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Evidence-based (S3) guideline

Evidence-based (S3) guideline for the treatment of androgenetic alopecia in women and in men

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Summary

Androgenetic alopecia is the most common hair loss disorder, affecting both men and women. Initial signs of androgenetic alopecia usually develop during teenage years leading to progressive hair loss with a pattern distribution. Moreover, its frequency increases with age and affects up to 80 % Caucasian men and 42 % of women.

Patients diagnosed with androgenetic alopecia may undergo significant impairment of quality of life. Despite the high prevalence and the variety of therapeutic options available, there have been no national or international evidence-based guidelines for the treatment of androgenetic alopecia in men and women so far. Therefore, the European Dermatology Forum (EDF) initiated a project to develop an evidence-based S3 guideline for the treatment of androgenetic alopecia. Based on a systematic literature research the efficacy of the currently available therapeutic options was assessed and therapeutic recommendations were passed in a consensus conference.

The purpose of the guideline is to provide dermatologists as well as general practitioners with an evidence-based tool for choosing an efficacious and safe therapy for patients with androgenetic alopecia.

I Introduction to the guideline

1.1 Needs/problems and issues in patient care

Androgenetic alopecia (AGA) is a common chronic dermatologic disease, affecting both men and women. It is characterized by progressive hair loss usually occurring in a pattern distribution. The frequency increases with age. In Caucasians, at the age of 70 or beyond 80 % of men and up to 42 % of women have signs of androgenetic alopecia. Though the prevalence is high in elderly patients, androgenetic alopecia often already starts at puberty.

Independent of age and gender, patients diagnosed with androgenetic alopecia undergo significant impairment in their quality of life. Hair is an important feature of image. Hair loss affects self-esteem, personal attractiveness and may lead to depression and other negative effects of life [1]. Androgenetic alopecia is clearly a burden for both sexes, but it is substantially more distressing for women [2].

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Patient's therapeutic experiences. Although there is a significant impairment in quality of life in many of the patients, Alfonso et al. revealed that three out of four men with androgenetic alopecia had never pursued therapy for hair loss [1]. On the other hand, lots of patients tried different therapies promising hair regrowth in vain and are dissatisfied with current therapeutic approaches when they first come to see a specialist. Consequently, their compliance is often poor. Men who had their hair loss successfully treated reported psychosocial benefits with improvements for self-esteem and personal attractiveness [1].

Patient's compliance. There are discrepancies between the interest for effective hair regrowth and willingness to perform a long-term therapeutic regimen. Limited efficacy, poor tolerance, fear and lack of information on treatment duration and possible adverse events lead to disappointment again.

Therapeutic concepts. The individual therapeutic concepts are still mainly based on physicians' personal experiences without taking into account the current evidence-based knowledge regarding the efficacy of the therapies.

1.2 Purpose of the guideline

The purpose of the guideline is to provide dermatologists with an evidence-based tool for choosing an efficacious and safe therapy for patients with androgenetic alopecia. The current guideline aims to prevent progressive hair loss and associated dermatological and psychosocial long-term complications by improving the individual therapeutic concept.

Improved patient care. The use of these evidence-based recommendations in clinical routine will lead to an improved patient care, as the physicians' personal experiences and traditional therapeutic concepts will be added and, if necessary, replaced by evidence-based assessments of the efficacy of the different therapeutic options.

Insure optimal usage of therapeutic regimen. In addition to the efficacy assessment the guideline provides details on administration and safety aspects of systemic, topical or surgical therapy. These instructions for use should decrease reservations of physicians and patients and assure that the therapy is provided in an optimal way. Initiation of the right therapy at the right time can prevent or at least slow down severe progression.

Improvement of patient's knowledge and compliance. Patients' compliance is most important in the individual response on a therapeutic concept. Good compliance is not only related with a balance of benefits, costs and adverse effects, but also requires the patient to be informed. By increasing the level of the patients' knowledge about the optimal use of each therapy and its possible complications, patients' compliance, response rates and satisfaction will increase. Information on administration and adverse reactions should serve to eliminate or reduce these and therefore will additionally improve compliance.

1.3 Directions for use of the Guideline

The current guideline is meant for dermatologists, general practitioners in clinics as well as in private practice and other specialists who are involved in the treatment of androgenetic alopecia. It should provide support in the development of individual therapeutic concepts. Each chapter summarizes the efficacy resulting from the evidence-based evaluation separately for men and women. Moreover, the experts provide information on practical aspects important for the different therapeutic regimens. The users of the guideline should be aware, that the listed aspects are not intended to be exhaustive. General obligations, which are part of every individual therapeutic decision, such as known allergies, potential intolerance reactions or contraindications are not conclusively individually listed.

Consequently, the users of the guideline have to consider additionally the manufacturer's product information and check the recommendations concerning completeness and up-to-datedness of dosages, contraindications or drug-interactions.

Although the authors took care that the guideline corresponds to the current state of the art at the time of completion, authors and publishers cannot take responsibility for dosages and therapeutic choices, as there may be changes and updates in between the actualisation cycles of the guideline. Therefore, the usage of the recommendations is at the reader's own responsibility and users are requested to keep informed about new knowledge regularly published parallel to the guidelines. The authors and publishers of the guideline would be pleased, if readers could inform them on potential inaccuracies.

1.4 Methodology

Literature research. A detailed description of methodology reflecting the process of developing the guidelines can be found in the method report of the guidelines. The methodology was defined as first step in development of the guideline. It was orientated on the standards of the AGREE instrument and on the methodology of the European S3 guideline for the treatment of psoriasis vulgaris.

To assess the efficacy of the individual therapeutic processes, a systematic search of literature in the databases Medline, Embase and Cochrane Library was conducted at 25th January 2007 and updated at 07th August 2008. Overall, 1 245 articles were found. Additional 51 articles were added by hand search. 125 articles were found by updating the literature research. Overall, after checking for doublets and relevance 396 articles were evaluated using the literature evaluation form (LEF) (see Appendix 1). 85 articles fulfilled the inclusion criteria of the guideline and built consequently the basis of the guideline. Figure 1 summarizes the process of literature research.

The evidence-based evaluations of these guidelines are restricted on the efficacy of the particular therapeutic options. All other issues outlined in the guideline, e.g. instructions for use, adverse events and contraindications, are based on opinions and personal experiences of the members of the guideline group.

Evidence assessment. The methodological quality of each study, which was included in the evidence-based analysis, was defined by the **grade of evidence**. We assessed the grade of evidence according to the following scheme:

- **A_1** Meta-analysis, which includes at least one randomized clinical trial of grade A_2 evidence with consistent results of the different studies.
- **A_2** Randomized, double-blind, comparative clinical studies of high-quality (e.g. sample size calculation, flow

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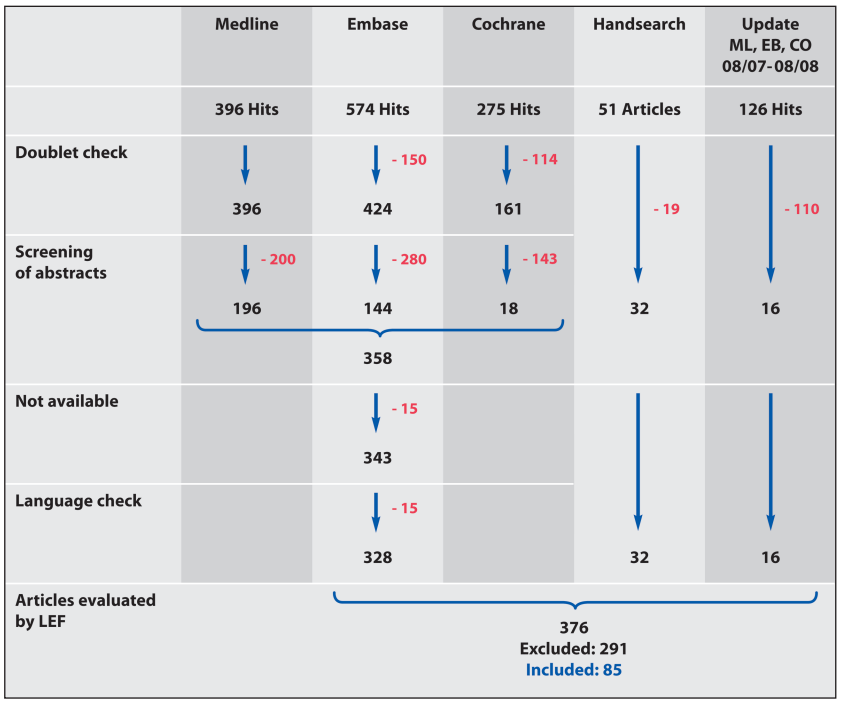

Figure 1: Schematic presentation of the process of systematic literature research.

chart of patient inclusion, ITT-analysis, sufficient size).

B Randomized, clinical studies of lesser quality or other comparable studies (not-randomized, cohort- or case-control-studies).

C Non-comparable studies.

D Expert opinion.

The grade of evidence was determined within the LEF form by the particular expert group and the staff member. The scheme for grading the evidence was used for assessment of monotherapies as well as combination therapies.

Level of evidence. After determining the grades of evidence of the individual studies, the grades of all studies belonging to a particular therapeutic regimen were summarized in a level of evidence. The **level of evidence** takes into account the methodological quality of the trials (grade of evidence) and the intertrial consistence of the results.

1 Studies grade A_1 evidence or studies with mainly consistent results grade A_2 evidence.

2 Studies grade A_2 evidence or studies with mainly consistent results grade B evidence.

3 Studies grade B evidence or studies with mainly consistent results grade C evidence.

4 Little to missing systematic evidence.

Therapeutic recommendation. Grades and levels of evidence were considered in the formal consensus process. The guideline group defined particularly relevant sections requiring consensus. These passages were discussed and approved at the consensus conferences. The resulting evidence-based therapeutic recommendations aim to optimize the therapeutic process and to support the practitioner in the individual decision on a suitable therapy. Nevertheless, the decision process on a particular therapy remains complex and limited on the individual case. It is not possible to define a strict clinical algorithm.

Strength of recommendation. This guideline summarizes the characteristics of the available drugs and their evidence-based therapeutic efficacies. The consented therapeutic recommendations were additionally weighted by the **strength of recommendation**. The strength of recommendation considers efficacy, evidence level, safety and practicability and was consented in a formal consensus process. The expert group agreed on a 5-point scale. This scale is illustrated by arrows:

↑↑ We recommend

↑ We suggest

→ Can be considered

↓ We do not suggest

↓↓ We do not recommend

II Introduction to androgenetic alopecia

Androgenetic alopecia is the most frequent form of alopecia in men and women. Today, in our societies, strong and dense hair is associated with youth, beauty, healthiness, attractiveness and success. Consequently, in patients presenting with androgenetic alopecia progressive thinning of hair often causes a psychological distress. Patients are looking for effective hair loss treatments in order to stop and prevent further thinning and to optimally stimulate regrowth. Knowledge on the efficacy of the different therapeutic options is essential for treating doctors and interest groups in the management of the disease and will lead to enhanced patient satisfaction.

2.1 Epidemiology

Androgenetic alopecia in men and women is present in populations of different ethnicities. Typically, frequency and severity increase with age. The highest prevalence is reported in the Caucasian population. At the age of 70 and beyond around 80 % of men and up to 42 % of women suffer from androgenetic alopecia [3, 4]. Usually, initial signs of the disease already develop in men at teenage [3–6]. Female pattern hair loss shows peaks of incidence at teenage and in postmenopause.

In the Asian population the frequency of androgenetic alopecia in male and female patients is lower compared to the Europeans. There is no information on the prevalence of the disease in African men and women.

2.2 Aetiology

Androgenetic alopecia is characterized by a non-scarring progressive miniaturization of the hair follicle in predisposed men and women, usually in a specific pattern distribution. Its aetiology is multifactorial and polygenetic [3].

In men androgenetic alopecia is an androgen-dependent trait. The terminal hair follicle becomes susceptible against dihydrotestosteron, which leads to a shortened anagen phase and miniaturization of terminal to vellus hair. The development of male androgenetic alopecia is predominantly hereditary. In men, family analyses showed strong concordance rates in twins and increased risk for sons with bald fathers [7]. Moreover,

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variant regions on the androgen receptor gene and at chromosome 20p11 are associated with the development of androgenetic alopecia in men [8, 9].

In female patients, little is known about the aetiology of androgenetic alopecia. Regarding inheritance, incidence of 54 % respectively 21 % are reported for women with male respectively female first degree relatives suffering from androgenetic alopecia [10, 11]. Possibly, early and late onset female androgenetic alopecia have different genetic traits. The androgen dependence is likewise uncertain in women, that is to say, other factors seem to be involved.

Nevertheless, it is important to consider that there is a subset of women with androgenetic alopecia and associated hormonal dysregulation. Detailed information on the steps in diagnostic procedure can be found in the S1 guideline for diagnostic evaluation in androgenetic alopecia in men, women and adolescents [3].

2.3 Clinical features

Clinically, androgenetic alopecia is characterized by a drift from terminal to vellus hairs and progressive thinning, usually in a pattern distribution. The different patterns can occur in men as well as in women, though the frequencies are gender-specific. Moreover, it is not rare to observe a diffuse thinning of the parietal and occipital areas in addition [3].

Male pattern, Hamilton-Norwood

The most frequent clinical pattern in men with androgenetic alopecia, only occasionally observed in women presents with a recession of the frontal hair line, mainly in a triangular pattern, later followed by a vertex thinning (Figure 2).

Female pattern, Ludwig

The so-called female pattern is characterized by a diffuse thinning of the centro-parietal region with preserving the frontal hair line (Figure 3). It is the most common type in women, occasionally also observed in men.

Christmas tree pattern

Similarly to the Ludwig pattern, the Christmas tree pattern shows diffuse centro-parietal thinning, but additionally, the frontal hair line is breached (Figure 4). The Christmas tree pattern is another common pattern in women.

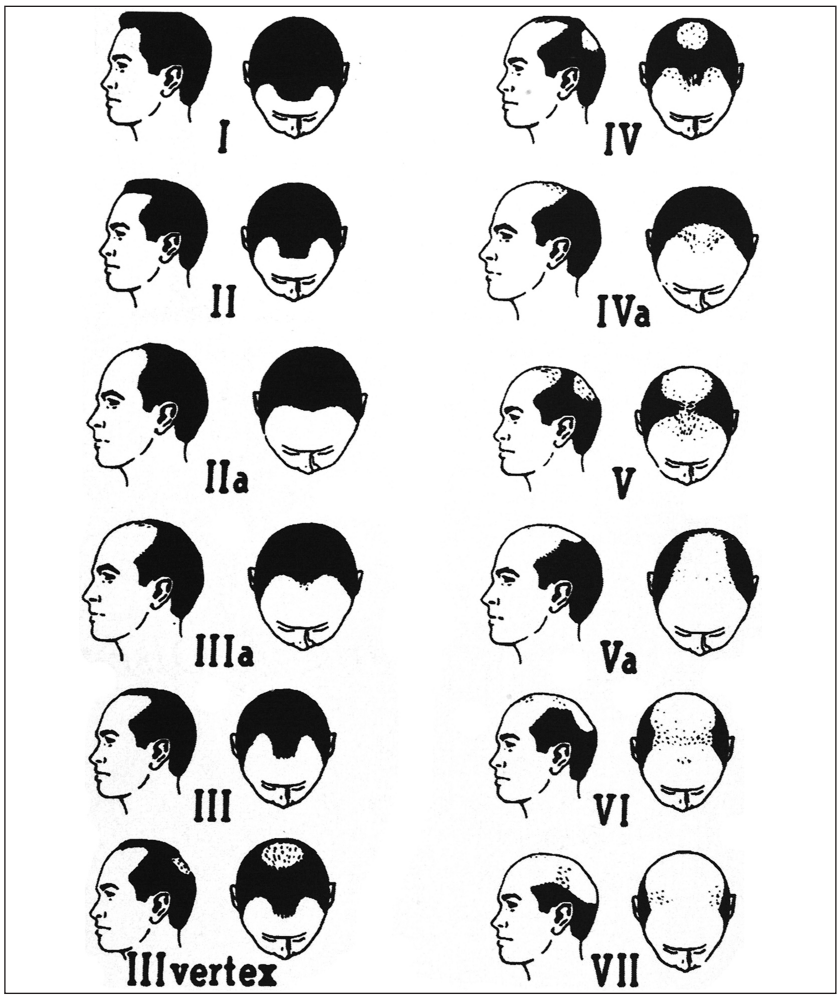

Figure 2: Hamilton-Norwood classification, male pattern (Norwood OT. Male pattern baldness: classification and incidence). South Med J 1975: 68(11); 1359–65.

