PRODUCT INFORMATION

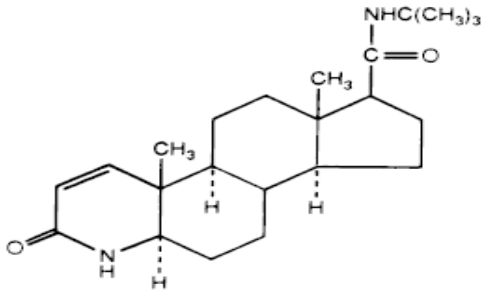
A&M - Fintab 1 (Finasteride Film-coated Tablets 1 mg)

NAME OF THE MEDICINE

Finasteride is described chemically as: *N*-(1,1-dimethylethyl)-3-oxo-4-aza-5α-androst-1-ene- 17βcarboxamide.

The CAS No is 98319-26-7.

Its empirical formula is C_{23} H_{36} N_2 O_2 and the molecular weight is 372.55. Its structural formula is:



DESCRIPTION

Finasteride is a white, crystalline solid with a molecular weight of 372.55. It is freely soluble in chloroform and in lower alcohol solvents but is practically insoluble in water.

Composition

Active:
Finasteride

Inactives: Lactose, microcrystalline cellulose, pregelatinised maize starch, lauroyl macrogolglycerides, sodium starch glycollate, magnesium stearate and Opadry 03F34739 Pink.

PHARMACOLOGY

Clinical pharmacology

Mechanism of action

Finasteride is a competitive and specific inhibitor of Type II 5α-reductase with which it slowly forms a stable enzyme complex. Turnover from this complex is extremely slow (t½ ~ 30 days). Finasteride has no affinity for the androgen receptor and has no androgenic, antiandrogenic, oestrogenic, antioestrogenic, or progestational effects. Inhibition of this enzyme blocks the peripheral conversion of testosterone to the androgen dihydrotestosterone (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 decreased scalp and serum DHT concentrations in these men. In addition, men with a genetic deficiency of the Type II 5α-reductase do not suffer from male pattern hair loss. These data and the results of the clinical studies confirm that finasteride inihibits the process responsible for miniaturisation of the scalp hair folliceles, leading to reversal of the balding process.

PHARMACOKINETICS

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 6-8 hours.

Distribution

Protein binding is approximately 93%. The volume of distribution of finasteride is approximately 76 litres.

There is a modest accumulation of finasteride in plasma after multiple dosing. 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 medicine does not appear to concentrate preferentially to the CSF. A very small amount of finasteride has also been detected in the seminal fluid of subjects receiving finasteride.

Metabolism

Finasteride is metabolised primarily via the cytochrome P450 3A4 enzyme subfamily. Following an oral dose of ^{14}C-finasteride in man, two metabolites of finasteride 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 medicine 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

In patients with chronic renal impairment whose creatinine clearance ranged from 9 to 55 mL/min, the disposition of a single dose of 14C-finasteride was not different from that in healthy volunteers. Protein binding also did not differ in patients with renal impairment. A

portion of the metabolites that normally is excreted renally was excreted in the faeces. It therefore appears that faecal excretion increases commensurate to the decrease in urinary excretion of metabolites. No adjustment in dosage is necessary in non-dialysed patients with renal impairment.

An open label, balanced, randomized, two-treatment, two period, two-sequence, single dose, two-way crossover, comparative oral bioavailability study of two formulations of Finasteride Film-coated Tablets 5 mg was conducted in 30 healthy adult human male subject under fasting condition. The study compared Finasteride Film-coated Tablets 5 mg with reference product Proscar® 5mg tablets.

Statistical comparisons of geometric means for Test v's Reference for finasteride C_{max} and $AUC_{0-\infty}$ were as follows:

Parameters (units)	**Geometric Least Squares Mean**			**90% Confidence Interval (Parametric)**
	Test Product B	Reference Product A	Ratio B/A%	
C_{max} (ng/mL)	52.717	47.293	111.5	106.28-116.91%
$AUC_{0-\infty}$ (ng.h/mL)	427.803	395.307	108.2	101.48-115.41%

This comparison of test product with reference product finasteride met the predefined criteria for bioequivalence, as the calculated 90% CI for all ratios of pre-specified In-Transformed PK parameters fell within the range 80.00%-125.00%.

