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Impact of Testosterone on Hair and Skin

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Abstract

Testosterone is the most important androgen. Testosterone production starts from adrenarche in both sexes: in males in the testes, in females in the ovaries and in both sexes to a lesser part in the adrenal cortex, too. Circulating in blood certain amounts of testosterone are bonded to Sexual Hormone Binding Globulin (SHBG). The free part represents the active steroid affecting various organs to a certain extent. Testosterone is responsible for the formation of the male-phenotype, development of muscular tissue, bone density, and in a certain way it influences the fat/sugar metabolism. It also has a big impact on skin, skin appendages, and hair in particular. Testosterone increases sebum production (seborrhoea) and controls hair growth as well as hair loss in certain areas of the body.

Keywords: Testosterone; Seborrhea; Acne; Androgenetic alopecia; Hirsutisma

Impact of Testosterone on the Skin

There is specific impact of steroid hormones in both sexes. Testosterone/estrogen ratio in males and females gives reason for differences in skin thickness and texture. Male epidermis for example is 20% thicker than female epidermis, being able to bind a larger amount of moisture, and containing more collagen in all ages, thus, making male skin more dense and vigorous [1].

Androgens stimulate sebum production. Therefore, due to higher androgen levels male seborrheic glands produce more sebum leading to fatty glow and coarser pores of the skin [2]. Consequently, acne, acneiform eruptions, and blemishes are more common in seborrheic areas (face, back, upper thorax, shoulders) (Figure 1). Due to androgen triggered follicular hyperkeratosis sebum is "trapped" within the follicle forming primary acne lesions (comedones). Comedones act as ideal breeding ground for habitual microbacteria like several cocci, propionibacterium acnes, and certain malassessia species, making them proliferate above physiological amounts. Hence, these microbacteria metabolize only a certain amount of sebum "leaving behind" free fatty acids. These "corrode" the follicular epithelium, and invading surrounding tissue they lead to irritation and immuno-chemical reactions. Chemotaxis causes "invasion" of granulocytes, inflammation, and development of secondary acne lesions (papules, pustules, nodes, and cysts). Severe manifestations may also involve sweat glands leading to hidradenitis or cause fistulae and gum boils in axillary, inguinal, or perineal locations. Early treatment of acne lesions aims at limitation of long-term sequels.

Wet shaving is an additional risk factor for male facial skin, causing dryness, irritation, redness, and exfoliation, as well as cutting damage caused by the razor blade followed by bleeding and scar formation. Decades back, male skin care consisted of washing and shaving only, since the beginning of the 21st century it has changed a lot. Today, skin care products particularly designed for male skin have revolutionized the market. A great variety of cosmeceuticals, aligned to the individual skin type, is now used by the young generation.

In general, water contact exhausts lipids from epidermal skin layers. Frequent water contact disturbing the barrier function of the skin, leads to dryness and eczema reactions. Moreover, testosterone has proven negative effect on the barrier function, making male skin particularly more sensitive than female skin [3]. Regular application of skin care products can reduce vulnerability of the skin and eczema probability by supplying the skin with lipids and moisture.

On one hand, male skin regenerates more quickly than female skin due to testosterone controlled higher cellular turnover, on the other hand wound healing turns out to occur more slow than in females, due to higher testosterone levels [4].