Figure 3: Ludwig classification, female pattern (Olsen EA. Female pattern hair loss. J Am Acad Dermatol 2001; 45(Suppl.): S70–80.

2.4 Diagnosis

Androgenetic alopecia is usually diagnosed clinically by examination of hair and scalp showing a non-scarring alopecia in the typical pattern distribution [3].

The clinical examination should also include a pull test and an examination of facial and body hair as well as nails to exclude differential diagnoses; diffuse telogen effluvium, alopecia areata and cicatrical alopecia in particular [3].

Due to the high prevalence of androgenetic alopecia its coincident appearance to other hair diseases should be taken into account. If a differential diagnosis cannot be excluded clinically, further

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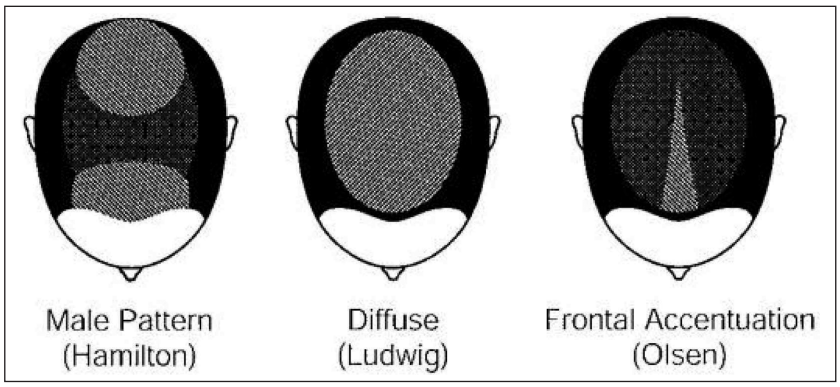

Figure 4: Christmas tree pattern (Olsen EA. Current and novel methods for assessing efficacy of hair growth promoters in pattern hair loss. J Am Acad Dermatol 2003; 48(2): 253–62).

diagnostics such as evaluations of hair roots, laboratory or histology can be helpful.

2.5 Hair growth assessment techniques

To document the extent of androgenetic alopecia in clinical practice, the different classifications of the pattern distribution are subdivided (Hamilton-Norwood I–VII, Ludwig I–III, Christmas tree pattern I–III). However, a generally applicable definition for the extent of androgenetic alopecia does not exist. Moreover, the documentation of the degree of the pattern distribution is often unsuitable to reflect the course of androgenetic alopecia.

As androgenetic alopecia is a ***naturally progressive disease***, therapy can have two required ***outcomes: cessation of hair loss and induction of hair regrowth***. In clinical practice, the evaluation and follow-up of hair growth is generally restricted to individual assessment of patient and physician. In clinical studies, the subjective hair growth assessment by patient and investigator are substantiated by objective hair count/density methods and assessment of standardized global photographs.

The global photographic assessment is a semi-objective tool in hair growth evaluation. Global photographs are assessed by experts blinded to treatment and time.

Automatic digitalized photographic systems are able to quantify hair density, hair thickness, anagen/telogen hair ratio, terminal/vellus hair ratio within an investigational area. A dot tattoo guaranties the analysis of the same area to ensure reproducibility in studies. The technique is limited by the size of the measured area. In clinical trials comparison to baseline and to placebo resp. another treatment is necessary for efficacy assessment of a therapeutic option.

Within the development of the S3 guideline the experts voted on a ***ranking of the different investigative methods*** and outcome parameters. The global photographic assessment was voted to be the most effective method in hair growth evaluation, as the whole scalp hair is evaluated in a standardized way. Patient's and investigator's perception can be excluded. According to the experts, global photographs should also be used in routine clinical practice for long-term follow-up.

2.6 Risk/benefit considerations

In routine clinical practice the individual decision for a particular treatment of androgenetic alopecia does not only depend on the efficacy, but also on practicability, risks and costs. The assessment of cost effectiveness has to be made by balancing the costs with the benefit attained. Consequently, expensive therapeutic options can also be cost-effective, if they are highly effective.

As the patient usually has to bear the full costs of the treatment, ***consideration of patient-relevant benefit is essential***. The benefit attained in the therapy of androgenetic alopecia is not only stabilization, prevention of progression and induction of hair growth, but also an improved quality of life.

The guideline offers evidence-based analyses of the existing therapeutic options that help to make suitable cost-benefit decisions in the assessment of the specific case.

III Therapeutic options and therapy assessment

The following chapters summarize the evidence-based efficacy assessment of the different therapeutic options in the treatment of androgenetic alopecia in men and women. Efficacy was evaluated separately for men and women.

Result tables. All studies that fulfilled the inclusion criteria of the guideline are listed in result tables (see Appendix 2). The evidence-based results of the trials are outlined in the particular chapter, but can be read in detail in the result tables, if required. Based on the result tables the expert group passed therapeutic recommendations for the different regimens by formal consensus process.

Overview of common therapeutic options. Table 1 shows a summary of evidence level, efficacy to prevent progression and/or improve androgenetic alopecia, safety aspects and practicability for the most common therapeutic interventions. Its intention is to provide a first rough orientation. Its exclusive use is not sufficient for individual therapeutic choices. Deeper observation of the individual factors of a given patient and its impact on the different therapeutic regimens are necessary.

3.1 Minoxidil

3.1.1 Introduction

Minoxidil was originally developed as an oral drug (trade name Loniten®) to treat high blood pressure. Its possible use in androgenetic alopecia was discovered when its side effect of increasing hair growth was observed.

Chemically, minoxidil is a pyrimidine derivate. It was the first product to be approved for the treatment of AGA in both men and women. The 2 % topical solution was first approved by the U.S. Food and Drug Administration (FDA) in1988 for the treatment of androgenetic alopecia in men and in 1991 in women. The 5 % solution was approved in 1997 for the treatment of androgenetic alopecia in men followed by approval of the 5 % foam in 2006 also for the treatment of androgenetic alopecia in men.

3.1.2 Mechanism of action

To exert its effect minoxidil needs to be transformed to its active metabolite,

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Table 1: Summary of evidence level, efficacy, safety aspects and practicability for the most common therapeutic interventions.

Therapy	Level of evidence	Efficacy to prevent progression	Efficacy to improve	Safety	Practicability (patient)	Practicability (physician)
Male patients						
Finasteride	1	+++	++	+++	++++	+++ / ++++
Minoxidil 5%	1	+++	++	++++	+ / ++	+++
Surgery	4	-	+++	++	+ intervention +++ long-term	+
Female patients						
Minoxidil 2%	1	+++	++	++++	+	+++
Hormones oral Hyperandrogenism normal hormones	3	+ + / -	+ + / -	+ +	+++ +++	++ ++
Surgery	4	-	++	++	+ intervention +++ long-term	+

minoxidil sulphate by the enzyme sulphotranspherase, which is present in the outer root sheath of anagen follicles. The exact mechanism by which minoxidil promotes hair growth is still unclear. Its active metabolite, minoxidil sulphate opens ATP-sensitive potassium channels in cell membranes, which conveys vasodilatory effect. Vasodilatation, however, does not appear to be responsible for minoxidil-induced hair growth. Studies on skin blood flow after topical minoxidil application produced inconsistent results.

Other possible effects of minoxidil on the hair follicles include:

a) increased expression of vascular endothelial growth factor (VEGF) mRNA in the dermal papilla. This indicates that the drug induces angiogenesis in the dermal papilla.
b) activation of cytoprotective prostaglandin synthase-1, a cytoprotective enzyme that stimulates hair growth.
c) increased expression of hepatocyte growth factor (HGF) m-RNA; HGF is an hair growth promoter.

3.1.3 Efficacy – males

34 studies assessing the efficacy of minoxidil in male patients with androgenetic alopecia met the inclusion criteria for the guideline [12–45]. 3 out of them treated male and female patients. 25 studies were placebo controlled. The majority of studies obtained grade A2 and B evidence (A2 = 17, B = 13, C = 3) resulting in **evidence level 1**.

In general most of the trials assessed the efficacy of minoxidil solution 2 % respectively 3 %, applied twice daily. In all trials that examined the effect of minoxidil 2 % solution, regular topical application resulted in hair regrowth.

Outcomes

The mean change from baseline *total hair count* ranged between 5.4 hairs/cm^2 and 29.9 hairs/cm^2 (11.0–54.8 %) at 4 to 6 months and between 15.5 hairs/cm^2 and 83.3 hairs/cm^2 (14.8–248.5 %) at 12 months [12, 13, 17, 19, 22, 23, 26, 27, 29, 30, 35, 38–40, 42, 43].

At 4 to 6 months the mean total hair count changes in the majority of studies were statistically significant compared to placebo (p between 0.074 and < 0.0001). At 12 months most of the older trials switched the placebo group also to minoxidil treatment.

Comparable to the results in total hair count the mean changes in *nonvellus hair counts* were also significantly different to placebo (p between < 0.05 and 0.001). There was a mean change in nonvellus hair counts between 4.7 hairs/cm^2 to 37.3 hairs/cm^2 (17.2–59.4 %) at 6 months, between 9.4 hairs/cm^2 to 41.8 hairs/cm^2 (8.8–443.8 %) at 12 months [12, 13, 17–22, 24, 26, 27, 29, 30, 33, 35, 43, 45].

The increases from total and nonvellus hair counts at 6 and 12 months did significantly differ from baseline hair counts (p between 0.01 > p < 0.0001).

It must be noted that the reported placebo rate in most of the minoxidil studies is very high. The mean increase from baseline total hair count of the placebo group ranged between 6.1 hairs/cm^2 and 22.4 hairs/cm^2 (9.3 and 48.8 %) at 4 to 6 months.

Dosage

Concentration. Minoxidil dosages below 2 % showed significant reduced mean changes from baseline total hair count in comparison to minoxidil 2 % at 6 months [30, 45]. The mean changes from nonvellus hair counts were not significantly different for minoxidil 0.1 %, 1 %, 2 % at 6 months.

Minoxidil 3 % solution, applied twice daily was not significantly different from minoxidil 2 %, twice daily (mean change from total hair count/nonvellus hair count at 4 respectively 12 months) [21, 25, 26, 29, 31, 32, 39, 40, 43]. Only Katz et al. reported a significance of p = 0.0464 at 4 months in mean change of nonvellus hair counts [24].

2 studies comparing minoxidil 2 % solution, twice daily and minoxidil 5 % solution, twice daily were included in the evidence-based analysis [33, 37]. In both studies the outcome of the minoxidil 5 % group was superior to minoxidil 2 % (mean change from baseline nonvellus hair count 18.6 hairs/cm^2 (12.3 %) vs. 12.7 hairs/cm^2 (8.8 %) at 12 months, p = 0.025, mean % change from baseline total hair count 30 % vs. 25 % at 24 months, p = 0.455).

Application frequency. Olsen et al. showed, that minoxidil 3 % applied twice daily was

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superior to application once daily (mean change from baseline total hair count 64.4 hairs/cm^2 vs. 44.1 hairs/cm^2 at 33 months, p = 0.015, mean change from baseline nonvellus hair count 4.4 hairs/cm^2 vs. –13.4 hairs/cm^2 at 36 months) [32].

Formulation

The standard formulation of minoxidil is a solution containing propylenglycol. Olsen et al. studied a foam formulation containing 5 % minoxidil [34]. The mean change from baseline nonvellus hair count was highly significant different from placebo at 16 weeks (20.9 hairs/cm^2 [13.4 %] vs. 4.7 hairs/cm^2 [3.4 %], p < 0.0001).

Piepkorn et al. examined minoxidil 2 % in a gel formulation and as solution [36]. Whereas placebo gel and solution reached comparable percentage improvement in subject's assessment (33 vs. 36 %), minoxidil 2 % gel, twice daily had 26 % improvement, minoxidil 2 % solution, twice daily 48 % improvement after 6 months in subject evaluation.

Minoxidil vs. finasteride

In comparison to finasteride 1 mg daily Arca et al. reported an 80 % improvement in global photographic assessment for minoxidil 5 %, twice daily and 52 % for finasteride at 12 months [14]. On the other hand, a study by Saraswat et al. reported superiority of minoxidil 2 % solution, applied twice daily, in comparison to finasteride 1 mg/d (mean change from baseline total hair count 36.1 hairs/cm^2 [29.1 %] vs. 19.6 [14.8 %] at 12 months, p = 0.003) [42].

3.1.4 Efficacy – females

11 studies that investigated the efficacy of topical minoxidil in female patients suffering from androgenetic alopecia could be included in the evidence-based evaluation [15, 39, 40, 46–53]. 3 studies treated male and female patients. 7 studies obtained grade B evidence, 4 studies grade A2 evidence, resulting in an **evidence level 1**.

Outcomes

Minoxidil 1 % solution, applied twice daily led to mean changes from baseline total hair count at 6 months from 15.2 hairs/cm^2 (8.0 %) [54]. Minoxidil 2 % solution showed mean changes from baseline nonvellus hair count at 6 months between 21.0 hairs/cm^2 and 50.1 hairs/cm^2 (12.4–31.3 %) [39, 47–51, 53].

Except the study by Whiting et al. all studies showed significant different mean changes from baseline hair counts in comparison to placebo (p between 0.02 and < 0.001).

Dosage

Concentration. The mean changes in nonvellus hair counts between minoxidil 5 % and 2 % in female patients were not statistically significant (p = 0.129). At 12 months the mean change from nonvellus hair count was 20.7 hairs/cm^2 (13.8 %) for minoxidil 2 %, twice daily, 24.5 hairs/cm^2 (17.3 %) for minoxidil 5 %, twice daily and 9.4 hairs/cm^2 (6.8 %) for placebo, twice daily (p < 0.001 vs. placebo).

Minoxidil vs. Alfatradiol

In comparison to topical alfatradiol 0.025 %, once daily, minoxidil 2 % solution, twice daily, led to increased hair counts after 6 months [46]. The mean change from baseline total hair count was –7.8 hairs/cm^2 (–4.3 %) for alfatradiol and 15.3 hairs/cm^2 (8.7 %) for minoxidil 2 % (p < 0.0005).

3.1.5 Instructions for use / Practicability

Treatment with minoxidil converts partially miniaturized (intermediate) hairs to terminal hairs and produces at least a partial normalization of the hair follicle morphology.

Minoxidil should be applied as 1 ml of solution with a pipette or half a cap of foam to dry hair and scalp once in the morning and again in the evening and left in place for at least four hours. When using spray applicator it has to be spread evenly over the affected areas. Hands should be washed with warm water after application.

Treatment efficacy should be assessed at least 6 months after initiation of therapy and treatment should be maintained as long as the patient is interested to maintain the efficacy.

Some patients may experience *increased hair shedding* during the first months of the treatment. This is transitory and only indicates that the drug is stimulating telogen follicles to re-enter anagen. It is important to inform the patient about a possible telogen shedding, before the treatment is started. If shedding occurs, therapy should be maintained. Usually, increased effluvium due to telogen shedding normalizes within a few weeks to months. Good patient-practitioner-relationship and detailed patient information are essential for good compliance.

Stopping topical minoxidil application is followed by increased shedding, usually 3 months after discontinuing the treatment.

The main reported side effect of topical minoxidil is *hypertrichosis*, which is more common with the 5 % concentration, but can also be due to incorrect application. To avoid contamination of the pillow with subsequent contact with face patients should be advised to apply the drug at least 2 hours before going to bed.

Irritant and allergic contact dermatitis may also occur. Irritation is more common with the 5 % solution due to its higher propylene glycole content. Contact dermatitis should be excluded by patch testing. If it is due to propylene glycole, an alternative vehicle can be used, whereas if irritation and contact dermatitis are due to minoxidil itself, drug interruption is unavoidable.

Minoxidil is contraindicated during pregnancy and lactation.

