THERAPY

Finasteride in the treatment of men with androgenetic alopecia

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Background: Androgenetic alopecia (male pattern hair loss) is caused by androgen-dependent miniaturization of scalp hair follicles, with scalp dihydrotestosterone (DHT) implicated as a contributing cause. Finasteride, an inhibitor of type II 5α-reductase, decreases serum and scalp DHT by inhibiting conversion of testosterone to DHT.
Objective: Our purpose was to determine whether finasteride treatment leads to clinical improvement in men with male pattern hair loss.
Methods: In two 1-year trials, 1553 men (18 to 41 years of age) with male pattern hair loss received oral finasteride 1 mg/d or placebo, and 1215 men continued in blinded extension studies for a second year. Efficacy was evaluated by scalp hair counts, patient and investigator assessments, and review of photographs by an expert panel.
Results: Finasteride treatment improved scalp hair by all evaluation techniques at 1 and 2 years ($P < .001$ vs placebo, all comparisons). Clinically significant increases in hair count (baseline = 876 hairs), measured in a 1-inch diameter circular area (5.1 cm^2) of balding vertex scalp, were observed with finasteride treatment (107 and 138 hairs vs placebo at 1 and 2 years, respectively; $P < .001$). Treatment with placebo resulted in progressive hair loss. Patients' self-assessment demonstrated that finasteride treatment slowed hair loss, increased hair growth, and improved appearance of hair. These improvements were corroborated by investigator assessments and assessments of photographs. Adverse effects were minimal.
Conclusion: In men with male pattern hair loss, finasteride 1 mg/d slowed the progression of hair loss and increased hair growth in clinical trials over 2 years.
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Androgenetic alopecia (male pattern hair loss) occurs in men with an inherited sensitivity to the effects of androgens on scalp hair.[1,2] It is marked by visible loss of hair in areas of the scalp caused by progressive miniaturization of hair follicles.[3-5] The condition does not occur in men with a genetic deficiency of the enzyme steroid 5α-reductase (5αR) type II, which converts testosterone to dihydrotestosterone (DHT), implicating DHT in its pathogenesis.[6] Of two 5αR isoenzymes in humans,[7-9] type I predominates in skin, including scalp,[10,11] whereas type II is present in hair follicles,[12] as well as the prostate.[11]

Oral finasteride, a type II 5αR inhibitor, lowers serum,[13] prostate,[14] and scalp [15,16] DHT. Develop-

Table IA. Baseline characteristics of men randomized in initial studies

	US phase III initial study		International phase III initial study	
	Finasteride 1 mg (n = 471)	**Placebo (n = 462)**	**Finasteride 1 mg (n = 308)**	**Placebo (n = 312)**
Age (y, mean ± SE)	33 ± 0.2	34 ± 0.2	31 ± 0.3	31 ± 0.3
Age at which hair loss began (y, mean ± SE)	24 ± 0.2	25 ± 0.2	24 ± 0.3	24 ± 0.3
No. (%) of patients with family history*	373 (80)	363 (80)	227 (74)	239 (77)
Baseline hair count (mean ± SE)†	864 ± 11	856 ± 12	916 ± 15	924 ± 14
No. (%) of patients with hair loss pattern‡				
II vertex	63 (13)	63 (14)	56 (18)	59 (19)
III vertex	150 (32)	129 (28)	76 (25)	75 (24)
IV	112 (24)	141(30)	83 (27)	84 (27)
V	146 (31)	129 (28)	93 (30)	94 (30)

*Family history: Parents or siblings with androgenetic alopecia.
†Measured in a 1-inch diameter circular area (5.1 cm^2) at anterior leading edge of vertex balding scalp.
‡According to a modified Norwood-Hamilton scale.

Table IB. Baseline characteristics of men randomized in extension studies

	US and International phase III extension studies combined			
	***Fin→Fin* (n = 547)**	***Fin→Pbo* (n = 65)**	***Pbo→Fin* (n = 543)**	***Pbo→Pbo* (n = 60)**
Age (y, mean ± SE)	33 ± 0.2	33 ± 0.7	33 ± 0.2	32 ± 0.7
Age at which hair loss began (y, mean ± SE)	24 ± 0.2	24 ± 0.2	24 ± 0.2	24 ± 0.6
No. (%) of patients with family history*	425 (78)	47 (72)	431 (80)	49 (82)
Baseline hair count (mean ± SE)†	876 ± 11	835 ± 26	878 ± 11	903 ± 27
No. (%) of patients with hair loss pattern‡				
II vertex	84 (15)	5 (8)	79 (14)	9 (15)
III vertex	156 (28)	21 (32)	153 (28)	12 (20)
IV	140 (26)	16 (25)	155 (29)	19 (32)
V	167 (30)	23 (35)	156 (29)	20 (33)

*Family history: Parents or siblings with androgenetic alopecia.
†Measured in a 1-inch diameter circular area (5.1 cm^2) at anterior leading edge of vertex balding scalp.
‡According to a modified Norwood-Hamilton scale.

ed for treatment of benign prostatic hyperplasia (BPH) at a dose of 5 mg/d, finasteride has an excellent safety profile.[13] Early studies showed that 1 mg/d was effective in men with male pattern hair loss.[16-19]

METHODS

Study population (Tables IA and IB)

Men 18 to 41 years of age, with mild to moderately severe vertex male pattern hair loss according to a modified Norwood/Hamilton classification scale (II vertex, III vertex, IV or V),[20,21] were enrolled. The principal exclusions were significant abnormalities on screening physical examination or laboratory evaluation, surgical correction of scalp hair loss, topical minoxidil use within 1 year, use of drugs with androgenic or antiandrogenic properties, use of finasteride or other 5αR inhibitors, or alopecia from other causes. Men were instructed not to alter their hair style or dye their hair during the studies.

