Clenbuterol Handbook

CLENBUTERAL FAQ: EVERYTHING YOU NEEDED TO KNOW ABOUT CLENBUTEROL

by BigAndy69

What is Clenbuterol?

Clenbuterol is a beta-2 agonist and is used in many countries as a bronchodilator for the treatment of asthma. Because of its long half life, Clenbuterol is not FDA approved for medical use. It is a central nervous system stimulant and acts like adrenaline. It shares many of the same side effects as other CNS stimulants like ephedrine. Contrary to popular belief, Clenbuterol has a half life of 35 hours and not 48 hours.

Dosing and Cycling

Clenbuterol comes in 20mcg tablets, although it is also available in syrup, pump and injectable form. It's also available as a powder in some areas. Doses are very dependent on how well the user responds to the side effects, but somewhere in the range of 4-8 tablets per day for men and 2-4 tablets a day for women is most common. Clenbuterol loses its thermogenic effects after around 8 weeks when body temperature drops back to normal. Its anabolic/anti-catabolic properties fade away at around the 18 day mark. Taking the long half life into consideration, the most effective way of cycling clen is 2 weeks on/ 2 weeks off for no more than 12 weeks. Ephedrine or Yohimbine can be used in the off weeks.

Clenbuterol vs. Ephedrine vs. DNP

Ephedrine will raise metabolic levels by about 2-3 percent and 200mg of DNP raises metabolic levels by about 30 percent. Clenbuterol raises metabolic levels about 10 percent and it can raise body temperature several degrees.

DNP is by far the most effective fat burner but many people will never use it because of the risks associated with it. It also offers no anti-catabolic benefit. Although it does have anti-catabolic effect, ephedrine's short half-life prevents it from being all that effective.

As far as side effects, Clenbuterol's are certainly milder than DNP's, and some would even say milder than an ECA stack. There is no ECA-style crash on Clenbuterol and many users find it easier on the prostate and sex drive. This may in part be due to the fact that Clen is generally used for only 2 weeks at a time.

Side effects

NAUSEA
NERVOUSNESS
DIZZINESS
DROWSINESS
DRY MOUTH
FACIAL FLUSHING
HEADACHE
HEARTBURN
INCREASED BLOOD PRESSURE
INCREASED SWEATING
INSOMNIA
LIGHTHEADEDNESS
MUSCLE CRAMPS
TREMORS
VOMITING
CHEST PAIN

The most significant side effects are muscle cramps, nervousness, headaches, and increased blood pressure.

Muscle cramps can be avoided by drinking 1.5-2 gallons of water and consuming bananas and oranges or supplementing with potassium tablets at 200-400mg a day taken before bed on an empty stomach. Taurine at 3-5grams is a necessity in minimizing cramps.

Headaches can easily be avoided with Tylenol Extra Strength taking at the first signs of a headache.

Common Uses

Post-Cycle Therapy: Clen is used post cycle to aid in recovery. It allows the user to continue eating large amounts of food, without worrying about adding body fat. It also helps the user maintain more of his strength as well as his intensity in the gym. Diet: Roughly the same as on cycle.

Fat loss: The most popular use for Clen, it also increases muscle hardness, vascularity, strength and size on a caloric deficit. For the most significant fat loss, Clen can be stacked with T3. Diet: A high protein (1.5g per lb of bodyweight), moderate carb (0.5g to 1g per lb of bodyweight), low fat diet (0.25g per lb of bodyweight) seems to work best with Clen.

Alternative to Steroids: Clenbuterol has mild steroid-like properties and can be used by non-AS using bodybuilder to increase LBM as well as strength and muscle

hardness. Diet: A moderate carb, high protein, moderate fat diet work well.

Stimulant/Performance Enhancement: It can be used as a stimulant, but an ECA stack may be a better choice because of its much shorter half-life. Diet: To take full advantage of the stimulatory effects of Clen, carbohydrates must be included in the diet. Ketogenic diets do not work well in this case.

Precautions: Is Clen for you?

The same precautions that apply to Ephedrine must be applied to Clen, although some people find ECA stacks are harsher than Clen. It should not be stacked with other CNS stimulants such as Ephedrine and Yohimbine. These combinations are unnecessary and potentially dangerous. Caffeine can be used in moderation before a workout for an extra quick. burst of energy.

