# SUMMARY OF PRODUCT CHARACTERISTICS

## 1 NAME OF THE MEDICINAL PRODUCT

Finasteride 1 mg Film-Coated Tablets

## 2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Each film-coated tablet contains 1 mg finasteride.

Excipient(s) with known effect:

Each film-coated tablet contains 95.55 mg of lactose monohydrate.

For the full list of excipients, see section 6.1.

## 3 PHARMACEUTICAL FORM

Film-coated tablet.

Round biconvex, reddish brown tablets 7 mm in diameter, marked “F1”.

## 4 CLINICAL PARTICULARS

### 4.1 Therapeutic indications

Finasteride is indicated for the treatment of men with male pattern hair loss (androgenetic alopecia) to increase hair growth and prevent further hair loss.

Finasteride is **not** indicated for use in women or children and adolescents.

### 4.2 Posology and method of administration

Posology

For oral use only
The tablet should be swallowed whole and must not be divided or crushed (see section 6.6).

*Androgenetic alopecia*
The recommended dosage is 1 mg daily. Finasteride may be taken with or without food.
There is no evidence that an increase in dosage will result in increased efficacy.

Efficacy and duration of treatment should continuously be assessed by the

treating physician. Generally, three to six months of once daily treatment are required before evidence of stabilisation of hair loss can be expected.
Continuous use is recommended to sustain benefit. If treatment is stopped, the beneficial effects begin to reverse by six months and return to baseline by 9 to12 months.

No data are available on the concomitant use of Finasteride and topical minoxidil in male pattern hair loss.

*Use in renal insufficiency*
No adjustment in dosage is required in patients with varying degrees of renal insufficiency (creatinine clearances as low as 9 ml/min), as pharmacokinetic studies did not indicate any change in the disposition of finasteride.

*Dosage in hepatic insufficiency*
There are no data available in patients with hepatic insufficiency (see section 4.4).

*Use in the elderly*
No dosage adjustment is required in elderly patients.

Method of administration
Crushed or broken tablets of 'Finasteride' should not be handled by women when they are or may potentially be pregnant because of the possibility of absorption of finasteride and the subsequent potential risk to a male foetus (see section 4.6 Fertility, pregnancy and lactation).
'Finasteride' tablets are coated to prevent contact with the active ingredient during normal handling, provided that the tablets are not broken or crushed.

## 4.3 Contraindications

Hypersensitivity to finasteride or to any of the excipients listed in section 6.1.

- Contraindicated in women: See 4.6 Fertility, pregnancy and lactation and section 5.1 'Pharmacodynamic properties'.
- Finasteride is not indicated for use in women or children and adolescents.
- Finasteride 1 mg should not be taken by men who are taking finasteride 5 mg or any other 5α- reductase inhibitor for benign prostatic hyperplasia or any other condition.

## 4.4 Special warnings and precautions for use

Paediatric population
Finasteride should not be used in children. There are no data demonstrating efficacy or safety of finasteride in children under the age of 18.

Effects on Prostate Specific Antigen (PSA)
In clinical studies with Finasteride in men 18-41 years of age, the mean value of serum prostate-specific antigen (PSA) decreased from 0.7 ng/ml at baseline to 0.5 ng/ml at month 12. Doubling the PSA level in men taking Finasteride should be considered before evaluating this test result.

Effects on fertility
See 4.6 Fertility, pregnancy and lactation.

Hepatic impairment

The effect of hepatic insufficiency on the pharmacokinetics of finasteride has not been studied.

Breast Cancer

Breast cancer has been reported in men taking finasteride 1 mg during the post-marketing period. Physicians should instruct their patients to promptly report any changes in their breast tissue such as lumps, pain, gynaecomastia or nipple discharge.

Mood alterations and depression

Mood alterations including depressed mood, depression and, less frequently, suicidal ideation have been reported in patients treated with finasteride 1 mg. Patients should be monitored for psychiatric symptoms and if these occur, treatment with finasteride should be discontinued and the patient advised to seek medical advice.

