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From PCOS to PMOS: Why the Name Change?

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Reproductive Endocrinology and Infertility
Laparoscopy and Hysteroscopy

170 Million Women Affected · 1 in 8 · 70% Undiagnosed · Based on: Teede HJ et al., The Lancet, 2026

Helena J Teede, Mahnaz Bahri Khomami*, Rachel Morman*, Joop S E Laven, Anju E Joham, Michael F Costello, Madhuri Patil, D Aled Rees, Lorna Berry, Melanie G Cree, Han Zhao, Robert J Norman*, Anuja Dokras*, Terhi Piltonen*, on behalf of the Global Name Change Consortium† *The Lancet. 2026. DOI: 10.1016/S0140-6736(26)00717-8*

Learning Objectives

1	**Understand**	why PCOS is being renamed and the global consensus process that produced PMOS
2	**Describe**	the multisystem pathophysiology — polyendocrine, metabolic, and ovarian dysfunction
3	**Apply**	the 2023 International Evidence-Based Diagnostic Criteria for PMOS in clinical practice
4	**Implement**	evidence-based lifestyle, pharmacological, and reproductive management strategies
5	**Appreciate**	the psychological burden and holistic care approach for women with PMOS

Source: Teede HJ et al. The Lancet. 2026; Teede HJ et al. Eur J Endocrinol. 2023

The Hidden Epidemic

170M

Women affected globally during reproductive years

1 in 8

Women affected — the most common endocrine disorder

~70%

Remain UNDIAGNOSED at any given time

2–3×

Average years delay to diagnosis after symptom onset

Source: Neven ACH et al. Hum Reprod Update. 2026; Gibson-Helm M et al. J Clin Endocrinol Metab. 2017

SECTION 1 · THE PROBLEM WITH PCOS

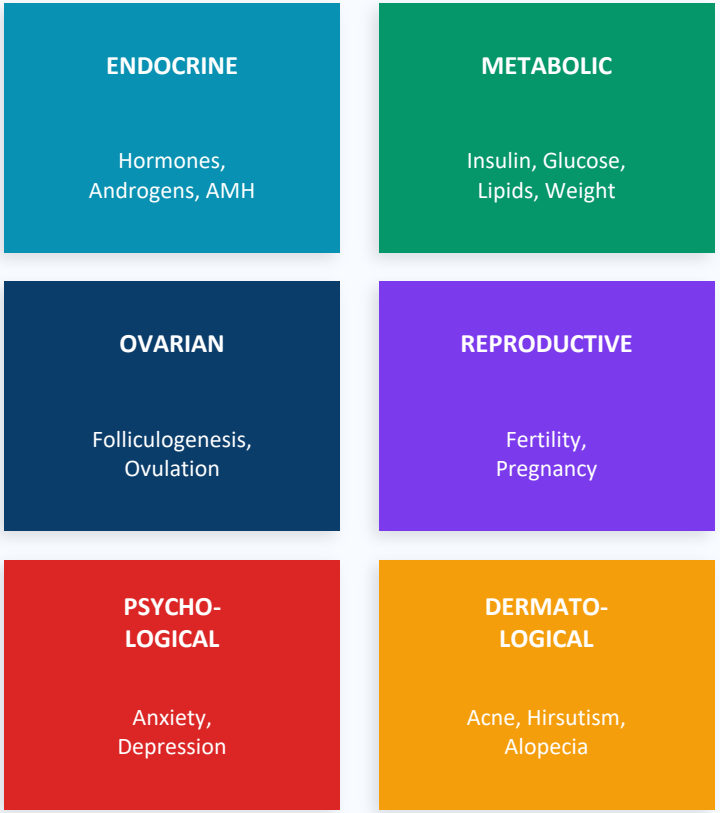
Why the Name Had to Change

Inaccuracy · Stigma · Fragmented Care · Missed Diagnoses

What Is Polyendocrine Metabolic Ovarian Syndrome?

PMOS (formerly PCOS) is a complex multisystem endocrine disorder characterized by:

1. **Oligoanovulation:** Irregular or absent ovulation & menstrual cycles
2. **Hyperandrogenism:** Excess androgens — clinical or biochemical
3. **Ovarian/AMH changes:** Polycystic morphology on USS or elevated AMH
4. **Insulin Resistance:** Present in ~85% — drives metabolic features
5. **Metabolic Dysfunction:** Dyslipidaemia, dysglycaemia, central obesity



Source: Stener-Victorin E et al. Nat Rev Dis Primers. 2024;10:27; Teede HJ et al. Eur J Endocrinol. 2023

The Misnomer: 'Polycystic' Ovary Syndrome

X What the Name Implies

- Pathological ovarian cysts are present
- A purely gynaecological/ovarian disorder
- Cysts are the primary pathological feature
- Condition resolves after menopause
- Fertility is the only concern

OK The Scientific Reality

+ Small antral follicles accumulate — NOT cysts
+ Multisystem polyendocrine disorder
+ Hyperandrogenism & insulin resistance are central
+ Features persist lifelong including post-menopause
+ Affects metabolism, psychology, cardiovascular health

Source: Piltonen T et al. JAMA Intern Med. 2026; Norman RJ et al. Fertil Steril. 2023;120:249–250

A Long History of Inaccuracy & Advocacy for Change

1935 — Stein & Leventhal describe syndrome of amenorrhea + enlarged ovaries

1990 — NIH Consensus: hyperandrogenism + oligomenorrhea — 'PCOS' entrenched

2003 — Rotterdam Criteria expand diagnosis to 3 phenotypes including polycystic morphology

