

Clenbuterol

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[Clenbuterol](#)

([Clen](#))

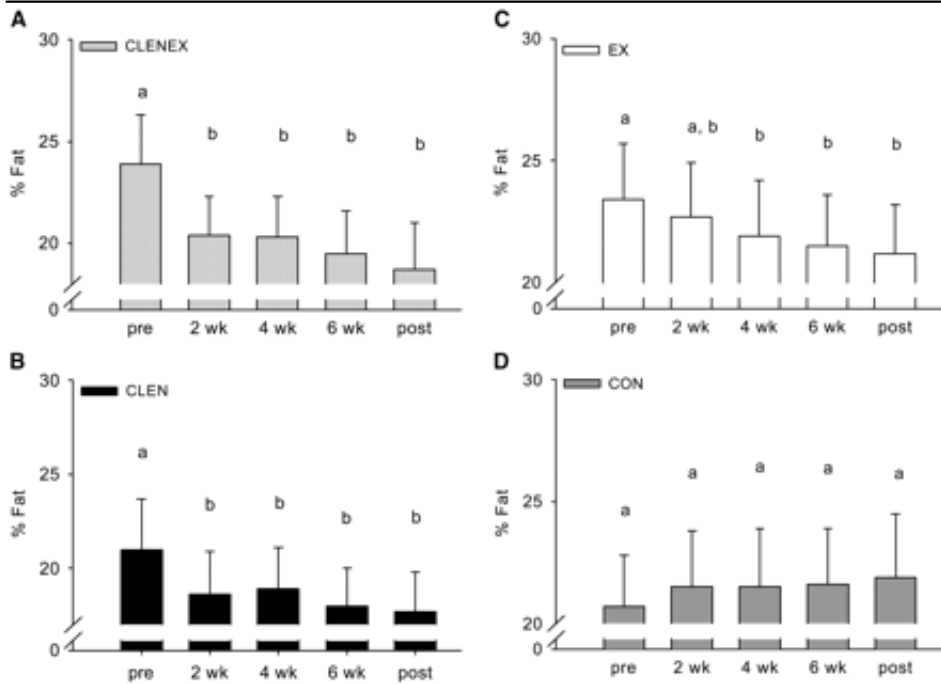
[Clenbuterol](#) ([Clen](#)) is a selective beta-2 agonist/antagonist and a bronchodilator. What this means, is that it stimulates your beta-2 receptors. Of great importance, is that [Clenbuterol](#) is a selective beta-2 agonist (because it works selectively on the beta-2-adrenergic-receptors), right? The thing is, [Clenbuterol](#) is selective...like hitting a tack (the tack being your beta-2 receptors) with a small hammer (the hammer being the [Clen](#))...thus, it hits the beta-2 receptors selectively. Sorry if that seems repetitious, but it ´s very important to understand that fact before we move on. Since [Clenbuterol](#) has very little beta-1 stimulating ability, it has the ability to reduce certain kinds of airway obstruction without much in the way of cardiovascular effect (more about that later), and this is why it is used as an asthma medication.

So what exactly dose a stimulant like [Clen](#) (or [Ephedrine](#)) do when it stimulates those Beta Receptors? Well, it serves to increase your body temperature a bit by increasing heat production in the Mitochondria, increase your basal metabolic rate, and decrease your appetite (1). This partly explains how Beta-2 agonists directly stimulate fat cells and increase lypolysis (fat-loss)(1)(13). And also, because it is a Beta-2 agent, [Clen](#) can decrease [Insulin](#) sensitivity (2), unfortunately.

