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Subject: original dutagen?

Posted by [demo99](#) on Tue, 03 Oct 2006 13:44:19 GMT

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hallo,

möchte einen dutagenvergleich machen. sind diese hier original?  
Ich glaube schon.

Bitte stellt auch eure fotos rein,

danke im voraus

mfg demo99

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### File Attachments

1) [blister\\_a.jpg](#), downloaded 441 times

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Dutasteride Soft

**DUTAGEN**<sup>TM</sup>

Each soft gelatin capsule contains:

Dutasteride 0.5 mg

COLOURS: Iron Oxide Black  
& Iron Oxide Red

DOSAGE: As directed by the Physician.

Store below 25°C. Protect  
from light & moisture.

**WARNING:** To be sold by retail on the

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2) [blister\\_a.jpg](#), downloaded 378 times

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3) [package insert.jpg](#), downloaded 366 times

For the use only of a straight or a bent

# DUTAGE

(Dutasteride Soft Gelatin)

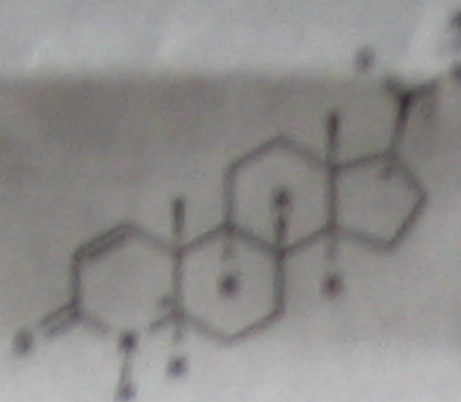
## COMPOSITION

Each soft gelatin capsule contains

Dutasteride 0.5 mg

## DESCRIPTION

Dutasteride is a synthetic 5- $\alpha$ -reductase inhibitor that is a selective inhibitor of both type 1 and type 2 5- $\alpha$ -reductase enzymes that convert testosterone to dihydrotestosterone (DHT). It is designated as (11*R*)-10-[(2*S*)-7-oxo-2-oxo-1,2,3,4-tetrahydro-1,4-benzoxepin-3-yl]-5- $\alpha$ -androstane-3-one, *N*,*N*,*N*,*N*-tetramethyl, representing a molecular weight of 426.5.



**Dutasteride**  
Dutasteride (Dutasteride)

## Pharmacokinetics

### Effectiveness of Dutasteride

Dutasteride inhibits the conversion of testosterone to the 5- $\alpha$ -reduced form, DHT, by the initial development and subsequent enlargement of the prostate gland. Two types of 5- $\alpha$ -reductase, which occur as 2 isoenzymes, type 1 and type 2. The type 2 isoenzyme is the major 5- $\alpha$ -reductase in the prostate gland and is responsible for testosterone conversion to the 5- $\alpha$ -reduced form. The type 1 isoenzyme is also responsible for testosterone conversion to the 5- $\alpha$ -reduced form. Dutasteride is a competitive and specific inhibitor of both type 1 and type 2 5- $\alpha$ -reductase isoenzymes. Inhibition of both isoenzymes has been demonstrated using <sup>3</sup>H-testosterone and <sup>3</sup>H-DHT as substrates.

The maximum effect of daily doses of dutasteride on the reduction of testosterone to DHT and DHT levels of daily dosing with dutasteride 0.5 mg, relative to placebo, respectively, in patients with BPH treated with dutasteride 0.5 mg/day for 1 year and 10% of 2 years. The maximum increase in total testosterone was 10% of the baseline range.

In BPH patients treated with 0.5 mg/day of dutasteride or placebo for up to 12 weeks, mean DHT concentrations in prostatic tissue were significantly lower in the 0.5 mg group, respectively, 2.0 (SD) and 1.0 (SD) ng/g, respectively. Mean prostatic tissue concentrations of testosterone group compared with placebo (2.0 (SD) and 1.0 (SD) ng/g, respectively) were not statistically different. Type 2 5- $\alpha$ -reductase deficiency also is sufficient to cause type 2 5- $\alpha$ -reductase gene throughout the prostate gland. However, present data do not show a statistically significant difference in testosterone deficiency. However, type 1 and type 2 5- $\alpha$ -reductase deficiency were indicated following 12 weeks