PIPADOR REVISION

MRCOG Part 1 Revision Notes

Androgens

ENDOCRINOLOGY • LECTURE 172

VISUAL EXAMINATION REVISION NOTES

2026 Edition

MRCOG PART 1 REVISION NOTES | ENDOCRINOLOGY

172 Androgens

Sources, transport and actions; hirsutism and virilisation; androgens in pregnancy; anti-androgen treatment

≈ 1–2%	≈ 50%	> 95%	< ≈ 5
Free testosterone	Women's testosterone from peripheral conversion	DHEAS from adrenal	Free androgen index (women)

■ KEY FACTS: Learning objectives

- Describe the androgens, their sources in women and men, transport (SHBG) and metabolism.
- Describe the androgen receptor and the actions of testosterone and DHT.
- Describe the evaluation of hirsutism and virilisation and causes of hyperandrogenism, including in pregnancy.
- Describe anti-androgens and testosterone therapy in women.

172.1 Androgens and their sources

Androgen	Potency	Notes
DHT	Most potent (≈ 2–3× T)	From T by 5α-reductase in target tissues (skin, hair follicle, prostate, external genitalia)
Testosterone	High	Men: Leydig cells (≈ 95%); women: ovary ≈ 25%, adrenal ≈ 25%, peripheral conversion ≈ 50%
Androstenedione	Weak (pro-hormone)	Ovary and adrenal ≈ 50:50; substrate for aromatase (oestrone) and 17β-HSD (testosterone)
DHEA / DHEAS	Very weak	Adrenal zona reticularis — DHEAS is a marker of adrenal androgen; falls with age

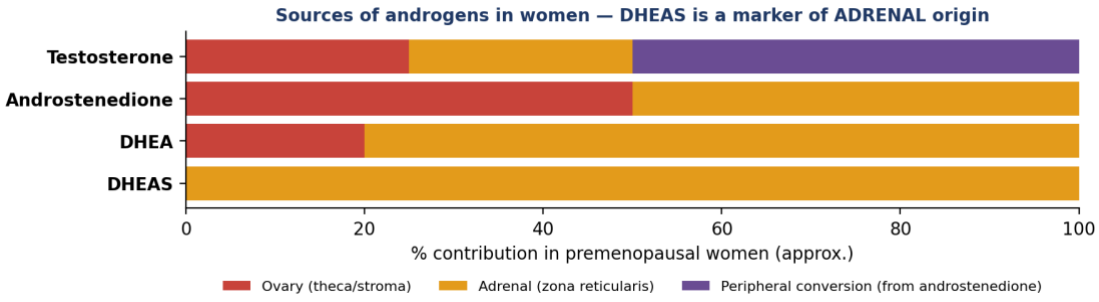


Figure 172.1 — Sources of androgens in women

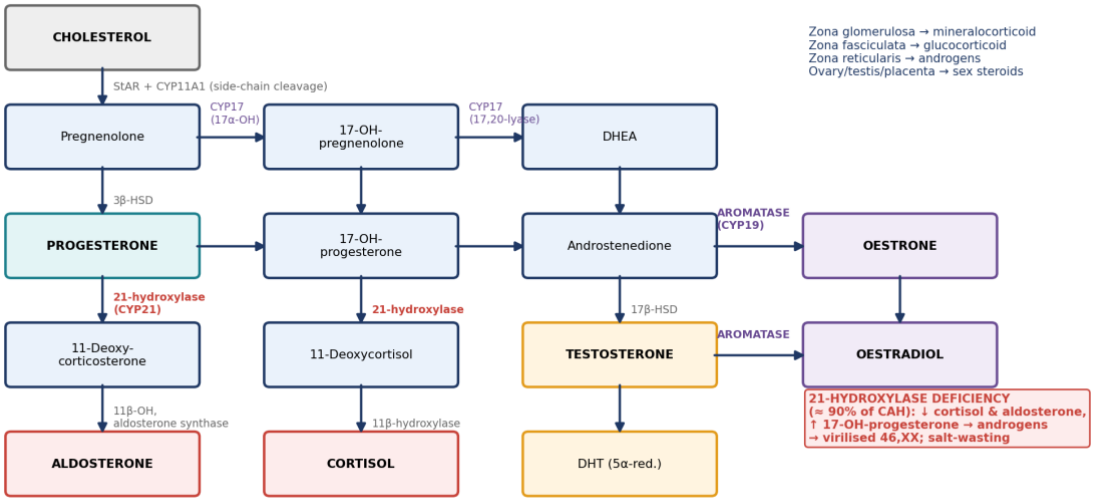


Figure 172.2 — Steroidogenic pathways (androgens are intermediates in oestrogen synthesis)

