Subject: Fin microdosen
Posted by DHT96 on Fri, 23 Jun 2017 16:36:28 GMT
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Hier ein Beitrag, den ich auf englisch geschrieben hab. Was haltet ihr davon?
Kann das jetzt leider nicht ohne viel Aufwand ordentlich formatieren.

After thinking way too much about finasteride the last few weeks, I decided that I will probably never even try the full dose, but will start microdosing immediately. I just got my most relevant blood levels tested and will start taking 0.03mg per day next week. Oh, before the haters start to cry: I don't think that reducing the chance of getting sides wont also reduce the benefits. It's a compromise, like it is so often in life. But I still think, it makes much more sense than doing nothing or cutting out creatine.
I actually think the ratio of good and bad effects almost stays the same for a given individual when taking any reasonable dose (0.02mg-5mg).

In this thread I want to collect everything about taking finasteride in the safest way possible. So please feel free to contribute. I will either edit this post or post it again, if I find out something new.

The practical steps can be found below.

This was my first thread about fin:
https://www.reddit.com/r/tressless/comments/6i1m57/my_thoughts_on_finasteride/

REASONS FOR MICRODOSING:

the relatively flat dose-response-curve is important, but it's not the reason
I don't want to risk PED, personally. I hate conspiracy theories and sensationalists, but in this case there really seems to be a discrepancy between studies and the real life, even if you take the "vocal minority" into account. Long-term a functioning dick is more important than hair. PED is inacceptable. Period. I also came up with the argument that you don't need a functioning dick, if you can't even use it because of your baldness, but this is sluthate-level incel pessimism. But I admit that I'd roid the fuck up if I'd suddenly go completely bald. But this is discussed in other threads.
Inhibiting DHT below a certain threshold will SLOW MPB down and is thus better than doing nothing. (I also started using POS, Biotin, Keto and Minox, but srs.. for most people those are analogous to creatine for muscle growth while 5ARI's are steroids)
there's hope that there will be an other treatment sometime, so slowing down MPB gives us a bit more time
even if you don't experience extreme sides that force you to stop the treatment, you might severely reduce your quality of life, energy levels, cognition etc. Blocking almost all DHT to stop MPB seems crazy.

DOSE-RESPONSE-STUDIES
Drake et al: The effects of finasteride on scalp skin and serum androgen levels in men with androgenetic alopecia - http://www.jaad.org/article/S0190-9622(99)80051-6/abstract
Steiner: Clinical Pharmacokinetics & Pharmacodynamics
https://link.springer.com/article/10.2165/00003088-199630010-00002

Roberts et al: Clinical dose ranging studies with finasteride, a type 2 5?-reductase inhibitor, in men with male pattern hair loss - http://www.sciencedirect.com/science/article/pii/S0190962299800528
Kaufmann: Finasteride, 1 mg (Propecia), Is the Optimal Dose for the Treatment of Men With Male Pattern Hair Loss - http://jamanetwork.com/journals/jamadermatology/article-abstract/477940
did I miss one?

DOSAGE
the studies are a bit contradicting IIRC
the most interesting study (Drake et al) shows that scalp skin DHT levels (hard to measure) were reduced by 14.9% and 61.6% with 0.01mg and 0.05mg, respectively and that serum DHT levels declined by 49.5% with 0.05mg. This was after 42 days of daily treatment, so the accumulative effect is included here, as opposed to the study measuring the effects of a SINGLE dose.
Problem: why did DHT decrease by 13% in the placebo group? However, this shows that after just a few weeks 0.05mg approaches the efficacy of 1mg. With 0.01mg serum DHT was only decreased by about 5% - not significant. -> 0.01mg is not enough, 0.02-0.04mg seem to be optimal for our goal.
Roberts et al: 0.01mg is definitely not enough, hair count mean change is negative (although slightly less [statistically significant] negative than placebo, so even 0.01mg slows MPB a little bit). Serum DHT levels decreased by 10.8 +- 4.2% with 0.01mg after 6 months. So 10% serum DHT reduction is not enough. (Since the next dose is 0.2mg and thus too big for microdosing the rest of the study isn't relevant. But it's interesting that after 12 months the mean hair count was almost 1/3 bigger in the 1mg group than in the 0.2mg group. Microdosing seems to be worse for regrowth).
If there's only a minimal accumulation going on (very likely), even 0.04mg could be too much, cause this dose would approach the efficacy of 0.05mg after some time, which long-term approaches the efficacy of 1mg.
The dose-response-relationship is actually pretty normal if you only consider a SINGLE dose: http://www.propeciahelp.com/forum/download/file.php?id=101&sid=d02fe4b5d83c0ef01fc6a0436f7e8884 Only the very slow production of new 5ar is responsible for the flat dose-response-curve, if you consider a longer period:
https://www.hairlosstalk.com/interact/proxy.php?image=http%3A%2F%2Fi.gyazo.com%2F8fe9b19135330e0d40b267304f770c54.png&hash=b13953d85bd4c8973aa73105d7b26247
For the effect on DHT supression, fin's half life isn't really important. Its blood levels peak 2-3hrs after taking it and then decrease rapidly, but DHT remains supressed.
Bigger microdoses do it faster, but they all approach the efficacy of 1mg after just 14 days.
I don't know from which study this chart is from, but it shows only 25% reduction for 0.125mg: https://www.hairlosstalk.com/interact/attachments/propchart-jpg.15285/
Supposedly there's also a study by Gormley et al with the (not so interesting) conclusion "that even a dose of 0.1 is likely to reach the same DHT inhibition as 1mg, but it would take longer to reach the level of suppression whereas 1mg reaches it after 1 dose. 0.2 mg reaches the same level as 1mg after two doses." I can't find it, but would appreciate, if somebody has a link for me.
While writing this, I realize that the extremely important graph, showing that even 0.04mg approach the efficacy of 1mg after 14 days is probably from that study.