2012 — NIH Workshop: recommends renaming PCOS to reflect its systemic nature

2023 — International Guideline: AMH added to criteria; name change mandate established

2026 — PMOS officially adopted: global consensus of 14,360 survey responses

Source: Azziz R. J Clin Endocrinol Metab. 2014; Dunaif A, Fauser BCJM. J Clin Endocrinol Metab. 2013; NIH Workshop Report. 2012

Patient Impact: Delayed Diagnosis & Knowledge Gaps

70%

of affected individuals remain UNDIAGNOSED

2–3 yrs

average diagnostic delay after seeking care

≥3

clinicians seen before correct diagnosis on average

84%

of patients endorse a name change in surveys

Source: Gibson-Helm M et al. J Clin Endocrinol Metab. 2017;102:604–612; Teede HJ et al. EClinicalMedicine. 2025

Stigma, Distress & Psychosocial Harm from the Name

The name 'polycystic ovary syndrome' causes measurable harm beyond its inaccuracy:

Fertility Stigma:	Overemphasis on 'ovary' and 'reproductive' features reinforces stigma in cultures where fertility is tied to a woman's social value and identity
Emotional Distress:	Many women report the diagnosis itself causes distress — the word 'cyst' implies serious pathology and generates fear disproportionate to clinical reality
Clinician Confusion:	Clinicians outside gynaecology often overlook PMOS due to its 'ovarian' framing — delaying metabolic, cardiovascular, and psychological care
Policy & Funding Gaps:	Reproductive-centric naming limits research funding, advocacy reach, and policy recognition of the condition's broad health burden
Cultural Harm:	Reproductive terminology carries different stigma in diverse cultures; translation often amplifies harmful implications

Source: Teede HJ et al. EClinicalMedicine. 2025;84:103287; Norman RJ et al. Fertil Steril. 2023

SECTION 2 · GLOBAL CONSENSUS PROCESS

From PCOS to PMOS

A Rigorous, Transparent, Patient-Led Renaming Initiative

The Global Consensus Process: An Unprecedented Initiative

1 Funding & Governance

NHMRC Australia funded; international steering group; 56 partner organisations

2 Delphi Survey A

14,360 responses (10,411 patients + 3,949 HCPs); 5 languages; 185 countries

3 Workshop A

Nov 2025: 90 attendees from all world regions; breakout groups; live voting

4 Marketing Analysis

Global branding agency + AI tools; evolutionary vs revolutionary rebranding assessed

5 Delphi Survey B

Jan 2026: 1,346 responses; refined term selection; ovarian vs ovulatory debate resolved

6 Workshop B & Final Name

Feb 2026: PMOS agreed by consensus; 8-stage implementation strategy launched

Source: Teede HJ et al. The Lancet. 2026. DOI: 10.1016/S0140-6736(26)00717-8

Survey Reach: A Truly Global Process

14,360	Total survey responses
10,411	People living with PCOS/PMOS
3,949	Multidisciplinary health professionals
56	Partner organisations across world regions
5	Languages: English, Chinese, German, Persian, Malaysian
90	Workshop attendees (Nov 2025 + Feb 2026)

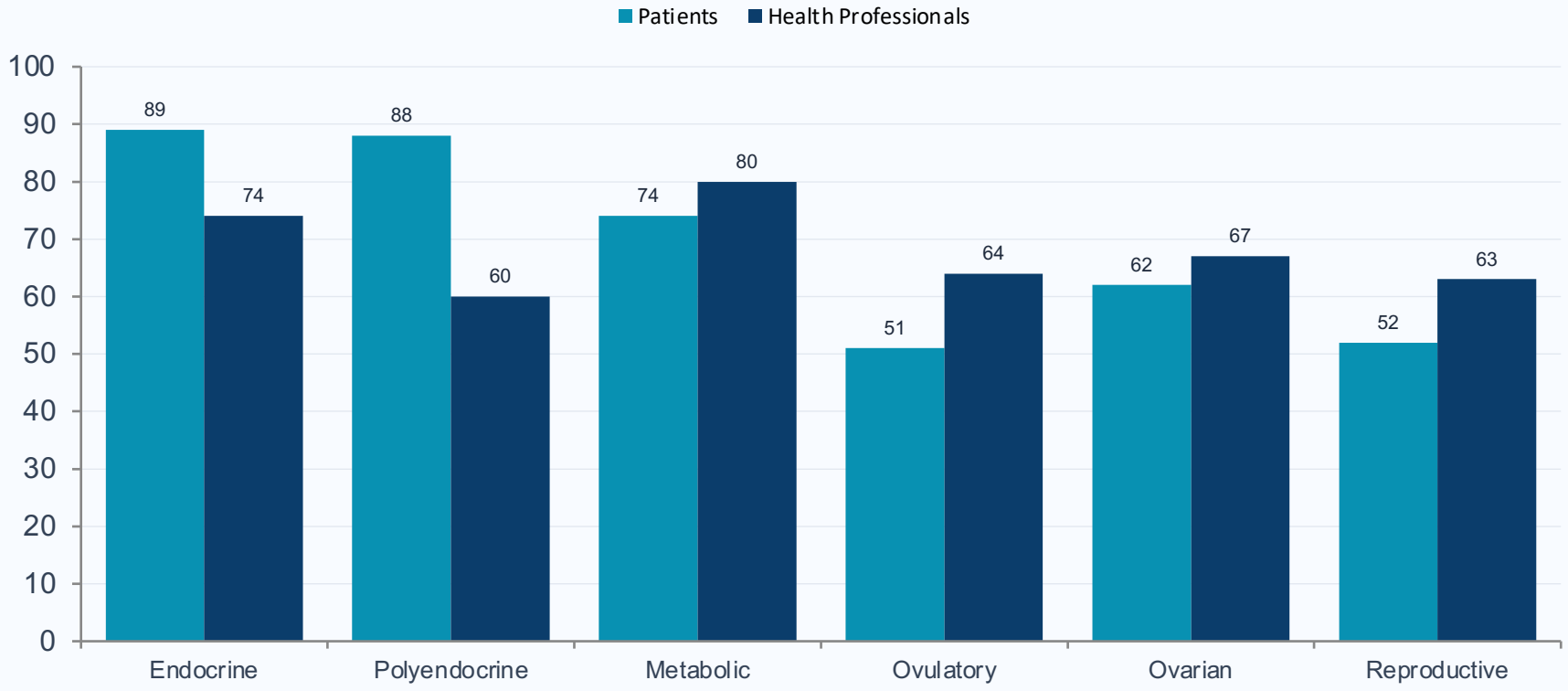
Source: Teede HJ et al. The Lancet. 2026. Supplementary data. 56 organisations, all world regions represented