We conducted 2 replicate, 1-year, double-blind, placebo-controlled, randomized, multicenter studies, which continued as 1-year, double-blind, placebo-controlled, randomized, extension studies to determine the effect of treatment for 2 years, the effect of withdrawal of treatment after 1 year, and the natural history of male pattern hair loss in men seeking treatment. Investigators at 33 US sites (US study) and 27 sites in 15 non-US countries (international study) participated. Institutional review board approval and written informed consent were obtained before patients were entered into each study.

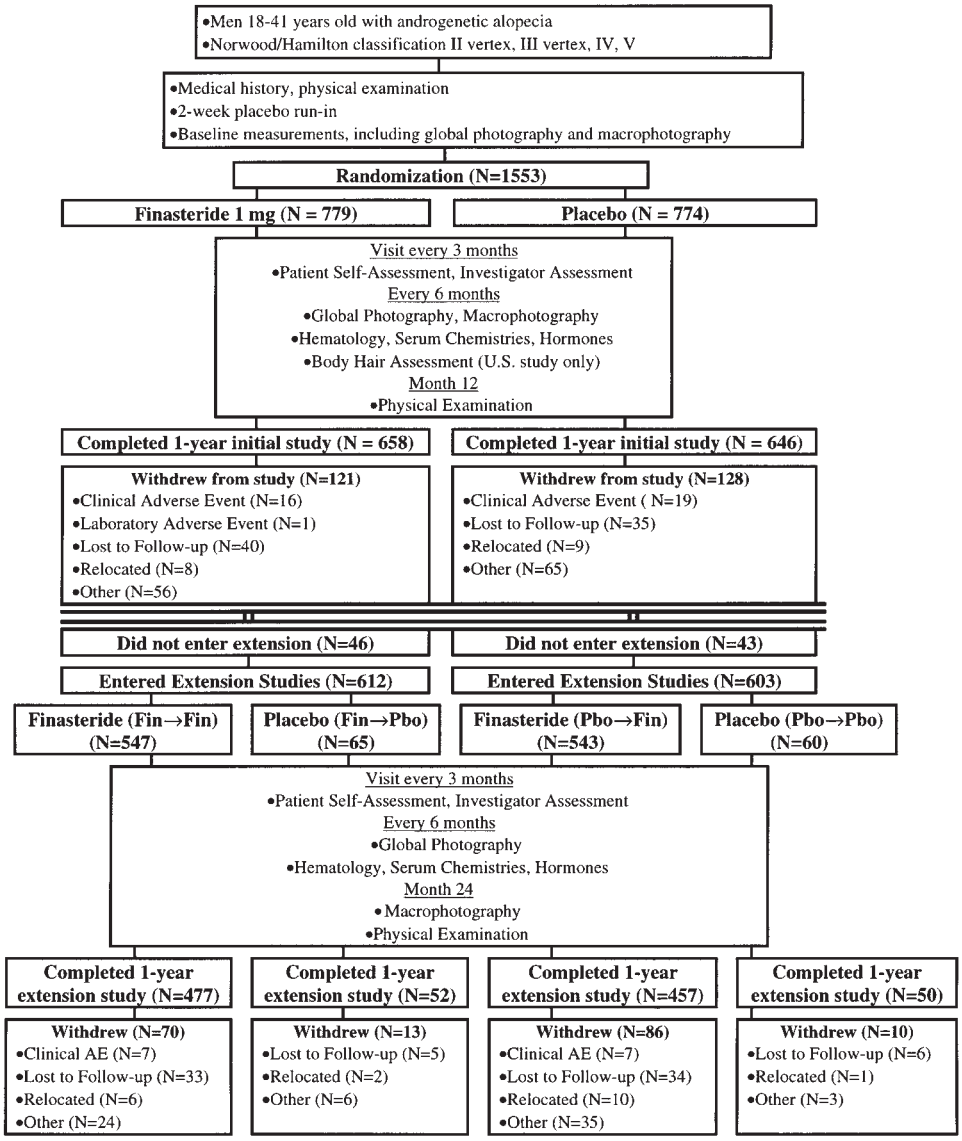

Fig 1. Trial profile. Main inclusion criteria and evaluation procedures, as well as the number of patients who were randomized, who completed the studies, and who discontinued prematurely, are shown for both original and extension studies by treatment group.

Study protocols (Fig 1)

Initial studies. After a screening procedure, patients entered a 2-week, single-blind placebo run-in. Patients received a study shampoo (Neutrogena T/Gel) for standardization of shampoo used and for prophylaxis of seborrheic dermatitis, which might affect scalp hair growth.[22] Patients (N = 1553) were then randomly assigned to treatment with either finasteride 1 mg/d or placebo for 1 year.