A word on Ketotifen

Ketotifen is safe antihistamine used extensively some European countries to treat asthma and allergies. It can up regulate beta-2-receptors that Clen down regulates. Basically, it allows users to extend their use of Clen for 6-8 weeks at a time. 2-3mg a day is ideal, 10mg as found in "superclen" can make users extremely drowsy. It also increases the effectiveness of Clen so doses must be adjusted accordingly. The downfall of this drug is its ability to induce extreme hunger is some people, which is not a desirable state to be in when dieting.

Cycling Clenbuterol

Most users that report bad side effects and discontinue use are those who use high doses right at the start of the cycle. The worst side effects occur within the first 3-4 days of use.

A first time user should not exceed 40mcg the first day. Increase by one tab until the side effects are not tolerable

Example of a first cycle:

Day1: 20mcg
Day2: 40mcg
Day3: 60mcg
Day4: 80mcg
Day5: 80mcg(Note: Increase the dose only when the side effects are tolerable)
Day6-Day12: 100mcg
Day13: 80 mcg (Tapering is not necessary, but it helps some users get back to normal gradually)
Day14: 60 mcgs

Day15: off
Day16: off
Day 17-Day 28: EC Stack

Example of a second cycle:

Day1: 60mcg
Day2: 80mcg
Day3: 80mcg
Day4: 100mcg
Day5: 100mcg
Day6-Day12: 120mcg
Day13: 100 mcg
Day14: 80 mcgs
Day15: off
Day16: off
Day 17-Day 28: EC Stack

What else do I need to know?

Taurine MUST be used with Clen at 3-5g daily. Clenbuterol depletes Taurine levels in the liver which stops the conversion of T4 to T3 in the liver. Taurine allows the user to avoid the dreaded rebound effect and painful muscle cramps. It's a must with Clen.

Clenbuterol should not be taken too close to a workout. It can interfere with your breathing and completely ruin your workout. When doing cardio, it's advisable to stay at a consistent pace and avoid HIIT style routines.

Do not take Clen Past 4pm and drink plenty of water; 1.5-2 gallons a day

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Steroid.com Info

Description: Is available in 10 - 20 mcg tablets or in the .016 mg/gram Ventapulmin Vet variety. Clenbuterol is known as a sympathomimetic. These hormones are taken to mimic adrenaline and noradrenalin in the human body. Clenbuterol is a selective beta-2 agonist that is used to stimulate the beta-receptors in fat and muscle tissue in the body. Clenbuterol exhibits most of its effects on the stimulation of both type 2 and 3 beta-receptors. Clenbuterol is really one of bodybuilding's most misunderstood performance enhancement drugs. It is true that it is effective in helping to burn body fat but it is often been stated that Clenbuterol is effective in causing anabolic gains and has in times even been compared to some of the weaker anabolic steroids. Books such as the World Anabolic Review, 1996, by P. Grunding and M. Bachmann state incorrectly that, "its effects, however, can by all means be compared to those of steroids. Similar to a

combination of Winstrol Depot and Oxandrolone...." These statements are inaccurate and misleading to say the least. A lot of these claims as to the anabolic effects of Clenbuterol are derived from studying the effects of Clenbuterol on livestock. Clenbuterol is effective in increasing muscle mass and decreasing fat loss in animals. The problem with the variation in anabolic effects between humans and livestock is that livestock have an abundance of the type 3 beta receptors whereas humans have little if any of the type 3 beta receptors. These beta-3 receptors increases insulin secretion and sensitivity, causing more glucose and amino acids to be transported into skeletal muscle thus causing the anabolic effects that we, humans, just aren't seeing. As Dan Duchaine stated in his Muscle Media article on Clenbuterol, "In those animal research studies showing an anabolic effect from Clenbuterol, it's my guess the anabolism happens specifically when the beta2 receptor stops working. At that point, the beta3 increases and causes the anabolic effect through insulin mechanisms." Since humans, again, have either very little or no beta-3 receptors, there is no chance of this anabolic effect. Just another of the studies where everyone assumed that what works in animals must work in humans. This is just simply not the case with Clenbuterol. Clenbuterol does work effectively as a fat burner though. It does this by slight increases in the body temperature. With each degree that the temperature in your body is raised from the use of Clenbuterol, you will burn up approximately an extra 5% of maintenance calories. This makes it effective as a fat burner. Your body will fight this by cutting down on the amount of active thyroid in the body as well as through beta-receptor down regulation, which explains why you only have a limited effective period to take Clenbuterol. While I am on the subject of beta-receptor down regulation, I would like to dispose of another myth. This involves the two on/two off cycling theory that I believe was originated by Bill Phillips in the Anabolic Reference Guide and has somehow made it's was into every other steroid book since then including the WAR and Physical Enhancement with an Edge. The two on-two off theory simply will not work because of one main reason: the half life of Clenbuterol. This 2-on/2-off idea was a THEORY ONLY, not by a doctor or scientist, and not based on specific knowledge of Clenbuterol, but derived by imitation from other drugs with shorter half lives.