3.1.6 Combination therapies

A study by Berger et al. failed to show, that combination of minoxidil 5 % solution and pyrithione zinc shampoo is superior to minoxidil monotherapy [16]. Minoxidil 5 % solution, twice daily combined with pyrithione zinc shampoo 1 x/d vs. minoxidil 5 % solution twice daily and placebo shampoo showed mean change from baseline total hair count of 6.2 hairs/cm^2 and 12.3 hairs/cm^2 respectively.

Bazzano et al. compared in a study of male and female patients minoxidil 0.5 % solution, 2 x/d, tretinoin 0.025 % solution, 2 x/d, placebo and the combination of minoxidil 0.5 % with tretinoin 0.025 % [15]. At 12 months, 58 % of the patients of the tretinoin group and 66 % of the patients with the combined treatment had at least 20 % or more increase from baseline total hair count.

Shin et al. failed to prove significance between minoxidil 5 % solution, twice daily and a combination of minoxidil 5 % and tretinoin 0.01 %, once daily [44]. The mean change from baseline total hair count at 18 weeks was 15.9 hairs/cm^2 (12.8 %) vs. 18.2 hairs/cm^2 (14.7 %) (p not significant).

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Topical minoxidil 2 % solution, 2 x/d in combination with an oral hormonal contraceptive led to a mean change from baseline total hair count of 16.1 hairs/cm^2 (8.6 %) at 6 months, 16.9 hairs/cm^2 (9.1 %) at 12 months, whereas cyproterone acetate 50 mg in combination with oral hormonal contraceptive led to decreased values (–2.8 hairs/cm^2 [–1.4 %] at 6 months, –7.8 hairs/cm^2 [–3.9 %] at 12 months [p < 0.001]) [52].

3.1.7 Summary

Minoxidil 2 % solution is effective to prevent progression and improve androgenetic alopecia in male and female patients (evidence level 1). Minoxidil 5 % solution is more effective than the 2 % solution in male patients (evidence level 2). Patients should be informed on telogen shedding within the first 8 weeks of therapy. Further studies are required to compare efficacy of minoxidil solution and foam formulation.

3.1.8 Therapeutic recommendation – Male

↑↑	Topical minoxidil 2 to 5 % solution 1 ml twice daily is recommended to improve or to prevent progression of AGA in male patients above 18 years with mild to moderate AGA (Hamilton-Norwood IIv–V).
↑	We suggest using 5 % solution for greater efficacy.
→	There is not enough data to recommend the 5 % minoxidil foam instead of the 5 % solution.
↑	The response to treatment should be assessed at 6 months. If successful, treatment needs to be continued to maintain efficacy.

3.1.9 Therapeutic recommendation – Female

↑↑	Topical minoxidil 2 % solution 1 ml twice daily is recommended to improve or to prevent progression of AGA in female patients above 18 years with AGA.
→	There is not enough data to recommend the 5 % minoxidil solution instead of the 2 % solution.
↑	The response to treatment should be assessed at 6 months. If successful, treatment needs to be continued to maintain efficacy.

3.2 5-alpha-reductase-inhibitors

3.2.1 Introduction

Androgenetic alopecia occurs after puberty in men with an inhered sensitivity to the effects of androgens on androgenetic sensitive scalp hair follicles. AGA does not develop in men without testosterone, and we know since 1974 that AGA does not occur in men with a genetic deficiency of the enzyme 5-alpha-reductase type II which converts testosterone to dihydrotestosterone (DHT) [55]. Two types of 5-alpha-reductase-inhibitors exist in humans. Type I predominates in liver, skin and scalp. Type II predominates in prostate and genitourinary tract, but also in the human hair follicle.

Initially, pharmaceutical 5-alpha-reductase-inhibitors were developed for the treatment of benign prostatic hyperplasia. Two drugs inhibiting the 5-alpha-reductase are available on the market: finasteride registered in Europe in 1992, and dutasteride registered in 2003. Finasteride is a type II 5-alpha-reductase-inhibitor which decreases DHT of about 65 % in serum, prostate and scalp. Dutasteride inhibits both type I and type II 5-alpha-reductase resulting in a decrease of the serum DHT level of about 90 %.

Two years after the registration of finasteride for the treatment of benign prostatic hyperplasia, first publications appeared concerning the efficacy of finasteride in androgenetic alopecia in male patients. At the same time, the drug was registered in the US (1993) and Europe (1994) for therapy of mild to moderate androgenetic alopecia in men.

First report on the use of dutasteride as a treatment for androgenetic alopecia was published in 2006, but up to now it is still only registered for treatment of benign prostatic hyperplasia.

3.2.2 Mechanism of action

A single oral administration of finasteride 1 mg decreases serum DHT as well as scalp DHT up to 70 % compared to baseline. Tachyphylaxis is not observed with long-term administration.

Finasteride is quickly absorbed after oral intake with peak plasma level occurring 1 to 2 hours after drug intake. The serum half-life of the drug is about 6 hours. 90 % of the drug is bound to plasma proteins. Finasteride is metabolised in the liver by hydroxylation and oxidation using P 450 3A4 pathway but without interaction with other drugs known to be metabolized by this cytochrome, such as warfarin, theophylline, digoxine and propanolol.

3.2.3 Efficacy – males

Finasteride

18 studies looking at the efficacy of finasteride in male patients with androgenetic alopecia met the inclusion criteria of the guideline [14, 42, 56–71]. 16 of them assessed the efficacy of finasteride monotherapy in male patients with androgenetic alopecia. 12 studies obtained grade A2 evidence, 5 grade B and 1 grade C. 12 studies were placebo controlled. Summarizing these results an **evidence level 1** can be attributed for finasteride.

Outcomes

In all of the included trials, the intake of finasteride 1 mg daily led to a significant increase in *total hair counts* compared to placebo. The mean change from baseline total hair count was 7.0 hairs/cm^2 (3.3 %) in the frontal/centroparietal region (p < 0.0001 vs. placebo) [60] and 13.5 hairs/cm^2 (7.3 %) in the vertex (p < 0.0001 vs. placebo) [64] at 6 months.

The mean increase from baseline total hair counts at 12 months was between 7.2 hairs/cm^2 (3.6 %) and 36.1 hairs/cm^2 (29.1 %) for the vertex (p between < 0.05 and 0.001 vs. placebo) [57, 60, 64, 65, 67, 68] and 9.3 hairs/cm^2 (4.9 %) and 9.6 hairs/cm^2 (4.6 %) in the frontal/centroparietal region (p between < 0.01 and 0.001 vs. placebo) [62, 72]. The placebo group showed at the same time mean changes from baseline total hair count between 2.4 hairs/cm^2 (1.4 %) and –10.1 hairs/cm^2 (–5.2 %).

At *global expert panel assessment* between 37 % and 54 % of the patients were rated as improved at 12 months (p < 0.001 vs. placebo) [57, 58, 60, 64, 65, 71]. In addition, subjective assessments by investigator and patients yielded significant improvements in the finasteride group [57, 58, 60, 64, 65, 69].

Long-term results were available for 24, 36, 48 and 60 months. The mean changes from baseline total hair counts were 13.0 hairs/cm^2 (6.2 %) at 24 months [60], 8.5 % at 36 months [63], 7.2 % at 48 months [63] and 7.5 hairs/cm^2 (4.3 %) at 60 months [71] respectively. The changes were statistically significant in comparison to placebo.

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Price et al. reported increase in hair weight at 12 to 48 months (20.4 % at 12 months, 21.5 % at 24 months, 19.5 % at 36 months and 21.6 % at 48 months vs. –5.2 %, –14.2 %, –14.8 % or –24.5 % in the placebo group, $p < 0.001$) [62, 63].

Dosage

Concentration. Two studies examining different finasteride dosages could be included in the evidence-based evaluation [58, 64]. Roberts et al. examined finasteride 0.01 mg, 0.2 mg, 1 mg and 5 mg versus placebo [64]. The mean change from baseline total hair counts under finasteride therapy (0.2 mg, 1 mg and 5 mg) was significantly different to placebo at 6 and 12 months ($p < 0.001$), whereas dosage of 0.01 mg showed progressing hair loss (difference to placebo not statistically significant). The differences in mean change from baseline total hair count between the finasteride groups (0.2–5 mg) did not reach significance.

Kawashima et al. reported 58 % respectively 54 % improvement in global expert panel assessments for finasteride 1 mg respectively 0.2 mg [58]. The efficacies in both groups were comparable and significantly different compared to placebo ($p < 0.001$).

Finasteride vs. Minoxidil

Only few data comparing finasteride 1 mg daily and minoxidil solution is available. Two of the included studies examined finasteride 1 mg against twice daily topical application of minoxidil 2 % solution [42, 59]. Both studies showed superiority for finasteride. At 12 months the mean change from baseline total hair count was 36.1 hairs/cm^2 (29.1 %) for finasteride 1 mg and 19.6 hairs/cm^2 (14.8 %) for minoxidil 2 %, twice daily application ($p = 0.003$) [42]. 87 % of the patients taking finasteride vs. 42 % of the minoxidil 2 % patients were rated as improved ($p < 0.001$) [59].

Arca et al. reported a better outcome for minoxidil 5 % solution applied twice daily against finasteride 1 mg daily at global photographic assessment of the frontal/parietal region at 12 months (80 % vs. 52 % improvement) [14].

Dutasteride

2 studies investigating dutasteride in androgenetic alopecia with grade A2 evidence were included in the evidence-based evaluation, resulting in a **level of evidence 2** [61, 73].

Outcomes

Stough et al. reported a significant mean increase from baseline total hair count of 6.8 hairs/cm^2 at 6 months and 16.5 hairs/cm^2 at 12 months for dutasteride 0.5 mg daily [73]. Olsen et al. showed in a study with 416 patients a significant increase from baseline total hair count for the different tested dutasteride dosages (dutasteride 0.1 mg 15.4 hairs/cm^2 (8.7 %), dutasteride 0.5 mg 18.6 hairs/cm^2 [10.2 %], dutasteride 2.5 mg 21.5 hairs/cm^2 [11.3 %]) at 24 weeks) [61]. Dutasteride 2.5 mg daily showed the best increase in hair count. The mean change from total hair count of finasteride 5 mg was significantly different to dutasteride 2.5 mg (14.8 hairs/cm^2 [8.4 %] vs. 21.5 hairs/cm^2 [11.3 %], $p = 0.009$). All dutasteride arms and finasteride 5 mg showed a significant difference of $p < 0.001$ vs. placebo.

Assessing the dutasteride results it is necessary to remark, that the most effective dosage of dutasteride 2.5 mg is 5 times higher than the standard dosage in the treatment of benign prostatic hyperplasia (dutasteride 0.5 mg corresponds to finasteride 5 mg). Trials comparing dutasteride to the standard dosage of finasteride 1 mg daily are required.

3.2.4 Efficacy – females

2 studies assessing the efficacy of finasteride 1 mg daily in female patients were included in the evidence-based evaluation [68, 74]. The grades of evidence were A2 and B, resulting in an **evidence level 2**.

Outcomes

Both studies showed a further progression of hair loss. The mean change from baseline hair count at 12 months was –14.6 hairs/cm^2 (–5.9 %) and –8.7 hairs/cm^2 (–5.8 %). Moreover, the mean decrease from baseline hair count in the finasteride group outvalued the placebo group (0 hairs/cm^2 [0 %] resp. –6.6 hairs/cm^2 [–4.0 %]). Trials with higher finasteride dosages or subgroup analyses in female patients of childbearing age were not available.

3.2.5 Instructions for use / Practicability

Finasteride can be taken with or without food and there is no known interaction with other drugs.

Finasteride is not indicated in women and is contraindicated in pregnant women, because of the risk of feminisation of a male foetus. Finasteride-treated men must avoid donating blood.

The level of finasteride in the semen of treated man is very low even with regular intake of finasteride 5 mg/day, and there is no risk in case of sexual relation with pregnant women. Use of a condom is not necessary for this reason.

The recommended dosage is 1 mg a day, but in a study with a lower dosage of 0.2 mg/day significant improvement compared to placebo was observed. If a patient forgets a pill, taking the double dosage the next day is therefore not recommended. In case of adverse events a dosage of 0.2 mg/ daily or a dosage of 0.5 to 1 mg every other day can be discussed, although no clinical studies are available.

Minimal period of use prior to assessing the efficacy is 6 months for reducing hair loss and 12 months for regrowth of hair.

If a patient intends to switch from minoxidil to finasteride we recommend a combination therapy for at least 3 but preferably 6 months before discontinuing minoxidil in order to avoid significant hair loss while finasteride action can take over.

Finasteride reduces PSA level. If treatment is started after the age of 45 years, monitoring of PSA level should be considered. The PSA levels should be double to compensate the reduction due to finasteride, resulting in an interpretation of the test remaining accurate.

Additional research is required on higher dosages of finasteride and different subgroups of female patients with pattern hair loss including females of childbearing potential and postmenopausal women as well as females with or without clinical signs of hyperandrogenism. Nevertheless, use of finasteride in females of childbearing potential can be considered only in combination with a safe contraceptive method due to the risk of malformation of genitals in male fetus (feminization). Overall, women under systemic finasteride should avoid donating blood.

3.2.6 Combination therapies

Leavitt et al. observed 79 male patients undergoing hair transplantation and reported that the combination with finasteride 1 mg daily led to increased hair counts after 12 months, whereas hair

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transplantation alone resulted in decreased hair count in the frontal area (mean change from total baseline hair count 18.5 hairs/cm^2 [2.6 %] vs. –13.5 hairs/cm^2 [–8.9 %], p = 0.019) [70].
Khandpur et al. compared the combination of finasteride 1 mg daily with minoxidil 2 % solution twice daily respectively ketoconazole 2 % shampoo, 3 x weekly to finasteride 1 mg daily and minoxidil 2 % solution twice daily as monotherapies [59]. At 12 months, 100 % of the patients of each combined therapy, 87 % for finasteride and 42 % for minoxidil 2 % solution were rated as improved by the investigator. The combination of minoxidil 2 % and finasteride 1 mg was statistically significant superior to finasteride or minoxidil monotherapies. Furthermore, Diani et al. showed an additive effect of finasteride and minoxidil in stumptail macaque [75].
Working mechanisms of minoxidil and finasteride are different. Thus, association of both drugs is possible and can be considered in motivated patients.

3.2.7 Summary
Finasteride 1 mg daily is effective in prevention of hair loss progression and induction of hair regrowth in androgenetic alopecia in male patients (evidence level 1).
Evaluation of the efficacy should be assessed 6 months after treatment initiation.
Patients should be aware of the reduction of prostate-specific antigen, which is important in prostate cancer screening in men older than 45 years.
Further studies comparing the efficacy of finasteride 1 mg vs. minoxidil 5 % are needed. If therapeutic approach is insufficient the combination of finasteride 1 mg and minoxidil 2 % or 5 % can be considered.
There is no reason to use dutasteride 0.5 mg instead of finasteride 1 mg, as higher dosages are needed to reach comparable efficacy and comparison studies vs. finasteride 1 mg daily are missing.
In postmenopausal female patients finasteride 1 mg failed to show efficacy (evidence level 2). Additional research is required at higher dosages and in different subgroups of female patients with androgenetic alopecia. If finasteride is used in women its use is off-label and at own responsibility; in particular in women of childbearing age, a safe contraceptive method is essential as finasteride may lead to feminisation of the male foetus.

3.2.8 Therapeutic recommendation – Male

Finasteride

↑↑ Oral finasteride 1 mg a day is recommended to improve or to prevent progression of AGA in male patients above 18 years with mild to moderate AGA (Hamilton-Norwood IIv–V).

↑ The response to treatment should be assessed at 6 months, although in some men it may not become evident until 12 months. If successful, treatment needs to be continued to maintain efficacy.

➔ There is insufficient evidence to support the use of topical finasteride.

➔ For greater efficacy the combination of oral finasteride 1 mg, 1 x/d and topical minoxidil 2 % to 5 % solution, 2 x/d can be considered.

Dutasteride

➔ Oral dutasteride 0.5 mg a day can be considered to improve or to prevent progression of AGA in male patients above 18 years with mild to moderate AGA (Hamilton-Norwood IIv–V).