Patients visited the clinic every 3 months, where they completed a hair growth questionnaire and investigators completed assessments of scalp hair growth. Every 6 months, photographs of scalp hair were taken for hair counts and for assessments of hair growth by an expert panel. Reports of adverse events were collected throughout the studies.

Extension studies. Patients completing the initial 1-year studies were eligible to enroll in 1-year extension studies. In the extension studies, patients (N = 1215) were randomly assigned treatment to either finasteride 1 mg or placebo (9:1), as determined at initial randomization (Table II). The protocol for the extension studies was similar to the initial studies, except that photography for hair count was done only at month 24.

Table II. Randomization of patients to treatment groups in original and extension studies

Original studies (first year)			Extension studies (second year)		
Treatment group	**% Subjects**		**Treatment**	**% Subjects**	**Treatment group**
Finasteride	50	→	Finasteride	45	*Fin→Fin*
		→	Placebo	5	*Fin→Pbo*
Placebo	50	→	Finasteride	45	*Pbo→Fin*
		→	Placebo	5	*Pbo→Pbo*

EVALUATION PROCEDURES

Four predefined efficacy end points provided a comprehensive assessment of changes in scalp hair from baseline.

Hair counts (co-primary end point)

Hair counts were obtained from color macrophotographs of a 1-inch diameter circular area (5.1 cm^2) of clipped hair (length, 1 mm) at the anterior leading edge of the vertex thinning area, centered with a dot tattoo to ensure reproducibility.[23]

Macrophotographs (Kodak KR-64 35-mm slide film) were taken with dedicated preset camera systems[23] at fixed focus, distance (primary magnification, 1:1.7), and exposure, with the use of a macroflash mounted on a scalp template,[23] with enlargement into 8- × 10-inch (20.3- × 25.4-cm) color transparencies (final magnification, 5.84:1). At the end of the initial studies, the baseline, month 6, and month 12 transparencies for each patient, and subsequently at the end of the extension studies the baseline and month 24 transparencies, were converted into dot maps of each visible hair by technicians (Canfield Scientific, Inc [CSI], Fairfield, NJ), who were blinded as to patient, treatment, and time. Dot maps were converted into hair counts by means of personal computer–based scanners and imaging software.

Patient self-assessment (co-primary end point)

Patients assessed their scalp hair using a validated, self-administered hair growth questionnaire,[24] consisting of 4 questions in the patient's language on treatment efficacy and 3 questions on satisfaction with appearance (Fig 2).

Investigator assessment

Investigators assessed patients at all time points, using a standardized 7-point rating scale of hair growth compared with baseline (–3 = greatly decreased, –2 = moderately decreased, –1 = slightly decreased, 0 = no change, +1 = slightly increased, +2 = moderately increased, +3 = greatly increased). Baseline patient global photographs (see below) were provided to the investigator for reference.

Global photographic assessment

Standardized color global photographs (Kodak KR-64 35-mm slide film) of the vertex scalp were taken with the head in a stereotactic positioning device.[23] Paired baseline and posttreatment slides were independently reviewed, with the use of the standardized 7-point rating scale (see above), by a panel of three dermatologists (E. Olsen, R. Savin, D. Whiting) blinded as to treatment and experienced in photographic assessments of hair growth. This technique has previously been demonstrated to have excellent test-retest reproducibility and interrater agreement.[25]

Safety measurements

Safety measurements included clinical and laboratory evaluations, adverse event reports, and patient body hair assessment via a self-administered questionnaire (US study only).

LABORATORY EVALUATION

Hematology, urinalysis, chemistry, and hormone measurements were performed at baseline and every 6 months. Serum chemistry, including prostate-specific antigen (PSA), and serum hormones, including testosterone, DHT, luteinizing hormone, and follicle-stimulating hormone, were assayed in central laboratories (Medical Research Laboratories, Highland Heights, Ky, and Endocrine Sciences, Calabasas Hills, Calif, respectively).

STATISTICAL ANALYSIS

A data analysis plan prespecified all primary and secondary hypotheses, including combining data from both initial studies, to improve precision of the estimates of treatment effect, and from both extension studies, because of the small size of the placebo groups in the extension phase.

The primary hypothesis for hair counts was assessed by the difference between the count at each time point and the accompanying baseline count, and mean values