Clenbuterol has been reported as having a half life of about 2 days, but that is not actually correct, since it has biphasic elimination, with the half-life of the rapid phase being about 10 hours, and the slower phase being several days. Supposedly, this is one of the reasons the FDA never approved Clenbuterol as an anti-asthmatic drug...the FDA frowns on drugs with long half-lives if drugs with more normal half-lives are available. So with a 2-on/2-off cycle you never have time to get enough of the Clenbuterol out of your system for this theory to be reasonable. In actuality, it probably hasn't even dropped to 50% of your peak concentration before you are taking the drug again. With this all taken into account, there is no reason to think that this cycling would significantly reduce the problem of receptor desensitization. A more reasonable approach would be either one week on, one week off, or alternately, two weeks on two weeks off. The two week cycle has the disadvantage of a "crash" period afterwards. This crash period can be helped with the use of ephedrine to lessen the lethargy that you will experience.

If you are interested in taking Clenbuterol for anything other than fat loss then you might

as well stay away from this compound. There is a lot of talk as to how Clenbuterol compares to ephedrine as well. Most "experts" feel that clen gives a better bang for the buck than the ECA stack. It should be noted that clenbuterol's results and effects are much shorter lived. They work through very similar mechanisms. Both products stimulate the beta-receptors but Clenbuterol seems to be a more refined version, called a second generation beta-agonist drug, than ephedrine. Clenbuterol targets the proper receptors, being the beta-2 and 3 receptors than ephedrine more specifically which should in theory make Clenbuterol more effective of a fat burner. Again, most of the so called "experts" say that Clenbuterol is more effective than ephedrine. I, personally, get worse results with clen vs. the good old ECA stack. Clenbuterol also didn't blunt my hunger either and I ate more while taking it as well. I also seem to get much better effects out of cytomel as a fat burner as well. Even better than the ECA stack or Clenbuterol. But, again, that is my personal opinion. Effective Dose: 80-140 mcgs. / Day in split doses throughout the day. Anything over 140 mcg a day is overkill since the beta receptors can only take so much of a product and then more is just wasteful.

Street Price: $.50 - 1.00 / tab. fairly inexpensive in Mexico though. Spiropent is currently going for about $7.50/box, Novegam for $5.25/box, and Oxyflux for about $3.30/box.

Effective Dose: A few drops under the tongue and not used for but a few weeks at a time.

Street Price: Not a clue. Too hard to find. Even if I could find it I would not buy it.

Pharmaceutical Phenotype Enhancement
by Loki

Clenbuterol Part I

First 'The Man' marginalized ephedra. Then we saw the realization of a second supplement ban—one which will effectively deprive the mainstream bodybuilding community of its most-preferred anti-catabolic ancillary: the pro-hormone or pro-steroid. So just how the hell is a dieter supposed to preserve lean body mass these days while languishing on laughably low calories? Well, aside from investing in our beloved LeptiGen and shipping out for real gear, it seems like a lot of would-be-chiseled chaps are taking a new interest in the age-old diet drug Clenbuterol (1-(4-Amino-3,5-dichlorophenyl)-2-tert-butyl-aminoethanol), a sympathomimetic beta2 adrenergic agonist most commonly used in veterinary therapeutics and livestock doping. But is this recent, glaringly rekindled curiosity in clen leading would-be and first-time users to the right drug? Or for that matter, is clen a safe drug? So, without further ado, I think it's time *M&M* gave ephedra's "wonder-cuz'" its full-on, discerning attention.

Clen in Context: Basic Pharmacology

I throw around the word sympathomimetic a lot to compensate for my chronically low self-esteem. In the forums, in my sleep, during sex—it's just such a dazzlingly erudite sounding word. And using it makes me feel special. And while you yourself may not feel the inclination to use it colloquially any time soon, if phrases like "nutrient partitioning" or "fat loss while preserving muscle" pique your interest, you should care about how this class of compounds works and what those workings result in.