High quality controlled clinical trials comparing dutasteride 0.5 mg to finasteride 1 mg are needed.

3.2.9 Therapeutic recommendation – Female

↓ Oral finasteride 1 mg daily is not suggested in the treatment of postmenopausal women with female pattern hair loss.

High quality controlled clinical trials with finasteride at different dosages on female patients are required.

3.3 Hormones

3.3.1 Introduction
The role of androgens in the aetiology of androgenetic alopecia has led to the widespread use of hormonal agents in its treatment. They fall into two broad groups: antiandrogens and oestrogenic (or anti-oestrogenic) drugs, although evidence of efficacy for any of these treatments is limited or absent.

3.3.2 Mechanism of action
Antiandrogens act primarily through blockade of the androgen receptor. Different agents may have other relevant effects on endocrine biology including inhibition of steroid synthesis and progestational activity. Antiandrogens have mainly been delivered systemically and used in women (they are contraindicated in men due to their feminizing action).
Topical oestrogens and anti-oestrogens have been used in both men and women. The rationale for their use is less clear than for antiandrogens as the effect, if any, of oestrogens on human hair growth is unknown. Oestrogens inhibit hair growth in several other mammals lending some support for the potential of antioestrogens to promote hair growth in humans.

3.3.3 Efficacy – males
Oral hormonal treatment
There is no evidence to support the use of oral oestrogens or antiandrogens to improve or prevent progression of androgenetic alopecia in male patients (**evidence level 4**).

Topical hormonal treatment
There are only two controlled trials of topical hormonal treatment that have employed modern methods of assessment.

Outcomes
Sovak et al. [76] studied the change of anagen hair count following daily topical application of the antiandrogen fluridil versus placebo. At 12 months, the response to fluridil was not significantly different from placebo. Thus, there is limited evidence that topical fluridil is ineffective in men [76].
Gassmueller et al. [77] compared topical application of fulvestrant (70 mg/ml twice daily), an oestrogen receptor antagonist, to minoxidil 2 % and placebo. At 16 weeks the mean change from baseline hair counts was not significantly different from placebo in the fulvestrant group, whereas there was a significant increase in hair counts in subjects treated with minoxidil. Thus, there is evidence

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that topical fulvestrant is ineffective in men.

There are three earlier studies on the efficacy of the topical oestrogen alfatradiol (= 17 alpha-oestradiol) in men. Unfortunately they either had no control group [78, 79] and/or the results were not reported separately for each sex [79, 80]. In one study, topical corticosteroid was included [79].

3.3.4 Efficacy – females

Oral hormonal treatment

Two studies met the inclusion criteria (grade of evidence B, **level of evidence 3**).

Outcomes

Peereboom-Wynia et al. compared a group of women treated for one year with Diane® (50 µg estradiol + 2 mg cyproterone acetate) + 20 mg cyproterone acetate days 1–14 with an untreated control group [81]. Trichogram data showed a mean change in anagen percentage from 49.7 at baseline to 74.4 after one year in the treated group compared to a fall from 60.4 to 48.8 in the controls. Subjects appeared not to be randomized to treatment or control groups and hair counts were not performed [81].

Vexiau et al. reported a mean change in total hair count of –2.8 hairs/cm²(–1,4) at 6 months and –7,8 % hairs/cm² (–3;9 %) at 12 months in subjects receiving oral contraceptive + 50 mg cyproterone acetate [52] whereas subjects treated with a combination of minoxidil 2 % solution twice daily and oral contraceptive showed a mean increase in hair count of 16.1 hairs/cm² (8.6 %) at 6 months and 16.9 hairs/cm² (9.1 %) at 12 months. The differences in total hair count at 12 months were statistically significant between groups ($p < 0.0001$).

In subgroup analysis, patients under treatment with cyproterone acetate with clinical signs of hyperandrogenism tended to show increased hair counts at 12 months compared to those without hyperandrogenism, although the results were not statistically significant. Consequently, there is insufficient evidence that oral hormonal treatment prevents progression or improves androgenetic alopecia in female patients. Nevertheless, subgroup analysis suggests that oral cyproterone acetate may improve androgenetic alopecia in female patients with hyperandrogenism.

Topical hormonal treatment

Outcomes

Blume-Peytavi et al. [46] reported a decreased total hair count after 6 months therapy with *alfatradiol* 0.025 % solution once daily (mean change from baseline –7.8 hairs/cm², –4.3 %, $p < 0,0005$). Subjects treated with minoxidil 2 % solution twice daily showed increased total hair counts at 6 months (15.3 hairs/cm²; 8,7 %). Non-vellus hair counts and cumulative hair thickness also showed a decrease in the alfatradiol group and an increase in the minoxidil group at 6 months (–6.0 hairs/cm² vs. 14.0 hairs/cm², $p < 0,001$; –0.5 mm/cm² vs. 1.8 mm/cm², $p < 0.0001$).

There are 3 earlier studies [78, 80, 81] on the efficacy of the *topical oestrogen* alfatradiol in women and one of topical oestrogen combined with corticosteroid [79]. All assessed response using a trichogram. Two studies did not analyse male and female subjects separately and are not considered further [79, 80]. Orfanos and Vogels reported a mean decrease in telogen rate of 24.4 % for patients treated with alfatradiol 0,025 % solution once daily at 30 weeks [78].

In a study by Georgala et al. the mean change from baseline anagen/telogen ratios at 12 and 24 weeks of treatment with alfatradiol solution once daily was 38.7 % and 44.6 % respectively [82]. The change in anagen/telogen ratio differed significantly from placebo treatment (–3.9 % at 24 weeks; $p < 0.01$).

As there are contrary results on the efficacy of topical alfatradiol, the evidence is insufficient to support its use in female patients with androgenetic alopecia. Further studies are needed to clarify the efficacy of alfatradiol.

Gassmueller et al. [77] compared fulvestrant (70 mg/ml twice daily) to minoxidil 2 % and placebo. The mean change in total hair count did not differ from placebo (14.7 hairs/cm², 6.9 % vs. 15.3 hairs/cm², 7.9 %). Therefore, we suggest that topical fulvestrant is not effective in women with androgenetic alopecia.

There is no evidence supporting the use of topical natural oestrogens, progestogens or antiandrogens in female androgenetic alopecia.

3.3.5 Instructions for use / Practicability

Oral antiandrogen therapy in women. Cyproterone acetate (25–50 mg per day, days 1–10) is generally prescribed together with an oral contraceptive e.g. Dianette®. Side effects of cyproterone acetate include depressive mood changes and liver toxicity. There is an increased risk of venous thromboembolism in patients taking oestrogen-containing oral contraceptives, which may be greater in those taking cyproterone acetate than other oral contraceptives.

Spironolactone 100–200 mg per day is taken continuously. Concurrent contraception is required in fertile women. Side effects include menstrual disturbance and hyperpotassemia.

3.3.6 Combination therapies

There are no instructive studies of combination therapy (e.g. minoxidil + antiandrogen).

3.3.7 Summary

There is little evidence to support the use of oral or topical hormonal treatment in men and women in androgenetic alopecia (evidence level 4) and limited evidence that oral cyproterone acetate may be helpful in women with AGA and hyperandrogenism.

3.3.8 Therapeutic recommendation – Male

⇓⇓	The use of oral oestrogens or androgen-receptor-antagonists is inappropriate to improve or prevent progression of AGA in male patients.
⇒	There is insufficient evidence to support the use of topical alfatradiol to improve or prevent progression of AGA in male patients.
⇓	We suggest that topical fluridil should not be used in male patients with AGA.
⇓	We suggest that topical fulvestrant should not be used in male patients with AGA.

3.3.9 Therapeutic recommendation – Female

⇒	There is no or insufficient evidence to support the use of oral antiandrogens (chlormadinone acetate, cyproterone acetate (CPA), drosperinone, spironolactone, flutamide) to improve or prevent progression of AGA in female patients.

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- ➔ Oral CPA can be considered in women with clinical or biochemical evidence of hyperandrogenism.
- ➔ There is insufficient evidence to support the use of topical alfatradiol to improve or prevent progression of AGA in female patients.
- ➔ There is no evidence to support the use of topical natural oestrogens or progesterones to improve or prevent progression of AGA in female patients.
- ➔ There is no evidence to support the use of topical fluridil to improve or prevent progression of AGA in female patients.
- ↓ We suggest that topical fulvestrant should not be used in female patients with AGA.

3.4 Surgery

3.4.1 Introduction

Hair restoration surgery involves hair transplantation, scalp reduction surgery or a combination of both.

Compared to scalp reduction surgery hair transplantation is less invasive. In androgenetic alopecia, hairless areas can be permanently covered again cosmetically, albeit with a decreased density. In areas with decreased hair density, the density can be at least temporarily improved.

Over the last decades, hair transplantation has evolved into a microsurgical procedure. Follicular units of 1 to 4 hairs are transplanted in large numbers and high densities.

3.4.2 Mechanism of action

The efficacy of hair transplantation is based on donor dominance, i.e. non-androgen-sensitive hair follicles keep their properties even when transplanted into scalp areas affected by androgenetic alopecia.

Follicles that are not affected by miniaturization are re-distributed over the scalp under local anaesthesia.

The outcome of hair transplantation result objectively depends on the number of transplanted hairs in relation to the area to be covered or densified, on the quality of hairs such as colour and caliber, and on the characteristics of the recipient area.

The technical success of this multi-step procedure is determined by the ability of the surgical team to successfully harvest, prepare and insert the grafts without impairing their viability. Another aspect is a minimal trauma to the recipient and donor areas.

The cosmetic effect greatly depends on the aesthetic skills of the surgeon, as well as patient selection, planning of the procedure considering an optimum life-long result, the creation of an authentic hairline design, the distribution of grafts with different numbers of hair and the natural creation of recipient sites with appropriate size, density and direction.

3.4.3 Efficacy – males

Although there are a lot of publications dealing with hair surgery, only 3 studies out of 77 analyzed publications fulfilled the inclusion criteria, resulting in an **evidence level 4**. This may be due to many reasons, such as high variation in techniques, multiple steps in the surgical process, problems in measuring hair growth, lack of financial support and difficult patient recruitment.

Outcomes

Bernstein et al. compared different preparation techniques for follicular unit transplantation [83]. The resulting mean harvested hairs were 17 % higher for preparation by dissecting microscope compared to preparation by magnifying loupe with transillumination (9.6 % more follicular units and 2.28 vs. 2.14 mean hairs per follicular unit).

Uebel et al. [84] showed that treatment of follicular units (FU) with platelet plasma growth factor before implantation could reduce the number of non-surviving FU grafts after follicular unit transplantation compared to follicular unit transplantation alone (mean change from baseline FU graft number: –25 (–17.6 %) vs. –40 (–28.2 %), $p < 0.001$).

In a study by Leavitt et al. [70], the combination of FU transplantation and finasteride 1 mg daily in patients with partially still existing hair in the recipient area resulted in an increase of hair density 12 months after transplantation, whereas the patients treated with FU transplantation alone had decreased hair counts. The mean change from baseline total hair count at 12 months was 18.5 hairs/cm^2 (12.6 %) and –13.5 hairs/cm^2 (–8.9 %) respectively ($p = 0.019$).

On frontal-superior global photography, 67 % of patients improved and 30 % did not improve after hair transplantation alone, versus 94 % and 6 % after combination therapy, respectively.

This is a considerably higher efficacy than previously reported in other studies with finasteride alone.

The differing results of hair counts and frontal-superior global photography in hair transplantation alone may partly be due to replacement and compensation of miniaturizing hairs by thicker permanent hair from the occipital area. Magnification should be used when making recipient sites in-between pre-existing hairs.

3.4.4 Efficacy – females

Only few of the 77 assessed publications concerning hair surgery studied efficacy in female patients. None of them fulfilled the inclusion criteria, resulting in an **evidence level 4**. This may be due to many reasons, such as high variation in techniques, multiple steps in the surgical process, problems in measuring hair growth, lack of funding and difficult patient recruitment.

3.4.5 Instructions for use / Practicability

While scalp reduction and flap surgery in combination with extenders is only successfully performed by a few skilled surgeons, hair transplantation is extensively conducted worldwide with further refined micro-techniques and larger graft numbers.

Hair transplantation in suitable candidates with a good donor hair supply, performed by a skilled team of a surgeon and several assistants, can permanently improve androgenetic alopecia by up to 3 stages on the Norwood-Hamilton scale.

In women, hair transplantation can be considered in the male pattern and the frontal accentuation subtypes and Ludwig stage II of stabilized androgenetic alopecia. This only applies if sufficient permanent donor hair is available and no overlying diffuse telogen effluvium is present.

In most cases, more than one surgical session is required and often only critical areas can be improved. Magnification should be used to cautiously insert the grafts in between pre-existing hair follicles.

The best long-term results can be achieved in medically controlled or spontaneously stabilized androgenetic alopecia.

In patients with progressive alopecia,

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hair transplantation should only be performed if additional surgery is possible in terms of donor hair reserve.
Patients should be extensively counselled regarding the possible outcome and the progressive nature of androgenetic alopecia which may require subsequent surgery and/or medical therapy.
Body dysmorphic disorder or unrealistic expectations are contraindications for this aesthetic surgery.
If hair transplantation is performed in early progressive AGA, a sufficient reserve of donor hair should be available for additional surgery, grafts should also be transplanted in-between miniaturizing hairs and the vertex area should not be transplanted initially.
Follicular unit transplantation (FUT) has become the standard technique in hair transplantation. Physiologic follicular units are smaller with less interfollicular tissue and can thus be placed denser into finer, less traumatic recipient sites. Larger grafts with multiple FU's should only be used in combination with FUT and in patients with a very good donor hair supply.
The harvesting of FU grafts from the donor area is usually performed by careful excision of a hair-bearing strip. Several techniques are used to minimize follicle transsection and scar formation during this step. The use of stereo-microscopes then allows for exact and fast dissection of large numbers of FU's with minimal trauma.
Individual extraction of FU's from the donor area is also possible but associated with a potentially higher risk of follicle injury and impairment of graft viability.
Recipient sites are prepared with different instruments. The creation of slits using micro-blades adapted to graft size enables achieving high densities.
In the frontal area, a transition zone of 1-hair-FU's is created with micro- and macro-irregularities for a more natural appearance.
Patients should be informed, that temporary post-operative telogen effluvium may appear if pre-existing hair is present. This may be minimized by making smaller incisions using magnification.
The final result can be evaluated at 9–12 months.

3.4.6 Combination therapies

As hair surgery has no efficacy to prevent further progression of androgenetic alopecia, a combination of medical and surgical therapy seems to be superior to surgery alone.
Leavitt et al. [70] reported a better clinical outcome for male patients treated with combination of finasteride 1 mg daily and hair surgery versus male patients treated with hair surgery alone 12 month after follicular unit transplantation (see section efficacy males).
In female patients there is lack of evidence concerning combination therapies. We suggest that combination therapy may reduce further post-operative progression of androgenetic alopecia.

3.4.7 Summary

3 studies concerning hair surgery fulfilled the inclusion criteria of the S3 guideline (evidence level 4). Hair transplantation can be considered to improve androgenetic alopecia in suitable patients with sufficient donor hair supply and medically controlled or spontaneously stabilized androgenetic alopecia, especially for the fronto-parietal area. As hair surgery does not influence progression of androgenetic alopecia, long-term results in early stages depend on spontaneous respectively medical stabilization. The result greatly depends on the skills of the surgical team and the adjustment of the surgical plan to individual patient characteristics. Preparation of follicular units using dissecting microscopes and pre-treatment of FU's with platelet growth factor lead to higher graft survival rates.
While follicular unit transplantation (FUT) can be considered a standard, especially when stereo-microscopic dissection is used by a skilled team, other components of the surgical technique require further evaluation.
Combination of finasteride 1 mg and follicular unit transplantation may reduce post-operative progression of androgenetic alopecia.

3.4.8 Therapeutic recommendation – Male

➔ Surgery, especially follicular unit transplantation (FUT) can be considered in male patients with sufficient donor hair.
↑ We suggest follicular unit transplantation (FUT) to be combined with finasteride 1 mg daily to achieve a better clinical outcome.

3.4.9 Therapeutic recommendation – Female

➔ Surgery, especially follicular unit transplantation (FUT) can be considered in female patients with sufficient donor hair.