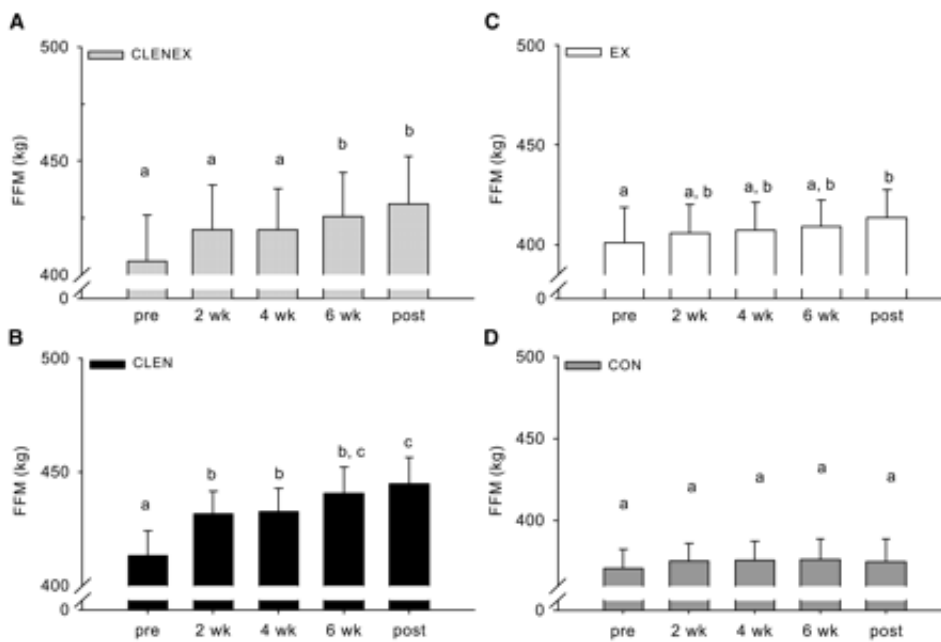
[Clenbuterol](#) Fat Loss

[Clenbuterol](#) is a very effective repartitioning agent, and this is what it ´s most often used for in athletic circles. It will increase your ratio of Fat Free Mass (FFM) to Fat Mass, by decreasing your Fat and possibly increasing your FFM (3). Lets quantify that a bit:

In one study, horses given a semi-reasonable dose of [Clen](#) (slightly over 1mcg/lb x2 a day) and exercised for 20mins, 3x a week (I suppose they were Mentzer disciples) had significant decreases in %fat (-17.6%) and fat mass (-19.5%) at week 2, which was similar to [Clen](#) given to horses who didn ´t exercise; in contrast, the exercised group had a different FFM response, which significantly increased (+4.4%) at week 6 (3). Week 6! Here ´s a chart illustrating the changes in % of Body Fat experienced in the various test groups, followed by a chart showing the increase in Fat Free Mass experienced by the same groups:



Changes in percent body fat (%fat) over time in [Clenbuterol](#) and exercise (ClenEx; A), [Clenbuterol](#) only (Clen; B), exercise only (Ex; C), and control (Con; D) groups. Means with different letters (A and B) are significantly different.



Changes in fat free mass (FFM) over time in ClenEx (A), [Clen](#) (B), Ex (C), and Con (D). Means with different letters (a-c) are significantly different.

I think those charts should clearly illustrate the repartitioning effects of [Clen](#), even though it is known that it's effects on animals are typically much more dramatic than in humans. There's still no doubt about it, in my mind [Clen](#) will help you lose fat and gain muscle.

So Let's re-examine that first point I made [Clen](#) vs. [Clen](#)+ exercise produce roughly the same results for the first 2 weeks! This tells me that the 2 weeks on and 2 weeks off schedule for [Clen](#) dosing is far from optimal, and if

you want the quasi-anabolic effect from the [Clen](#), it´ll take more than 2weeks on (6 weeks apparently). In addition, since [Clen](#) alone is similar to [Clen](#)+ exercise for those first 2 weeks...why would you ever use a 2on/2off protocol? Keep in mind that animal responses to beta-agonist/antagonists differ a bit from ours but I´m sure that you get the idea that 2on/2off is not a great dosing protocol. If I were using [Clen](#), I´d be using it for 6-12 weeks at a time, if I expected to get maximum results from it, but certainly, the most dramatic effects on fat loss appear to be in weeks 1-2. The reasons for the further increase in FFM around week 6 despite no changes in %fat or fat mass is not easily explained... It might be that [Clenbuterol](#) can increase FFM through another nonreceptor-mediated pathway, which would be very good for us, since the anabolic effects would also be applicable in humans, despite the fact that animals often respond more dramatically to beta-agonist/antagonists, due to receptor properties.. However, [Clenbuterol](#) is highly lipophilic and can also enter muscle tissue (12), so that could indicate a possible mechanism of work. Maybe that would explain the significant increase in FFM of 13 kg in at 8 wks in the study? Certainly, muscle protein synthesis (MPS) must be a part of it, since [Clen](#) will increase MPS in your body (17)& But it has even been speculated that the growth-promoting effect of [Clenbuterol](#) may be specific to muscle and that the drug may act in a not-yet-understood manner which circumvents (!) the physiological mechanisms responsible for the control of muscle growth (13). This may mean that [Clenbuterol](#) can help blast you past "sticking points" in your training by circumventing the usual mechanisms by which anabolism is experienced! It is of note that both muscle composition and fiber size has been shown to increase with administration of [Clen](#) (14).

In any case, Clearly the results you want to reproduce for yourself are those to be gained by [Clen](#) + exercise, for 6 weeks or more. This type of dramatic anabolic effect hasn´t been confirmed in human studies (8), but the anabolic effects of [Clen](#) in animal (specifically equine and rodent) studies are clearly quite astounding.