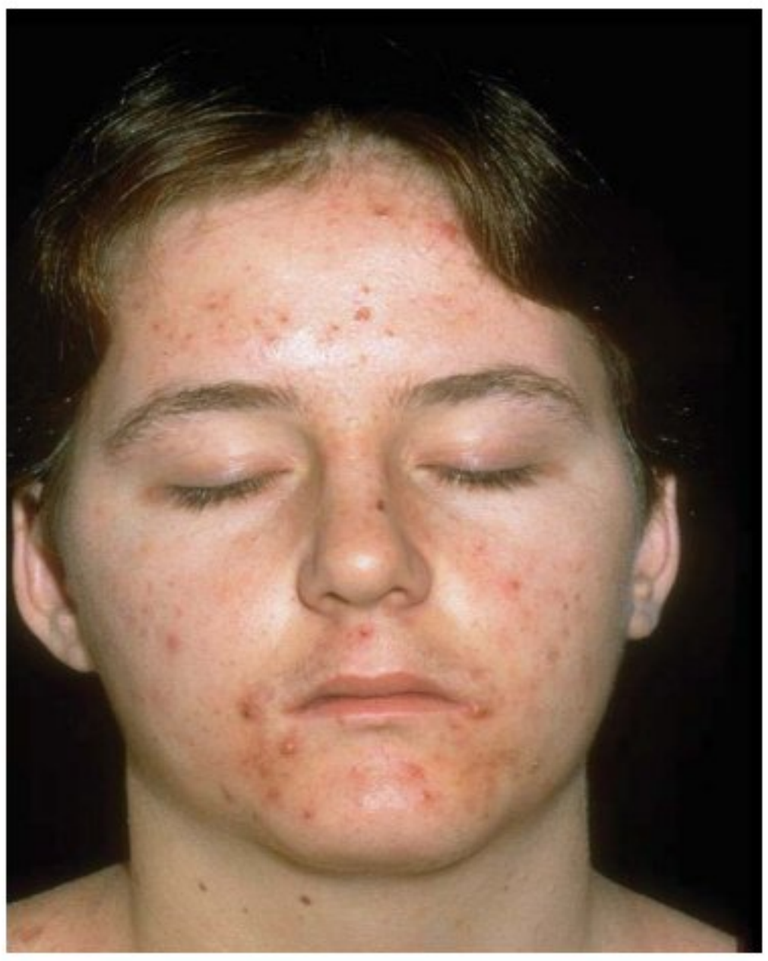

Figure 1: Acne papulopustulosa (source: Derma, Graz).

In females hyperandrogenism causes characteristic changes of the skin and impacts on hair growth, resulting in fatty skin and hair by increased sebum production (seborrhea), acne, hirsutism, androgenetic alopecia, as when all together, they can be summarized under the term SAHA-syndrome and hyperpigmentation in skin folds, referred to as acanthosis nigricans. Severity may vary, but exclusion of polycystic ovary syndrome should be accomplished [5].

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There is a lack of evidence-based literature on the context of testosterone and skin aging [6]. Sparse trials have shown that there is no specific difference in skin aging processes in both sexes. Presumably, males tend to develop a larger number of seborrheic keratosis and females preferably solar lentigines [7]. UV-exposure and Fitzpatrick-skin-type are predispositions for developing wrinkles and rhytides in both sexes [8]. Males may develop melanomas more easily than women [9,10].

Impact of Testosterone on Hair Growth

Hair follicles develop during the first trimester of fetal development. At that time the whole skin except palms and soles, lips and semi-mucosal areas is covered with fine lanugo hair follicles. Each hair follicle is "loaded" with an individual genetic code, telling when during the lifespan, where on the skin, due to which trigger a hair will grow or fall out. Androgens play an important role in androgenic hair loss, even if androgen levels are within normal ranges, as androgen sensitivity of the hair follicles varies individually due to genetic determination.

Effect of Testosterone on Hair Growth in Males

Androgenetic alopecia male type

Di-hydro-testosterone DHT, synthesised by the activity of the enzyme 5-α-reductase is the most important metabolite of testosterone concerning hair growth and hair loss. Whereas DHT is responsible for hair loss in certain areas of the scalp, it represents the main trigger for hair growth in androgen sensitive areas in (elderly) males (Figure 2), and hirsute females. On the scalp hair follicles miniaturize according to the androgen-sensitivity of the individual, thus, leading to the clinical appearance of androgenetic alopecia presenting a receding frontal hairline as well as vertex and total balding [11]. Severity of androgen dependent balding can be scored according to the Norwood-Hamilton scale [12] (Figure 3). The transformation of testosterone into DHT can be reduced by finasteride, a 5-α-reductase inhibitor. Finasteride is used successfully in the prevention and treatment of androgenetic alopecia [13].