PHARMACODYNAMICS

Finasteride had no effect on circulating levels of cortisol, oestradiol, prolactin, thyroid-stimulating hormone, or thyroxine, nor did it affect the plasma lipid profile (e.g., total cholesterol, low density lipoproteins, high density lipoproteins, and triglycerides) or bone mineral density. In studies with finasteride, no clinically meaningful changes in luteinising hormone (LH) and follicle-stimulating hormone (FSH) were detected. Gonadotropin-releasing hormone (GnRH) stimulated levels of LH or FSH were not altered, indicating that regulatory control of the hypothalamic-pituitary-testicular axis was not affected. There was no effect on semen parameters in men treated with finasteride 1 mg/day for 48 weeks.

Finasteride appeared to inhibit both C19 and C21 steroid metabolism and hence appeared to have an inhibitory effect on both hepatic and peripheral Type II 5α-reductase activity. The serum DHT metabolites androstenediol glucuronide and androsterone glucuronide were also significantly reduced. This metabolic pattern is similar to that observed in individuals with a genetic deficiency of Type II 5α- reductase who have markedly decreased levels of DHT and who do not suffer from male pattern hair loss.

CLINICAL TRIALS

Studies in men

The efficacy of finasteride was demonstrated in men (88% Caucasian) with mild to moderate androgenetic alopecia (male pattern hair loss) between 18 and 41 years of age. There were three double-blind, randomised, placebo-controlled studies of 12-month duration. The two primary endpoints were hair count and patient self-assessment; the two secondary endpoints were investigator assessment and ratings of photographs. The three studies were conducted in 1,879 men with mild to moderate, but not complete, hair loss. Two of the studies enrolled men with predominantly mild to moderate vertex hair loss (n=1,553). The third enrolled men having mild to moderate hair loss in the anterior mid-

scalp area with or without vertex balding (n=326).

Two studies on vertex baldness

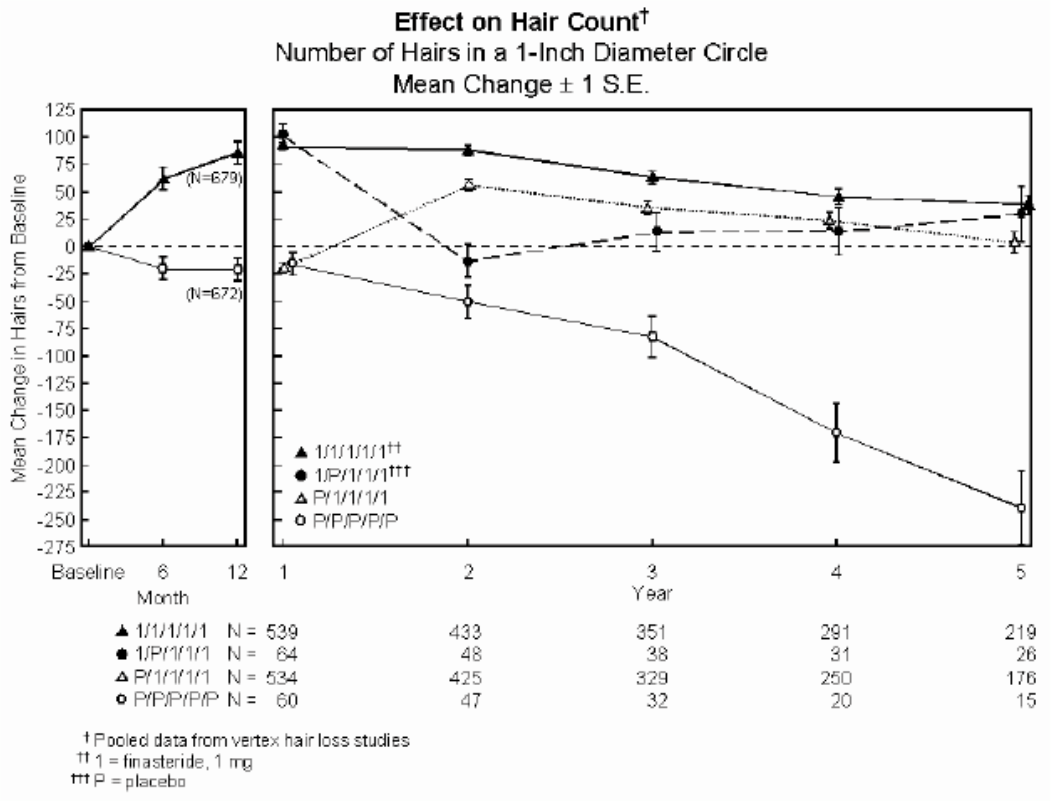
Of the men who completed the first 12 months of the two vertex baldness trials, 1,215 elected to continue in double-blind, placebo-controlled, 12-month extension studies. There were 547 men receiving finasteride for both the initial study and first extension periods (up to 2 years of treatment) and 60 men receiving placebo for the same periods. The extension studies were continued for 3 additional years, with 323 men on finasteride and 23 on placebo entering the fifth year of the study.