Sexual dysfunction that may contribute to mood alterations, including suicidal ideation, has been reported in some patients. Patients should be informed to seek medical advice in case they experience sexual dysfunction. Treatment discontinuation should be considered (see section 4.8).

Sexual dysfunction has also been reported without psychological effects, such as mood alterations or suicidal ideation.

A patient card reminding of the above is provided with the package of Finasteride.

Lactose intolerance

Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine.

Sodium

This medicinal product contains less than 1 mmol sodium (23 mg) per tablet, that is to say essentially ‘sodium free’.

## 4.5 Interaction with other medicinal products and other forms of interaction

Finasteride is metabolized primarily via, but does not affect, the cytochrome P450 3A4 system. Although the risk for finasteride to affect the pharmacokinetics of other drugs is estimated to be small, it is probable that inhibitors and inducers of cytochrome P450 3A4 will affect the plasma concentration of finasteride. However, based on established safety margins, any increase due to concomitant use of such inhibitors is unlikely to be of clinical significance.

Interaction studies have only been performed in adults.

## 4.6 Fertility, pregnancy and lactation

*Pregnancy*
Finasteride is contra-indicated for use in women due to the risk in pregnancy.
Because of the ability of type II 5α-reductase inhibitors to inhibit conversion of testosterone to dihydrotestosterone (DHT) in some tissues, these drugs, including finasteride, may cause abnormalities of the external genitalia of a male foetus when administered to a pregnant woman.

*Exposure to finasteride: risk to male foetus*
A small amount of finasteride, less than 0.001% of the 1 mg dose per ejaculation, has been detected in the seminal fluid of men taking finasteride. Studies in Rhesus monkeys have indicated that this amount is unlikely to constitute a risk to the developing male foetus (see Section 5.3).
During continual collection of adverse experiences, post-marketing reports of exposure to finasteride during pregnancy via semen of men taking 1 mg or higher doses have been received for eight live male births, and one retrospectively-reported case concerned an infant with simple hypospadias. Causality cannot be assessed on the basis of this single retrospective report and hypospadias is a relatively common congenital anomaly with an incidence ranging from 0.8 to 8 per 1000 live male births. In addition, a further nine live male births occurred during clinical trials following exposure to finasteride via semen, during pregnancy, and no congenital anomalies have been reported.

*Breast-feeding*
It is not known whether finasteride is excreted in human milk.

*Fertility*
Long-term data on fertility in humans are lacking, and specific studies in sub-fertile men have not been conducted. The male patients who were planning to father a child were initially excluded from clinical trials. Although, animal studies did not show relevant negative effects on fertility, spontaneous reports of infertility and /or poor seminal quality were received post-marketing. In some of these reports, patients had other risk factors that might have contributed to infertility. Normalisation or improvement of seminal quality has been reported after discontinuation of finasteride.

## 4.7 Effects on ability to drive and use machines

Finasteride has no or negligible influence on ability to drive or use machines.

## 4.8 Undesirable effects

The adverse reactions during clinical trials and/or post-marketing use are listed in the table below.
Frequency of adverse reactions is determined as follows:

Very Common (≥ 1/10); Common (≥ 1/100, ≤1/10); Uncommon (≥ 1/1,000, ≤ 1/100); Rare (≥ 1/10,000, ≤ 1/1,000); Very rare (< 1/10,000); not known (cannot be estimated from the available data).

The frequency of adverse reactions reported during post-marketing use cannot be determined as they are derived from spontaneous reports.

| **System Organ Class** | **Frequency: adverse reaction** |
|---|---|
| Immune system disorders | *Not known:* Hypersensitivity reactions, including rash, pruritus, urticaria and angioedema (swelling of the lips, tongue, throat and face). |
| Psychiatric disorders | *Uncommon*: decreased libido*<br>*Uncommon: depression†.*<br>*Not known: Anxiety. suicidal ideation* |
| Cardiac disorders | *Not known:* palpitation |
| Hepatobiliary disorders | *Not known:* increased hepatic enzymes |
| Reproductive system and breast disorders | *Uncommon *:* Erectile dysfunction, ejaculation disorder (including decreased volume of ejaculate).<br>*Not known:* Breast tenderness and enlargement, testicular pain, haematospermia, infertility**.<br>**See section 4.4. |

* Incidences presented as difference from placebo in clinical studies at Month 12.