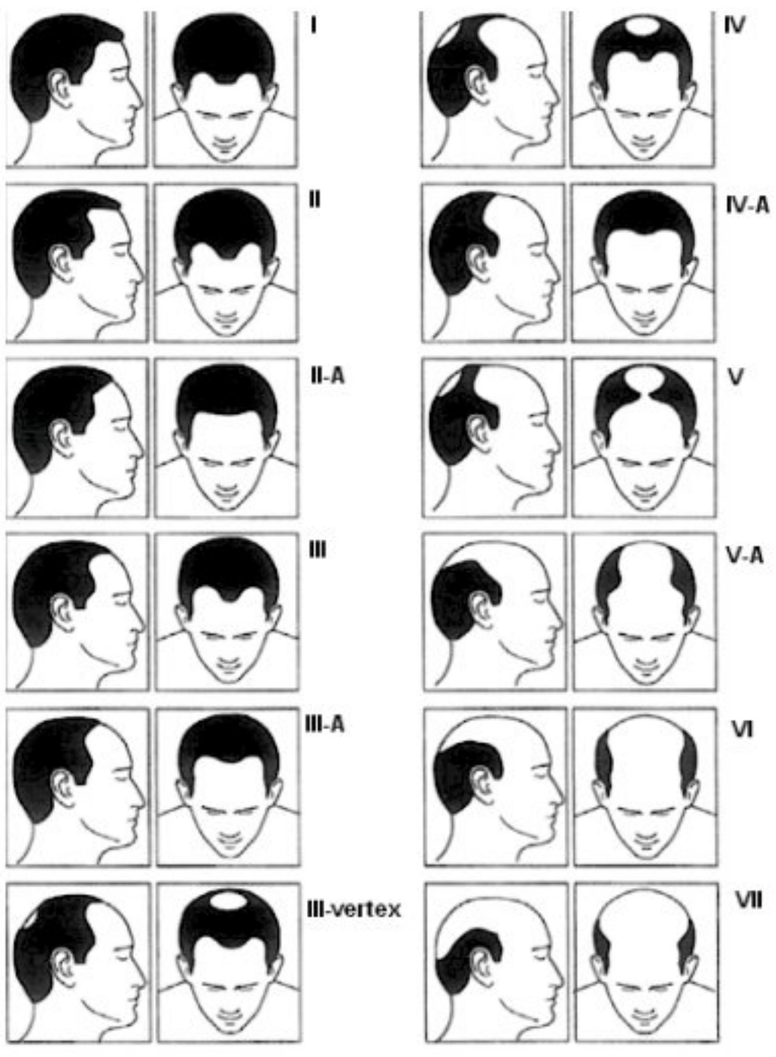

Figure 2: Norwood-Hamilton Score for male type androgenetic alopecia.

Androgen induced hair loss has an individual impact on psychosomatic status and quality of life has been evaluated in a multinational telephone survey: more than 60% of over 1.500 individuals reported negative impact of hair loss on their self-confidence, 21% showed depressive symptoms [14].

Effect of Testosterone on Hair Growth in Females

Androgenetic alopecia female type

DHT plays an inferior role in hormonal hair loss in women, androgen metabolites like dihydro-epi-androsterone DHEA and androstendione are more important in female androgenetic alopecia, causing a certain phenotype gradually developing sparse hair in parietal areas, scored according to the Ludwig scale (Figure 4). A ndrogentic female hair loss is most likely retaining the frontal hairline [15].

Antiandrogens like Cyproterone-Acetate (CPA) may reduce hormonal hair loss in females. Finasteride should not be used for the treatment of AGA in premenopausal women, as male fetus´ may feminize, whereas several studies have shown that finasteride is able to stop further hair loss in post-menopausal females [16,17].

Hirsutism

The amount of hair growth in certain body areas differs according to culture and ethnicity. Thus, the differentiation between androgen-induced Hair growth in hirsute women has to be differentiated from increased vellus hair growth in females with mediterranean or indogenic background. Hirsutim defines a male phenotype pattern of hair growth in females, 5% of all women are affected (Figure 5 and 6).

From internal-medical view hirsutism may have various reasons: ovary and adrenal tumors are rare, congenital adrenal hyperplasia is even more so, insulin-resistance-syndrome, Mb. Cushing and Polycystic