THOUGHTS & THEORY
The three main cause of sides are probably 1) too low DHT, 2) permanently impaired levels of

neuroactive steroids (Dihydroprogesteron, Allopregnanolone, Tetrahydroprogesteron, Isopregnanolone, whose synthesis is an other important function of 5ar!) and the resulting ms-like spinal cord injury (impaired myelination or whatever, google 'neurological damages of fin') 3) other fucked up hormones (too high prolactin, E2, hypogonadism...)
impaired levels of neuroactive steroids is seen in most PED patients - http://onlinelibrary.wiley.com/doi/10.1111/jsm.12269/abstract#
thus sexual function could depend on the brain as seen in MS patients. There are psychogenic erections and erections that originate from physical contact (without being aroused). That's why maybe myelin damage is responsible for PED and not hormonal changes. More on this neurological damage theory (don't know if it's the right thread): http://www.propeciahelp.com/forum/viewtopic.php?t=2577
it might make sense to additionally microdose dut too a few times a week
topical application is not sensible, since fin needs to go systemic to block 5ar pathways
It might be possible to lessen the neurological damage a SMALL bit with: NAC, acetyl-l-carnitine, minocycline, l-theanine, taurine, curcumin, green tea (EGCG), CoQ10, nicotinamide/niacinamide, creatine.
Maybe it's possible to supplement the neuroactive steroids. At least, I think they definitely deserve more attention in the future.
5ar isn't destroyed by fin, but the inhibiton-complex lasts longer than the 5ar enzyme, so when it is dissociated, it's already dead
there's a treshold level of DHT, below which MPB is not triggered, which probably depends on the scalp's AR sensitivity to DHT.
Quote: "Scalp DHT is not that important either. It's the follicular DHT levels that are more contributor towards miniaturization. scalp DHT levels are difficult to measure properly. finasteride molecules have higher affinity towards prostate tissue, compared to scalp tissue. So you would clearly need much higher doses (10 times if I'm not mistaken) to get the same amount of suppression. So even though micro doses achieve almost the same serum DHT level inhibition, due primarily to the slow 5-AR II turnover rate, the finasteride molecules might not actually reach the scalp area including the follicles as well as the prostate. So in that sense, serum DHT levels might not mean much in terms of hair efficacy. Don't get me wrong, 0.05-0.12 should still hold off male pattern baldness quite well in some individuals, assuming it cuts off DHT levels just enough below the threshold level of androgenization."

QUESTIONS
do you have something to add?
Is there anything wrong with this approach?
What's the optimal frequency? Some knowledgable people were guessing that taking fin twice daily could be better. Relevant for determining this isn't only the relatively short half-life (4-6 hrs) of fin, but much more the fact that its effects last much longer than the fin itself, because 5ar production is very slow. After taking it, blood levels of fin peaks after 2-3hrs and then decrease rapidly, but: Even after a single dose, DHT levels wont be back to baseline before 1-2 weeks. The above linked study by Steiner also says that a slow accumulation takes place, but I don't know how that she be possible I guess the long DHT-suppressing effect of fin made him think that. The EFFECTS accumulate: when starting fin there is more 5ar in the body then a the time when you take the next dose of fin. So after a few days there's much less 5ar to block for the same dose of fin. "The turnover for the enzyme complex is slow (t1/2 approximately 30 days for the Type II enzyme complex and 14 days for the Type I complex)." If a higher frequency or slower absorption

would be beneficial to keep higher blood levels of fin, one could maybe slow down the metabolism of fin with grapefruit juice (probably large doses or rather the extracted active ingredient, which is responsible for this [CYP3a4?])
Does it even make sense to only reduce DHT a bit? Or: Where's the threshold? If you only have a bit of DHT left, but this small amount preferably binds to the androgen receptors in the scalp, you'd be fucked, too. But I know that at least the prostata is more sensitive/affine. I guess the threshold depends on the AR sensitivity to DHT in the individual's scalp and the speed of 5ar enzyme production.
Is it possible to just dissolve pills in alcohol to make microdosing possible? Do the pills lose potency? How long would such a solution be sustainable? Do you need to filter out the non-soluble fillers? Can the coating of the pill be destroyed? (I read that its not enteric coated, only protected against absorption through scalp.) Would it make sense to put the tincture in a capsule? "studies published have shown it to work better with glycerol, propylene glycol, and polyethylene glycol 400, while vehicles like oleic acid make its permeability decrease"

PRACTICAL STEPS:
get relevant blood levels tested
buy propecia, ethanol (not 100% sure about this) and equipment and create an alcohol tincture with the desired fin concentration. To be completely safe you could also keep some cabergoline and clomid at hand.
Store the solution air- and light-excluded
start with 0.01mg daily and up the dosage every 4 days (till 0.03-0.05mg)
usage: Shake the bottle. Don't take it sublingually. Take it before bed, since it can make you dizzy (?).
test relevant blood levels again after 4 weeks of taking at least 0.03mg
evaluate success of the treatment no earlier than after 6 months
if you decide to stop, taper off gradually