3.5 Miscellaneous

3.5.1 Introduction

Besides the pharmacologic therapeutic options minoxidil, 5-alpha-reductase-inhibitors, hormonal preparations and hair surgery, which were already assessed in the previous chapters, the patient diagnosed with androgenetic alopecia faces a confusing array of products claiming to be efficient in androgenetic alopecia.
The range of products is wide and reaches from topical to systemic modalities; it includes cosmetic to pharmaceutical products, natural products, functional food and even electrostatic/-magnetic or laser treatment.
Though scientific investigations are rare in the majority of cases, the patient is attracted by hair growth promoting claims of advertisement or distribution of myths, rumours and assumptions provided in different internet fora.
Within the consultation the practitioner will be confronted with questions concerning the efficacy of some of the following products. It is therefore important for the development of a stable patient-physician-relationship to be informed on these products, to know their properties and their potentials as well as limits.

3.5.2 Mechanism of action

The assumed mechanisms of action in androgenetic alopecia are as various as the number of products. Though it remains unclear how these products mediate their effects, most of them claim at least one of the following mechanisms:

a) Promotion of hair regrowth by activation of the dermal papillae and consequently induction of anagen hair regrowth.
b) Comparable to minoxidil promoting hair regrowth by improving the perifollicular vascularisation.
c) Hormonal effects, mainly inhibition of 5-alpha-reductase and reducing the activity of dihydrotestosterone (DHT).
d Anti-inflammatory activity.
e) Improvement of hair follicle nutrition.

In Table 2 we aimed to group the different therapeutic options based on their assumed main mechanism of action.

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Table 2: Overview on the assumed main mechanism of action of miscellaneous treatments.

Promotion of hair regrowth	• Aminoacids • Iron supplements in absence of iron deficiency • Vitamines (biotin, niacin derivates) • Proanthocyanidines • Millet seed (silic acid, aminoacids, vitamines, minerals) • Marine extract and silicea component • Chinese herbals • Ginkgo biloboa • Aloe vera • Ginseng • Bergamot • Hibiscus • Sorphora • Caffeine • Melatonin • Retinoids • Ciclosporine • Electromagnetic/-static field • Low level laser
Improved perifollicular vascularisation	• Prostaglandines (viprostol, latanoprost) • Aminexil • Glyceroloxyesters and silicium • Minerals • Niacin derivates • Mesotherapy
DHT-inhibitory activity	• Saw palmetto • ß-sitosterol • Polysorbate 60 • Green tea • Cimicifuga racemosa
Anti-inflammatory activity	• Ketoconazol • Zinc pyrithione • Corticosteroids
Improved hair nutrition	• Vitamines (biotin, niacin derivates) • Trace elements (zinc, copper)
Others	• Botulinum toxin

3.5.3 Efficacy – males and females

Contrary to the previous chapters the efficacy of the miscellaneous therapies is summarized together for males and females, as evidence proving the efficacy of the particular therapies in androgenetic alopecia is mainly not available.

For almost 50 % of the therapeutic agents no literature fulfilling the inclusion criteria of the guideline was found. Furthermore, the evaluation is limited, as most of the tested products contain multiple different substances, e.g. food supplements with aminoacids and trace elements or different herbal preparations. Only 11 of the 20 trials that were included into the guideline examined a single therapeutic agent.

The available evidence is therefore insufficient resulting in **evidence level 4** for the different therapeutic options.

Hereafter, a short overview on the different therapeutic agents is provided.

Aminoacids, especially cysteine is supposed to lead to increased growth factors involved in hair growth. Morganti et al. report a significant mean change in total hair count in male and female patients after a 50-week-treatment with an oral supplement containing cysteine, histidine, copper and zinc taken 4 times daily (29 % vs. 11 % placebo, $p < 0.005$) [85]. A combination of cysteine, calcium pantothenate and millet seed twice a day for 6 months in 40 female patients showed increased anagen rate that was significantly different to placebo ($p = 0.0225$) [86].

Trace elements like ***copper*** and ***zinc*** are suggested to improve hair nutrition, though studies examining the link between serum and hair follicle concentration of trace elements, vitamins etc. fail to show correlation. As zinc and copper were only studied in combination with other agents, evidence is missing.

Different opinions exist concerning the supplementation of ***iron*** in absence of iron deficiency in androgenetic alopecia patients. Various observational studies discussed the relationship between hair loss and decreased serum ferritin levels with controversial results [87]. There is insufficient evidence for iron supplementation in absence of iron deficiency in patients with androgenetic alopecia.

Vitamins, especially ***biotin*** and ***niacin***, are also claimed to have hair growth promoting properties and have positive influence on hair nutrition. Draelos et al. studied the effect of topically applied niacin derivates once daily in 60 female patients. After 6 months 69 % were rated as improved in global photographic assessment ($p = 0.04$ vs. placebo) [88]. Prager et al. reported 60 % improvement rated by investigator in 26 male patients after 18–24 weeks treatment with an oral combination containing biotin and niacin, but also ß-sitosterol and saw palmetto [89].

Proanthocyanidines like procyanidine B belongs to the group of flavonoids, which are antioxidants. Their mechanism of action may be inhibition of transforming growth factor TGF-ß and conversion of telogen follicles to anagen hair follicles. Kamimura et al. showed, that the topical application of procyanidine B 1 % twice daily leads to significant mean changes from total hair count in male patients after 6 months ($p < 0.0005$ vs. placebo) [90].

Millet seed is a natural product that contains silicic acid, aminoacids, vitamines and minerals. An oral supplement composed of millet seed extract, cysteine and calcium pantothenate (Priorin®) taken

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twice daily for 6 months led to increased anagen rates female patients (p = 0.0225 vs. placebo) [86]. Viviscal® is a similar oral supplement composed of marine extracts and a silicea component. Lassus et al. studied this supplement in comparison to fish extract and in combination with topical and oral use [91, 92]. Due to lack of placebo respectively standard for comparison the results are deficient.

Two trials examining the efficacy of topical applied ***herbal preparations*** fulfilled the inclusion criteria of the guideline [93, 94]. As the ingredients of the particular herbal preparations significantly differ, they have to be evaluated separately. Kessels et al. reported modest increase in hair regrowth in 396 male patients, who applied a Chinese herbal preparation twice daily for 6 months. The mean change in nonvellus hair counts was 26.6 hairs/cm^2 vs. 21.8 hairs/cm^2, p = 0.02 vs. placebo) [94]. Greenberg et al. reported a mean change from baseline total hair count of 77.4 % in 24 men after 40 weeks usage of herbal extract containing fennel, polygonum, menthe, chamomile, thuja, hibiscus) (p = 0.003) [93].

No trials were found concerning the ***natural products gingko biloboa, aloe vera, ginseng, bergamot, hibiscus or sorphora***. Animal models and resp. or in vitro studies suggest hair growth promoting properties. The agents are used in cosmetic hair care products.

There are different hair care products with ***caffeine*** claiming to be effective in the treatment of androgenetic alopecia in men and women. In vitro studies with caffeine showed higher transfollicular penetration rates [95]. Caffeine is suggested to prevent progression and induce hair regrowth in androgenetic alopecia. Studies investigating this hypothesis are missing/not available.

Topical application of ***melatonin*** leads to the induction of anagen hair in animal models. A small trial, which did not fulfil the inclusion criteria of the guideline, reported significantly increased anagen hair rates in trichogram in women with androgenetic alopecia or diffuse effluvium after topical use of melatonin 0.1 % for 6 months [96].

Retinoids modulate proliferation, differentiation of keratinocytes and the T-cell immune response. Moreover, its usage as pharmaceutical excipient to improve minoxidil resorption is discussed. Two trials fulfilled the inclusion criteria of the guideline [15, 44]. Bazzano et al. reported in 58 % of the male and female patients, who treated their scalp twice daily with tretinoin 0.025 % solution, at least 20 % increase from baseline hair count at 12 months [15]. It is conspicuous that the placebo and the minoxidil 0.5 % group reached no improvement at all. The trial was not blinded or randomized. Shin et al. failed to show superiority of minoxidil 5 % solution combined with tretinoin 0.01 % once daily vs. minoxidil 5 % solution applied twice daily in 31 male patients [44]. The mean changes from baseline total hair count did not differ significantly at 18 weeks, though the combination of minoxidil and tretinoin led to slightly elevated values (15.9 hairs/cm^2 vs. 18.2 hairs/cm^2).

The induction of hypertrichosis is well known as adverse event in systemic treatment with ciclosporin. Experimental models could demonstrate this effect for topical application of ciclosporin, too. In a small study by Gilhar et al. 2 patients out of 8 responded to topical application of ciclosporin for 12 months [97].

Besides cosmetic and pharmaceutic agents ***physical treatments*** such as pulsed electromagnetic/-static field and low level laser therapy are also suggested to be efficient in androgenetic alopecia.

Four trials for pulsed electrostatic field could be included into the evidence-based evaluation of the guideline [98–101]. Though the trials showed modest increase in total, anagen or nonvellus hair counts, the use in clinical routine is doubtful due to unfavourable cost-benefit ratio.

Satino et al. examined the effects of a low level laser comb in androgenetic alopecia [102]. They reported a mean change from baseline total hair count of 14.1 hairs/cm^2. A control group for comparison was missing.

Cimicifuga racemosa is a natural product with positive influence on the oestrogenic level. It is mainly used for perimenopausal complaints. No evidence was found for its efficacy in androgenetic alopecia, though it is likely that elevation of oestrogen levels in menopause can improve androgenetic alopecia in female patients.

Other therapeutic agents may act by inhibition of the activity of dihydrotestosterone (DHT), namely ***saw palmetto, ß-sitosterol, green tea or polysorbate 60***.

The application of a lotion containing saw palmetto extract twice a day showed statistically significant improvement in mean change from baseline total hair count at 50 weeks (p < 0.005 vs. placebo) [85]. A combination of saw palmetto, ß-sitosterol, nicacin and biotin in an oral softgel taken twice daily also led to an improvement significantly different compared to placebo treatment (investigator assessment 60 % improved vs. 11 %) [89].

A trial by Groveman et al. failed to prove efficacy of the non-ionic detergent polysorbate 60 applied twice daily topically in 174 male patients [103]. After 16 weeks global photographic assessment for the polysorbate group was below the placebo group.

Whereas the previous agents claim to have mechanism of action comparable to 5-alpha-reductase-inhibitors, others may improve perifollicular vascularization similar to minoxidil. ***Aminexil*** is a vasodilatator chemically similar to minoxidil. Studies providing evidence for its efficacy are missing.

Another promising group of substances were the prostaglandine analogues such as ***viprostol or latanoprost***. They mediate vasodilatatory effects and latanoprost leads in topical use as eye drops to induction of prolonged eyelashes. Unfortunately, the topical application of viprostol for 24 weeks in male patients did not show significant difference compared to placebo or vehicle treatment [104].

Minerals and niacin derivates should have positive effects on perifollicular vascularisation in addition to their hair growth promoting properties. Reygagne et al. assessed the efficacy of a topical combination of glycerol oxyesters and silicium (Maxilene®) [38]. In comparison to standard minoxidil treatment Maxilene® led to statistically significant hair loss.

Another therapeutic regimen that claims to improve androgenetic alopecia by improvement of vascularization and hair nutrition is the ***mesotherapy***. Different agents, e.g. vitamins are intracutaneously injected. There was no evidence of efficacy found.

The injection of botulinum toxin is also suggested to improve androgenetic alopecia. A traction component by tension of the musculus occipitofrontalis is discussed, but studies are missing/not available.

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A further therapeutic approach is to act on the inflammatory component of androgenetic alopecia. ***Ketoconazole or zinc pyrithione*** are antimicrobial and are effective agents in the treatment of seborrhoic dermatitis. As concomitant seborrhoeic dermatitis is common in androgenetic alopecia and may aggravate hair loss, impact on androgenetic alopecia is difficult to evaluate. Berger et al. showed significant improvement for 1 % pyrithione zinc shampoo, minoxidil 5 % solution or the combination of both compared to placebo treatment at 26 weeks [16]. The mean change from baseline hair count for the 1 % pyrithione zinc shampoo group was significantly below the standard therapy with minoxidil. Combination of minoxidil and pyrithione zinc was inferior to minoxidil monotherapy.

3.5.4 Instructions for use / Practicability
For instructions for use the reader is asked to consult the product information of the particular product.

3.5.5 Combination therapies
Patients often ask for one of the particular miscellaneous therapies in combination with another treatment. As evidence is insufficient to missing for the therapies mentioned above, they cannot be recommended in combination. Additional use depends on the individual case and decision of the patient and the physician.

3.5.6 Summary
Plenty of oral and topical miscellaneous therapies are claimed to be effective in the treatment of androgenetic alopecia in men and women. There is insufficient to missing evidence for this assumption (evidence level 4).

3.5.7 Therapeutic recommendation – Male and Female

➔ There is no or insufficient evidence that the molecules summarized in Table 2, substances and interventions improve or prevent progression of AGA in male and female patients.

Conflicts of Interest
Blumeyer A: No conflict of interest declared.
Blume-Peytavi U: Advisor to Johnson & Johnson (J & J), Almirall, Follica and Dr. Hans Karrer. Received honorarium as speaker from Procter & Gamble. Participated as employee of Charité in clinical and research trials from the companies mentioned above and L'Oréal, Galderma and Innéov. Received unrestricted research grant from J & J.
del Marmol V: Has given lectures for and received honorarium as speaker for MEDA, Merck and Medipress.
Finner A: Received honorarium as speaker for Merz Pharmaceutics GmbH.
Kiesewetter F: No conflict of interest declared.
Messenger A: Has acted in the past as a consultant for Johnson & Jonhnson (actually Pfizer) and MSD (Merck, Sharp & Dohme). Currently investigator in a Johnson & Johnson sponsored clinical trial.
Reygagne P: Has given lectures for Bailleul-Biorga, Galderma, Innéov, Janssen Cilag, MSD and Pierre Fabre Dermatologie. Is a member of the advisory board to Galderma. Is or has been investigator for Ducray, Galderma, Innéov, Johnson & Johnson and L'Oréal recherché.
Rzany B: No conflict of interest declared.
Spuls PI: No conflict of interest declared.
Tosti A: Has given lectures and received honorarium as speaker for MSD (Merck, Sharp&Dohme), received honorarium for advisory board from Procter&Gamble, participated in clinical trials for Inneov.
Trakatelli M: Has given lectures for the European Skin College sponsored by MEDA.
Trüeb R: Has given lectures and received honorarium as speaker for Merz Pharmaceutics GmbH and Spirig AG. Is or has been member of the advisory board/consultant to the following companies: ASATONA, Lexington.