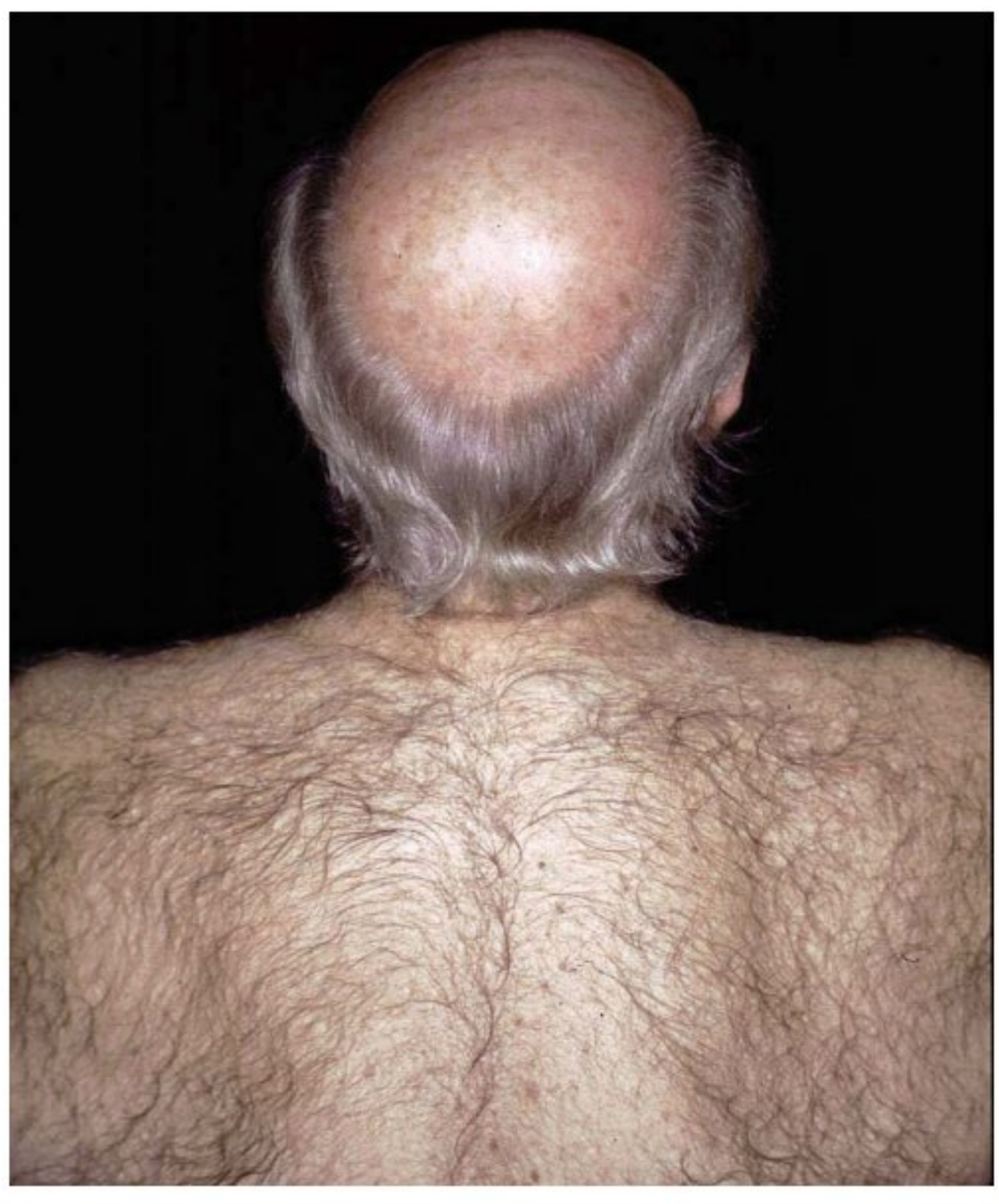

Figure 3: Variable androgen sensitivity in certain body areas: hair-loss vs. hair-growth (source: Derma, Graz).

Figure 4: Ludwig score for female type androgenetic alopecia.