In order to evaluate the effect of discontinuation of therapy, there were 65 men who received finasteride for the initial 12 months followed by placebo in the first 12-month extension period. Some of these men continued in additional extension studies and were switched back to treatment with finasteride, with 32 men entering the fifth year of the study. Lastly, there were 543 men who received placebo for the initial 12 months followed by finasteride in the first 12-month extension period. Some of these men continued in additional extension studies receiving finasteride, with 290 men entering the fifth year if the study (see Figure below).

Hair counts were assessed by photographic enlargements of a representative area of active hair loss. In these two studies in men with vertex baldness, significant increases in hair count were demonstrated at 6 and 12 months in men treated with finasteride, while significant hair loss from baseline was demonstrated in those treated with placebo. At 12 months there was a 107-hair difference from placebo ($p<0.001$, finasteride [n=679 evaluable men] vs placebo [n=672 evaluable men]) within a 1-inch diameter circle (5.1 cm2). Hair count was maintained in those men taking finasteride for up to 2 years resulting in a 138-hair difference between treatment groups ($p<0.001$, finasteride [n=433 evaluable men] vs placebo [n=47 evaluable men]) within the same area. In men treated with finasteride, the maximum improvement in hair count compared to baseline was achieved during the first two years, and hair count was maintained above baseline throughout the 5 years of the studies. The difference between treatment groups also continued to increase throughout the studies, resulting in a 277-hair difference ($p<0.001$, finasteride [n=219 evaluable men] vs placebo [n=15 evaluable men]) at 5 years. Thus, compared to baseline, hair loss did not progress further in the majority of men treated with finasteride; in contrast, hair loss progressively worsened in all men in the placebo group (see Figure below).

Patients who switched from placebo to finasteride (n=426 evaluable men) had a decrease in hair count at the end of the initial 12 month placebo period, followed by an increase in hair count after 1 year of treatment with finasteride. This increase in hair count was less (56 hairs above original baseline) than the increase (91 hairs above original baseline) observed after 1 year of treatment in men initially randomised to finasteride. Although the increase in hair count, relative to when therapy was initiated, was comparable between these two groups, a higher absolute hair count was achieved in patients who were started on treatment with finasteride in the initial study. This advantage was maintained throughout the 5 years of the studies. A change of treatment from finasteride Tablets 1 mg to placebo (n=48 evaluable men) at the end of the initial 12 months resulted in reversal of the increase in hair count 12 months later, at 24 months (see Figure below).

At 12 months, 58% of men in the placebo group had further hair loss (defined as any decrease in hair count from baseline) compared with 14% of men treated with finasteride. In men treated for up to 2 years, 72% of men in the placebo group demonstrated hair loss, compared with 17% of men treated with finasteride Tablets 1 mg. At 5 years, 100% of men in the placebo group demonstrated hair loss, compared with only 35% of men treated with finasteride.



Patient self-assessment was obtained at each clinic visit from a self-administered questionnaire, this included questions on their perception of hair growth, hair loss, and appearance. This self-assessment demonstrated an increase in amount of hair, a decrease in hair loss, and improvement in appearance in men treated with finasteride. Overall improvement compared with placebo was seen as early as 3 months ($p<0.05$), with continued improvement over 5 years.

