Introduction

The term “hirsutism” means presence of excessive terminal hair in androgen-dependent areas of the female body. Hirsutism is a frequent medical complaint that usually results from relatively benign functional disorders albeit, rarely, hirsutism may be the presenting sign of a life-threatening disorder. The present chapter aims to provide a comprehensive review on the diagnostic and therapeutic management of hirsutism based almost entirely on the author’s experience and usual practices. The reader expecting evidence-based guidelines is kindly referred to a recent publication sponsored by the Androgen Excess and Polycystic Ovary Syndrome Society [1].

Pathophysiology of hirsutism

The pilosebaceous unit is a highly dynamic system that changes throughout the lifespan. Before puberty, the pilosebaceous unit includes a vellus hair – which is soft, short, and has no medulla – and a small sebaceous gland. In androgen-responsive areas, the increase in androgens characteristic of puberty induces the pilosebaceous unit to produce terminal hair – which is coarse, pigmented, and has a medulla – and the size of the sebaceous unit increases markedly.

Hair grows in asynchronous cycles that comprise three phases consisting of: (i) an anagen or growing phase that accounts for 85–90% of the duration of the hair cycle and may last for a few months in the case of terminal hair; (ii) a catagen or rapid involution phase that accounts for 2–3% of the hair cycle; and (iii) a telogen or resting phase accounting for 10–15% of cycle at which the end hair is ejected and anagen starts again (Figure 1.1) [2]. Because androgens stimulate the growth of terminal hair by prolonging its anagen phase, the clinical effects of androgen excess and its amelioration necessarily take months to be apparent to both the patient and her physician. Being aware of this fact is of capital importance for the correct management of hirsutism.

Because androgens play a definite role in the transformation of vellus into terminal hair during puberty, and in the growth of terminal hair in androgen-dependent areas of the female body, hirsutism is considered to be a clinical marker of androgen excess [3]. However, some hirsute patients do not show any other evidence of androgen excess, such as hyperandrogenemia or ovarian dysfunction, and often receive a diagnosis of “idiopathic” hirsutism [4].

To understand this apparent paradox, it is important to know some facts about female androgen metabolism. In women, the adrenals and the ovaries secrete androgens into the circulation, because these are the only organs in the female body expressing the biosynthetic enzymes needed for the synthesis of androgens. Peripheral tissues, such as fat, also contribute to circulating androgen levels by converting other steroid precursors. Testosterone is the most important androgen and circulates mostly bound to serum albumin (low affinity, but large capacity) and to sex-hormone-binding globulin (SHBG) (high affinity, but small capacity). Given its high affinity for testosterone, SHBG actually regulates the amount of testosterone that reaches target tissues, even if its binding capacity is much less than that of albumin. Therefore, the lower the SHBG concentration, the larger the fraction of free or unbound testosterone that may reach target tissues.

However, testosterone is a prohormone that undergoes conversion into dihydrotestosterone in target cells before entering the cell nucleus and binding the androgen receptor. Both the conversion rate of...
testosterone – mediated by 5α-reductase – and the binding of dihydrotestosterone to the androgen receptor are subject to individual variation, and current hypotheses explain idiopathic hirsutism as the result of increased 5α-reductase activity and/or increased sensitivity of the androgen receptor to normal amounts of testosterone [4]. An alternate hypothesis is that women with idiopathic hirsutism have mild steroidogenic abnormalities that go undetected by the relatively insensitive biochemical tests applied in routine clinical practice [5]. Considering the well-known limitations of the assays of serum androgens currently available for clinical practice [6], it is my personal opinion that the presence of hirsutism should be considered an accurate marker of androgen excess, irrespective of serum androgen concentrations. In other words, most if not all hirsute patients are hyperandrogenic, and our limitation in detecting androgen excess is the actual culprit that we cannot confirm this diagnosis by analytical tools.

Quantification and epidemiology of hirsutism

The definition of hirsutism implies that the amount of terminal hair must be quantified before establishing such a diagnosis. After the initial attempts to standardize the quantification of body hair made by S. M. Garn (who developed his score to assess male hairiness) [7], D. Ferriman and J. D. Gallwey [8], and E. Moncada Lorenzo [9], in 1981, Hatch and colleagues [10] published the modification of the original Ferriman–Gallwey score that is currently the “gold-standard” for the quantification of hirsutism. The modified Ferriman–Gallwey score (mFG) estimates the presence of terminal hair in nine areas of the female body – upper lip, chin, chest, abdominal region above and below the navel, upper and lower back, arms and thighs – and assigns a score from 0 (absent) to 4 (complete cover) to each of these areas for a total score ranging from 0 to 36 [10]. Most clinicians and researchers use a cut-off value of 8 or above to diagnose hirsutism, and grade it as mild up to a score of 15, moderate from 16 to 25, and severe above 25.