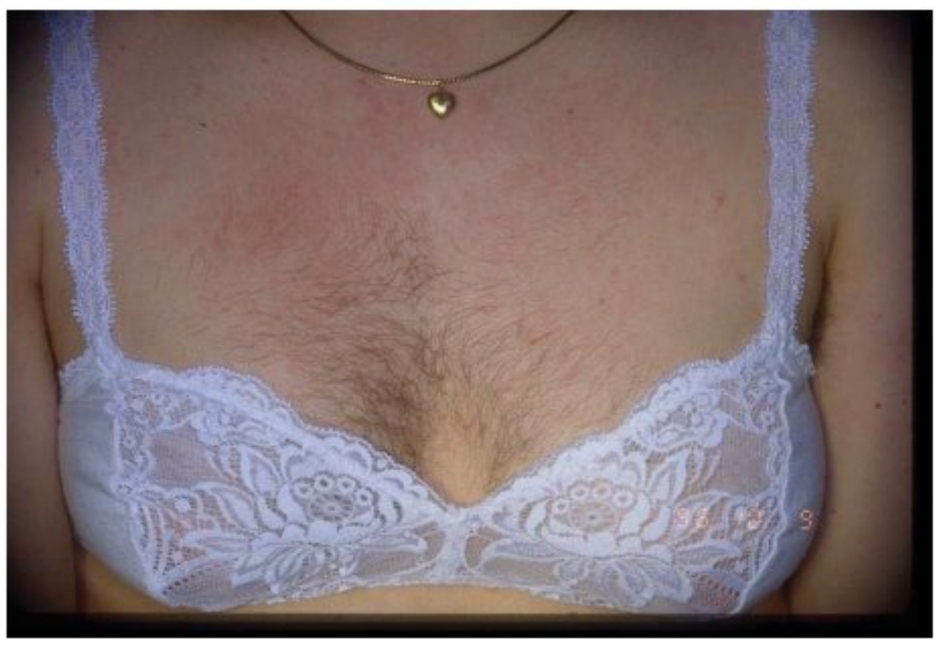

Figure 5: Hirsutism: androgen-induced hair growth (source: Kopera).

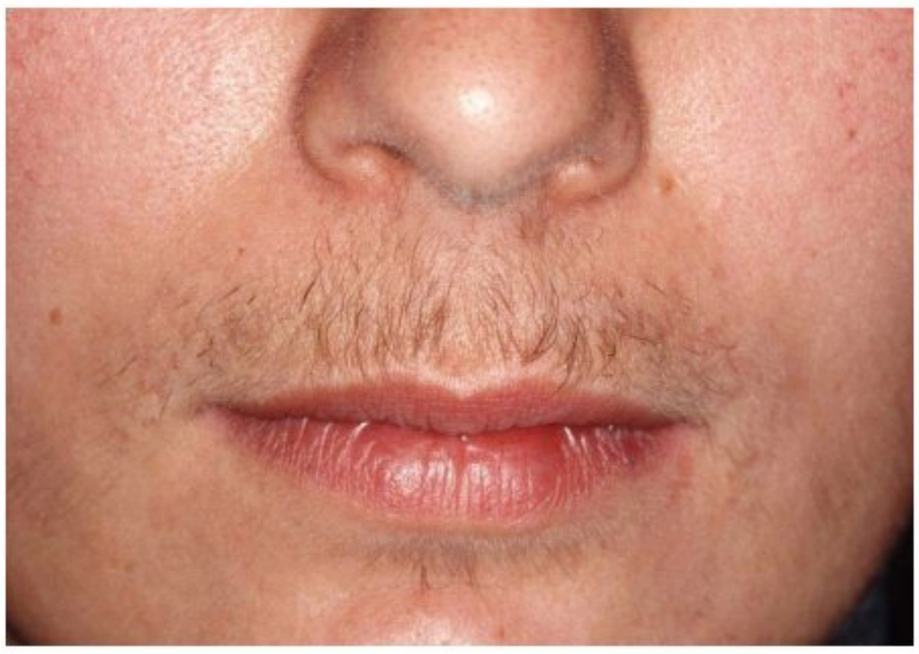

Figure 6: Undesired hair growth in androgen-sensitive area (source: Derma, Graz).

Ovary Syndrome (PCOS), representing the most common endocrine disorder in 5% to 10% of all females. More than 70% of hirsute women feature PCOS – occurring also in females with normal menstrual cycle.

Hirsute females must not necessarily show increased Testosterone levels, but there may be a "masked" hyperandrogenism. It is a very sophisticated mechanism that is controlled by increased LH/FSH-ratio, increased GnRH-excretion and the insulin metabolism in females with PCOS. Nevertheless, in severe cases elevated androgen levels lead to manifest hyperandrogenism. Moreover, the LH-synergistic effect of

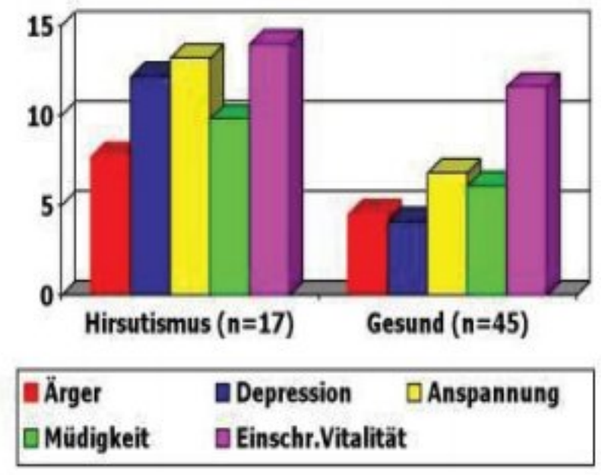

Figure 7: Psychosocial comorbididy and hirsutism.

insulin on theca-cells in the ovaries also attributes to increased androgen production. On the other hand, it inhibits SHBG production, which should bind testosterone, thus, leading to elevated free testosterone levels [18].