Guiding Principles: What the New Name Must Achieve

		Support %
1	**Scientific Accuracy** Must reflect true pathophysiology — polyendocrine + metabolic + ovarian — and REMOVE the misleading 'polycystic cyst' implication	86% HCPs / 60% patients
2	**Clarity & Communication** Readily understood by patients, clinicians, researchers, and policy makers globally	85% HCPs / 62% patients
3	**Avoidance of Stigma** Terms linked to reproduction or fertility should be avoided to prevent social harm in diverse cultural contexts	85% HCPs / 66% patients
4	**Cultural Appropriateness** Acceptable and interpretable across cultural, linguistic, and regional contexts worldwide	80% HCPs / 53% patients
5	**Implementation Feasibility** Must allow practical transition across clinical, research, and policy environments	–

Source: Teede HJ et al. The Lancet. 2026. Table 3 — Naming principles endorsed in surveys and workshops

Consensus Voting: How the Key Terms Were Selected



Source: Teede HJ et al. The Lancet. 2026. Table 3 — Survey A support % for key terms (patients n=9,358; HCPs n=3,656)

GLOBAL CONSENSUS · FEBRUARY 2026

The New Name Is:

Polyendocrine Metabolic Ovarian Syndrome

PMOS

66% Survey B support · Workshop B: unanimous (98% of attendees)

"The condition formerly known as PCOS now has a new name: polyendocrine metabolic ovarian syndrome." — Teede HJ et al., The Lancet, 2026

Decoding PMOS: What Each Word Reflects

POLY-ENDOCRINE

- Multiple interacting endocrine abnormalities — NOT an isolated ovarian disorder
- Insulin, androgens, LH, FSH, AMH, adipokines — all dysregulated
- Polygenic origins across neuroendocrine, metabolic & reproductive pathways

METABOLIC

- Insulin resistance in ~85% of women with PMOS
- Obesity, dysglycaemia, dyslipidaemia, MASLD, T2DM all inherent features
- OR 1.68 for composite CVD vs. women without PMOS

OVARIAN SYNDROME

- Ovarian dysfunction is a defining feature — folliculogenesis impaired
- Elevated AMH from disordered follicular maturation
- Menstrual irregularity, anovulation, infertility all captured

Source: Teede HJ et al. The Lancet. 2026; Stener-Victorin E et al. Nat Rev Dis Primers. 2024;10:27