The broad application of this scoring system provided researchers with a common language for the definition of hirsutism and was followed by significant advances in the study of hirsutism and related conditions, such as polycystic ovary syndrome (PCOS). However, the mFG score has evident limitations, the most notable being the subjective nature of the assessment – yet, it appears that inter-observer variation is acceptable [11] – the possibility that substantial terminal hair in one or
Table 1.1 Summary of studies addressing the prevalence of hirsutism in women

<table>
<thead>
<tr>
<th>Author, year</th>
<th>Country</th>
<th>Race</th>
<th>Ethnicity</th>
<th>Score</th>
<th>Cut-off</th>
<th>Method of sample selection</th>
<th>Sample size</th>
<th>Prevalence (95% CI)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diamanti-Kandarakis et al., 1999 [12]</td>
<td>Greece</td>
<td>White</td>
<td>Mediterranean</td>
<td>FG</td>
<td>≥ 6</td>
<td>Invitation of free medical examination</td>
<td>192</td>
<td>38% (31–45)</td>
<td>Possible selection self-referred bias</td>
</tr>
<tr>
<td>Asarian-Asarian et al., 2000 [13]</td>
<td>Spain</td>
<td>White</td>
<td>Mediterranean</td>
<td>mFG</td>
<td>≥ 8</td>
<td>Unselected female blood donors from general population clinic</td>
<td>154</td>
<td>10.5% (6.9–14.4)</td>
<td>Includes postmenopausal women</td>
</tr>
<tr>
<td>Zargar et al., 2002 [14]</td>
<td>India</td>
<td>Asian</td>
<td>Kashmiri/Dardic</td>
<td>FG</td>
<td>≥ 6</td>
<td>Hospital outpatient clinic</td>
<td>4780</td>
<td>10.5% (9.6–11.4)</td>
<td>Includes postmenopausal women</td>
</tr>
<tr>
<td>Sagsoz et al., 2004 [15]</td>
<td>Turkey</td>
<td>White</td>
<td>Middle Eastern</td>
<td>mFG</td>
<td>≥ 8</td>
<td>Regular check-up in outpatient clinic</td>
<td>204</td>
<td>8.3% (4.5–12.1)</td>
<td></td>
</tr>
<tr>
<td>Cheewadhanaraks et al., 2004 [16]</td>
<td>Thailand</td>
<td>White</td>
<td>Thai and Chinese</td>
<td>mFG</td>
<td>≥ 3</td>
<td>Regular cervical smear check</td>
<td>531</td>
<td>2% (0.8–3.3)</td>
<td></td>
</tr>
<tr>
<td>DeOroy et al., 2006 [17]</td>
<td>USA</td>
<td>White</td>
<td>Caucasian and Hispanic, African American</td>
<td>mFG</td>
<td>≥ 8</td>
<td>Pre-employment physical exam</td>
<td>300</td>
<td>5% (2.8–8.0)</td>
<td></td>
</tr>
<tr>
<td>De Ugarte et al., 2006 [18]</td>
<td>USA</td>
<td>White, Black, Caucasian and Hispanic, African American</td>
<td>African American</td>
<td>mFG</td>
<td>≥ 8</td>
<td>Pre-employment physical exam</td>
<td>204 350</td>
<td>5.4% (2.8–8.0) 4.3% (2.2–6.4)</td>
<td>Possible selection self-referred bias 97.5% reproductive age</td>
</tr>
<tr>
<td>Noorbala and Kefai, 2010 [18]</td>
<td>Iran</td>
<td>White</td>
<td>Middle Eastern</td>
<td>mFG</td>
<td>≥ 8</td>
<td>Randomized cluster-sampling proportional to population size</td>
<td>900</td>
<td>10.6% (8.8–12.8)</td>
<td>Included only teenagers</td>
</tr>
<tr>
<td>March et al., 2010 [19]</td>
<td>Australia</td>
<td>White</td>
<td>Caucasian</td>
<td>mFG</td>
<td>≥ 8</td>
<td>Unselected population cohort</td>
<td>728</td>
<td>21.2% (18.2–24.2)</td>
<td>3% were not white</td>
</tr>
<tr>
<td>Sanchez et al., 2012 [20]</td>
<td>Spain</td>
<td>White</td>
<td>Mediterranean</td>
<td>mFG</td>
<td>≥ 8</td>
<td>Unselected female blood donors from general population clinic</td>
<td>393</td>
<td>11.7% (8.5–14.9)</td>
<td></td>
</tr>
<tr>
<td>Sanchez et al., 2012 [20]</td>
<td>Italy</td>
<td>White</td>
<td>Mediterranean</td>
<td>mFG</td>
<td>≥ 8</td>
<td>Unselected female blood donors from general population clinic</td>
<td>199</td>
<td>13.1% (8.4–17.7)</td>
<td></td>
</tr>
<tr>
<td>Gabrielli and Aquino, 2012 [21]</td>
<td>Brazil</td>
<td>Mixed</td>
<td>Mixed</td>
<td>mFG</td>
<td>≥ 6</td>
<td>Premenopausal women during cervical cancer screening</td>
<td>859</td>
<td>12.5% (10.4–14.8)</td>
<td>88.5% were black</td>
</tr>
</tbody>
</table>