Investigator assessment was based on a 7-point scale evaluating increases or decreases in scalp hair at each patient visit. This assessment showed significantly greater increases in hair growth in men treated with finasteride compared with placebo as early as 3 months ($p<0.001$). At 12 months, the investigators rated 65% of men treated with finasteride as having increased hair growth compared with 37% in the placebo group. At 2 years, the investigators rated 80% of men treated with finasteride Tablets 1 mg as having increased hair growth compared with 47% of men treated with placebo. At 5 years, the investigators rated 77% of men treated with finasteride as having increased hair growth, compared with 15% of men treated in the placebo group.

An independent panel rated standardised photographs of the head in a blinded fashion based on increases or decreases in scalp hair, using the same 7-point scale as the investigator assessment. At 12 months, 48% of men treated with finasteride had an increase as compared with 7% of men treated with placebo. At 2 years, an increase in hair growth was demonstrated in 66% of men treated with finasteride compared with 7% of men treated with placebo. At 5 years, an increase in hair growth was demonstrated in 48% of men treated with finasteride compared with 6% of men treated with placebo.

Based on this assessment, 10% of men treated with finasteride for 5 years were rated as having lost hair, compared with 75% of men in the placebo group. These results

demonstrate that 90% of the men treated with finasteride had no further visible progression of hair loss, compared with 25% of men treated with placebo, based on ratings of either no change or increased hair growth.

In one of the two vertex baldness studies, patients were questioned on non-scalp body hair growth. Finasteride did not appear to affect non-scalp body hair.

Study on hair loss in the anterior mid-scalp area

A study of 12-month duration, designed to assess the efficacy of finasteride in men with hair loss in the anterior mid-scalp area, also demonstrated significant increases in hair count compared with placebo. Increases in hair count were accompanied by improvements in patient self-assessment, investigator assessment, and ratings based on standardised photographs. Hair counts were obtained in the anterior mid-scalp area, and did not include the area of bitemporal recession or the anterior hairline.

Phototrichogram study

A 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, telogen, and anagen 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 ($p<0.001$) and 27 hairs ($p<0.001$), 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% ($p<0.001$) at 48 weeks for men treated with finasteride, compared to placebo.

Summary of Clinical Studies

Clinical studies were conducted in men aged 18 to 41 with mild to moderate degrees of androgenetic alopecia. Clinical improvement was seen as early as 3 months in the patients treated with finasteride and led to a net increase in scalp hair count and hair regrowth. In clinical studies for up to 5 years, treatment with finasteride prevented the further progression of hair loss observed in the placebo group. In general, the difference between treatment groups continued to increase throughout the 5 years of the studies. There were no studies comparing finasteride with other medicines for androgenetic alopecia.

Ethnic analysis of clinical data

In a combined analysis of the two studies on vertex baldness, mean hair count changes from baseline were 91 vs –19 hairs (finasteride vs placebo) among Caucasians (n=1,185), 49 vs –27 hairs among North American Blacks (n=84), 53 vs –38 hairs among Asians (n=17), 67 vs 5 hairs among North American Hispanics (n=45) and 67 vs -15 hairs among other ethnic groups (n=20). Patient self- assessment showed improvement across racial groups with finasteride treatment, except for satisfaction of the frontal hairline and vertex in North American Black men, who were satisfied overall.

A sexual function questionnaire was self-administered by patients participating in the two vertex baldness trials to detect more subtle changes in sexual function. At Month 12, statistically significant differences in favour of placebo were found in 3 of 4 domains (sexual interest, erections, and perception of sexual problems). However, no significant difference was seen in the question on overall satisfaction with sex life.

Studies in women

Lack of efficacy was demonstrated in postmenopausal women with androgenetic alopecia who were treated with finasteride in a 12-month, placebo-controlled study (n=137). These women showed no improvement in hair count, patient self-assessment, investigator assessment, or ratings based on standardised photographs, compared with the placebo group (see INDICATIONS).

INDICATIONS

A&M - Fintab 1 is indicated for the treatment of male pattern hair loss (androgenetic alopecia) to increase hair growth and prevent further hair loss in men 18 years or older. Efficacy has not been demonstrated in men over the age of 41 years.

A&M - Fintab 1 is not indicated for use in women (see Use in Pregnancy and Clinical Studies) or children.

