~9 months for these effects to become maximal. Hirsutism results from an interaction between the plasma androgens and the apparent sensitivity of the hair follicle to androgen. The sensitivity of the hair follicle is determined in part by the local metabolism of androgens, particularly by conversion of testosterone to dihydrotestosterone by the enzyme 5α-reductase and subsequent binding of these molecules to the androgen receptor. The hirsutism score does not correlate well with the androgen level (16, 17), apparently because the androgen-dependent pilosebaceous follicle response to androgen varies considerably.

Etiology of hirsutism

The majority of hirsutism is due to androgen excess (≥80%), and the majority of women with hirsutism (70% to 80%) have PCOS (18, 19). PCOS is defined by the presence of a combination of two of three symptoms or findings: otherwise unexplained chronic hyperandrogenism, oligoovulation, and ultrasonographic polycystic ovarian morphology (20). Gonadotropin-dependent functional ovarian hyperandrogenism is the source of the hyperandrogenemia in the majority of PCOS cases (18, 20). This may be accompanied by a related mild adrenocorticotropic hormone–dependent functional adrenal hyperandrogenism, and in a minority of instances this form of adrenal hyperandrogenism may occur in isolation (20, 21). PCOS is frequently associated with a metabolic syndrome that results from insulin resistance and/or central obesity and that requires considerations distinct from those for hirsutism itself. Obesity may worsen or cause features of PCOS (20, 22, 23).

Many women have hirsutism without hyperandrogenemia. We term this "idiopathic hirsutism" in eumenorrheic women who have no other clinical evidence suggesting PCOS or other hyperandrogenic endocrine disorder (19), although some may have polycystic ovary morphology on ultrasound and thus meet a Rotterdam criterion for "ovulatory PCOS" (24). Idiopathic hirsutism constitutes 5% to 20% of hirsute women (19, 24). Available data suggest that among eumenorrheic women with mild hirsutism (a Ferriman–Gallwey hirsutism score of 8 to 15 in the United States), approximately half have idiopathic hirsutism (16). However, the percentage of women with idiopathic hirsutism who meet Rotterdam criteria for "ovulatory PCOS" remains unclear, as studies using transvaginal ultrasound and/or high-quality androgen assay technologies in this population have not yet been performed.

It is unclear whether idiopathic hirsutism is due to altered androgen mechanism of action within the hair follicle (referred to as cutaneous hyperandrogenism) or to other alterations in hair biology (15, 17, 25). The routine assay of androgenic steroids other than testosterone has proven to be of little further diagnostic utility in most, but not all, populations (16, 26–30) (Section 5, Androgen Testing Remarks). Serum total testosterone is similar to and correlates well with serum androgenic bioactivity in young women with and without PCOS ($r = 0.7$ to 0.8) (31). Thus, hirsutism cannot currently be considered synonymous with "clinical evidence of hyperandrogenism" if serum total and free testosterone are normal. However, the possibility exists that previously unsuspected circulating androgens contribute to idiopathic hirsutism (21, 30, 32) (Section 5, Androgen Testing Remarks).

Further workup of the eumenorrheic patient for mild hirsutism and a normal serum total testosterone is only clinically indicated if there is other clinical evidence to suggest the etiology is a hyperandrogenic endocrine disorder.

As noted, among women with mild hirsutism, approximately half have idiopathic hirsutism. Plasma total and/or free testosterone levels are elevated in the remainder of cases of mild hirsutism and in most cases of more severe hirsutism (16, 26, 27) (Section 5, Androgen Testing Remarks). Most women with a twofold or greater elevation of serum androgen levels have some degree of hirsutism or an alternative pilosebaceous response, such as excessive acne vulgaris, seborrhea, or female- or male-pattern alopecia.

Other causes of androgen overproduction are infrequent (24, 26). NCCAH, the most common of these disorders, is present in 4.2% of hyperandrogenic women worldwide, although specific ethnic groups are at lower or higher risk (Section 5, Androgen Testing Remarks) (33). Androgen-secreting tumors are present in ~0.2% of hyperandrogenic women; over half are malignant (34). Clinicians must consider Cushing syndrome, acromegaly, hypothyroidism, and (rarely) hyperprolactinemia in the differential diagnosis of hirsutism, but patients typically will present with the features specific to these disorders. Clinicians must consider topical androgen use by a partner (35), exogenous androgens or anabolic steroids (36), or valproic acid when evaluating patients with hirsutism.

1.0 Diagnosis of Hirsutism

1.1. We suggest testing for elevated androgen levels in all women with an abnormal hirsutism score (2 |⊕⊕OO). In those cases where serum total testosterone levels are normal, if sexual hair growth is moderate/severe or sexual hair growth is mild but there is clinical evidence of a hyperandrogenic endocrine disorder (such as menstrual disturbance or progression in spite of therapy), we suggest measuring an early morning serum total and free testosterone by a reliable specialty assay. (2 |⊕⊕OO)