FG, Ferriman–Gallwey score; mFG, modified Ferriman–Gallwey score.

a Invitation of free medical examination.
b Only 66% of invited women participated.
c Only 53% of invited women participated, and patients self-assessed their hirsutism scores.

Modifié from Escobar-Morreale et al. [1], by permission of Oxford University Press.
Section 1: Managing the basics

The prevalence of hirsutism varies according to the mFG score cut-off value and the population under study [12–21]. This prevalence is relatively homogeneous across the world with the exception of women of Asian ancestry, in whom hirsutism is much less frequent (Table 1.1). In American women, 7.6%, 4.6%, and 1.9% demonstrated a score of 6 or more, 8 or 10, and there was no significant racial difference, with hirsutism prevalences of 8.0%, 2.8%, and 1.6% in white women, and 7.1%, 6.1%, and 2.1% in black women, respectively, according to the chosen cut-off [22]. Similarly, we found that 7.1% of unselected blood donors in Spain had hirsutism as defined by an mFG score above 7 [13]. These and other studies addressing the prevalence of hirsutism, as defined by a pre-defined mFG score cut-off value in different populations according to their race and ethnicity, are summarized in Table 1.1. However, because race and ethnicity greatly influence the amount of body hair, ideally the cut-off values of the mFG score should be obtained from the particular population under study. Table 1.2 includes proposed mFG score cut-off values based on the 95th percentile of selected female populations of fertile age [11,13,15–18,20,23–26]. Broad application of these cut-off values would render a uniform 5% worldwide prevalence of hirsutism.

### Diagnosis of hirsutism

After establishing the presence of hirsutism by an increased mFG score, or if a history of hirsutism is strongly suggested by the finding of some evidence of terminal hair in androgen-dependent areas in women successfully treated for this condition, the most likely etiology should be established in all patients.

Functional causes account for most cases [27–31]: PCOS, as defined by the combination of hyperandrogenism with ovarian dysfunction (oligo-ovulation or polycystic ovarian morphology), is the most frequent diagnosis, accounting for approximately 60% of cases, followed by idiopathic hyperandrogenism (when there is no evidence of ovarian dysfunction) in approximately 25% of cases, idiopathic hirsutism (when there is no evidence of hyperandrogenemia or ovarian dysfunction) in approximately 10% of cases, and nonclassic congenital adrenal hyperplasia in approximately 3–5% of cases (Table 1.3). Exceptionally, hirsutism derives from benign or malignant adrenal or ovarian tumors, from hyperplasia of ovarian cells, from androgenic medications or drugs that