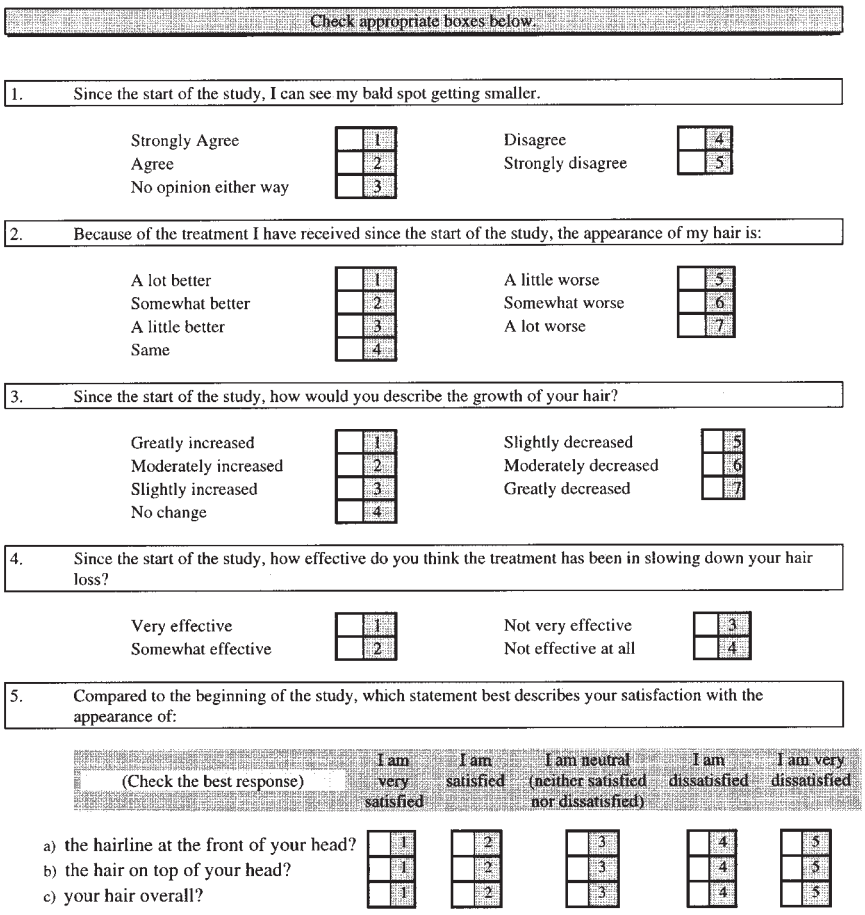

Fig 2. Male hair growth questionnaire used for patient self-assessment of changes in scalp hair.

for each treatment group were determined by means of SAS computed Least Mean Squares. The primary hypothesis for patient self-assessment was assessed by a global test across all 7 questions, by means of a generalized least squares procedure that accounts for different scales of, and covariance among, the questions.[26,27] For investigator and global photographic assessments, hypotheses were assessed by comparison of mean rating scores for each treatment group at each time point, based on the 7-point rating scale (minimum value = –3.0; maximum value = 3.0). Hypothesis testing for hair counts, individual patient self-assessment questions, and investigator and global photographic assessments was performed by means of analysis of variance (ANOVA).

Efficacy analyses were based on the intention-to-treat principle; that is, analysis of the initial studies included all men with at least one measurement post-randomization, and analysis of the extension studies included all men with at least one measurement in the second year. In each study, the last observation was carried forward where appropriate to impute missing data.

The focus of the safety analyses was on the biochemical parameters, with the use of ANOVA, and on adverse event reports. Comparison of the proportion of patients with an adverse event was done between groups with Fisher's exact test.

RESULTS

Patient accounting is summarized in Fig 1.

Hair counts

Initial studies. In each study, finasteride treatment produced progressive increases in hair count at months 6 and 12, whereas treatment with placebo resulted in significant hair loss (all P values < .001 vs baseline). At month 12, the difference between groups (mean ± SE) was 106 ± 5.6 and 107 ± 7.0 hairs in the target area in the US and international studies, respectively (both, $P < .001$). Combining data from both studies (mean baseline hair count = 876 hairs) demonstrated an increase

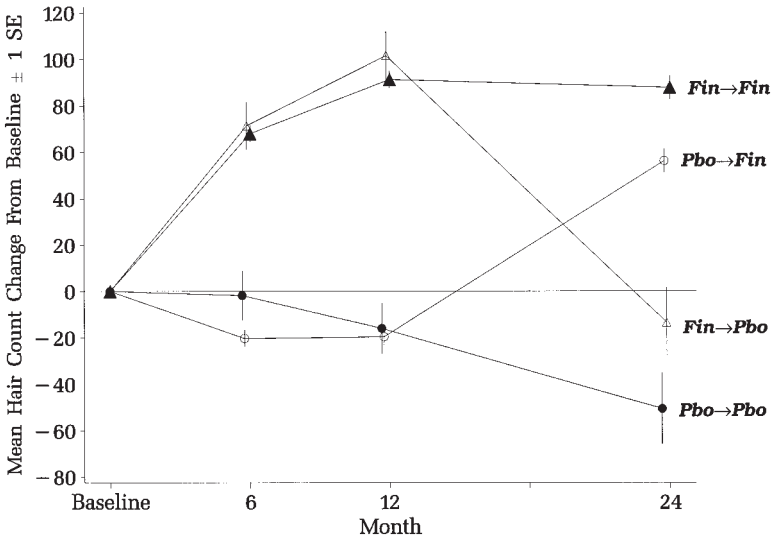

Fig 3. Hair count mean change from baseline (± 1 SE) from the combined US and international studies for men who entered the extension studies.

of 86 ± 3.4 hairs (11% ± 0.5%) for finasteride, a decrease of –21 ± 3.4 hairs (–2.7% ± 0.5%) for placebo, and a difference of 107 ± 4.4 hairs (14% ± 0.6%) between groups (all *P* values < .001). By month 12, 58% of patients receiving placebo had fewer hairs than at baseline, compared with only 14% receiving finasteride.