CONTRAINDICATIONS

A&M - Fintab 1 is contraindicated in the following:

• Use in women when they are or may potentially be pregnant (See Use in Pregnancy)
• Hypersensitivity to any component of this product

A&M - Fintab 1 is not indicated for use in women or children.

PRECAUTIONS

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. When finasteride is used for treatment of male pattern hair loss in older men who also have benign prostatic hyperplasia (BPH), consideration should be given to the fact that, in older men with BPH, PSA levels are decreased by approximately 50%.

There are no data to suggest that finasteride affects the ability to drive or use machines.

Carcinogenicity

In a 24 month carcinogenicity study in rats there was an increase in the incidence of thyroid follicular adenomas in male rats receiving 160 mg/kg/day finasteride (statistically significant trend test). This oral dose produced an exposure in rats of more than 800 times that observed in humans at the recommended dose (based on AUC (0-24 hrs) values). The effect of finasteride on the thyroid in rats appears to be due to an increased rate of thyroxine clearance and not a direct effect of the medicine. These observations seen in the rat are thought not relevant to man.

In a 19-month carcinogenicity study in mice, a statistically significant increase in the incidence of testicular Leydig cell adenoma was observed at an oral dose of 250 mg/kg/day (estimated exposure of more than 1700 times that observed in humans at the recommended dose); no adenomas were seen in mice given 2.5 or 25 mg/kg/day.

In mice at an oral dose of 25 mg/kg/day (estimated exposure about 90 times that in humans at the recommended dose) and in rats at an oral dose of =40 mg/kg/day, (estimated exposure about 300 times that in humans at the recommended dose) an increase in the incidence of Leydig cell hyperplasia was observed. A positive correlation between the proliferative changes of the Leydig cells and the increase in serum luteinising hormone (LH) levels (2-3 fold above control) has been demonstrated in both rodent species treated with high doses of finasteride. This suggests the Leydig cell changes are secondary to elevated serum

LH levels and not due to a direct effect of finasteride.

No medicine-related Leydig cell changes were seen in either rats or dogs treated with finasteride for one year at respective oral doses of 20 mg/kg/day (estimated exposure more than 220 times that in humans at the recommended dose) and 45 mg/kg/day (estimated exposure more than 2600 times that in humans at the recommended dose) or in mice treated for 19 months at an oral dose of 2.5 mg/kg/day (estimated exposure about 9 times that in humans at the recommended dose).

Genotoxicity

No evidence of mutagenicity was observed in an in vitro bacterial mutagenesis assay, a mammalian cell mutagenesis assay, or in an in vitro alkaline elution assay. In an in vitro chromosome aberration assay, when Chinese hamster ovary cells were treated with high concentrations (450-550 µmol) of finasteride, there was a slight increase in chromosome aberrations. These concentrations are in excess of the peak plasma concentrations in men given a total dose of 1 mg and are not achievable in a biological system. In an in vivo chromosome aberration assay in mice, no treatment-related increases in chromosome aberration were observed with finasteride at the maximum tolerated dose.

Effect on Fertility

Oral treatment of male rabbits with finasteride up to 80 mg/kg/day (estimated exposure more than 4000 times that in humans at the recommended dose) did not impair fertility. In male rats, oral treatment for up to 24 or 30 weeks with 80 mg/kg/day (estimated exposure approximately 440 times that in humans at the recommended dose) resulted in an apparent decrease in fertility associated with a significant decrease in weight of seminal vesicles and prostate. All of these effects were reversible within 6 weeks of discontinuation of treatment. This decrease in fertility in rats was secondary to the effect of finasteride on the accessory sec organs, resulting in failure to form a seminal plug, which is essential for fertility in rats, but is not relevant to man.

Developmental studies

Hypospadias was observed in the male offspring of pregnant rats given finasteride at oral doses ranging from 100µg/kg/day to 100 mg/kg/day (= 5 times the recommended human dose) at an incidence of 3.6 to 100%. Additionally, pregnant rats produced male offspring with decreased prostatic and seminal vesicular weights, delayed preputial separation, and transient nipple development when given finasteride at oral doses =30 µg/kg/day (=1.5 times the recommended human dose), and decreased anogenital distance when given finasteride in oral doses =3 µg/kg/day (approximately one- fifth the recommended human dose). The critical period during which these effects can be induced has been defined in male rats as Days 16-17 of gestation.