<table>
<thead>
<tr>
<th>Table 1.2</th>
<th>Suggested cut-offs for the modified Ferriman–Gallwey hirsutism score (mFG) according to the 95th percentile in different unselected populations of premenopausal women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Author, year</td>
<td>Year</td>
</tr>
<tr>
<td>Sagsoz et al., 2004 [15]</td>
<td>2004</td>
</tr>
<tr>
<td>Cheewadhanarasiks et al., 2004 [16]</td>
<td>2004</td>
</tr>
<tr>
<td>Tellez and Frenkel, 1995 [23]</td>
<td>2005</td>
</tr>
<tr>
<td>DeUgarte et al., 2006 [17]</td>
<td>2006</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Zhao et al., 2007 [24]</td>
<td>2007</td>
</tr>
<tr>
<td>Api et al., 2009 [11]</td>
<td>2009</td>
</tr>
<tr>
<td>Moran et al., 2010 [25]</td>
<td>2010</td>
</tr>
<tr>
<td>Noorbala and Kefaie, 2010 [18]</td>
<td>2010</td>
</tr>
<tr>
<td>Kim et al., 2011 [26]</td>
<td>2011</td>
</tr>
<tr>
<td>Sanchón et al., 2012 [20]</td>
<td>2011</td>
</tr>
</tbody>
</table>

¹ As defined by the 95th percentile of an unselected population of premenopausal women. Modified from Escobar-Morreale et al. [1], by permission of Oxford University Press.
## Table 1.3 Frequencies of the etiologies of androgen excess in large clinical series

<table>
<thead>
<tr>
<th>Author, year</th>
<th>Sample size (n)</th>
<th>PCOS (n)</th>
<th>Idiopathic hyperandrogenism (n)</th>
<th>Idiopathic hirsutism (n)</th>
<th>NCCAH (n)</th>
<th>Tumors (n)</th>
<th>Miscellaneous (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Azziz et al., 2004 [27]</td>
<td>873</td>
<td>749</td>
<td>59a</td>
<td>39</td>
<td>18</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>Glintborg et al., 2004 [28]</td>
<td>340</td>
<td>134</td>
<td>86b</td>
<td>115</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Unluhizarci et al., 2004 [29]</td>
<td>168</td>
<td>96</td>
<td>29c</td>
<td>27</td>
<td>12</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Carmina et al., 2006 [30]</td>
<td>950</td>
<td>685</td>
<td>150</td>
<td>72</td>
<td>41</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Escobar-Morreale et al., 2008 [31]</td>
<td>270</td>
<td>171</td>
<td>61d</td>
<td>24</td>
<td>6</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td><strong>Total no. (%)</strong></td>
<td><strong>2601 (100)</strong></td>
<td><strong>1835 (71)</strong></td>
<td><strong>385 (15)</strong></td>
<td><strong>277 (10)</strong></td>
<td><strong>79 (3)</strong></td>
<td><strong>8 (0.3)</strong></td>
<td><strong>17 (0.7)</strong></td>
</tr>
</tbody>
</table>

NCCAH, nonclassic congenital adrenal hyperplasia; PCOS, polycystic ovary syndrome.

- a The original article considered 33 patients with the hyperandrogenism, insulin resistance, and acanthosis nigricans (HAIR-AN) syndrome as a separate disorder from PCOS, reducing this figure to 716.
- b Polycystic ovarian morphology was not considered for the diagnosis of PCOS in these studies, which relied on the 1990 National Institute of Child Health and Human Development criteria [32]. Some of these patients might have been diagnosed with PCOS if ovarian morphology had been considered.
- c This study considered polycystic ovarian morphology for PCOS diagnosis, and 147 of the 685 PCOS patients who had regular ovulatory cycles would have been included in the idiopathic hyperandrogenism subgroup if ovarian morphology had not been considered.

Reproduced from Escobar-Morreale et al. [1], by permission of Oxford University Press.
interfere with ovarian steroidogenesis such as valproate, or from gestational hyperandrogenism secondary to placent al aromatase deficiency or Krukenberg tumors. 

By and large, the most important tool for the etiological diagnosis of hirsutism is a detailed clinical history and a complete physical examination. Functional causes almost always show a peripubertal onset and a slow progression over years, a family history of hyperandrogenism is frequent, and signs of virilization, such as clitoromegaly or balding, or of defeminization, such as mammary atrophy, are extremely rare. In contrast, androgen-secreting tumors usually show a sudden onset – rarely coincidental with puberty – and a rapid progression with severe virilization and defeminization, usually accompanying hirsutism.

Moreover, clinical evaluation provides valuable clues to discriminate between functional causes of hirsutism. Oligo- or amenorrhea and infertility may indicate the ovarian dysfunction associated with PCOS. Because this disorder is frequently associated with insulin resistance, its diagnosis is also suggested by the presence of abdominal obesity or acanthosis nigricans. 

