonstrated human safety, and given that its mechanism involves mitochondrial uncoupling, I would be particularly cautious about experimenting with it.

What the Forum and Bodybuilding Evidence Actually Tells Us

The important distinction throughout this chapter is between evidence that the individual compounds work and evidence that a particular stack is superior. For several of these drugs, there is legitimate human research demonstrating effects on thermogenesis, energy expenditure, lipolysis or body composition. What is largely missing are controlled studies comparing the exact combinations used by competitive bodybuilders at very low body-fat levels.

This is where bodybuilding experience fills part of the gap. Combinations such as clenbuterol and yohimbine have been discussed and used within contest preparation for years, whereas combinations involving mirabegron are considerably less established. The useful approach is to separate the two: the research tells us what these compounds can do physiologically, while bodybuilding experience tells us how competitors have actually attempted to apply those effects when trying to reach extreme levels of conditioning.

CONCLUSION - FAT BURNERS AND CONTEST PREPARATION

Fat burners work, but they are not all doing the same thing and they are not all supported by the same level of evidence. Clenbuterol has decades of bodybuilding use alongside increasingly strong human evidence. Yohimbine has a clear mechanism and human research supporting its ability to increase fat mobilisation, particularly when insulin is low. Salbutamol has legitimate human evidence but less contest-prep experience, while mirabegron has demonstrated increased thermogenesis and energy expenditure in humans but remains less established within bodybuilding.

SANA, BAM15 and ATX-304 are different. They represent potentially interesting future directions in fat-loss pharmacology, but interesting research does not automatically mean something is ready for bodybuilding use. SANA and ATX-304 need considerably more human data, while BAM15 currently has nowhere near the human safety evidence I would want to see before considering experimentation with a mitochondrial uncoupler.

That distinction between published research and bodybuilding experience has been the point of this article. The absence of studies involving bodybuilders at 5-6% body fat does not mean decades of contest-prep experience should simply be ignored, but anecdotal evidence should not be presented as clinical proof either.

Fat burners also need to be viewed in the correct context. They are not a substitute for getting the diet right, doing the cardio and allowing enough time to get lean. Their real value in bodybuilding is generally toward the harder stages of a cut, when the athlete is already lean and relatively small reductions in remaining body fat can make a significant difference to conditioning.

More drugs also don't automatically mean better results. Understanding what a compound does, why it is being used and what evidence actually supports it is far more useful than blindly copying somebody else's stack.

Want to Discuss Your Own Situation?

If you want help with bodybuilding coaching, contest prep, cutting strategies, PED education or understanding your bloodwork in the context of enhanced bodybuilding, you can reach out to me through KG Physique.

For coaching and consultations, visit kgphysique.com.au.

You can also find me on YouTube and Instagram @kg.physique for more content covering PEDs, bloodwork, training and competitive bodybuilding.

stay strong. stay brown.

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