The changes described above are expected pharmacological effects of Type II 5α-reductase inhibitors. Many of the changes, such as hypospadias, observed in male rats exposed in utero to finasteride are similar to those reported in male infants with a genetic deficiency of Type II areductase. No effects were seen in female offspring exposed in utero to any dose of finasteride.

Administration of finasteride to rats during the late gestation and lactation period resulted in slightly decreased fertility in first generation male offspring (3 mg/kg/day). No developmental abnormalities have been observed in first generation male or female offspring resulting from mating finasteride- treated male rats (80 mg/kg/day) with untreated females.

No evidence of malformations has been observed in rabbit foetuses exposed to finasteride in

utero from Days 6-18 of gestation at doses up to 100 mg/kg/day.

The in utero effects of finasteride exposure during the period of embryonic and foetal development were evaluated in the rhesus monkey (Gestation Days 20-100), a species more predictive of human development than rats or rabbits. Intravenous administration of finasteride to pregnant monkeys at doses up to 800 ng/day (at least 750 times the highest estimated amount, on a bodyweight basis, of finasteride in semen to which a pregnant woman might be exposed) resulted in no abnormalities in male foetuses. In confirmation of the relevance of the rhesus model for human foetal development, oral administration of a very high dose of finasteride (2 mg/kg/day; 100 times the recommended human dose or approximately 12 million times the highest estimated amount, on a bodyweight basis, of finasteride in semen to which a pregnant woman might be exposed) 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.

Use in pregnancy

Category X: Medicines which have such a high risk of causing permanent damage to the foetus that they should not be used in pregnancy or when there is a possibility of pregnancy.
A&M - Fintab 1 is contraindicated for use in women when they are or may potentially be pregnant. Because of the ability of Type II 5α-reductase inhibitors to inhibit conversion of testosterone to DHT in some tissues, these medicines, including finasteride, may cause abnormalities of the external genitalia of a male fetus when administered to a pregnant woman.

Women who are or may potentially be pregnant should not handle crushed or broken tablets of A&M - Fintab 1 mg, or handle tablets with wet hands, because of the possibility of absorption of finasteride and the subsequent potential risk to a male foetus. Whole tablets are coated to prevent contact with the active ingredient during normal handling.

Use in lactation

A&M - Fintab 1 is not indicated for use in women and should not be used by lactating women. It is not known whether finasteride is excreted in human milk

Use in children

A&M - Fintab 1 is not indicated for use in children.

Use in the elderly

Clinical studies with A&M - Fintab 1 have not been conducted in elderly men with male pattern hair loss.

Interactions with other medicines

No medicine interactions of clinical importance have been identified. Compounds that have been tested in man have included antipyrine, digoxin, glyburide, propranolol, theophylline, and warfarin and no interactions were found. Increases in P-450 medicine-metabolising activity were observed in animal studies (in rats, mice and dogs) receiving doses of >80, 250 and 45 mg/kg/day respectively. Finasteride is metabolised primarily via, but does not affect, the cytochrome P450 3A4 system. Although the risk for finasteride to affect the pharmacokinetics of other medicines 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.

Although specific interaction studies were not performed, in clinical studies finasteride doses of 1 mg or more were used concomitantly with ACE inhibitors, paracetamol, alpha blockers, benzodiazepines, beta blockers, calcium-channel blockers, cardiac nitrates, diuretics, H 2 antagonists, HMG-CoA reductase inhibitors, prostaglandin synthetase inhibitors (NSAIDs), and quinolones, without evidence of clinically significant adverse interactions.

ADVERSE EFFECTS

A&M - Fintab 1 is generally well tolerated. Side effects, which usually have been mild, generally have not required discontinuation of therapy.

Clinical trial data

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, multicenter studies of comparable design, the overall safety profiles of finasteride and placebo were similar.

Discontinuation of therapy due to any clinical adverse experience occurred in 1.7% of 945 men treated with finasteride and 2.1% of 934 men treated with placebo.

Table 1 presents the only clinical adverse effects considered possibly, probably or definitely medicine- related by the investigator, for which the incidence on finasteride was =1% and greater than placebo over the 12 months of the study.