† This adverse reaction was identified through post-marketing surveillance but the incidence in randomized controlled Phase III clinical trials (Protocols 087, 089 and 092) was not different between finasteride and placebo.

Side effects, which usually have been mild, generally have not required discontinuation of therapy.

Finasteride for male pattern hair loss has been evaluated for safety in clinical studies involving more than 3,200 men. In three 12-month, placebo-controlled, double-blind, multicentre studies of comparable design, the overall safety profiles of Finasteride 1 mg and placebo were similar. Discontinuation of therapy due to any clinical adverse experience occurred in 1.7% of 945 men treated with Finasteride 1 mg and 2.1% of 934 men treated with placebo.

In these studies, the following drug-related adverse experiences were reported in ≥1% of men treated with Finasteride 1 mg: decreased libido (Finasteride 1mg, 1.8% vs. placebo, 1.3%) and erectile dysfunction (1.3%, 0.7%). In addition, decreased volume of ejaculate was reported in 0.8% of men treated with Finasteride 1 mg and 0.4% of men treated with placebo. Resolution of these side effects occurred in men who discontinued therapy with Finasteride 1mg and in many who continued therapy. The effect of Finasteride 1 mg on ejaculate volume was measured in a separate study and was not different from

that seen with placebo.

By the fifth year of treatment with Finasteride 1 mg, the proportion of patients reporting each of the above side effects decreased to <0.3%.

Finasteride has also been studied for prostate cancer risk reduction at 5 times the dosage recommended for male pattern hair loss. In a 7-year placebo-controlled trial that enrolled 18,882 healthy men, of whom 9060 had prostate needle biopsy data available for analysis, prostate cancer was detected in 803 (18.4%) men receiving finasteride 5 mg and 1147 (24.4%) men receiving placebo. In the finasteride 5 mg group, 280 (6.4%) men had prostate cancer with Gleason scores of 7-10 detected on needle biopsy vs. 237 (5.1%) men in placebo group. Of the total cases of prostate cancer diagnosed in this study, approximately 98% were classified as intracapsular (stage T1 or T2). The relationship between long-term use of finasteride 5 mg and tumours with Gleason scores of 7-10 is unknown.

In addition, the following have been reported in post-marketing use: persistence of sexual dysfunction (decreased libido, erectile dysfunction and ejaculation disorder) after discontinuation of treatment with Finasteride 1 mg; male breast cancer (see 4.4 Special warnings and precautions for use).

Drug-related sexual undesirable effects were more common in the finasteride 1 mg-treated men than the placebo-treated men, with frequencies during the first 12 months of 3.8% vs. 2.1%, respectively. The incidence of these effects decreased to 0.6% in finasteride 1 mg-treated men over the following four years. Approximately 1% of men in each treatment group discontinued due to drug related sexual adverse experiences in the first 12 months, and the incidence declined thereafter.

**Reporting of suspected adverse reactions**

Reporting suspected adverse reactions after authorisation of the medicinal product is important. It allows continued monitoring of the benefit/risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions via the Yellow Card Scheme at:www.mhra.gov.uk/yellowcard or search for MHRA Yellow Card in the Google Play or Apple App Store.

## 4.9 Overdose

In clinical studies, single doses of finasteride up to 400 mg and multiple doses of finasteride up to 80 mg/day for three months (n=71) did not result in dose-related undesirable effects.

No specific treatment of overdosage with Finasteride is recommended.

# 5 PHARMACOLOGICAL PROPERTIES

## 5.1 Pharmacodynamic properties

Pharmacotherapeutic group: 5α-reductase inhibitor, ATC code: D11AX10.

Mechanism of action

Finasteride is a competitive and specific inhibitor of type II 5α-reductase. Finasteride has no affinity for the androgen receptor and has no androgenic, anti-androgenic, oestrogenic, anti-oestrogenic, or progestational effects. Inhibition of this enzyme blocks the peripheral conversion of testosterone to the androgen DHT, resulting in significant decreases in serum and tissue DHT concentrations. Finasteride produces a rapid reduction in serum DHT concentration, reaching significant suppression within 24 hours of dosing.