Now that I told you how great [Clen](#) is; I´ll tell you how to take it&

[Clen](#) has a biphasic elimination, which means that it is technically reduced in your body in 2 different stages. This isn´t particularly important, as a recent study has shown that for most intents and purposes [Clen](#) concentrations in the body decline with a ½ life (approximately) equivalent to 7-9.2hours and again up to as much as 35 hours later(4)(5). If you´re really interested, though [Clen](#) technically declines biphasically at 10 and then 36 hours. But really, in our little world, where we use ½ life to tell us when to take our next dose, who the hell is going to take [Clen](#), then a dose 10 hours later, then a dose 36 hours later? We´ll stick with the earlier 7-9 hour ½ life for dosing purposes, and take our [Clen](#) every 3.5-4.5 hours that we´re awake, stopping early enough to still be able to get to bed. [Clen](#) can, in some people, cause insomnia (and as with all stimulants, can cause anxiety in some). Recently, it´s become popular to take a whopping dose of [Clen](#) in the morning, and that´s it for the day. There´s nothing wrong with this, I guess, but I´d rather not go through that kind of roller-coaster of sweating and shaking until it wore off.

[Clenbuterol](#) Cycle

Based on its rate of elimination from the body, and how much is usually needed to be effective for athletes, my recommendations are the same for both men and women. You´ll need to take 20mcgs upon rising, and then repeat that same dose again later in the day, and then once again in that day (if you find you can tolerate the effects). So you´ll start with 20mcgs, and then repeat that dose 2 more times that same day if you can tolerate it ([Side Effects](#) will determine this hand shaking, sweating, etc& classic stimulant sides). Then you can start increasing the dose gradually. Personally, I wouldn´t work my way up to more than 200mcg/day. 60-120mcg/day is an average dose. And keep your Blood Pressure at (or under) 140/90, while on [Clen](#), just to be safe. If you go over that, lower the dose. You´ll also want to know your body temperature, upon rising, for the week before you start taking your [Clen](#), and then monitor it (again, as soon as you wake up) throughout your [Clen](#) regimen. When it returns to the level it was at before you began taking the [Clen](#), you´ll need to start taking your Benadryl or [Ketotifen](#), as the decrease in Body Temperature back to original levels indicates the thermogenic effect is beginning to decline.

[Clenbuterol](#) can also cause a downregulation in testicular androgen receptors and in pulmonary, cardiac and central nervous system beta-adrenergic receptors(6.) possibly making steroids less effective (if there is androgen receptor downregulation elsewhere as well, then it´s highly probable) while you are on [Clen](#); but definitely making [Clen](#) less effective as time goes on and you keep taking it. To counteract this, you can take some [Ketotifen](#) every

3rd or 4th week that you remain on [Clen](#). It's a prescription anti-histamines, so it'll make you drowsy (take before bedtime). Basically, the way this works is to reduce beta-2 receptor activity, and restore receptor function (15).

Another option, if you are worried about receptor downgrade, is taking Benadryl, at around 50-100mgs/night before bed (every 3rd week or so, for that week). Benadryl is sold as an anti-histamine in the United States, and/or a sleep aid elsewhere in the world. However, Beta receptors are embedded in the cell's outer phospholipid membrane. The stability of the membrane has a lot to do with the proper function of the receptors. Methylation of the phospholipids is stimulated by the binding of beta agonists to their receptors. Methylated phospholipids are foreign to the body, and when the body recognizes them as foreign, it breaks them down with phospholipase A2. This changes the structure of the outer membrane which results in desensitization of the beta receptors. On the other hand, agents that inhibit phospholipase A2 slow desensitization.

Cationic amphiphilic drugs are known for their ability to inhibit phospholipase A2. Benadryl (diphenhydramine) is a cationic amphiphilic drug.

Ergo, Benadryl slows desensitization of Beta receptors (i.e. Upgrades them) by inhibiting phospholipase A2, which is the enzyme that breaks down methylated phospholipids, and this action in turn keeps the phospholipid membrane stable, and thus keeps the receptors functioning properly. (7). This will allow you to use [Clen](#) for much longer and it'll still have the same effects. Also, since Benadryl is an anti-histamine, and histamines have a direct effect on beta-adrenoreceptors (not just Beta-2's but all of them), using an anti-histamine will have a direct effect on reducing beta-receptor stimulation (16), and thus upregulating your beta-receptors.