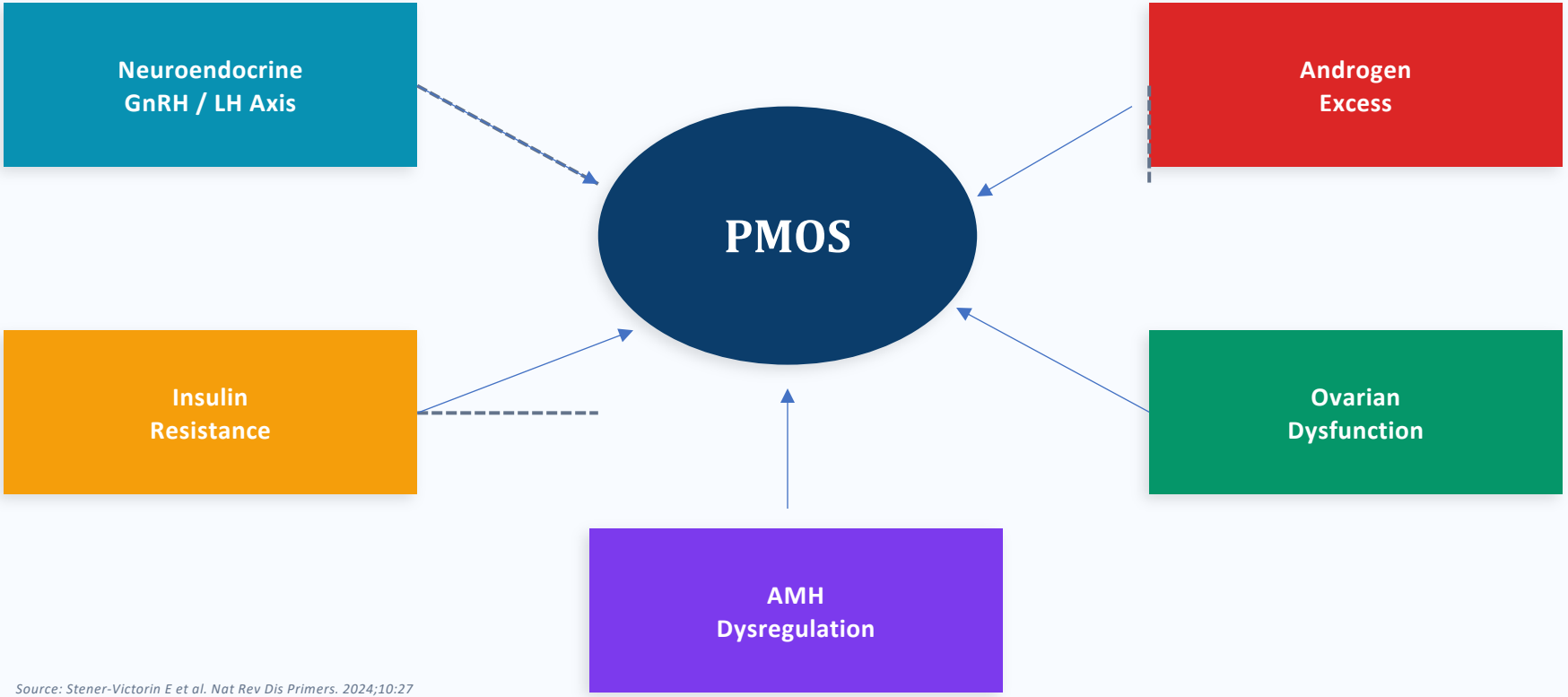
SECTION 3 · PATHOPHYSIOLOGY

Understanding PMOS at the Molecular Level

Polyendocrine · Insulin Resistance · Ovarian Dysfunction

The Polyendocrine Model: Multiple Interacting Systems

PMOS is driven by overlapping dysregulation across at least five endocrine axes:



Source: Stener-Victorin E et al. Nat Rev Dis Primers. 2024;10:27

Genetic Origins: A Complex Polygenic Disorder

Polygenic Architecture

- GWAS identify loci across neuroendocrine, metabolic, and reproductive pathways
- Multi-ancestry GWAS (2025): largest study to date — 57 risk loci identified
- Key genes: LHCGR, THADA, DENND1A, YAP1, RAB5B, HMGA2, TOX3
- Variants in neuroendocrine (FSHB), metabolic (RAB5B), and androgen (CYP11A1) pathways
- Gene-environment interaction: obesity amplifies polygenic risk expression

Neuroendocrine Pathway

FSHB, LHCGR
GnRH pulsatility

Metabolic Pathway

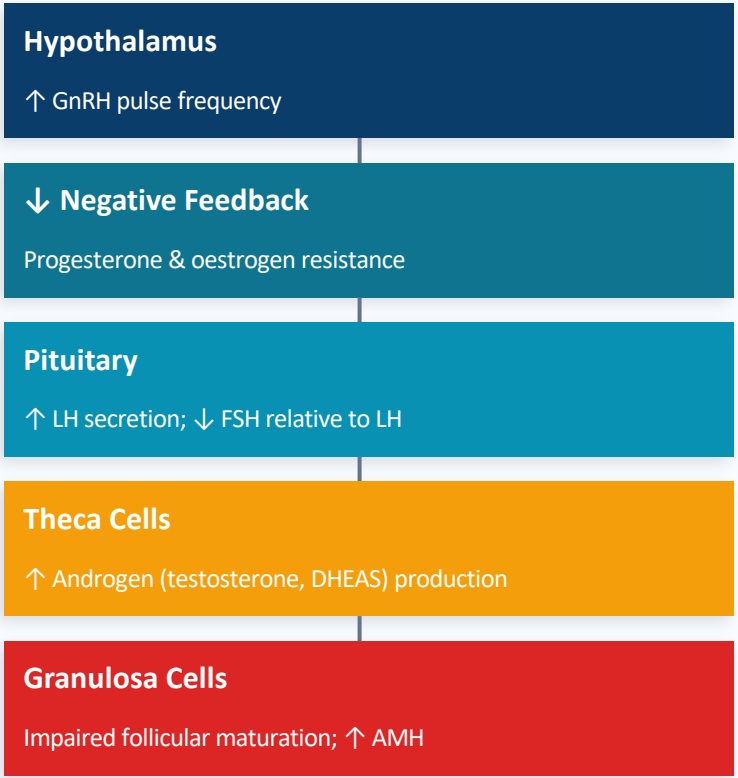
THADA, HMGA2
Insulin signalling

Reproductive Pathway

DENND1A, YAP1
Folliculogenesis

Source: Zhao H et al. Nat Genet. 2025;57:2669–2681; Hiam D et al. J Clin Med. 2019;8:1606

Neuroendocrine Dysfunction: The GnRH–LH Axis



Clinical Consequences

- Elevated LH:FSH ratio (>2:1) — hallmark finding on testing
- Excess androgen → hirsutism, acne, androgenic alopecia
- Arrested follicular development → anovulation & infertility
- Elevated AMH (>3.4 ng/mL) from excess small antral follicles
- Irregular or absent menstrual cycles (oligomenorrhea/amenorrhea)
- Deficient oestrogen-negative feedback → self-perpetuating cycle