Hair follicles contain type II 5α-reductase. In men with male pattern hair loss, the balding scalp contains miniaturised hair follicles and increased amounts of DHT. Administration of finasteride decreases scalp and serum DHT concentrations in these men. Men with a genetic deficiency of type II 5α-reductase do not suffer from male pattern hair loss. Finasteride inhibits a process responsible for miniaturisation of the scalp hair follicles, which can lead to reversal of the balding process.

Clinical efficacy and safety

*Studies in men*

Clinical studies were conducted in 1879 men aged 18 to 41 with mild to moderate, but not complete, vertex hair loss and/or frontal/mid-area hair loss. In the two studies in men with vertex hair loss (n=1553), 290 men completed 5 years of treatment with Finasteride vs. 16 patients on placebo. In these two studies, efficacy was assessed by the following methods: (i) hair count in a representative 5.1cm2 area of scalp, (ii) patient self-assessment questionnaire, (iii) investigator assessment using a seven-point scale, and (iv) photographic assessment of standardised paired photographs by a blinded expert panel of dermatologists using a seven point scale.

In these 5-year studies men treated with Finasteride improved compared to both baseline and placebo beginning as early as 3 months, as determined by both the patient and investigator assessments of efficacy. With regard to hair count, the primary endpoint in these studies, increases compared to baseline were demonstrated starting at 6 months (the earliest time point assessed) through to the end of the study.

In men treated with Finasteride these increases were greatest at 2 years and gradually declined thereafter to the end of 5 years; whereas hair loss in the placebo group progressively worsened compared to baseline over the entire 5 year period. In Finasteride treated patients, a mean increase from baseline of 88 hairs [p <0.01; 95% CI (77.9, 97.80; n=433] in the representative 5.1 cm2 area was observed at 2 years and an increase from baseline of 38 hairs [p <0.01; 95% CI (20.8, 55.6); n=219] was observed at 5 years, compared with a decrease from baseline of 50 hairs [p <0.01; 95% CI (-80.5, -20.6); n=47] at 2 years and a decrease from baseline of 239 hairs [p <0.01; 95% CI (-304.4, -173.4); n=15] at 5 years in patients who received placebo. Standardised photographic assessment of efficacy demonstrated that 48% of men treated with finasteride for 5 years were rated as improved, and an additional 42% were rated as unchanged. This is in comparison to 25% of men treated with placebo for 5 years who were rated as improved or unchanged. These data demonstrate that treatment with

Finasteride for 5 years resulted in a stabilisation of the hair loss that occurred in men treated with placebo.

An additional 48-week, placebo-controlled study designed to assess the effect of Finasteride on the phases of the hair-growth cycle (growing phase [anagen] and resting phase [telogen]) in vertex baldness enrolled 212 men with androgenetic alopecia. At baseline and 48 weeks, total, anagen and telogen hair counts were obtained in a 1-cm2 target area of the scalp. Treatment with Finasteride led to improvements in anagen hair counts, while men in the placebo group lost anagen hair. At 48 weeks, men treated with Finasteride showed net increases in total and anagen hair counts of 17 hairs and 27 hairs, respectively, compared to placebo. This increase in anagen hair count, compared to total hair count, led to a net improvement in the anagen-to-telogen ratio of 47% at 48 weeks for men treated with Finasteride, compared to placebo. These data provide direct evidence that treatment with Finasteride promotes the conversion of hair follicles into the actively growing phase.

*Studies in women*
Lack of efficacy was demonstrated in post-menopausal women with androgenetic alopecia who were treated with Finasteride in a 12-month, placebo-controlled study (n=137). These women did not show any improvement in hair count, patient self-assessment, investigator assessment, or ratings based on standardised photographs, compared with the placebo group.