Extension studies. For the two groups continued on the original study therapy (*Fin→Fin* and *Pbo→Pbo*), the combined analysis (Fig 3) demonstrated a difference of 107 ± 11 hairs between groups at month 12 ($P < .001$), which was identical to the result observed in all patients in the initial studies. At month 24, the *Fin→Fin* group maintained the hair count observed at month 12, whereas the *Pbo→Pbo* group demonstrated further hair loss (–37 ± 13 hairs vs month 12, $P < .01$). The difference between groups ($P < .001$) was 138 ± 16 hairs (16% ± 2.1%), which was significantly greater ($P < .05$) than the difference at month 12. By month 24, 72% of patients continuing placebo had fewer hairs than at baseline, compared with only 17% continuing finasteride.

For the group switched from finasteride to placebo at month 12 (*Fin→Pbo*), reversibility of the finasteride effect was demonstrated at month 24 (–117 ± 13 hairs vs month 12, $P < .001$), whereas the group switched from placebo to finasteride (*Pbo→Fin*) demonstrated improvement (76 ± 4.3 hairs vs month 12, $P < .001$).

Patient self-assessment

Initial studies. In each study, finasteride was superior to placebo as early as month 3 ($P < .05$), the first efficacy time point, and at all subsequent timepoints ($P < .001$). For individual questions, finasteride was superior to placebo for 6 of 7 questions (except Q5a) by month 6, and for all questions at all subsequent time points (all *P* values < .001).

Extension studies. In the combined analysis, continued treatment with finasteride (*Fin→Fin*) was superior to continued treatment with placebo (*Pbo→Pbo*) at each time point ($P < .001$), and the treatment effect was significantly greater ($P < .05$) at month 24 than at month 12. At month 24, the *Fin→Fin* group demonstrated further improvement for each question ($P < .05$ vs month 12), whereas the *Pbo→Pbo* group demonstrated a negative trend.

For the *Fin→Pbo* group, partial or complete loss of effect of finasteride was observed for 6 of 7 questions (except Q4), whereas the *Pbo→Fin* group demonstrated improvement for each question ($P < .001$ vs month 12).

Another way of demonstrating the treatment effect is the percentage of patients who reported improvement for each of the 7 questions (Table III). For each question, a greater percentage of finasteride-treated than placebo-treated patients reported improvement, with the difference between groups increasing with time.

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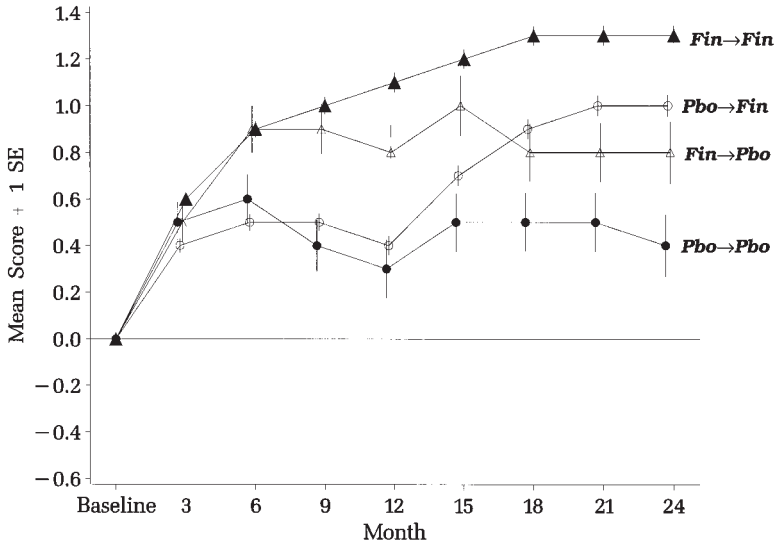

Fig 4. Investigator assessment mean rating score (± 1 SE) from the combined US and international studies for men who entered the extension studies.

Table III. Percentage of men with improvements in scalp hair

	Original studies (month 12)			Extension studies (month 24)		
	Finasteride (n = 720-751)	Placebo (n = 709-749)	Difference (± 95% CI)	*Fin→Fin* (n = 508-535)	*Pbo→Pbo* (n = 55-60)	Difference (± 95% CI)
Patient self-assessment (questionnaire)						
Q1: Size of bald spot	42	21	21 (17, 26)	58	17	41 (31, 52)
Q2: Appearance of hair	58	35	23 (18, 28)	71	31	40 (28, 53)
Q3: Growth of hair	56	33	23 (18, 27)	69	27	42 (30, 54)
Q4: Slowing hair loss	68	45	23 (18, 28)	81	46	35 (22, 48)
Q5a: Satisfaction with frontal hairline	29	17	12 (8, 16)	36	12	24 (15, 33)
Q5b: Satisfaction with hair on top	36	20	16 (12, 21)	49	17	32 (21, 42)
Q5c: Satisfaction with hair overall	39	22	17 (12, 21)	51	25	26 (14, 37)
Investigator assessment						
Increased hair growth	65	37	32 (23, 33)	80	47	33 (21, 47)
Global photographic assessment						
Increased hair growth	48	7	41 (36, 45)	66	7	59 (51, 67)

Investigator assessment

Initial studies. In each study, finasteride was superior to placebo at all time points ($P < .001$). By month 12, 65% of finasteride-treated patients were rated as improved by the investigators versus 37% of placebo-treated patients (Table III).