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Appendix 1: Guideline's Inclusion Criteria

Excerpt from the Literature Evaluation Form (LEF)

A. Inclusion/Exclusion of an article:
This serves as a prescreening. If the article is not included, neither the quality nor the results will be assessed. (tick a grey box = exclusion of the article)

1. Author + Year:
   ……

2. Language:
   ……

3. Type of the study: prospective study? Yes ☐ No ☐

4. Number of patients ≥20? Yes ☐ No ☐
   Exception in identical twin studies no minimal patient number required.

5. Age: Adults (≥18 years) ☐
   Adolescents (≥12 years) ☐
   (Studies of adolescents will be evaluated separately)
   Children / n.d ☐

6. Confirmed diagnosis of androgenetic alopecia male versus female pattern, respectively
   (diagnosis either clinically or by further diagnostic evaluations e.g. trichogram, TrichoScan, biopsy)?
   Yes ☐ No/n.d. ☐

7. Type of therapy?
   monotherapy ☐ *or* combination therapy ☐ distinct classification not possible ☐

8. Objective outcome measure of efficacy described? Yes ☐ No ☐

   For drug therapy:
   Mean change from baseline hair count in target area OR
   Measurement of hair growth/loss in target area by global photography

   For surgical therapy:
   Mean change from baseline hair count in target area OR
   Measurement of hair growth/loss in target area by global photography OR
   Graft survival and global photography

9. Exception:
   Category of the article: Review ☐ Safety study ☐ Other ☐
   Article should be quoted: Yes ☐ No ☐

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Appendix 2

Appendix 2: Literature Results Tables.
Table 3: Literature Table. Minoxidil.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
Minoxidil																
41	Roenigk	1987	777	male	6m	Arm1: minoxidil 2.8%, 2x/d, topical							46.6%			C
28	Montagnani	1990	33	male	24w	Arm1: Minoxidil 2% solution, 2x/d, topical							37.5%			C
23	Karam	1993	195	male	48w	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**44.7 (82.1%)** p < 0.001 vs. Baseline	**47.8 (119.7%)** p < 0.0001 vs. Baseline						vertex	C
Minoxidil vs. Placebo																
Male																
34	Olsen	2007	352	male	16w	Arm1: minoxidil 5% foam, 2x/d, topical	Phototrichogram	**20.9 (13.4%)** p < 0.0001 vs. Baseline				**improved: 38.3% unchanged: 52.2%** p < 0.0001 vs. Placebo			vertex	A2
						Arm2: placebo foam, 2x/d, topical		**4.7 (3.4%)**				**5.2% unchanged: 77.9%**				

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Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
17	Califano	1991	132	male	24w	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**18.8 (47.6%)**	**17.3 (59.4%)**						vertex	A2
						Arm2: placebo solution, 2x/d, topical		**17.9 (48.8%)**	**12.3 (46.2%)**							
13	Anderson	1988	154	male	24w	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**25.4 (40.0%)** p = 0.0001 vs. Placebo	**25.4 (69.9%)** p = 0.002 vs. Placebo						vertex	A2
						Arm2: placebo, 2x/d, topical		**10.3 (15.5 %)**	**12.1 (31.8%)**							
22	Dutree-Meulenberg	1988	151	male	24w	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**29.9 (35.3%)** p = 0.02 vs. Placebo	**17.5 (38.0%)** p = 0.0003 vs. Placebo						vertex	A2
						Arm2: placebo, 2x/d, topical		**18.4 (20.4%)**	**5.9 (10.7%)**							
35	Petzold	1988	201	male	24w	Arm1: Minoxidil 2% solution, 2x/d, topical	manual hair count	**16.1 (39.2 %)** p = 0.0076 vs. Placebo	**10.8 (33.1%)** p = 0.0104 vs. Placebo						vertex	A2
						Arm2: placebo, 2x/d, topical		**6.1 (14.9%)**	**2.5 (7.3%)**							

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Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
30	Olsen	1986	100	male	6m	Arm1: minoxidil 0.01% solution, 2x/d, topical	manual hair count	**16.5 (15.3%)**	**9.4 (14.2%)**						vertex	A2
						Arm2: minoxidil 0.1% solution, 2x/d, topical		**18.0 (18.9%)**	**15.9 (27.1%)**							
						Arm3: minoxidil 1% solution, 2x/d, topical		**29.2 (27.8%)**	**35.3 (53.3%)**							
						Arm4: minoxidil 2% solution, 2x/d, topical		**34.3 (33.1%)** significant vs. Arm1-3, 5	**37.3 (52.1%)**							
						Arm5: placebo, 2x/d, topical		**17.6 (17.1%)**	**15.9 (23.1%)**							
45	Shupack	1987	58	male	6m	Arm1: placebo, 2x/d, topical	phototrichogram		**2.9 (14.4%)**						vertex	B
						Arm2: minoxidil 0.01% solution, 2x/d, topical			**0.3 (1.2%)** p > 0.2 vs. Placebo							
						Arm3: minoxidil 0.1% solution, 2x/d, topical			**5.1 (16.3%)** p < 0.01 vs. Placebo, p > 0.1 vs. Arm4/5							
						Arm4: minoxidil 1% solution, 2x/d, topical			**7.8 (39.6%)** p < 0.01 vs. Placebo, p > 0.1 vs. Arm3/5							
						Arm5: minoxidil 2% solution, 2x/d, topical			**10.6 (58.2%)** p < 0.01 vs. Placebo, p > 0.1 vs. Arm3/4							

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Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
33	Olsen	2002	393	male	48w	Arm1: minoxidil 5% solution, 2x/d, topical	phototrichogram		**18.6 (12.3%)** p < 0.001 vs. Placebo, p = 0.025 vs. Arm2			**improved: 57.9% unchanged: 30.3%**			vertex	A2
						Arm2: minoxidil 2% solution, 2x/d, topical			**12.7 (8.8%)** p < 0.013 vs. Placebo			**improved: 40.8% unchanged: 48.6%**				
						Arm3: placebo, 2x/d, topical			**3.9 (2.6%)**			**improved: 23.2% unchanged: 60.6%**				
18	Civatte	1987	247	male	24w, 48w	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count		**24w: 13.3 (47.2%)** p < 0.01 vs. Baseline **48w: 32.0 (78.2%)** p = 0.02 vs. Baseline						vertex	A2
						Arm2: 24w placebo, 2x/d, topical, 25-48w minoxidil 2% solution, 2x/d, topical			**24w: 13.3 (42.0%)** p < 0.01 vs. Baseline **48w: 31.4 (99.0%)**							

Continued

Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
27	Lopez-Bran	1990	114	male	24w, 48w	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**24w: 5.4 (12.1%)** **48w: 15.5 (34.6%)**	**24w: 4.7 (17.2%)** **48w: 14.2 (52.4%)**						vertex	A2
						Arm2: 24w placebo, 2x/d, topical, 25-48w minoxidil 2% solution, 2x/d, topical		**24w: 4.7 (10.4%)** **48w: 12.2 (27.1%)**	**24w: 2.9 (10.8%)** **48w: 10.5 (39.4%)**							
19	Civatte	1988	428	male	24w, 48w	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**24w: 23.8 (54.8%)** p = 0.0611 vs. Arm2 **48w: 36.1 (83.0%)**	**24w: 13.3 (48.6%)** p = 0.0187 vs. Arm2 **48w: 25.0 (91.3%)**						vertex	A2
						Arm2: 24w placebo, 2x/d, topical, 25-48w minoxidil 2%, 2x/d, topical		**24w: 19.9 (47.5%)** **48w: 34.6 (82.7%)**	**24w: 9.7 (39.6%)** **48w: 22.6 (91.8%)**							
12	Alanis	1991	240	male	24w, 48w	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**24w: 13.7 (25.6%)** **48w: 25.5 (47.8%)** p = 0.000 (24w, 48w) vs. Baseline							vertex	A2
						Arm2: 24w placebo, 2x/d, topical, 25-48w minoxidil 2% solution, 2x/d, topical		**24w: 5.1 (9.3%)** **48w: 16.9 (31.0%)** p = 0.000 (24w, 48w) vs. Baseline								

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Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
36	Piepkorn	1988	72	male	6m	Arm1: minoxidil 2% gel, 2x/d, topical	manual hair count						**26%** p < 0.05 vs. Arm2		vertex	B
						Arm2: placebo gel, 2x/d, topical							**33%**			
						Arm3: minoxidil 2% solution, 2x/d, topical							**48%** p < 0.05 vs. Arm4			
						Arm4: placebo solution, 2x/d, topical							**36%**			
29	Olsen	1985	153	male	4m, 12m	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**4m: 29.6 (53.6%)** **12m: 63.5 (114.9%)** n.s. vs. Arm3 (4m), n.s. vs. Arm2 (4m, 12m)	**4m: 24.7 (100.0%)** **12m: 42.8 (173.0%)**						vertex	A2
						Arm2: minoxidil 3% solution, 2x/d, topical		**4m: 32.6 (52.0%)** p = 0.04 vs. Arm3 **12m: 66.3 (106.0%)**	**4m: 27.5 (95.2%)** **12m: 53.5 (185.7%)**							
						Arm3: 4m placebo, 2x/d, topical, 5-12m minoxidil 3% solution, 2x/d, topical		**4m: 22.4 (39.3%)** **12m: 61.4 (108.0%)**	**4m: 11.6 (44.7%)** **12m: 42.9 (165.9%)**							

Continued

Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
43	Savin	1987	96	male	4m, 12m	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**4m: 14.9** p = 0.0076 vs. Baseline p = 0.013 vs. Arm3 **12m: 16.4** p < 0.001 vs. Baseline	**4m: 11.4** **12m: 22.0**						vertex	A2
						Arm2: minoxidil 3% solution, 2x/d, topical		**4m: 11.9** **12m: 28.3** p < 0.001 vs. Baseline	**4m: 9.6** **12m: 30.9**							
						Arm3: 4m placebo, 2x/d, topical, 5-12m minoxidil 3% solution, 2x/d, topical		**4m: 9.3** **12m: 24.7** p < 0.001 vs. Baseline	**4m: 0.8** **12m: 33.2**							
24	Katz	1987	153	male	4m, 12m	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count		**4m: 20.4 (216.7%)** p = 0.0002 vs. Arm3, p = 0.0464 vs. Arm2 **12m: 41.8 (443.8%)**						vertex	A2
						Arm2: minoxidil 3% solution, 2x/d, topical			**4m: 14.5 (168.2%)** p = 0.0833 vs. Arm3 **12m: 39.6 (459.1%)**							
						Arm3: 4m placebo, 2x/d, topical, 5-24m minoxidil 3% solution, 2x/d, topical			**4m: 9.8 (116.3%)** **12m: 33.1 (393.0%)**							

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Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
21	De Villez	1987	64	male	4m, 12m	Arm1: minoxidil 3% solution, 2x/d, topical	phototrichogram		**4m: 1.2** p < 0.05 vs. Arm3, n.s. vs. Arm2 **12m: 9.4**						vertex	B
						Arm2: minoxidil 2% solution, 2x/d, topical			**4m: 1.2** p < 0.05 vs. Arm3 **12m: 8.6**							
						Arm3: 4m placebo, 2x/d, topical, 5-12m minoxidil 3% solution, 2x/d, topical			**4m: -0.2** **12m: 4.9**							
31	Olsen	1987	127	male	132w	Arm1: minoxidol 3% solution, 2x/d, topical	phototrichogram	**64,4**	**65.7**						vertex	B
						Arm2: 24m minoxidol 3% solution, 2x/d, topical, 25 33m minoxidil 3% solution, 1x/d, topical		**44,1**	**46.1**							
12	Alanis	1991	240	male	24w, 48w	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count		**24w: 9.9 (27.7%)** **48w: 18.2 (50.9%)**						vertex	B
						Arm2: 24w placebo, 2x/d, topical, 25-48w minoxidil 2% solution, 2x/d, topical			24w: 4.8 (12.6%) 48w: 12.3 (32.4%)							

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Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
25	Koperski	1987	72	male	12m, 30m	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**12m: 46.9 (191.7%)** **30m: 22.5 (91.7%)** p < 0.001 (4m, 12m) vs. Baseline n.s. vs. Arm2							vertex	A2
						Arm2: minoxidil 3% solution, 2x/d, topical		**12m: 36.7 (163.6%)** **30m: 26.5 (118.2%)** p < 0.001 (4m, 12m) vs. Baseline								
						Arm3: 4m placebo, 2x/d, topical, 5m-30m minoxidil 3% solution, 2x/d, topical		**12m: 44.9 (200.0%)** **30m: 36.7 (163.6%)** p < 0.01 (4m), p < 0.001 (12m) vs. Baseline								
32	Olsen	1990	31	male	3y	Arm1: minoxidil 3% solution, 2x/d, topical	phototrichogram		4.4 p = 0.015 vs. Arm2						vertex	B
						Arm2: 1st y minoxidil 3% solution, 2x/d, topical, 2nd-3rd y, 1x/d, 4th-5th y 2x/d			-13.4							

Continued

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Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
26	Kreindler	1987	150	male	4m, 12m	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**4m: 15.9 (47.4%)** **12m: 83.3 (248.5%)** p < 0.0001 vs. Baseline	**4m: 12.6 (91.4%)** p = 0.0018 vs. Placebo **12m: 40.4 (294.3%)**						bald area	A2
						Arm2: minoxidil 3% solution, 2x/d, topical		**4m: 16.1 (50.9%)** **12m: 72.9 (231.1%)** p < 0.0001 vs. Baseline	**4m: 10.2 (85.3%)** p = 0.0167 vs. Placebo **12m: 34.9 (291.8%)**							
						Arm3: 4m placebo 2x/d, topical, 5-12m minoxidil 3% solution, 2x/d, topical		**4m: 11.0 (37.1 %)** **12m: 80.4 (271.5%)** p < 0.0001 vs. Baseline	**4m: 3.1 (23.2%)** **12m: 33.1 (244.9%)**							
20	Connors	1990	169	male	24w, 48w	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count		**24w: 7.7 (30.6%)** p < 0.05 vs. Placebo **48w: 3.3 (13.1%)** p = 0.07 (24-48w)						bald area	A2
						Arm2: 24w placebo, 2x/d, topical, 25-48w minoxidil 2% solution, 2x/d, topical			**24w: 0.4 (1.6%)** **48w: 7.8 (34.1%)** p < 0.01 (24-48w)							

Continued

Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
37	Price	1999	36	male	96w	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**25%** p = 0.074 vs. Placebo, p = 0.455 vs. Arm2			**hair weight 15%** p < 0.005 vs. Placebo, p = 0.437 vs. Arm2				frontal/ parietal	B
						Arm2: minoxidil 5% solution, 2x/d, topical		**30%** p = 0.010 vs. Placebo			**22%** p = 0.000 vs. Placebo					
						Arm3: placebo, 2x/d, topical		**9%**			**-15%**					
						Arm4: no treatment		**10%**			**-10%**					
Male and Female																
39	Rietschel	1997	149	female and male	4m, 12m	Arm1: 1sty minoxidil 2% solution, 2x/d, topical, 2ndy minoxidil 3% solution, 2x/d, topical	manual hair count	**4m: 4.8 (38.7%)** n.s. vs. Baseline **12m: 23.0 (184.4%)** p < 0.001 vs. Baseline, n.s. vs. Arm2+3	**4m: 1.5 (16.8%) 12m: 16.0 (175.9%)**						vertex	B
						Arm2: minoxidil 3% solution, 2x/d, topical		**4m: 5.9 (49.3%)** n.s. vs. Baseline **12m: 23.3 (194.9%)** p < 0.001 vs. Baseline, n.s. vs. Arm3	**4m: 1.7 (19.5%) 12m: 16.9 (195.2%)**							
						Arm3: 4m placebo, 2x/d, topical, 5m-24m minoxidi 3% solution 2x/d, topical		**4m: 4.7 (36.5%)** n.s. vs. Baseline **12m: 24.7 (194.0%)** p < 0.001 vs. Baseline	**4m: 1.3 (13.9%) 12m: 17.7 (193.4%)**							

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Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
40	Roberts	1987	60	female and male	4m, 12m	Arm1: minoxidil 2% solution, 2x/d, topical	manual hair count	**4m: 22.0 (70.8%)** n.s. vs. Arm2+3 **12m: 50.5 (162.7%)** p = 0.0001 vs. Baseline							vertex	B
						Arm2: minoxidil 3% solution, 2x/d, topical		**4m: 25.6 (83.3%)** n.s. vs. Arm3 **12m: 57.2 (186.4%)** p = 0.0001 vs. Baseline								
						Arm3: 4m placebo, 2x/d, topical, 5-12m minoxidil 3% solution, 2x/d, topical		**4m: 16.5 (51.8%)** **12m: 60.5 (189.7%)** p = 0.0001 vs. Baseline								
Female																
51	Tsuboi	2007	280	female	24w	Arm1: minoxidil 1% solution, 2x/d, topical	phototrichogram	**15.2 (8.0%)** p < 0.001 vs. Placebo	**8.2 (6.1%)** p < 0.001 vs. Placebo						bald area	A2
						Arm2: placebo, 2x/d, topical		**2.9 (1.5%)**	**2.0 (1.5%)**							
48	Jacobs	1993	346	female	32w	Arm1: minoxidil 2% solution, 2x/d, topical	phototrichogram		**33.1 (24.2%)** p = 0.0148 vs. Placebo						bald area	A2
						Arm2: placebo, 2x/d, topical			**19.1 (13.7%)**							

