

## Finasteride (toremifene citrate)

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### [Finasteride](#)

#### [\(Toremifene Citrate\)](#)

[Finasteride](#) was available for years as Proscar (5mg tabs), but only recently became available as Propecia (1mg tabs). It is in a class of chemicals known as 5alpha-reductase inhibitors. It is based on the progesterone skeleton (4) and has a high inhibitory activity for the enzyme 5alpha-reductase (5-AR). 5-AR, as you may recall, is the enzyme responsible for converting certain steroids into 5-Alpha Reduced versions of themselves (such as turning testosterone into dihydrotestosterone). [Finasteride](#) and similar compounds are used for the treatment of androgen dependent diseases such androgenic alopecia (hair loss), benign prostatic hyperplasia (prostate enlargement) and prostate cancer. Dihydrotestosterone is a 5alpha-reduced metabolite of testosterone and has been implicated as a causative factor for the onset and progression of these problems. This was discovered when males who are genetically deficient of the enzyme steroid 5alpha-reductase were shown to have much lower incidences of these problems (1)(2). Of course, these problems can be a major annoyance, and nothing to toy around with, but by using [Finasteride](#) you risk reducing your gains on a given cycle, and can even suppress reproductive function (3). I'm not a big fan of this, as you could guess. However, if you are worried about your hairline, or have incidences of prostate issues in your family, then 1mg/day of [Finasteride](#) may be the answer you've been looking for.

It needs to be noted that there are actually 2 different 5-AR enzymes, and [Finasteride](#) specifically blocks the type-II variety. The type-II 5-AR enzyme is the one responsible primarily for hairloss and prostate enlargement, while type-I is often the culprit behind acne and hirsutism. In either case, type-II is responsible for around 2/3rds of the circulating DHT in your body, so it's no surprise that [Finasteride](#) typically reduces your total DHT levels by around 65%.

There is also some novel information about this compound regarding the conversion of testosterone into DHT via the 5-Alpha-reductase enzyme. It's come to my attention that the actual conversion process of testosterone into DHT via this enzyme may act in some way to inhibit luteinizing hormone release (and ergo would inhibit your HPTA and natural testosterone production). Check this out:

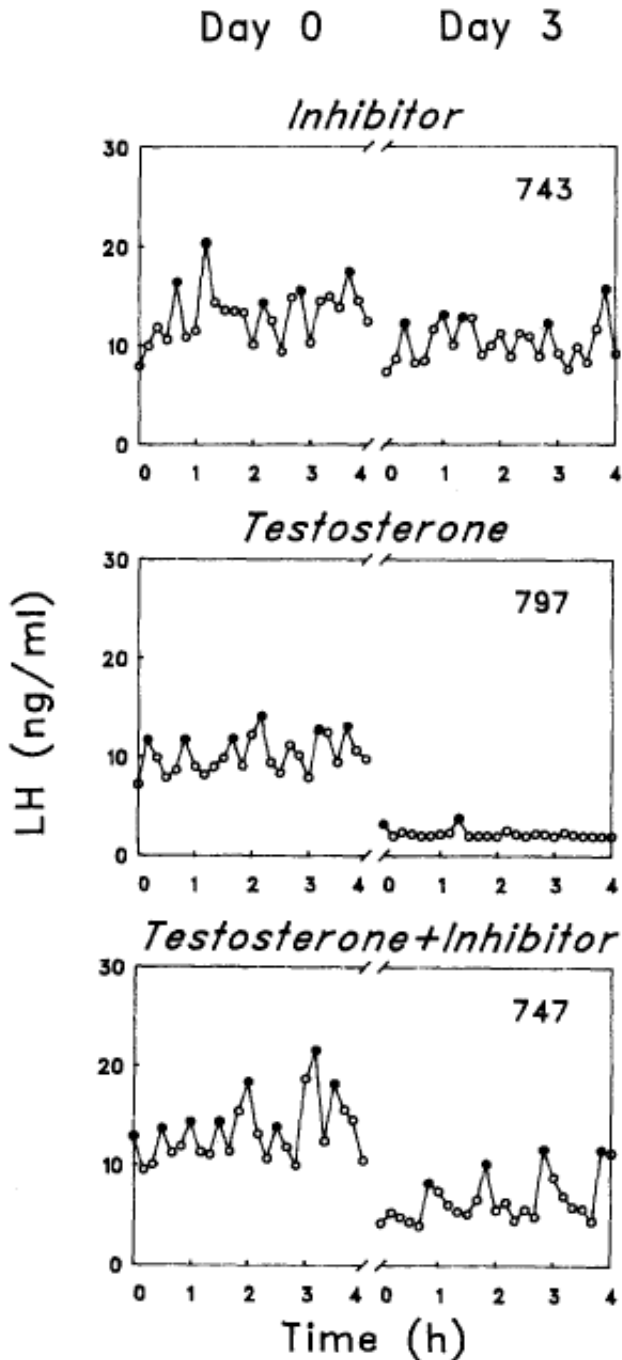


FIG. 2. LH secretory profiles for representative animals before (Day 0) and after 3 days (Day 3) of treatment with either 0.6 mg/kg/day of RI (top panel), 768 µg/kg/day of T (middle panel), or T+RI (bottom panel). Peaks of LH pulses, as identified by PULSAR, are indicated by the solid circles.

Basically, this chart above shows the baseline level of LH in male sheep given a 5-Alpha-Reductase inhibitor (such as [Finasteride](#)), then one showing the LH levels in sheep given [Testosterone Propionate](#), and finally a chart showing LH levels of sheep given [Testosterone Propionate](#) + the inhibitor (graph 3).(5) You'll note that although using the inhibitor alone produced no discernable effects on LH, when administered with testosterone, it seems to have allowed LH pulsatility to continue nearly unaffected. This may indicate that you can use [Finasteride](#) on a cycle (1mg/day) and possibly keep your LH levels normal (and thus your HPTA), ergo making recovery much easier. This is, of course only my speculation.