Extension studies. In the combined analysis (Fig 4), continued treatment with finasteride (*Fin→Fin*) was superior to continued treatment with placebo (*Pbo→Pbo*) at each time point ($P < .001$). At month 24, the *Fin→Fin* group demonstrated further improvement and, as anticipated, the *Pbo→Fin* group improved (both, $P < .001$ vs month 12). In contrast to the other end points, neither the *Pbo→Pbo* nor the *Fin→Pbo* groups significantly worsened. By month 24, the investigators rated as improved 80% of patients continuing finasteride versus 47% continuing placebo (Table III).

Global photographic assessment

Initial studies. In each study, finasteride was

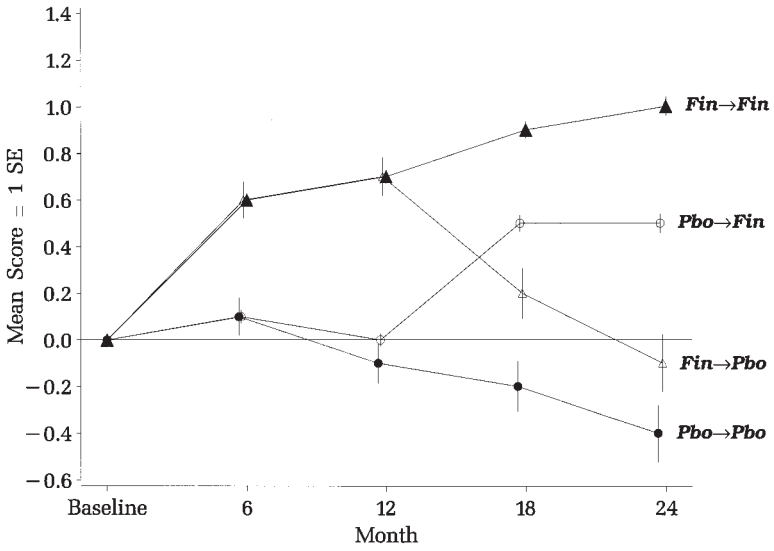

Fig 5. Global photographic assessment mean rating score (± 1 SE) from the combined US and international studies for men who entered the extension studies.

superior to placebo at all time points ($P < .001$). By month 12, the expert panel rated as improved 48% of finasteride-treated patients (30% slightly improved, 18% moderately or greatly improved) versus 7% of placebo-treated patients (Table III).

Extension studies. In the combined analysis (Fig 5), continued treatment with finasteride (*Fin→Fin*) was superior to continued treatment with placebo (*Pbo→Pbo*) at each time point ($P < .001$), and the treatment effect at month 24 was significantly greater ($P < .001$) than at month 12. At month 24, the *Fin→Fin* group demonstrated further improvement, whereas the *Pbo→Pbo* group demonstrated further worsening (both *P* values < .01 vs month 12). For the *Fin→Pbo* group, the effect of finasteride was lost gradually over 12 months ($P < .001$, month 24 or month 18 vs month 12). As anticipated, the *Pbo→Fin* group improved ($P < .001$ vs month 12).

By month 24, two thirds of patients continuing finasteride were rated as improved by the expert panel (30% slightly improved, 36% moderately or greatly improved) versus 7% continuing placebo (Table III). Only 1% of patients continuing finasteride worsened versus one third continuing placebo. Figs 6 and 7 show baseline, month 12, and month 24 global photographs of 2 representative finasteride-treated patients rated at 12 and 24 months as slightly, moderately, or greatly improved.

Serum hormones and PSA

Finasteride markedly reduced serum DHT from a median of 44.0 ng/dL at baseline (normal range = 30-85 ng/dL) to 14.0 ng/dL at month 12 (median percent change ± SE = –68.4% ± 1.2%; $P < .001$ vs placebo), and slightly increased serum testosterone from a median of 510 ng/dL at baseline (normal range = 350-1030 ng/dL) to 559 ng/dL at month 12 (median percent change ± SE = 9.1% ± 1.5%; $P < .001$ vs placebo). Finasteride treatment had no significant effects on serum luteinizing hormone or follicle-stimulating hormone, whereas serum PSA (normal range < 4.0 ng/mL) fell slightly (baseline mean ± SE = 0.78 ± 0.04 ng/mL; month 12 = 0.52 ± 0.02 ng/mL; mean change vs placebo = –0.23 ± 0.04 ng/mL, $P < .001$).

ADVERSE EVENTS

Clinical adverse events considered by the investigator to be possibly, probably, or definitely drug-related that occurred in 1% of men or more are shown in Table IV. In the first year, a slightly higher proportion of finasteride-treated than placebo-treated patients reported adverse events related to sexual function (4.2% vs 2.2%, $P < .05$; see Table IV for details). Only 11 men (1.4%) in the finasteride group and 8 (1.0%) in the placebo group discontinued the study because of sexual adverse events, which resolved after discontinuation. These adverse events also resolved in many of the