TABLE 1
Medicine-Related Adverse Effects

	Treatment	YEAR 1 (%)
Decreased Libido	Placebo	1.3
	Finasteride	1.8
Erectile Dysfunction	Placebo	0.7
	Finasteride	1.3

In addition, in the 12-month controlled studies, decreased volume of ejaculate was reported in 0.8% of men treated with finasteride and 0.4% of men treated with placebo. Resolution of these side effects occurred in men who discontinued therapy with finasteride and in many who continued therapy. In a separate study, the effect of finasteride on ejaculate volume was measured and was not any different from that seen with placebo.

The incidence of each of the above side effects decreased to < 0.3% by the fifth year of treatment with finasteride.

Table 2 presents the other most common clinical adverse experiences reported in the initial 12 month phase III clinical studies occurring in >2% of men treated with finasteride or placebo. A causal relationship to treatment with finasteride has not been established.

Table 2: Other Adverse Experiences

Incidence ≥2% in Any Treatment Group Without Regard to Causality
Phase III Controlled Studies

	Finasteride 1mg (N=945)	Placebo (N=934)
Body as a Whole		
Trauma	17 (1.8)	21 (2.2)
Musculoskeletal Disorders		
Pain, back	24 (2.5)	23 (2.5)
Nervous System and Psychiatric Disorders		
Headache	114 (12.1)	96 (10.3)
Respiratory System Disorders		
Bronchitis	26 (2.8)	21 (2.2)
Infection, respiratory, upper	139 (14.7)	145 (15.5)
Influenza	70 (7.4)	72 (7.7)
Pharyngitis	45 (4.8)	34 (3.6)
Sinusitis	36 (3.8)	26 (2.8)

Post marketing experience

The following adverse experiences have been reported in post marketing use. Because these reactions are reported voluntarily from a population of uncertain size, it is not always possible to reliably estimate the frequency or establish a causal relationship to drug exposure.

Immune system disorders: hypersensitivity reactions including rash, pruritus, urticaria, and swelling of the lips and face.

Psychiatric disorders: depression; decreased libido that continued after discontinuation of treatment.

Reproductive system and breast disorders: sexual dysfunction (erectile dysfunction and ejaculation disorders) that continued after discontinuation of treatment; breast tenderness and enlargement; male breast cancer; testicular pain; male infertility and/or poor seminal quality. Normalisation or improvement of seminal quality has been reported after discontinuation of finasteride.

DOSAGE AND ADMINISTRATION

The recommended dosage is one 1-mg tablet daily. A&M - Fintab 1 may be taken with or without food.

In general, daily use for 3 months or more is necessary before increased hair growth and/or prevention of further hair loss is observed. Continued use is recommended to obtain maximum benefit.

OVERDOSAGE

In clinical studies, single doses of finasteride up to 400 mg and multiple doses of finasteride up to 80 mg/day for three months did not result in side effects.

No specific treatment for overdosage with A&M - Fintab 1 is recommended.

The Poisons Information Centre, telephone number 13 11 26 should be contacted for advice on the management of an overdose.

PRESENTATION AND STORAGE CONDITION

A&M - Fintab 1 (AUST R 173216)

A&M - Fintab 1 comes as a reddish brown, round, biconvex, film-coated tablets, marked 'F1' on one side and plain on the other. Each tablet contains finasteride 1mg. A&M – Fintab 1 tablets are available in packs containing 28, 30,120 and 126 tablets in aluminium/aluminium foil blisters.

Storage

Store below 25°C.

NAME AND ADDRESS OF THE SPONSOR

Pharmacor Limited
Suite 401/7 Oaks Avenue,
Dee Why, NSW, 2099
Australia

NAME AND ADDRESS OF THE DISTRIBUTOR

AM Pharmaceuticals Pty Ltd
44 Parliament Place
West Perth WA 6005
Australia

POISON SCHEDULE OF THE MEDICINE

Prescription Only Medicine (Schedule 4).

DATE OF FIRST INCLUSION IN THE AUSTRALIAN REGISTER OF THERAPEUTIC GOODS (the ARTG)

29th November 2010

DATE OF MOST RECENT AMENDMENT

25 July 2014