Source: Stener-Victorin E et al. Nat Rev Dis Primers. 2024;10:27; Azziz R et al. Nat Rev Dis Primers. 2016;2:16057

Hyperandrogenism: The Defining Endocrine Feature

OVARIAN SOURCE

- Theca cells: ↑ LH-driven androgen synthesis
- ↑ 17α-hydroxyprogesterone & testosterone
- Amplified by hyperinsulinaemia
- Contributes 60–70% of excess androgens in PMOS
- Dexamethasone suppression does NOT fully suppress

ADRENAL SOURCE

- ↑ DHEA-S in ~25–50% of women with PMOS
- Adrenal androgen excess overlaps with PMOS phenotype
- Amplified by cortisol–insulin interaction
- Differentiate from non-classical CAH (17-OHP)
- ACTH stimulation test if NC-CAH suspected

Source: Bizuneh AD et al. Eur J Endocrinol. 2025;192:228–239; Azziz R et al. Nat Rev Dis Primers. 2016;2:16057

Insulin Resistance: The Central Metabolic Pathogen

Insulin resistance affects ~85% of women with PMOS

75% of LEAN women (BMI ≤25 kg/m²)

Insulin Resistance

Compensatory Hyperinsulinaemia

↑ Androgen Synthesis

↑ Adipokine Dysregulation

Impaired glucose tolerance → T2DM (RR 4× vs controls)

Amplified LH-driven theca cell androgen production

↑ Free androgens (suppresses SHBG synthesis in liver)

Disrupted granulosa cell function → impaired folliculogenesis

Source: Cassar S et al. Hum Reprod. 2016;31:2619–2631; Stepto NK et al. Hum Reprod. 2013;28:777–784

Ovarian Dysfunction: Impaired Folliculogenesis

NORMAL FOLLICULOGENESIS

✓ GnRH pulse → balanced FSH:LH

✓ FSH recruits cohort of ~5–7 follicles

✓ Dominant follicle selected → ovulation

✓ Normal oestrogen progression

✓ AMH within reference range

✓ Corpus luteum formed → progesterone

PMOS FOLLICULOGENESIS

✗ ↑ LH → excess androgen from theca cells

✗ Arrested follicular maturation at 5–9mm

✗ ≥20 follicles/ovary accumulate (USS)

✗ ↑ AMH from >30 small antral follicles

✗ No dominant follicle selected → anovulation

✗ No corpus luteum → progesterone deficiency

Source: Pea J et al. Hum Reprod Update. 2024;30:109–130; Stener-Victorin E et al. Nat Rev Dis Primers. 2024;10:27

Anti-Müllerian Hormone: Biomarker & Pathogenic Role

AMH is now included in the 2023 adult diagnostic criteria for PMOS

Elevated in PMOS

AMH >3.4–4.0 ng/mL (lab-specific thresholds) indicates polycystic ovarian morphology. Correlates with follicle number and androgen levels.

Pathophysiology

↑ Small antral follicles each secrete AMH → aggregate level elevated. AMH suppresses FSH sensitivity, further impairing follicular selection.

Clinical Utility

Can replace ultrasound for diagnosis in adolescents and women on OCP. Correlates with androgen levels and disease severity.

Metabolic Correlation

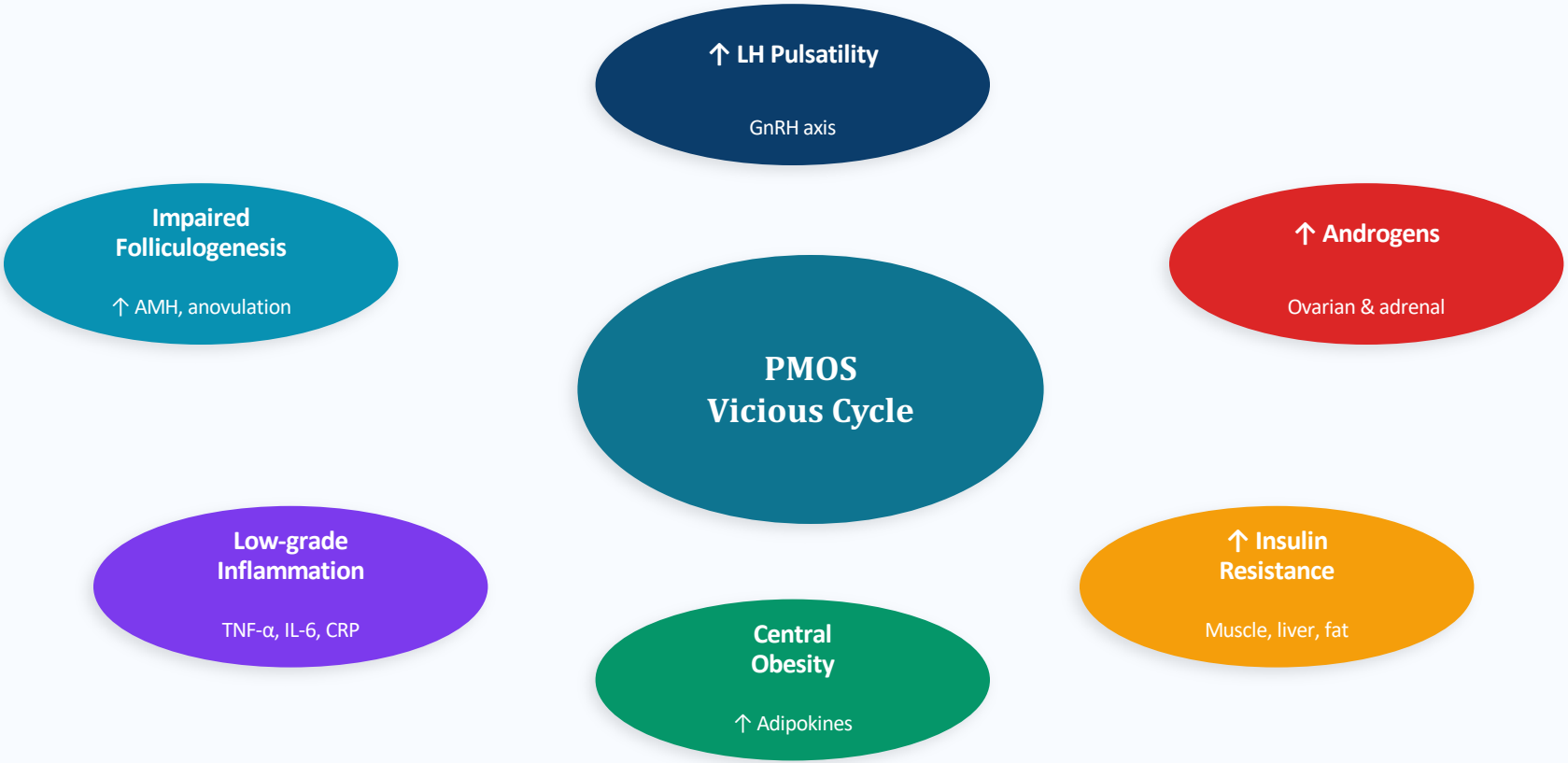
AMH levels correlate with insulin resistance and androgen excess — supporting polyendocrine model.