## 5.2 Pharmacokinetic properties

***Absorption***
Relative to an intravenous reference dose, the oral bioavailability of finasteride is approximately 80%. The bioavailability is not affected by food. Maximum finasteride plasma concentrations are reached approximately two hours after dosing and the absorption is complete after six to eight hours.

***Distribution***
Protein binding is approximately 93%. The volume of distribution of finasteride is approximately 76 litres. At steady state following dosing with 1 mg/day, maximum finasteride plasma concentration averaged 9.2 ng/ml and was reached 1 to 2 hours postdose; AUC (0 24 hr) was 53 ng•hr/ml.
Finasteride has been recovered in the cerebrospinal fluid (CSF), but the drug does not appear to concentrate preferentially to the CSF. A small amount of finasteride has also been detected in the seminal fluid of subjects receiving the drug.

***Biotransformation***
Finasteride is metabolised primarily via the cytochrome P450 3A4 enzyme subfamily.
Following an oral dose of $^{14}$C-finasteride in man, two metabolites of the drug were identified that possess only a small fraction of the 5α-reductase inhibitory activity of finasteride.

***Elimination***

Following an oral dose of $^{14}$C-finasteride in man, 39% of the dose was excreted in the urine in the form of metabolites (virtually no unchanged drug was excreted in the urine) and 57% of total dose was excreted in the faeces.
Plasma clearance is approximately 165 ml/min.
The elimination rate of finasteride decreases somewhat with age. Mean terminal half-life is approximately 5-6 hours in men 18-60 years of age and 8 hours in men more than 70 years of age. These findings are of no clinical significance and hence, a reduction in dosage in the elderly is not warranted.

***Renal impairment***
No adjustment in dosage is necessary in non-dialysed patients with renal impairment.

### 5.3 Preclinical safety data

In general, the findings in laboratory animal studies with oral finasteride were related to the pharmacological effects of 5α-reductase inhibition.

Intravenous administration of finasteride to pregnant rhesus monkeys at doses as high as 800 ng/day during the entire period of embryonic and foetal development resulted in no abnormalities in male foetuses. This represents at least 750 times the highest estimated exposure of pregnant women to finasteride from semen. In confirmation of the relevance of the Rhesus model for human foetal development, oral administration of finasteride 2 mg/kg/day (100 times the recommended human dose or approximately 12 million times the highest estimated exposure to finasteride from semen) to pregnant monkeys resulted in external genital abnormalities in male foetuses. No other abnormalities were observed in male foetuses and no finasteride-related abnormalities were observed in female foetuses at any dose.

## 6 PHARMACEUTICAL PARTICULARS

### 6.1 List of excipients

Tablet core
Lactose monohydrate
Cellulose, microcrystalline
Pregelatinised starch
Lauroyl macrogolglycerides
Sodium starch glycolate - Type A
Magnesium stearate

Tablet coating
Hypromellose 6 cps.

Titanium dioxide (E171)
Iron oxide yellow E 172
Iron oxide red E 172
Macrogol 6000

### 6.2 Incompatibilities

Not applicable.

### 6.3 Shelf life

3 years

### 6.4 Special precautions for storage

This medicinal product does not require any special storage conditions

### 6.5 Nature and contents of container

Blister packs: Aluminium/PVC or Aluminium/Aluminium.
Pack size: 28 and 84 tablets.

Plastic bottles (HDPE) with cap.
Pack size: 28 tablets.

Not all pack sizes may be marketed.

### 6.6 Special precautions for disposal

Women who are pregnant or may become pregnant should not handle crushed or broken finasteride tablets because of the possibility of absorption of finasteride and the subsequent potential risk to a male foetus (see section 4.6, Fertility, pregnancy and lactation).
Any unused medicinal product or waste material should be disposed of in accordance with local requirements.

## 7 MARKETING AUTHORISATION HOLDER

Relonchem Limited,
Cheshire House,
Gorsey Lane,
Widnes,
Cheshire,
WA8 0RP,
United Kingdom.

## 8 MARKETING AUTHORISATION NUMBER(S)

PL 20395/0068

## 9 DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

11/09/2008

## 10 DATE OF REVISION OF THE TEXT

12/03/2026