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Table 3: Literature Table. Minoxidil continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
47	De Villez	1994	308	female	24w, 32w	Arm1: minoxidil 2% solution, 2x/d, topical	phototrichogram		**24w: 21.8 (15.5%)** **32w: 22.7 (16.2%)** p = 0.02 vs. Placebo						bald area	A2
						Arm2: placebo, 2x/d, topical			**24w: 9.9 (7.1%)** **32w: 10.1 (7.3%)**							
53	Whiting	1992	33	female	24w, 32w	Arm1: minoxidil 2% solution, 2x/d, topical	phototrichogram		**21 (12.4%)** n.s. vs. Placebo						bald area	B
						Arm2: placebo, 2x/d, topical			**17 (10.6%)**							
50	Olsen	1991	30	female	32w	Arm1: minoxidil 2% solution, 2x/d, topical	phototrichogram		**50.1 (31.3%)** p = 0.006 vs. Placebo						frontopa-rietal	B
						Arm2: placebo, 2x/d, topical			**20.6 (13.4%)**							
49	Lucky	2004	381	female	48w	Arm1: minoxidil 5% solution, 2x/d, topical	phototrichogram		**24.5 (17.3%)** p < 0.001 vs. Placebo, p = 0.129 vs. Arm2						fronto-/occipitopa-rietal	A2
						Arm2: minoxidil 2% solution, 2x/d, topical			**20.7 (13.8%)** p < 0.001 vs. Placebo							
						Arm3: placebo, 2x/d, topical			**9.4 (6.8%)**							

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Table 3: Literature Table. Minoxidil and Finasteride and Other Therapies.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
14	Arca	2004	65	male	12m (52w)	Arm1: minoxidil 5% solution, 2x/d, topical	7-point-rating scale					**80%**			frontal/ parietal	B
						Arm2: finasteride 1mg, 1x/d, oral						**52%**				
42	Saraswat	2003	99	male	12m	Arm1: finasteride 1 mg, 1x/d, oral	manual hair count	**36.1 (29.1%)** p = 0.003 vs. Minoxidil				**62%** p = 0.19 vs. Arm2			bald area	B
						Arm2: minoxidil 2% solution, 2x/d, topical		**19.6 (14.8%)**				**56%**				
16	Berger	2003	200	male	26w	Arm1: minoxidil 5% solution, 2x/d, topical + pyrithione zinc 1% shampoo, 1x/d, topical	phototrichogram	**6,2**							vertex	A2
						Arm2: minoxidil 5% solution, 2x/d, topical + placebo shampoo, 1x/d, topical		**12,3**								
						Arm3: pyrithione zinc 1% shampoo, 1x/d, topical		**5,7**								
						Arm4: placebo shampoo, 1x/d, topical		**-0.6**								

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Table 3: Literature Table. Minoxidil and Other Therapies.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
38	Reygagne	1997	72	male	6m	Arm1: minoxidil 2% solution, 2x/d, topical	phototrichogram	**6.8 (11.0%)** p < 0.001 vs. Arm2		**anagen: 4.5 (9.8%)** p = 0.06 vs. Arm2					vertex	B
						Arm2: maxilene lotion (glycerol oxyesters and organic silicium), 1x/d, topical		**-3.0 (-4.8%)**		**-1.8 (-3.7%)**						
44	Shin	2007	31	male	18w	Arm1: minoxidil 5% solution, 2x/d, topical	phototrichogram	**15.9 (12.8%)** p < 0.05 vs. Baseline	**14.0 (41.9%)** p < 0.05 vs. Baseline	**anagen ratio -0.014 (-2.5%)** n.s. vs. Baseline	**hair diameter: m 2.6 (7.5%)** p < 0.05 vs. Baseline				transitional zone to balding area	A2
						Arm2: minoxidil 5% + tretinoin 0.01% solution, 1x/d, topical, morning placebo solution, 1x/d, topical, evening		**18.2 (14.7%)** p < 0.05 vs. Baseline, n.s. vs. Arm1	**6.1 (14.3%)** p < 0.05 vs. Baseline, n.s. vs. Arm1	**-0.033 (-6.0%)** n.s. vs. Baseline	**1.9 (5.3%)** n.s. vs. Baseline					
15	Bazzano	1996	56	female and male	12m	Arm1: placebo, 2x/d, topical	manual hair count	**0%**							bald area	B
						Arm2: minoxidil 0.5%, 2x/d, topical		**0%**								
						Arm3: tretinoin 0.025% solution 2x/d, topical		**58%**								
						Arm4: minoxidil 0.5% + Tretinoin 0.025% solution, 2x/d, topical		**66%**								

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Table 3: Literature Table. Minoxidil and Hormones.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
46	Blume-Peytavi	2007	103	female	6m, 12m	Arm1: minoxidil 2% solution, 2x/d, topical	TrichoScan	**6m: 15.3 (8.7%)** p = 0.0025 vs. Baseline, p < 0.0005 vs. Arm2 **12m: 17.3 (9.9%)** n.s. vs. 6m			**hair thickness (mm/cm²) 6m: 1.8** p < 0.0001 vs. Baseline **12m: 2.1**				centro-parietal	B
						Arm 2: 0-6m: alfatradiol 0.025% solution, 1x/d, topical, 7-12m: minoxidil 2% solution, 2x/d, topical		**6m: -7.8 (-4.3%)** n.s. vs. Baseline **12m: 9.8 (5.4%)** p < 0.0001 vs. 6m			**6m: -0.5** n.s. vs. Baseline **12m: 0.4**					
52	Vexiau	2002	66	female	6m, 12m	Arm1: minoxidil 2% solution, 2x/d, topical + oral contraceptive (ethinyl oestradiol 30 µg and gestodene 75 µg), 1x/d, 21 of 28 days, oral	phototrichogram	**6m: 16.1 (8.6%) 12m: 16.9 (9.1%)** p < 0.001 vs. Baseline and Arm2	**6m: 14.0** p = 0.003 vs. Baseline, p < 0.001 vs. Arm2 **12m: 14.9**	anagen: (hairs/cm²) 25 p < 0.001 vs. Arm2					fronto-parietal	B
						Arm2: cyproterone acetate 50 mg, 1x/d, 20 of 28 days, oral + oral contraceptive (ethinyl estradiol 35 µg and cyproterone acetate 2 mg), 1x/d, 21 of 28 days, oral		**6m: -2.8 (-1.4%) 12m: -7.8 (-3.9%)** p = 0.85 vs. Baseline	**6m: -6.0** n.s. vs. Baseline **12m: 5.9**	**-5.6**						

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Table 3: Literature Table. Finasteride.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
Finasteride																
66	Trüeb	2001	265	male	6m	Arm1: finasteride 1 mg, 1x/d, oral	n.a.					**39.9%**				C
Finasteride vs. Placebo																
65	Stough	2002	18	male	1y	Arm1: finasteride 1mg, 1x/d, oral	phototrichogram	**16 (9.8%)** p < 0.05 vs. placebo				44% p < 0.01 vs. placebo			vertex	A2
						Arm2: placebo, 1x/d, oral		**-4 (-2.5%)**				**11%**				
67	van Neste	2000	212	male	48w	Arm1: finasteride 1 mg, daily, oral	phototrichogram	**7.2 (3.6%)** p < 0.001 vs. placebo		**anagen, hairs/cm² (%): 18 (14.5%)** p < 0.001 vs. placebo					vertex	A2
						Arm2: placebo, daily, oral		**-10.1 (-5.2%)**		**anagen, hairs/cm² (%): -9.0 (-7.6%)**						
58	Kawashima	2004	414	male	48w	Arm1: finasteride 1 mg, 1x/d, oral						**improved: 58%** p < 0.001 vs. placebo **unchanged: 40%**			vertex	A2
						Arm2: finasteride 0.2 mg, 1x/d, oral						**improved: 54%** p < 0.001 vs. placebo **unchanged: 41%**				
						Arm3: placebo, 1x/d, oral						**improved: 6% unchanged: 72%**				

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Table 3: Literature Table. Finasteride continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
57	Kaufman	1998	1553	male	12m	Arm1:b finasteride 1 mg, 1x/d, oral	phototrichogram	**16.9 (11.0%)** p < 0.001 vs. placebo				**48% (12m)** **66% (24m)** p < 0.001 (12m) vs. placebo			vertex	A2
						Arm2: placebo 1x/d, oral		**-4.1 (-2.7%)**				**7% (12m)** **7% (24m)**				
						Arm3: 1st y finasteride 1mg, 1x/d, oral, 2nd y placebo, 1x/d, oral										
						Arm4: 1st y placebo, 1x/d, oral, 2nd y finasteride 1 mg, 1x/d, oral										
64	Roberts	1999	693	male	6m, 12m	Arm1: finasteride 5mg, 1x/d, oral	phototrichogram	**6m: 12.9 (7.7%)** **12m: 18.2 (10.8%)** p < 0.001 (6,12m) vs. baseline and placebo				**51% (6m)** **48% (12m)** p < 0.001 vs. placebo			vertex	A2
						Arm2: placebo, 1x/d, oral		**6m: -3.9 (-2.1%)** **12m: -3.9 (-2.1)**				**10% (6m)** **3% (12m)**				
						Arm3: finasteride 1 mg, 1x/d, oral		**6m: 13.5 (7.3%)** **12m: 16.7 (9.0%)** p < 0.001 (6,12m) vs. baseline and placebo				**52% (6m)** **54% (12m)** p < 0.001 vs. placebo				
						Arm4: finasteride 0.2 mg, 1x/d, oral		**6m: 10.8 (6.1%)** **12m: 12.7 (7.2%)** p < 0.001 (6,12m) vs. baseline and placebo				**38% (6m)** **38% (12m)** p < 0.001 vs. placebo				
						Arm5: finasteride 0.01 mg, 1x/d, oral		**6m: -1.8 (-1.0%)** **12m: -3.5 (-2.0 %)**				**12% (6m)** **10% (12m)**				

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Table 3: Literature Table. Finasteride continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
69	Whiting	2003	424	male	2y	Arm1: finasteride 1 mg, 1x/d, oral						**improved: 39%** p < 0.001 vs. placebo **unchanged: 55%**			vertex	A2
						Arm2: placebo, 1x/d, oral						**improved: 4% unchanged: 73%**				
71	The Finasteride Male Pattern Hair Loss Study Group	2002	1553	male	24m, 60m	Arm1: finasteride 1mg, 1x/d, oral	phototrichogram	**7.5 (4.3%)** p < 0.001 vs. baseline and placebo				**improved: 48%** p < 0.001 vs. placebo **unchanged: 42%** p < 0.001 vs. placebo			vertex	A2
						Arm2: placebo, 1x/d, oral		**-46.9 (-26.5%)** p < 0.001 vs. baseline				**improved: 6% unchanged: 19%**				
						Arm3: 1sty finasteride 1mg, 1x/d, oral, 2nd-y placebo, 1x/d, oral, 3rd-y-5th-y finasteride 1 mg, 1x/d, oral										
						Arm4: 1st-y placebo, 1x/d oral, 2nd-y-5th-y finasteride 1 mg,1x/d, oral										

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Table 3: Literature Table. Finasteride continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
62	Price	2002	66	male	48w, 96w	Arm1: finasteride 1 mg, 1x/d, oral	manual hair count	**48w: 12.4%** **96w: 9.1%** p < 0.001 (48, 96w) vs. baseline p < 0.01 (48w), p < 0.001 (96w) vs. placebo			**hair weight:** **48w: 20.4%** **96w: 21.5%** p < 0.001 (48w, 96w) vs. placebo				fronto-parietal	A2
						Arm2: placebo, 1x/d, oral		**48w: 3.2%** **96w: -6.3%** p = 0.11 (48w) p < 0.05 (96w)			**hair weight:** **48w: -5.2%** **96w: -14.2%**					
60	Leyden	1999	326	male	6m, 12m, 24m	Arm1: finasteride 1 mg, 1x/d, oral	phototrichogram	**6m: 7.0 (3.3%)** **12m: 9.6 (4.6%)** **24m: 13.0 (6.2%)** p < 0.001 vs. placebo				**improved: 37%** p < 0.001 vs. placebo **unchanged: 62%**			frontal/ centro-parietal	A2
						Arm2: placebo, 1x/d, oral, 2nd y finasteride 1 mg, 1x/d, oral		**6m: -4.0 (-1.8%)** **12m: -2.0 (-0.9%)** **24m: 8.0 (3.7%)**				**improved: 7%** **unchanged: 86%**				
72	Brenner	1999	28	male	6m, 12m, 24m	Arm1: finasteride 5 mg, 1x/d, oral	manual hair count	**6m: 6.4 12m: 16.7** **24m: 34.6** p < 0.05 (6,12,24m) vs. baseline p < 0.01 (12,24m) vs. placebo							bald area	B
						Arm2: placebo, 1x/d, oral		**6m: -2.6** **12m: -1.3** **24m: -14.1**								

Continued

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Table 3: Literature Table. Finasteride and Other Therapies.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
63	Price	2006	28	male	36m (144w), 48m (192w)	Arm1: finasteride 1 mg, 1x/d, oral	manual hair count	**36m: 8.5%** **48m: 7.2%** p = 0.119 (36m) vs. placebo p < 0.05 (48m) vs. placebo			**weight (%)** **36m: 19.5%** **48m: 21.6%** p < 0.001 (36m, 48m) vs. placebo				fronto-parietal	A2
						Arm2: placebo, 1x/d, oral		**36m: -1.6%** **48m: -13.0%**			**weight (%)** **36m: -14.8%** **48m: -24.5%**					
Finasteride vs. Other Therapies																
14	Arca	2004	65	male	12m (52w)	Arm1: minoxidil 5% solution, 2x/d, topical						**80%**			frontal/ parietal	B
						Arm2: finasteride 1mg, 1x/d, oral						**52%**				
42	Saraswat	2003	99	male	12m	Arm1: finasteride 1 mg, 1x/d, oral	manual hair count	**36.1 (29.1%)** p = 0.003 vs. minoxidil				**62%** p = 0.19 vs. Arm2			bald area	B
						Arm2: minoxidil 2% solution, 2x/d, topical		**19.6 (14.8%)**				**56%**				

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Table 3: Literature Table. Finasteride and Other Therapies/Combination Therapies.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
59	Khandpur	2002	100	male	12m	Arm1: finasteride 1 mg, 1x/d, oral							**87%** p < 0.03 vs. Arm2 p < 0.001 vs. Arm3 p = 0.3 vs. Arm4			B
						Arm2: finasteride 1 mg, 1x/d, oral + minoxidil 2% solution, 2x/d, topical							**100%** p < 0.001 vs. Arm3 p = 1 vs. Arm4			
						Arm3: minoxidil 2% solution, 2x/d, topical							**42%** p < 0.0014 vs. Arm4			
						Arm4: finasteride 1 mg, 1x/d, oral + ketoconazole 2% shampoo, 3x/w, topical							**100%**			
Combination Therapies																
70	Leavitt	2005	79	male	12m	Arm1: hairtransplantation + finasteride 1mg, 1x/d, oral	macrophotograph	**18.5 (12.6%)** p = 0.019 (Arm1 vs. Arm2)							frontal	A2
						Arm2: hairtransplantation + placebo, 1x/d, oral		**-13.5 (-8.9%)**								

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Table 3: Literature Table. Finasteride in Female Patients.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
Finasteride – Female Patients																
68	Whiting	1999	163	female and male	12m	Arm1: finasteride 1mg, 1x/d, oral, male	biopsy	**17.7 (5.5%)**	**41.5 (34.8%)**						vertex men, frontal/ parietal women	B
						Arm2: placebo, 1x/d, oral, male		**-0.8 (-0.3%)**	**7.7 (5.8%)**							
						Arm3: finasteride 1mg, 1x/d, oral, female		**-14.6 (-5.9%)**	**1.5 (1.0%)**							
						Arm4: placebo, 1x/d, oral, female		**0.0 (0.0%)**	**8.5 (5.3%)**							
74	Price	2000	137	female	1y	Arm1: finasteride 1 mg, 1x/d, oral	phototrichogram	**-8.7 (-5.8%)** p < 0.01 vs. baseline n.s. vs. placebo		**A/T ratio 1.8 (29.5%)**		**improved: 11% unchanged: 77%**			frontal/ parietal	A2
						Arm2: placebo, 1x/d, oral		**-6.6 (-4.0%)** p < 0.05 vs. baseline		**A/T ratio 1.7 (29.8%)**		**improved: 16% unchanged: 77%**				