Limitations

Assay variability between labs (Gen II vs Elecsys). Levels decline with age. Not diagnostic alone — clinical context essential.

Source: van der Ham K et al. Fertil Steril. 2024;122:727–739; Teede HJ et al. Eur J Endocrinol. 2023

The Metabolic–Endocrine Interplay: A Vicious Cycle



Source: Shorakae S et al. Clin Endocrinol. 2018;89:628–633; Stener-Victorin E et al. Nat Rev Dis Primers. 2024

Pathophysiology Summary: Why 'Polyendocrine'?

Neuroendocrine

- ↑ GnRH pulsatility → ↑ LH
- Reduced progesterone & oestrogen feedback
- Self-perpetuating hypothalamic dysregulation

Androgenic

- ↑ Theca cell androgens (LH-driven)
- ↑ Adrenal DHEAS in ~30–50%
- ↓ SHBG from hyperinsulinaemia → ↑ free T

Metabolic

- Insulin resistance in 85%
- ↑ Risk T2DM, MASLD, CVD
- Causal role of obesity (Mendelian randomisation)

Ovarian

- Arrested folliculogenesis
- ↑ AMH from 30+ small antral follicles
- Anovulation → infertility

Inflammatory

- Low-grade chronic inflammation
- ↑ CRP, TNF-α, IL-6
- Worsens insulin signalling & adiposity

Adipokine

- ↓ Adiponectin (insulin-sensitising)
- ↑ Leptin resistance
- Gut-hormone dysregulation

Source: Stener-Victorin E et al. Nat Rev Dis Primers. 2024;10:27; Joham AE et al. Lancet Diabetes Endocrinol. 2022

SECTION 4 · CLINICAL FEATURES

The Multisystem Clinical Spectrum

Reproductive · Metabolic · Dermatological · Psychological

Multisystem Clinical Manifestations of PMOS

REPRODUCTIVE

- Oligomenorrhea / amenorrhea
- Chronic anovulation
- Subfertility / infertility
- Recurrent pregnancy loss
- Gestational diabetes
- Pre-eclampsia

METABOLIC

- Insulin resistance / T2DM
- Central obesity
- Dyslipidaemia
- MASLD (fatty liver)
- Hypertension
- Sleep apnoea

DERMATOLOGICAL

- Hirsutism (70% of cases)
- Acne vulgaris
- Androgenic alopecia
- Acanthosis nigricans
- Seborrhoea

PSYCHOLOGICAL

- Depression (3× risk)
- Anxiety (5× risk)
- Eating disorders
- Poor quality of life
- Body image distress
- Sexual dysfunction

CARDIOVASCULAR

- ↑ CVD risk (OR 1.68)
- Endothelial dysfunction
- Subclinical atherosclerosis
- Vascular stiffness

ENDOMETRIAL

- ↑ Endometrial cancer risk
- Endometrial hyperplasia
- Anovulation → oestrogen excess without progesterone
- Annual USS if amenorrhoea >3 months

Source: Teede HJ et al. Eur J Endocrinol. 2023; Stener-Victorin E et al. Nat Rev Dis Primers. 2024;10:27

Reproductive Features: Menstrual, Fertility & Pregnancy

Menstrual Dysfunction

- Oligomenorrhea: cycles >35 days
- Amenorrhoea: absence >90 days
- <8 cycles per year = pathological
- Results from chronic anovulation
- Not all PMOS = irregular cycles (phenotype D)