172.2 Transport, receptor and actions

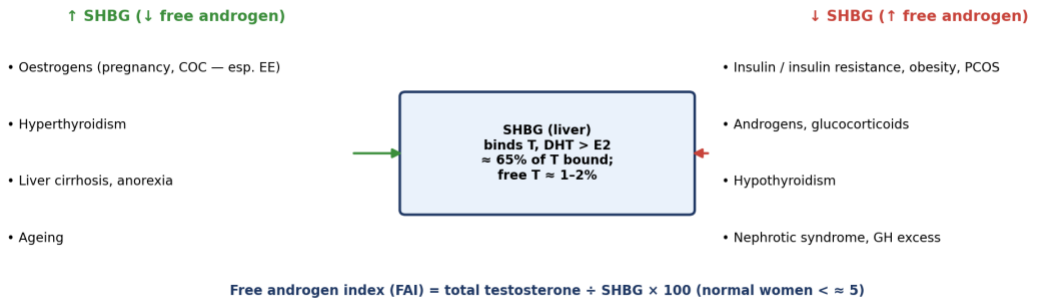


Figure 172.3 — Regulation of SHBG

- Testosterone is bound to **SHBG** (≈ 65%, high affinity) and albumin (≈ 33%, low affinity); free ≈ 1–2%. 'Bioavailable' = free + albumin-bound.
- **Androgen receptor** (nuclear; gene on the **X chromosome**) — mutations cause androgen insensitivity (Lecture 150).
- Metabolism: aromatase → oestrogens; 5α-reductase → DHT; hepatic conjugation → urinary 17-ketosteroids.

Actions in women	Actions in men
Pubic/axillary hair (adrenarche), sebum, libido, muscle and bone mass; precursor for oestradiol	Wolffian duct development (fetus), external genitalia (DHT), spermatogenesis, secondary sexual characteristics, muscle, bone, erythropoiesis (↑ Hb), libido

172.3 Hyperandrogenism

Sign	Features
Hirsutism	Terminal hair in male pattern; modified Ferriman–Gallwey score (≥ 4–8 depending on ethnicity); often with acne, androgenic alopecia
Virilisation (red flag)	Clitoromegaly, deepening voice, temporal balding, ↑ muscle mass, breast atrophy, rapid onset — suggests androgen-secreting tumour or severe CAH

Cause	Clues / investigation
PCOS (≈ 70–80%)	Oligo-ovulation, PCO morphology/AMH; mildly ↑ testosterone, ↓ SHBG

Cause	Clues / investigation
Idiopathic hirsutism	Regular ovulatory cycles, normal androgens
Non-classic CAH (21-hydroxylase)	↑ 17-OH-progesterone (follicular phase; Synacthen test)
Androgen-secreting tumours	Ovary (Sertoli–Leydig), adrenal carcinoma: testosterone > ≈ 5 nmol/L or ↑↑ DHEAS (adrenal) — imaging
Cushing's syndrome	Cushingoid features — dexamethasone suppression / 24-h urinary cortisol
Hyperprolactinaemia, acromegaly, hypothyroidism	Prolactin, IGF-1, TSH
Drugs	Testosterone, danazol, anabolic steroids, valproate
Postmenopausal ovarian hyperthecosis	Severe hyperandrogenism, ↑ testosterone; bilateral enlarged ovaries

172.4 Androgens in pregnancy and the fetus

- Maternal **total testosterone ↑** (↑ SHBG; ↑ ovarian/placental precursors) but free testosterone remains low; **placental aromatase** converts maternal and fetal androgens to oestrogens — **protects a female fetus** from virilisation.
- Maternal virilisation in pregnancy: **luteoma of pregnancy** (solid, regresses postpartum; maternal virilisation in ≈ 25–35%, and a female fetus may be virilised when the mother is), **hyperreactio luteinalis** (bilateral theca-lutein cysts with high hCG — molar pregnancy, multiples), aromatase deficiency (placental), androgen-secreting tumours.
- **Fetal sexual differentiation**: placental hCG drives fetal Leydig testosterone at **8–12 weeks** → Wolffian ducts; DHT → external genitalia (Lecture 150). **21-hydroxylase deficiency** virilises a 46,XX fetus.

172.5 Treatment

Agent	Mechanism	Notes
COC	↓ LH-driven ovarian androgens; ↑ SHBG (EE)	First line for hirsutism/acne in PCOS; ≥ 6 months for hair
Spironolactone	Androgen receptor antagonist; ↓ 5α-reductase	Hyperkalaemia; teratogenic (feminises male fetus) — contraception required
Cyproterone acetate	AR antagonist + progestogen	Co-cyprindiol (VTE); hepatotoxicity at high doses
Finasteride	5α-reductase inhibitor	Teratogenic — contraception
Eflornithine cream; laser/electrolysis	Local hair growth inhibition / removal	Adjuncts
Testosterone for women	Transdermal (off-label in UK)	Hypoactive sexual desire disorder in postmenopausal women when HRT alone ineffective (NICE NG23); monitor levels

172.6 Must-know summary

■ **EXAM TIP**

- In women: T = 25% ovary, 25% adrenal, 50% peripheral; **DHEAS = adrenal marker**.
- SHBG binds T > E2; **↑ by oestrogen/thyroid**, **↓ by insulin/obesity/androgens** — FAI = T/SHBG × 100.
- DHT (5α-reductase) is most potent; androgen receptor gene on X.
- Virilisation or T > 5 nmol/L → exclude **tumour**; ↑ 17-OH-progesterone → NCCAH.
- Placental aromatase protects a female fetus; anti-androgens are **teratogenic**.

■ MNEMONIC / MEMORY AID

Virilisation — 'Deep voice, Big clitoris, Bald temples' = tumour until proven otherwise.