Chances are you wouldn't be reading this article if you didn't, so quickly: sympathomimetics (take "sympath" from 'sympathetic nervous system,' throw an 'o' on there, and add 'mimetic' [i.e. 'to mimic'] and you're in business) are a class of drugs that affect the sympathetic nervous system (SNS), either by prompting central catecholamine (NE/NA and E/A) release or by peripherally mimicking the effects of those same endogenous hormone/neurotransmitters. Most of a sympathomimetic's pharmalogical interplay occurs via interaction with beta andrenoreceptors. Ephedrine is a sympathomimetic, norephedrine is a sympathomimetic, H.E.A.T. is a sympathomimetic, and boy is clenbuterol ever a sympathomimetic. There are also many others.

With clenbuterol specifically, what we see is **both** forms of sympathomimetic activity. Clen synthetic mimics some of norepinephrine's and epinephrine's effects in specific outreaches of the SNS, **and** centrally exerts these effects in skeletal muscle, adipose tissue, and in an often-overlooked third area: the brain. Yes, that's right: clenbuterol crosses the blood brain barrier, and is able to readily activate certain central adrenoreceptors (1).

Virtually all of this peripheral mimicking occurs at the beta2 adrenergic receptor, which is the reason clenbuterol is characterized as a beta2 specific agonist. When it interacts with these receptors in muscle, clenbuterol is able to catalyze Cyclic Adenosine Monophosphate (cAMP) production, a second-messenger signal transducer which regulates rates of glycogen decomposition, protein synthesis, and lipolysis (among many other things). What distinguishes clenbuterol prominently from ephedrine is its specificity, potency, and duration of effect.

Ephedrine, whether you already knew it or not, has very little direct activity in muscle or fat. Rather, it stimulates central sympathetic nerve terminals, thereby inciting an indiscriminate release of NE/NA (and to a lesser extent, epinephrine/adrenalin), which then relays across the entirety of the SNS. This makes ephedrine a primarily indirect and non-specific sympathomimetic, as it effectively delivers a mild 'catecholamine carpet-bombing' to all your various beta receptors (beta1, beta2, atypical beta3, and putative, atypical beta4). It is also this mechanism that gives ephedrine its long-term pharmacological viability: although not very set-point friendly, it will nonetheless

continue to indirectly agonize adrenergic receptors along your SNS, even after months of continual use.

Clenbuterol is somewhat of a different beast. As mentioned earlier, it is able to prompt a small degree of catecholamine release from central adrenoceptors, as well as interact directly with the beta2 receptor in a dose-dependent manner with a potency that far exceeds the resultant effects of ephedrine administration.

More Technical Stuff on Clen's Workings than You Could Ever Possibly Want to Know: Lipolysis

Nutrient-partitioning junkies, your patience is about to be rewarded. Now that we've established some context, it's time to move on and discuss 'the goods.' It's time to discuss the bad-ass lipolytic and repartitioning effects of clenbuterol in vivo. And it goes a little something like this. Following administration, clenbuterol avoids first-pass metabolism (it's oral bioavailability ranges between 89-98%) and doses typically reach peak plasma levels roughly two hours after a dosage is ingested. This peak will then stabilize and continue for four additional hours (2). Eventually, roughly 50% of ingested clenbuterol will undergo metabolization into its four primary metabolites; the remaining half will be excreted intact, without metabolic breakdown (3). This biphasic elimination lends clenbuterol a veritable half-life that clocks in at just under thirty six hours.

Once it gets to work clenbuterol, as I already mentioned, binds to cellular beta2 receptors. Intracellularly this will increase cAMP (4), which then binds to regulatory subunits of protein kinase A, causing the release of its catalytic subunit. This process activates the enzyme HSL (hormone sensitive lipase), which hydrolyzes triglycerides, breaking them down into glycerol and fatty acids to allow for beta oxidation.

Now, as you can probably guess, one of the facets to clenbuterol that makes it such a potent lipolytic drug is that it exerts its beta-agonism steadily and continuously. If ephedrine is 'hit-it-and-quit-it,' clenbuterol is a friggin' marathon man when it comes to stimulation. Clen isn't very cuddly though, so all you high-maintenance bodybuilders are just plum out of luck.

Clenbuterol is undeniably potent at its target receptor. However, clenbuterol cannot be said to own ephedrine (particularly when combined with caffeine) outright for fat loss. Remember, since clen is primarily direct-acting on a cellular level, it can't prompt the same kind of NE-induced hypophagia (loss of appetite) as ephedrine, which has proved to be an essential pharmacological component in its ability to further weight loss (5). Individuals looking to use clenbuterol for weight loss need to keep this in mind:

clenbuterol partitions energy intake, but it will not aide in regulating or helping to decrease it, hence my recommendation that those planning to cut on clen also look into ancillary appetite suppressants.