[Ephedrine](#)

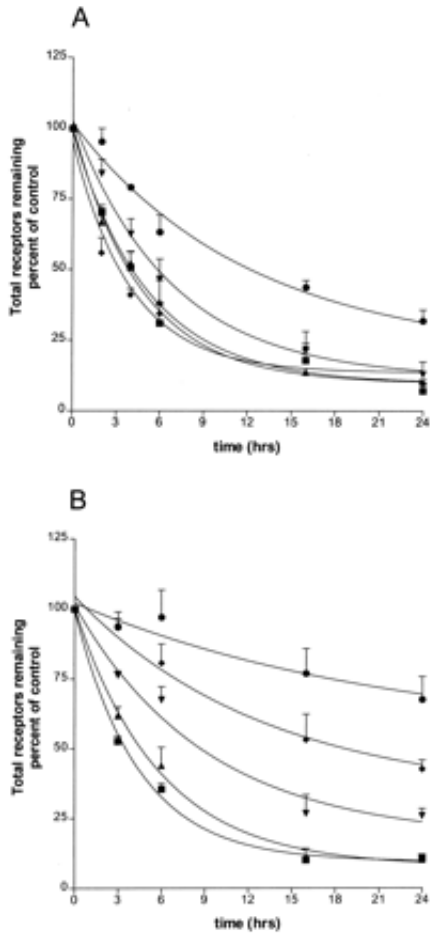
Since we're speaking about beta-receptors and upregulation, here, let me address the claim that you can use [Ephedrine](#) (or the ECA stack), alternating with [Clen](#), in order to avoid receptor downgrade. I'm not sure where this rumor came from, but it is totally incorrect.

To dispel this myth, let's examine [Ephedrine](#) for a second. Remember when I said that using [Clenbuterol](#) to stimulate the beta-2 receptors is like hitting a tack with a hammer? Well, [Ephedrine](#) is like a sledge-hammer, it hits the beta-2's and everything around them. That's because it's not selective, but rather it stimulates other receptors to a great degree as well.

Anyway, one of those receptors that [Ephedrine](#) hits is the Beta-2 (yeah...the same one as [Clen](#)). As you can see from the graph below ([Ephedrine](#) is represented by the the solid circles), it reduced Beta-2-AdrenergicReceptor (what we call, in laymens terms, the "Beta-2 receptor") levels to 32% of the control level after 24 hours. Read this again:

[Ephedrine](#), in this study, reduces Beta-2 receptor levels to 32% of control after 24 hours.

(see the solid circles in this graph represent [Ephedrine](#))



Granted, it's not perfect, it's not in vivo, etc...But there's no denying [Epitadrine](#) will downregulate beta-2 receptors....ergo you will not be able to use it on the weeks in between your [Clen](#) to upgrade your receptors.

[Clenbuterol Side Effects](#)

Also, bear in mind that [Clenbuterol](#) has some [Side Effects](#). It isn't great for your heart, and can cause some issues there (enlargement of ventricles, etc,) but most studies showing [Clen](#) to cause heart problems are with animals, and even though the dosing is almost similar to what humans take (in some studies it's within the range of what would be double of a large human dose...) it's important to remember that animals have more beta-2 receptors and they cause certain event chains that humans' beta-2 receptors may not. [Clen](#) causes cardiac hypertrophy and cardiac necrosis (cell death) to some degree, in some cases. Again though, many studies showing the more significant, possibly irreversible, heart problems are with mg dosing. We humans take [Clen](#) in mcg doses.

If we want to duplicate those "therapeutic" levels of [Clen](#) seen in the more conservative studies, we'd still be taking just over 1mcg/lb of bodyweight, twice a day. I'd suggest a bit less than half of that dose, however.

Performance issues with [Clen](#) also vary. Some studies show reduced exercise (cardiovascular) performance with [Clen](#) (9), while some show that [Clen](#) can alleviate exercise induced asthma (10)! Clearly, this compound will have different effects on different people, and I suspect that a lot of it is sports specific. Many bodybuilders claim that [Clen](#) makes it difficult for them to do cardio, yet I can play a full game of rugby on it. You need to figure out how it affects you, and tailor your dose personally.

Finally, this brings me to the issue of cramps while on [Clen](#). I don't get them. My friends don't get them. Most of us are athletes who use [Clen](#) during the season as well as the off season, and one of my friends even claims that it gives him more "wind" (cardiovascular stamina). Take on enough water every day and you should be fine. If

you ´re really concerned, you can take some extra minerals and taurine, since [Clen](#) depletes taurine (11) as do most if not all beta-agonists. I don ´t take anything more than my usual vitamins and minerals.

1st Graph Reference:

ASPET Journals, Vol. 58, Issue 2, 421-430, August 2000

Kinetic Analysis of Agonist-Induced Down-Regulation of the 2-Adrenergic Receptor in BEAS-2B Cells Reveals High- and Low-Affinity Components

Bruce R. Williams, Roger Barber, and Richard B. Clark

2nd set of Graph references:

J Appl Physiol 91: 2064-2070, 2001; 8750-7587/01

Chronic administration of therapeutic level [Clenbuterol](#) acts as a repartitioning agent

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