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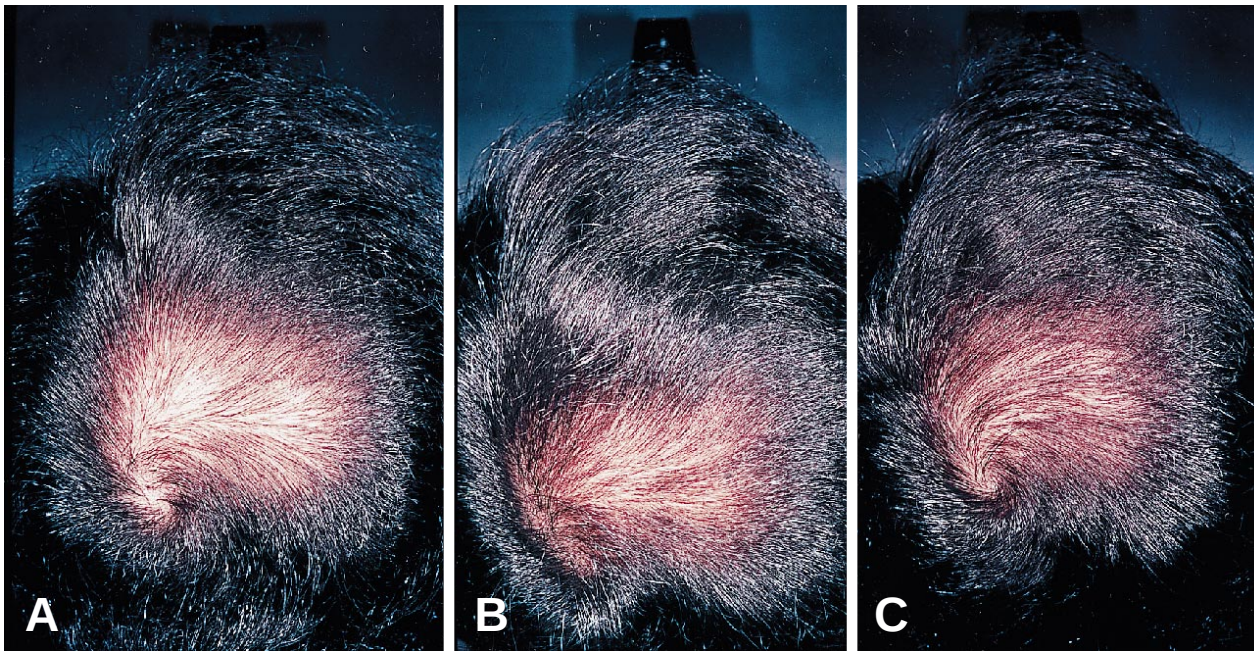

Fig 6. Patient 1. **A**, Baseline. **B,** Month 12: Slightly increased hair growth. **C,** Month 24: Moderately increased hair growth.

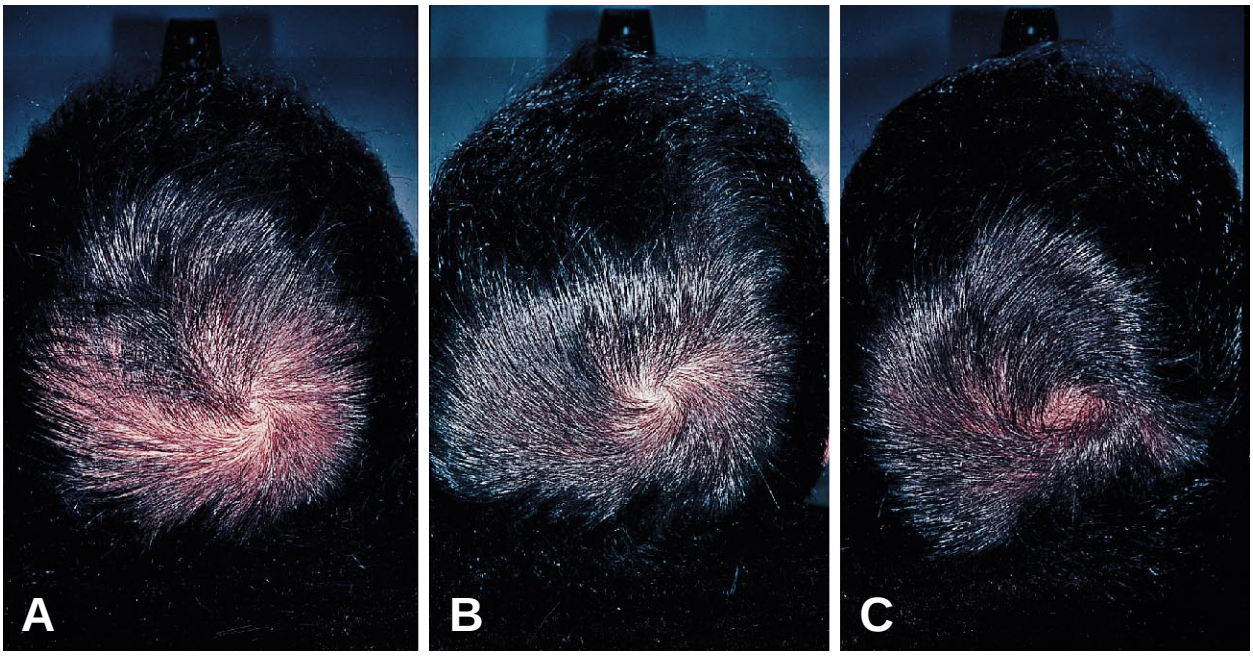

Fig 7. Patient 2. **A,** Baseline. **B,** Month 12: Moderately increased hair growth. **C,** Greatly increased hair growth.

patients who reported them but who remained on the finasteride regimen and continued in the study. An equal number (n = 4; 0.4%) of patients in each treatment group reported adverse events related to the breast. The adverse event profile for patients continuing in the second year was similar. In each treatment group, small increases, slightly greater for placebo-treated than finasteride-treated patients, were reported in nonscalp body hair by patient body hair assessment.