There is also the prevailing theory in a lot of bodybuilding circles that clenbuterol actually raises metabolic rate by increasing endogenous thermogenesis. So let's explore the purported calorie-burning properties of clenbuterol. In animals, although clenbuterol increases thermogenesis in mutant rats (genetically obese Zucker rats), multiple studies have demonstrated that in normal rats—even those administered rather hefty dosages of the drug--- clenbuterol "did not affect energy intake [or] energy expenditure" (6,7).

In human studies, the direct infusion of the related beta2-specific agonists salbutamol and terbutaline in lean men caused a modest increase in whole body energy expenditure and respiratory exchange ratio—an increase of 0.6 kJ/min in terms EE adjusted for fat-free mass (8). In other words, the guys getting heavy-duty beta2 adrenergic stimulation would have ended up burning about 200 extra kcals over a twenty four hour period. So in other words, thermogenesis was enhanced, but not so much to suggest that clenbuterol has strong calorie-burning properties of its own. Interestingly enough, in the same study the researchers noted that:

during beta2-adrenergic stimulation, the increases in energy expenditure and plasma nonesterified fatty acids and glycerol concentrations were reduced in the obese group. Furthermore, lipid oxidation significantly increased in the normal weight group, but remained similar in the overweight group… [this] data suggests that beta2-adrenoceptor-mediated increases in thermogenesis and lipid utilization are impaired in the obese (8).

In other words, if you're still in plus-size pants, you're out of luck: clenbuterol isn't going to help you lose a whole lot of weight, because your obesity-train-wrecked metabolism just ain't havin' that (9). Plus, given the effects of beta adrenergic agonists on heart rate contraction, the use of clenbuterol in significantly overweight individuals may pose significant danger to the user (10,11).

And again, the take home message is the same: when dieting, nutrient-partitioning definitely matters, but in the end it still comes down largely to energy expenditure vs. intake, and clen is a calorie re-distributor, not a burner.

Clenbuterol and its Interaction(s) With Your Mammoth Guns

If you get one sentence out of this section on clenbuterol and skeletal muscle, please let it be this: clen is never going to get you big, but it is extremely good at keeping you big once you get there. Yes, I know clenbuterol is wicked-anabolic in Dawley-Sprague hyperphagic wombats, but you are a human, and the amount of clenbuterol it would take for you to see a genuine anabolic effect would also put you in a coffin, so let's just let that one go.

Now, I said clen's not anabolic, but it certainly does have positive ramifications for protein synthesis, primarily through the beta2s, cAMP, and its ability to mitigate Ca2+-dependent proteolysis in skeletal muscle (12). A critical component to its full effect is its repartitioning properties. As stated earlier, clenbuterol is exceedingly good at liberating fatty acids from adipose tissue. But, more than that, clenbuterol exerts this effect in tandem with large scale, itself-induced skeletal muscular insulin resistance (13).

Now, when you're a type II diabetic, this isn't so hot. However, in a healthy bodybuilder using a strong sympathomimetic you basically have the best of all worlds: plenty of free-fatty acids getting released for oxidation in muscle, plenty of insulin-resistant muscle to feast on them, and pretty much all consumed calories getting spared for muscle retention and protein synthesis. Granted, there's very little good research on human skeletal muscle in the presence of clenbuterol (particularly when it comes to athletes), but reasoned inference and extrapolation certainly paints a pretty convincing picture that clenbuterol is significantly anti-catabolic.

For starters, human research with ephedrine and caffeine has demonstrated that indiscriminate, weaker beta-adrenergic agonism significantly improves protein deposition and preserves lean body mass during periods of caloric restriction (14). Also interesting was the researchers' discovery that the ephedrine and caffeine mixture wasn't attenuating skeletal muscular breakdown, but was in fact *accelerating* protein synthesis. This was proved clinically by 3-methylhistidine examination, an index for skeletal muscle breakdown.

In the sympathomimetic group, an increase in nitrogen balance was demonstrated independent of 3-methylhistidine, which means the ephedrine was actually helping to synthesize lean tissue at a faster rate, and thereby counteracting the increase in diet-induced catabolism (15). See? I wasn't lying when I said clenbuterol could be anabolic; you just can't take a high enough dose to get an anabolic degree of protein synthesis augmentation without ending up in the ER long before you could get your shaky ass anywhere near a squat rack.