Infertility & Fertility

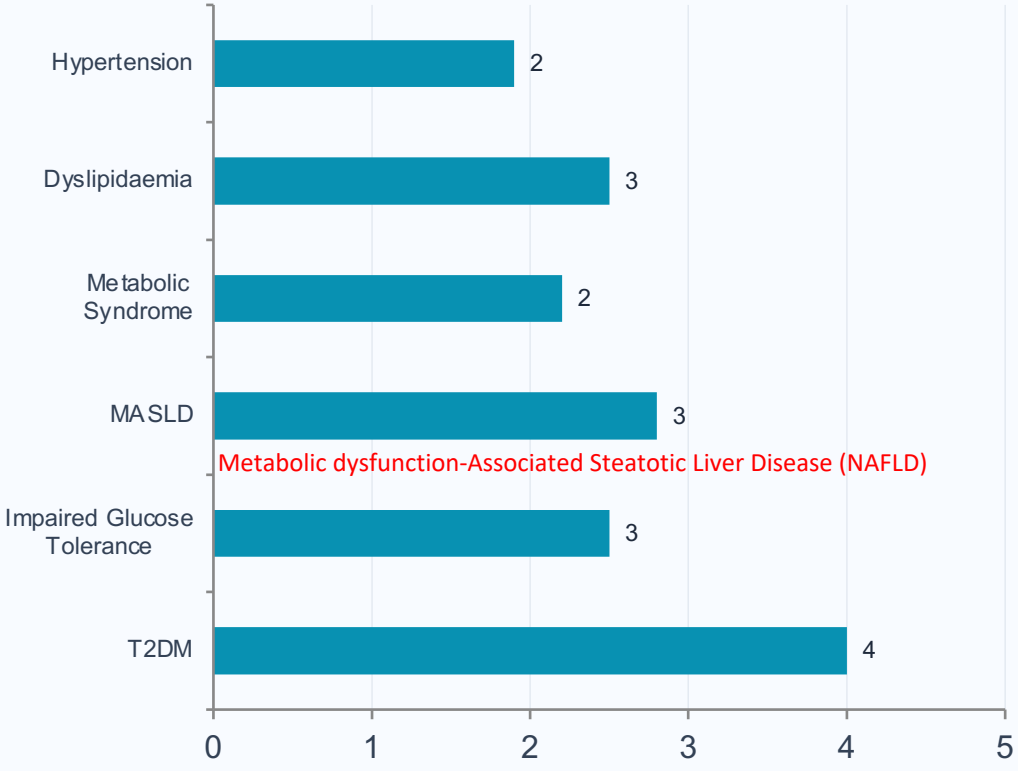
- Most common cause of anovulatory infertility
- 70–80% respond to ovulation induction
- Letrozole = first-line (superior to clomiphene)
- Metformin adjunct improves ovulation rates
- IVF: ↑ OHSS risk — antagonist protocols preferred

Pregnancy Complications

- Gestational diabetes: OR 2.8
- Hypertensive disorders: OR 3.7
- Pre-eclampsia: OR 4.3
- Preterm birth: OR 1.6
- Large for gestational age: OR 2.1
- Miscarriage: OR 1.5

Source: Bahri Khomami M et al. Nat Commun. 2024;15:5591–5592; Teede HJ et al. Eur J Endocrinol. 2023

Metabolic Complications: Inherent Features of PMOS



Key Metabolic Facts

- OR 1.68 for composite CVD vs women without PMOS
- OR 2.50 for myocardial infarction
- OR 1.71 for stroke
- 40% lifetime risk of T2DM or prediabetes
- Obesity exacerbates all features — causal (Mendelian RCT)
- Risk persists post-menopause

Source: Tay CT et al. J Am Heart Assoc. 2024;13:e033572; Moran LJ et al. Hum Reprod Update. 2011;17:138

Dermatological Features: Androgen-Mediated Manifestations

Hirsutism

70%

Mechanism: ↑ Free testosterone acts on pilosebaceous unit; 5α-reductase converts T→DHT in skin

Rx: OCP (cyproterone acetate preferred), spironolactone, eflornithine cream

Acne Vulgaris

15–30%

Mechanism: ↑ Sebaceous gland activity driven by androgens; often resistant to standard topical therapy

Rx: OCP + anti-androgens; isotretinoin for severe cases

Androgenic Alopecia

~10%

Mechanism: DHT-mediated miniaturisation of terminal hair follicles; frontal recession pattern

Rx: Minoxidil + spironolactone; OCPs; low-level laser therapy

Acanthosis Nigricans

Variable

Mechanism: Insulin-driven keratinocyte proliferation via IGF-1; marker of severe insulin resistance

Rx: Treat underlying insulin resistance: metformin, weight loss, GLP-1 agonists

Source: Bizuneh AD et al. Eur J Endocrinol. 2025;192:228–239; Teede HJ et al. Eur J Endocrinol. 2023

Psychological Burden: Often Underestimated, Always Important

3×	Risk of DEPRESSION vs women without PMOS
5×	Risk of ANXIETY vs general population
Elevated	Risk of eating disorders (binge eating, restrictive)
Reduced	Quality of life across all domains (SF-36 scores)

Drivers of Psychological Distress

- Diagnostic delay and uncertainty
- The name itself: 'cysts' causes fear
- Hirsutism, weight changes, acne → body image
- Fertility concerns and reproductive pressure
- Chronic condition with limited cure