However, certain tests are needed in order to properly ascertain the etiology of hirsutism. It always is my practice to obtain blood samples to measure serum androgens, SHBG, and other hormones to rule out secondary causes of androgen excess, although this practice is debated when dealing with mild cases of hirsutism [1,33]. In my opinion, a correct etiological diagnosis is essential, because of the lifelong consequences of some of the disorders associated with hirsutism, such as polycystic ovary syndrome and nonclassic congenital adrenal hyperplasia. These disorders cannot be reliably ruled out simply because hirsutism is mild and menstrual periods are normal; in as many as 30% of hirsute patients, regular menstrual cycles are actually anovulatory [34], and nonclassic congenital hyperplasia may even be asymptomatic in some women – the so-called “cryptic” cases – who carry one severe allele needing genetic counseling. 

Therefore, I measure serum total testosterone and SHBG during the follicular phase of the menstrual cycle to calculate free testosterone concentrations in every woman with hirsutism and monitor ovulation by measuring luteal phase progesterone concentrations and/or body temperature. In those patients presenting with normal free testosterone concentrations and regular ovulatory menstrual cycles, I obtain an ovarian ultrasound scan to rule out ovulatory PCOS. In addition, I always measure serum thyrotropin, prolactin, and basal 17-hydroxyprogesterone levels, followed by a 1–24 adrenocorticotropic stimulation test when basal 17-hydroxyprogesterone levels are above 1.7 ng/mL (5.1 nmol/L) [31] to definitively rule out 21-hydroxylase deficiency (nonclassic 11β-hydroxylase deficiency is extremely rare in Spain).

In those patients whose clinical evaluation induces me to suspect PCOS, or when this diagnosis is confirmed after the initial evaluation, I order an oral glucose tolerance test for glucose and insulin to establish their glucose tolerance and to obtain a dynamic estimate of their insulin sensitivity. I also obtain a lipid profile, including cholesterol fractions and triglycerides.

In the rare instance that a patient presents with a clinical history suggestive of an androgen-secreting tumor, I start evaluation by ordering an adrenal computed tomography scan and a transvaginal ultrasound of the ovaries, because, in my limited experience with this kind of tumors, imaging, and not serum androgen measurements, is the most effective technique in these cases. In the rare cases when these techniques show negative results, or when in doubt, simultaneous selective venous sampling of adrenals and ovaries may be needed to ascertain the non-functional source of androgen excess [35].

Management of hirsutism

The goals of the correct management of hirsutism are ameliorating hirsutism and reproductive complaints, preventing and/or treating the possible metabolic derangements associated and, if possible, treating the underlying cause. To be truly effective, the management of hirsutism must follow a few, but quite important principles: (i) treatment must be chronic; (ii) the effects of drugs are not evident before 6 to 12 months of administration; (iii) treatment should change depending on the characteristics and expectations of the individual patient; and (iv) treatment must be monitored by an expert [1]. Unfortunately, during the past decade there have been very few, if any, advances in the tools available to the practitioner to accomplish these goals. These tools include cosmetic procedures and drugs.

Cosmetic procedures are essential for the correct management of hirsutism, and may be used alone in mild cases or in combination with drug treatment. These procedures include temporary and “permanent” methods of hair reduction and topical eff ornithine, but I rarely recommend the latter because its effects take weeks to be apparent and its economic cost is high.
Bleaching and temporary methods of hair removal such as shaving, plucking, waxing, or the use of chemical depilatory agents are invaluable tools in the first months of treatment while waiting for drug treatment to be noticed, or may be used even as single procedures in mild cases [1]. The patient must be assured that even shaving does not increase the growth and thickness of hair, a common and incorrect belief among women, because the blunt tip of shaved hair is more visible than the tapered tip of uncut hair. Local discomfort is the common drawback of these procedures.

Among the methods of permanent hair reduction, I favor a competently performed galvanic electrolysis for small areas, such as the face, and laser or intense pulsed light (IPL) photoepilation for larger areas of the body, because, although real scientific evidence is lacking [33], I have the suspicion that galvanic electrolysis actually prevents hair regrowth in treated areas, whereas methods based on thermal destruction of the hair follicle, such as thermolysis or photoepilation, do not. Nevertheless, inexperienced electrolysis may cause considerable local side effects and even scarring, and this possibility should be weighed carefully. In some countries, including Spain, many women consider the presence of terminal hair, even in normal areas like the axilla and the pubic region,
unacceptable. Photoepilation is the only realistic choice for these women.