Table IV. Adverse events occurring in 1% of patients or more*

	Original studies (first year)		Extension studies (second year)			
	Finasteride (n = 779)	**Placebo (n = 774)**	***Fin→Fin* (n = 547)**	***Pbo→Fin* (n = 543)**	***Fin→Pbo* (n = 65)**	***Pbo→Pbo* (n = 60)**
Genitourinary system						
Urinary frequency	0	0	0	0	0	1 (1.7)
Sexual function						
Libido decreased	15 (1.9)	10 (1.3)	6 (1.1)	7 (1.3)	0	1 (1.7)
Erectile dysfunction	11 (1.4)	7 (0.9)	4 (0.7)	6 (1.1)	0	0
Decreased ejaculate volume	8 (1.0)	3 (0.4)	1 (0.2)	0	0	0
Skin and skin appendages						
Body hair growth increased	7 (0.9)	7 (0.9)	1 (0.2)	4 (0.7)	0	3 (5.0)

DISCUSSION

In these studies, finasteride treatment produced significant improvements in scalp hair in men with male pattern hair loss. The efficacy of finasteride was evident within 3 months of therapy. Hair count, first measured at 6 months, progressively increased over 1 year in the finasteride group, and the improvement was maintained through the second year. In contrast, the placebo group progressively lost hair, consistent with the miniaturization process and the natural history of male pattern hair loss. As is often observed in long-term studies, finasteride-treated patients who entered the extension studies had a slight tendency toward greater efficacy in hair count than those who did not (an increase of 92 vs 86 hairs from baseline at 1 year). Regardless, the net improvement for finasteride compared with placebo for continuing patients increased with time (107 hairs at 1 year and 138 hairs at 2 years).

Significantly more patients in the finasteride group reported improvements in scalp hair growth and appearance, as well as satisfaction with appearance, compared with the placebo group. Satisfaction with the frontal hairline was also improved compared with placebo, although the area of bitemporal recession was not specifically assessed. As is typical of patient questionnaire data, a placebo effect was observed, probably caused in part by recall bias, as each question assessed change from baseline. Nevertheless, patients in the placebo group perceived the loss documented by hair counts, as indicated by their responses to the question on slowing hair loss. Conversely, responses for finasteride-treated patients indicated that therapy led to slowing of further hair loss.

Assessment of hair growth by investigators also demonstrated the benefit of finasteride treatment, with a placebo effect also observed. Recall or other bias by investigators appeared to obscure detection of ongoing hair loss, documented by other end points, in placebo-treated patients.

In contrast, the blinded comparison of paired pretreatment and posttreatment global photographs by the expert panel, which also assessed change from baseline but was not subject to recall bias, demonstrated minimal, if any, placebo effect. By this assessment, finasteride treatment produced progressive improvement in hair growth for 2 years, whereas placebo-treated patients worsened. Because significant improvement was observed in finasteride-treated patients between months 12 and 24 while hair count was stable, the continued use of finasteride appears to improve the quality (ie, thickness, pigment, length and/or growth rate) of hair.

The safety and excellent tolerability of finasteride at 5 times the dose used in the present studies has been abundantly documented through large clinical trials and postmarketing surveillance for more than 5 years in men with BPH.[13,28] As expected from this body of experience, a few men in the current studies experienced reversible impairment of sexual function, but only 11 men receiving finasteride, compared with 8 men in the placebo group, discontinued treatment for this reason, with resolution in all. No other significant adverse effects of finasteride were observed. Based on observations of men with 5αR deficien-

cy, finasteride treatment might be expected to decrease body hair,[6] but no such effect was observed based on the patient body hair assessment administered in this study. The reduction observed in serum PSA is well understood, and for men in whom serum PSA is used as part of a screening evaluation for prostate cancer, guidelines have been published for interpretation in patients receiving finasteride treatment.[29,30] An ongoing 10-year study in 18,000 men[31] will test the hypothesis that finasteride 5 mg/d will reduce the risk of prostate cancer by reducing intraprostatic DHT.

As a type II 5α-reductase inhibitor, finasteride is contraindicated in women who are or may potentially be pregnant because of the risk that inhibition of conversion of fetal testosterone to DHT could impair virilization of a male fetus. Finasteride treatment has recently been shown to lack efficacy in postmenopausal women with androgenetic alopecia in a 1-year, placebo-controlled trial.[32]

Finasteride 1 mg/d improved scalp hair in men with male pattern hair loss within 3 months, with the benefit increasing with continued treatment. In contrast, men receiving placebo lost hair. These results confirm that DHT is a key factor in those men genetically predisposed for development of male androgenetic alopecia. Adverse events caused by finasteride treatment were minimal. Finasteride 1 mg represents a new oral therapy for men with male pattern hair loss.

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