Nonetheless, because of its pharmacology, clenbuterol is currently recognized in the

scientific community as a valid remedial treatment for muscle-wasting conditions (16,17). In rodents, clenbuterol also actively inhibits glucocorticoid-induced muscle atrophy, and one can speculate that it may also exert similar anti-glucocorticoid properties in humans as well (18). Clenbuterol, by virtue of its beta-agonism, may also even be more effective at reducing glucocorticoid activity than that though, as it has been demonstrated that beta-receptor antagonism increases the release of adrenocorticotrophin (ACTH) in humans subjected to stress (19). For those unaware, ACTH is a pituitary hormone that stimulates cortisol secretion, which means there is a possibility that beta-receptor *agonism* may in fact be able to prompt the opposite: a decrease in ACTH release in response to stress.

All told, clenbuterol is pretty much the bomb and the shiznit as far drugs go for preserving muscle during periods of energy restriction through a number of different pathways. Oh but there are just a few more things…

But I'm Too Sexy To Die… (Side Effects and Precautions Pt. I)

By now it should be clear that clenbuterol is a powerful drug. And with all powerful drugs, there are consequences, 'cause life just sucks like that. So for those of you about to get all 'clenbutaholic' with your research chemicals, here's a little info I counsel you to take to heart. In fact, speaking of hearts, let's examine yours in relation to clenbuterol, because there definitely is some cause for concern.

For starters, there are more rodent studies under the sun that show clenbuterol use can cause significant cardiac hypertrophy—so many in fact that I'm not even going to bother citing them. Just type "clenbuterol" and "cardiac hypertrophy" into Google if you don't believe me; no lie, it's a little unsettling. However, clenbuterol also kills fat cells (adipocyte apoptosis) in rodents too, and it sure doesn't in humans, so take that animal data for what it's worth. Unfortunately though, things don't look too much better when we move up the evolutionary chain and start looking at hearts in good-ole' human beings either.

For example in 1998, the internal medicine outpatient clinic at the University of Alabama Birmingham received a walk-in from a previously healthy 26-year-old bodybuilder complaining of significant chest pains. The man, who had a history of moderate anabolic steroid use but who had not used any steroid preparations in the weeks leading up to his visit, revealed that he had continuously been using clenbuterol for nearly a month [Loki's note: idiot]. During check-up, the man turned out to be completely fit and healthy with the only exception being a significant amount of left ventricle (heart) hypertrophy and cardiac dyskinesias (meaning distortion of muscle [in this case smooth muscle] activity)(20).

In fact, between 1988 and 1998, eight cases of medically-diagnosed cardiac hypertrophy have been reported in drug-using bodybuilders within the United States (21,22). We can assume many more went overlooked or unreported. Still, because of the steroid outlier (which could also be a potential factor in the pathology—or perhaps even the outright cause), the medical community has been unable to isolate clenbuterol's true role in contributing to these instances of myocardial infarcation (20). Still, the researchers who have examined this phenomenon arrived at a conclusion that should give most clen user's pause of thought. Namely that:

We suspect there may have been a synergistic role between the anabolic steroid and clenbuterol. Hypothetically, the anabolic steroid may have caused cardiac hypertrophy, coronary artery spasm, or thrombosis. The clenbuterol may have precipitated ischemia by producing intermittent tachycardia. Alternately, clenbuterol may have contributed primarily to the cardiac hypertrophy...(20)

Furthermore, clenbuterol ingestion (particularly excessive ingestion) has also been documented to cause tachycardia (sudden, rapid racing of the heart)(20,23,24), hypokalemia (23), hypophosphatemia (23), potassium depletion (24), taurine depletion (25), headaches (24), tremors (24), and vertigo (24). Now, it should be noted that the more severe of the aforementioned conditions have only been demonstrated in instances of clenbuterol overdose and are thus not directly applicable to carefully monitored doses within the 20-100mcg range. Nonetheless, clenbuterol is definitively a "big kid sympathomimetic," and not a drug that lends itself to immoderation, recklessness, or just outright stupidity.

And for now, I'm afraid that's just going to have to do it for Part I of our comprehensive look at Clenbuterol. Next issue we'll get into receptor down-regulation, clenbuterol, the brain, and neuroprotection (for all my Chemically Correct homies), cycling, stacking recommendations, and potential novel uses for clenbuterol in the treatment of injuries and various diseases and conditions.

Questions or comments on this article? Post them in the Avant Labs Forums for live feedback from the author, as well as the Mind and Muscle staff and fellow readers!

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