Hirsutism may cause negative impact on psychosocial health and behaviour [19] (Figure 7). In order to achieve optimal results, management of hirsute females requires optimal interdisciplinary care, combining endocrinologic, dermatologic, gynecologic, and aesthetic know how for the application of individual treatment options like antiandrogens, insulin sensitizers, and light-assisted hair reduction.

References

1. Markova MS, Zeskand J, McEntee B, Rothstein J, Jimenez SA, et al. (2004) A role for the androgen receptor in collagen content of the skin. J Invest Dermatol 123: 1052-1056.
2. Baumann L (2002) Acne, in: Cosmetic Dermatology: Principles and Practice , Baumann L, Weisberg E, Mcgraw-Hill, New York.
3. Kao JS, Garg A, Mao-Qiang M, Crumrine D, Ghadially R, et al. (2001) Testosterone perturbs epidermal permeability barrier homeostasis. J Invest Dermatol 116: 443-451.
4. Ashcroft GS, Mills SJ (2002) Androgen receptor-mediated inhibition of cutaneous wound healing. J Clin Invest 110: 615-624.
5. Dalamaga M, Papadavid E, Basios G, Vaggopoulos V, Rigopoulos D, et al. (2013) Ovarian SAHA syndrome is associated with a more insulin-resistant profile and represents an independent risk factor for glucose abnormalities in women with polycystic ovary syndrome: a prospective controlled study. J Am Acad Dermatol 69: 922-930.
6. Mercurio MG (1998) Gender and dermatology. J Gend Specif Med 1: 16-20.
7. Chung JH, Lee SH, Youn CS, Park BJ, Kim KH, et al. (2001) Cutaneous photodamage in Koreans: influence of sex, sun exposure, smoking, and skin color. Arch Dermatol 137: 1043-1051.
8. Akiba S, Shinkura R, Miyamoto K, Hillebrand G, Yamaguchi N, et al. (1999) Influence of chronic UV exposure and lifestyle on facial skin photo-aging--results from a pilot study. J Epidemiol 9: S136-142.
9. U.S. Cancer Statistics Working Group (2010) United States Cancer Statistics: 1999-2007 Incidence and Mortality Web-based Report. Atlanta (GA): Department of Health and Human Services, Centers for Disease Control and Prevention, and National Cancer Institute.
10. Stoebner-Delbarre A, Thezenas S, Kuntz C, Nguyen C, Giordanella JP (2005) Sun exposure and sun protection behavior and attitudes among the French population]. Ann Dermatol Venereol 132: 652-657.
11. Alsantali A, Shapiro J (2009) Androgens and hair loss. Curr Opin Endocrinol Diab Obes 16: 246-253.
12. Hamilton JB (1951) Patterned loss of hair in man; types and incidence. Ann N Y Acad Sci 53: 708-728.
13. Kaufman KD, Girman CJ, Round EM, Johnson-Levonas AO, Shah AK, et al.

(2008) Progression of hair loss in men with androgenetic alopecia (male pattern hair loss): long-term (5-year) controlled observational data in placebo-treated patients. Eur J Dermatol 18: 407-411.

14. Alfonso M, Richter-Appelt H, Tosti A, Viera MS, García M (2005) The psychosocial impact of hair loss among men: a multinational European study. Curr Med Res Opin 21: 1829-1836.

15. Ludwig E (1977) Classification of the types of androgenetic alopecia (common baldness) occurring in the female sex. Br J Dermatol 97: 247-254.

16. Iorizzo M, Vincenzi C, Voudouris S, Piraccini BM, Tosti A (2006) Finasteride treatment of female pattern hair loss. Arch Dermatol 142: 298-302.

17. Van Zuuren EJ, Fedorowicz Z, Carter B (2012) Evidence-based treatments for female pattern hair loss: a summary of a Cochrane systematic review. Br J Dermatol 167: 995-1010.

18. Kopera D, Wehr E, Obermayer-Pietsch B (2010) Endocrinology of hirsutism. Int J Trichology 2: 30-35.

19. Barth JH, Catalan J, Cherry CA, Day A (1993) Psychological morbidity in women referred for treatment of hirsutism. J Psychosom Res 37: 615-619.