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Table 3: Literature Table. Dutasteride and Finasteride.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
Dutasteride																
73	Stough	2007	34	male	6m, 12m	Arm1: dutasteride 0.5 mg, 1x/d, oral	phototrichogram	**6m: 6.8** **12m: 16.5** significant (6m) vs. placebo p = 0.14 (12m) vs. placebo							vertex	A2
						Arm2: placebo, 1x/d, oral		**6m: -11.0** **12m: -3.8**								
61	Olsen	2006	416	male	24w	Arm1: dutasteride 0.1 mg, 1x/d, oral	phototrichogram	**15.4 (8.7%)** p < 0.001 vs. placebo				**39%**			vertex	A2
						Arm2: dutasteride 0.5 mg, 1x/d, oral		**18.6 (10.2%)** p < 0.001 vs. placebo				**63%** p = 0.026 vs. Arm 4				
						Arm3: dutasteride 2.5 mg, 1x/d, oral		**21.5 (11.3%)** p < 0.001 vs. placebo				**78%** p < 0.001 vs. Arm4				
						Arm4: finasteride 5 mg, 1x/d, oral		**14.8 (8.4%)** p < 0.001 vs. placebo p = 0.009 vs. Arm3				**57%**				
						Arm5: placebo, 1x/d, oral		**-6.3 (-3.5%)**				**2%**				

Continued

Table 3: Literature Table. Hormones Oral.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
Hormones Oral																
81	Peereboom-Wynia	1989	30	female	12m	Arm1: ethinyl oestradiol 50 m + CPA 2 mg 1x/d, oral, day 5-27 of menstrual cycle, CPA 20 mg, 1x/d, oral day 5-20 of menstrual cycle	trichogram			**anagen / telogen ratio 2.8 (164.7%)**					centro-parietal	B
						Arm2: no treatment				**-1.2 (-50%)**						
52	Vexiau	2002	66	female	6m, 12m	Arm1: minoxidil 2% solution, 2x/d, topical + oral contraceptive (ethinyl oestradiol 30 µg and gestodene 75 µg), 1x/d, 21 of 28 days, oral	phototrichogram	**6m: 16.1 (8.6%) 12m: 16.9 (9.1%)** p < 0.001 (12m) vs. Baseline and Arm2		**anagen (hairs/cm²) 25** p < 0.001 vs. Arm2					fronto-parietal	B
						Arm2: cyproterone acetate 50 mg, 1x/d, 20 of 28 days, oral + oral contraceptive (ethinyl estradiol 35 µg and cyproterone acetate 2 mg), 1x/d, 21 of 28 days, oral		**6m: -2.8 (-1.4%) 12m: -7.8 (-3.9%)** p 0 0.85 (12m) vs. Baseline		**-5.6**						

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Table 3: Literature Table. Hormones Topical.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
Hormones Topical																
77	Gassmueller	2008	172	male and female	16w	Arm1: Fulvestrant 70 mg/ml, 2x/d, topical, men	TrichoScan	**8.2** (4.4%) p 0 0.815 vs. Placebo p < 0.001 vs. Minoxidil			**hair thickness mm/cm²** **0.84** (5.4%) p = 0.968 vs. Placebo p < 0.001 vs. Minoxidil				vertex	A2
						Arm2: placebo, 2x/d, topical, men		**8.0** (4.4%)			**0.7** (4.4%)					
						Arm3: minoxidil 2% solution, 2x/d, topical, men		**25.4** (13.3%)			**2.9** (17.3%)					
						Arm4: fulvestrant 70 mg/ml, 2x/d, topical, women		**14.7** (6.9%) p 0 0.340 vs. Placebo			**1.54** (7.2%) p = 0.227					
						Arm5: placebo, 2x/d, topical, women		**15.3** (7.9%)			**1.58** (8.1%)					
46	Blume-Peytavi	2007	103	female	6m, 12m	Arm1: minoxidil 2% solution, 2x/d, topical	TrichoScan	**6m: 1 5.3** (8.7%) p = 0.0025 vs. Baseline p < 0.0005 vs. Arm2 **12m: 17.3** (9.9%)	**6m: 14.0** p = 0.003 vs. Baseline **12m: 14.9** p < 0.001 vs. Arm2		**hair thickness mm/cm²** **6m: 1.8** p < 0.0001 vs. Baseline **12m: 2.1**				centro-parietal	B
						Arm 2: 0-6m: alfatradiol 0.025% solution, 1x/d, topical, 7-12m: minoxidil 2% solution, 2x/d, topical		**6m: -7.8** (-4.3%) n.s. vs. Baseline **12m: 9.8** (5.4%) p < 0.0001 vs. 6m	**6m: -6.0** n.s. vs. Baseline **12m: 5.9**		**6m: -0.5** n.s. vs. Baseline **12m: 0.4**					

Continued

Table 3: Literature Table. Hormones Topical continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
82	Georgala	2004	75	female	12 - 24w	Arm1: Alfatradiol lotion 0.03%, 1x/d, topical, 12w	trichogram			**A/T ratio 0.65 (38.7%)** p < 0.01 vs. Placebo, n.s. vs. Arm2					bald area	B
						Arm2: Alfatradiol lotion 0.03%, 1x/d, topical, 24w				**0.7 (44.6%)** p < 0.01 vs. Placebo						
						Arm3: placebo lotion, 1x/d, topical, 24w				**-0.06 (-3.9%)**						
80	Wozel	2005	233	female and male	30w	Arm1: alfatradiol 0.025% solution, 1x/d, topical, men	trichogram			**telogen rate -6.7 (-21.1%)**					frontal	C
						Arm2: alfatradiol 0.025% solution, 1x/d, topical, women				**-5.8 (-24.4%)**						
78	Orfanos	1980	69	female and male	6m	Arm1: Alfatradiol 0.025% solution, 1x/d, topical	trichogram			**%improvement from baseline telogen rate**** 63%**					triangular	B
						Arm2: placebo, 1x/d, topical				**37%**						

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Table 3: Literature Table. Hormones Topical continued/Surgery.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
79	Wüstner	1974	50	female and male	6m	estrogen and corticoid, 1x/d, topical	trichogram	52%		%improvement from baseline telogen rate**** 52%					triangular/ parietal	C
76	Sovak	2002	43	male	3m, 9m	Arm1: fluridil, 1x/d, topical	phototrichogram			anagen hairs/cm² 3m: 9.3 (13.5%) 9m: 10.6 (15.5%)						B
						Arm2: placebo, 1x/d, topical				3m: 6.5 (10.1%) 9m:13.1 (20.5%)						
Surgery																
84	Uebel	2006	20	male	7m	Arm1: follicular unit transplantation	manual hair count	follicular units (%) ‘-40 (-28.2%) p < 0.001 (Arm1 vs. Arm2)							parietal	C
						Arm2: follicular unit transplantation + platelet plasma growth factor added before implantation		follicular units (%) ‘-25 (-17.6%)								
70	Leavitt	2005	79	male	12m	Arm1: hairtransplantation + finasteride 1mg, 1x/d, oral	macrophotograph	18.5 (12.6%) p = 0.019 (Arm1 vs. Arm2)				improved: 94% unchanged: 6%			frontal	A2
						Arm2: hairtransplantation + placebo, 1x/d, oral		-13.5 (-8.9%)				improved: 67% unchanged: 30%				

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Table 3: Literature Table. Surgery/Miscellaneous Oral.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
83	Bernstein	1998	41	male		Arm1: follicular unit transplantation, preparation by dissecting microscope		**mean hairs/ follicular unit 2,28**							parietal	B
						Arm2: follicular unit transplantation, preparation by magnifying loupe with transillumination		**mean hairs/ follicular unit 2,14**								
Miscellaneous Oral																
89	Prager	2002	26	male	18–24w	Arm1: active oral softgel supplement (ß-sitosterol 50 mg + saw palmetto extract 200 mg, lecithin 50 mg, inisotol 100 mg, phosphatidyl choline 25 mg, niacin 15 mg, biotin 100 g), 2x/d, oral	7-point-rating scale						**60%**			B
						Arm2: softgel placebo, 2x/d, oral							**11%**			
91	Lassus	1992	40	male	6m	Arm1: viviscal (marine extracts and silicea component), 2x/d, oral	manual hair count	**4.8 (38.1 %)** p < 0.001 vs. Arm2							bald area	A2
						Arm2: fish extract, 2x/d, oral		**0.3 (2.1%)**								

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Table 3: Literature Table. Miscellaneous Topical.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
Miscellaneous Topical																
90	Kamimura	2000	30	male	6m	Arm1: procyanidin 1% solution, 2x/d, topical	phototrichogram	**26,7** p < 0.005 vs. Placebo	**8.0** p < 0.02 vs. Placebo						vertex	B
						Arm2: placebo, 2x/d, topical		**0,3**	**-3.3**							
88	Draelos	2005	60	female	6m	Arm1: Niacin derivates (octyl nicotinate 0.5% and myristyl nicotinate 5.0%), 1x/d, topical	7-point-rating scale					**69%** p = 0.04 vs. Placebo			vertex	A2
						Arm2: placebo, 6 drops, 1x/d, topical						**33%**				
94	Kessels	1991	396	male	6m	Arm1: herbal preparation, 2x/d, topical	manual hair count		**26,6** p = 0.02 vs. Placebo						bald area	A2
						Arm2: placebo, 2x/d, topical			**21,8**							

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Table 3: Literature Table. Miscellaneous Topical continued.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
Miscellaneous Topical																
93	Greenberg	1996	24	male	40w	Arm1: herbal extract cream (7.5% extract of fennel, polygonum, mentha, chamomile, thuja, hibiscus), 1x/d, topical	manual hair count	**77,4%** p = 0.003 vs. Placebo	**169.4%** p = 0.01 vs. Placebo						triangular	B
						Arm2: placebo cream 1x/d, topical		**3%**	**33.9%**							
104	Olsen	1990	72	male	6m	Arm1: viprostol solution 120 µg, 2x/d, topical	phototrichogram		**-3.9 (-4.0%)** n.s. vs. Arm2/3						vertex	A2
						Arm2: vehicle, 2x/d, topical			**-9.1 (-9.5%)** n.s. vs. Arm1/3							
						Arm3: placebo, 2x/d, topical			**-3.1 (-3.3%)**							
38	Reygagne	1997	72	male	6m	Arm1: minoxidil 2% solution, 2x/d, topical	phototrichogram	**6.8 (11.0%)** p < 0.001 vs. Arm2		**anagen (hairs/cm²) 4.5 (9.8%)** p = 0.06 vs. Arm2					vertex	B
						Arm2: maxilene lotion (glycerol oxyesters and organic silicium), 1x/d, topical		**-3.0 (-4.8%)**		**-1.8 (-3.7%)**						

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Table 3: Literature Table. Miscellaneous Topical continued/Combination Therapies.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
103	Groveman	1985	174	male	16w	Arm1: polysorbate 60 25% lotion, 2x/d, topical						**2,6%****			full view	A2
						Arm2: placebo, 2x/d, topical						**4,7%****				
86	Gehring	2000	40	female	6m	Arm1: millet seed extract, L-cysteine and calcium pantothenate, 2x/d, oral	phototrichogram			**anagen rate 12.1 (16.0%)** p = 0.0225 vs. Placebo					centro-parietal	B
						Arm2: placebo, 2x/d, oral				**8.4 (11.3%)**						
Combination Therapies																
92	Lassus	1994	30	male	8m	Arm1: viviscal (polysaccharides and salt), 2x/d 80 kg, 3x/d > 80 kg, oral + viviscal shampoo 2-3x/w								**93.3%**		B

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Table 3: Literature Table. Miscellaneous Topical/Combination Therapies.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
16	Berger	2003	200	male	26w	Arm1: minoxidil 5% solution, 2x/d, topical + pyrithione zinc 1% shampoo, 1x/d, topical	phototrichogram	**6,2**							vertex	A2
						Arm2: minoxidil 5% solution, 2x/d, topical + placebo shampoo, 1x/d, topical		**12,3**								
						Arm3: pyrithione zinc 1% shampoo, 1x/d, topical		**5,7**								
						Arm4: placebo shampoo, 1x/d, topical		**-0.6**								
15	Bazzano	1986	56	female and male	12m	Arm1: placebo, 2x/d, topical	manual hair count	**% > 20% increase from baseline 0%**							bald area	B
						Arm2: minoxidil 0.5%, 2x/d, topical		**0%**								
						Arm3: tretinoin 0.025% solution 2x/d, topical		**58%**								
						Arm4: minoxidil 0.5% + Tretinoin 0.025% solution, 2x/d, topical		**66%**								

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Table 3: Literature Table. Miscellaneous Topical/Combination Therapies.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
85	Morganti	1998	60	female and male	50w	Arm1: lotion containing saw palmetto extract, 2x/d, topical	manual hair count	**27%** p < 0.005 vs. Baseline			**hair weight 32%**				frontal/ parietal	B
						Arm2: placebo lotion, 2x/d, topical		**13%**			7%					
						Arm3: diet supplement containing l-cistin, lmethionin, Cu, Zn, 4x/d, oral		**29%** p < 0.005 vs. Baseline			**42%**					
						Arm4: placebo supplement, 4x/d, oral		**11%**			**-8%**					
						Arm5: lotion containing saw palmetto extract, 2x/d, topical + diet supplement containing l-cistin, lmethionin, Cu, Zn, 4x/d, oral		**38%** p < 0.005 vs. Arm1/3			**60%**					

Continued

Table 3: Literature Table. Miscellaneous Topical/Combination Therapies.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
Pulsed Electromagnetic Field																
101	Policarpi	1993	30	female and male	18w, 36w	Arm1: pulsed electrostatic field, 1-2 sessions per week	phototrichogram			**anagen: 18w: 4.5 % 36w: 6.2%**					bald area	B
						Arm2: placebo sessions, 1-2x/week				**18w: 0.9% 36w: 1.0%**						
99	Maddin	1990	73	male	36w	Arm1: pulsed electrostatic field, 1x/w, week 1, 17, 33 2x/w	manual hair count		**11.8 (66.1%)** p = 0.0298 vs. Arm2						vertex	A2
						Arm2: sham treatment, 1x/w, week 1, 17, 33 2x/w			**5.6 (25.6%)**							
100	Maddin	1992	34	male	12w, 34w	Arm1: pulsed electrostatic field, 1x/w, pretreatment 36w pulsed electrostatic field	manual hair count	**12w: 5.9 (20.0%) 34w: 24.7 (84.0%)**							vertex	B
					16w, 30w	Arm2: pulsed electrostatic field, 1x/w, pretreatment 36w sham electrostatic field		**16w: -0.8 (-2.5%) 30w: 17.5 (55.6%)**								

Continued

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Table 3: Literature Table. Miscellaneous Topical/Combination Therapies.

ArtNumber	Author	Year	Number of patients	Gender	Time of evaluation	Dosage	Method	mean change from baseline hair count (total) hairs/cm² (%)	mean change from baseline hair count (nonvellus) hairs/cm² (%)	mean change from baseline hair count (anagen, telogen, anagen/telogen ratio)	mean change from baseline hair weight / thickness	global expert panel assessment	investigator assessment	patient assessment	Investigational area	Degree of evidence
98	Bureau	2003	93	female and male	6m	Arm1: herbal solution (pimenta racemosa, myrtus communis, cedrus atlantica, laurus nobilis, pogostemon patchouli, rosmarinus officinalis, salvia sclarae, thymus satureioides, cananga odorata), 3x/w, topical + pulsed electromagnetic field 3x/w, topical	phototrichogram	**34 (22.4%)** p = 0.003 vs. Baseline, p = 0.01 vs. Arm2							bald area	A2
						Arm2: placebo, 3x/w, topical + pulsed electromagnetic field 3x/w, topical		**9 (5.5%)** n.s. vs. Baseline								
102	Satino	2003	35	female and male	6m	Arm1: HairMax Laser Comb (low level laser therapy), comb hair 1x/d 5-10 minutes manual hair count	manual hair count	**14.1 (76.2%)** n.s. vs. Baseline	**12.3 (60.0%)**						vertex/ temporal	C

* blinding till 12m, afterwards open study design
** mean of 3 reviewers
*** investigator = outcome assessor

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