Drug treatment of hirsutism includes oral contraceptive pills (OCP), antiandrogens, and the 5α-reductase inhibitor finasteride. Insulin sensitizers, on the contrary, are not effective for hair reduction, although they may be useful in hirsute patients with PCOS to ameliorate insulin resistance and improve the metabolic conditions frequently associated with this disorder [36]. However, there is no sound scientific evidence to recommend insulin sensitizers solely for hirsutism [1].

For most hirsute patients, an OCP containing a neutral (gestodene or desogestrel) or antiandrogenic (cyproterone or clomadinone acetate, or drospierone) progestin is the drug of choice. OCPs not only decrease circulating testosterone levels by reversibly suppressing gonadotropin secretion, but also induce a marked increase in SHBG concentrations, thereby decreasing free testosterone concentrations to levels below those observed in healthy women (Figure 1.2). Additional advantages are the regularization of menstrual bleeding in women with menstrual dysfunction, avoiding endometrial hyperplasia, and the safe contraception needed for the combination with antiandrogens.

Drawbacks of these third generation OCPs include their deleterious effects on coagulation, especially among smokers, and an increased risk of non-fatal venous thromboembolism when compared with second generation OCPs containing the androgenic progestin levonorgestrel [38]. Although the increased risk of venous thromboembolism with third generation OCPs is small, it must be noted that even older formulations containing androgenic progestins may also ameliorate hirsutism [39]. However, these older OCPs may be less effective on hirsutism and might increase body mass index, compared with OCPs containing neutral progestins [40]. Therefore, the choice of an OCP for the treatment of hirsutism must carefully balance the greater efficacy of third generation OCPs against the safer coagulation profile of second generation OCPs, especially in adolescents, hypertensive women, and smokers.

Because of my clinical and research experience [37,41–44], I am particularly fond of a 2 mg cyproterone acetate plus 35 μg ethinylestradiol combination (CPA + EE) that has been available for decades in Europe, and I am still to be convinced that newer formulations containing drospierone are of similar efficacy. The CPA + EE OCP ameliorates hirsutism and suppresses hyperandrogenemia (Figure 1.2), regularizes menstrual bleeding, and has an overall safe metabolic profile in hyperandrogenic women, showing a neutral effect on glucose tolerance and insulin sensitivity and a beneficial effect on the lipid profile consisting of an increase in Apo-A and high-density lipoprotein phospholipids [37]. In women presenting with moderate to severe hirsutism, I add 50 mg of CPA during the first 10 days of each CPA + EE cycle to obtain a faster amelioration of hirsutism. After attaining a satisfactory amelioration of hirsutism, I withdraw the additional CPA and continue maintenance therapy with the CPA + EE pill for years.

Because the CPA + EE combination may induce a small, but significant increase in blood pressure in PCOS patients [41], in hypertensive women I am currently recommending combinations of ethinylestradiol with a neutral progestin plus spironolactone as antiandrogen, with excellent tolerability and clinical results comparable to those of the CPA + EE pill.

Only in those hirsute patients in whom oral estrogens are contraindicated do I favor the use of finasteride over other antiandrogens, and only after ensuring safe non-hormonal contraception to avoid fetal male pseudohermaphroditism in case of unadverted pregnancy. The efficacy of finasteride is similar to that of CPA, spironolactone, and flutamide [45], but lacks the menstrual disturbances associated with CPA and spironolactone when used as single agents [46,47], and the potential for severe hepatotoxicity of flutamide [48].

Treatment has to be monitored at least annually, and must always consider additional measures related to the etiology of hirsutism, such as diet and lifestyle recommendations in women with PCOS or mineralo- and/or glucocorticoid replacement therapy in women with classic congenital adrenal hyperplasia. Of note, quitting smoking must be strongly advised because this habit worsens the undesirable effects of almost all the drugs available for hirsutism and related conditions [43]. Drug treatment for hirsutism may be stopped in patients seeking fertility and reinstated after delivery and lactation.

**Conclusions**

Hirsutism is a frequent complaint in women of fertile age and usually results from functional disorders of androgen excess. The diagnosis of hirsutism requires quantifying the amount and distribution of terminal hair and establishing the most likely underlying etiology. Treatment of hirsutism is effective, but should be chronic and multidisciplinary. Cosmetic measures can be used in every case and, while treatment with an OCP is indicated for most patients, an antiandrogen may be
added in moderate or severe cases. Finally, successful management of hirsutism should also consider the treatment of any reproductive or metabolic comorbidity.

Acknowledgments

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References

Section 1: Managing the basics