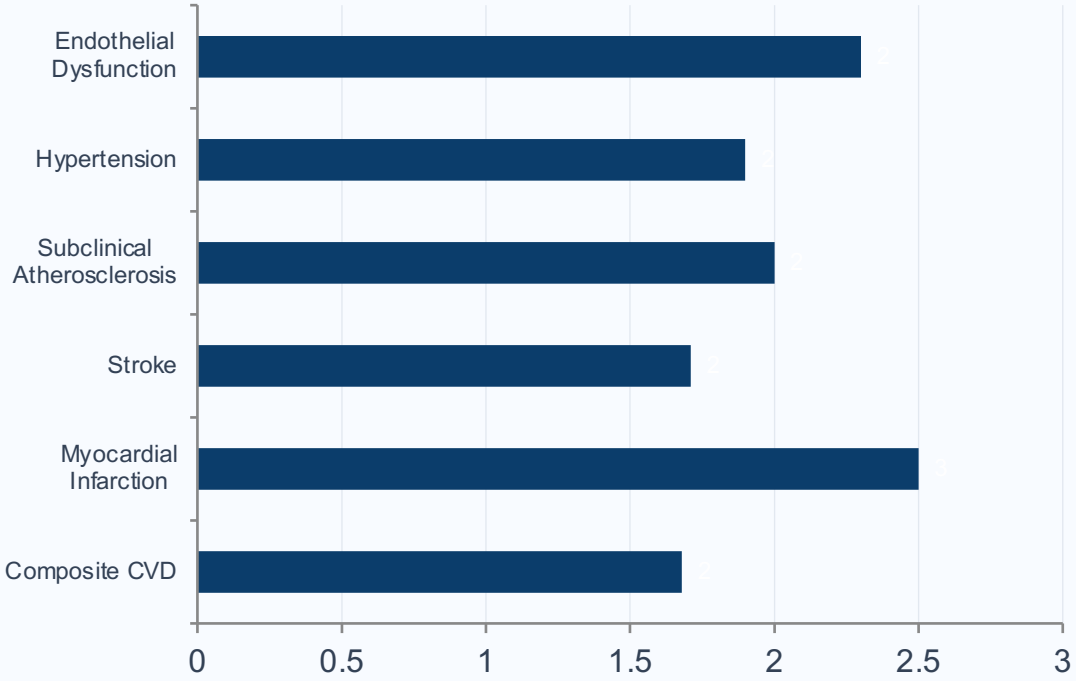
Management Essentials

- Screen ALL patients: PHQ-9, GAD-7
- CBT — strong evidence base
- Mindfulness-based interventions
- Peer support groups and patient communities
- Address PMOS features to reduce distress

Source: Teede HJ et al. Eur J Endocrinol. 2023;189:G43–G64; Stener-Victorin E et al. Nat Rev Dis Primers. 2024

Cardiovascular Risk Profile in PMOS

Evidence from predominantly premenopausal women shows significantly elevated cardiovascular risk:



Key Drivers of CV Risk

- Insulin resistance & T2DM
- Dyslipidaemia (↑ LDL, ↓ HDL)
- Hypertension
- Central obesity (↑ visceral fat)
- Chronic low-grade inflammation
- Endothelial dysfunction
- Obstructive sleep apnoea

Source: Tay CT et al. J Am Heart Assoc. 2024;13:e033572; Thong EP et al. Lancet Diabetes Endocrinol. 2020;8:134–149

SECTION 5 · DIAGNOSIS

Diagnosing PMOS in Clinical Practice

2023 International Evidence-Based Criteria · Investigations · Differential

International Diagnostic Criteria for PMOS (Adults ≥20 yrs)

Diagnosis requires EXCLUSION of other causes PLUS at least 2 of 3 criteria:

1 Oligo-Anovulation

- Cycles <21 or >35 days
- <8 menstrual cycles per year
- Amenorrhoea (>90 days without menstruation)
- Both irregular cycles AND anovulation required

2 Clinical or Biochemical Hyperandrogenism

- Clinical: hirsutism (mFG score ≥4–6), acne, alopecia
- Biochemical: ↑ free testosterone, ↑ FAI, ↑ total testosterone
- SHBG should be measured — low SHBG → elevated free T
- Ethnicity-specific thresholds apply (Ferriman-Gallwey)

3 Polycystic Ovarian Morphology or Elevated AMH

- Ultrasound: ≥20 follicles/ovary OR ovarian volume ≥10 mL
- AMH: ≥3.4–4.0 ng/mL (assay-specific thresholds)
- Either USS OR AMH qualifies — not both required

⚠ **EXCLUDE: Thyroid disease, hyperprolactinaemia, non-classical CAH, Cushing syndrome, androgen-secreting tumour**

Source: Teede HJ et al. Eur J Endocrinol. 2023;189:G43–G64; Pea J et al. Hum Reprod Update. 2024;30:109–130

Diagnosing PMOS in Adolescents (Ages 10–19)

Stricter criteria required — both criteria 1 AND 2 must be met:

Criterion 1 — OLIGO-ANOVULATION

- Persistent oligomenorrhoea ≥2 years post-menarche
- Cycle length <21 or >45 days at Year 1–2; >35 days thereafter
- Primary amenorrhoea >90 days after expected menarche

Criterion 2 — CLINICAL HYPERANDROGENISM

- Persistent hirsutism (mFG score ≥4–6) — NOT peripubertal normal
- Moderate-severe inflammatory acne unresponsive to treatment
- ↑ Free testosterone or FAI (age-specific norms)

Why Stricter Criteria? Key Considerations for Adolescents

- Normal puberty includes transient oligo-anovulation (1–2 yrs post-menarche) — must be persistent
- Polycystic ovarian morphology is common in normal adolescent ovaries — USS not used for adolescent diagnosis
- AMH utility in adolescents — not yet validated for diagnostic use; reference ranges differ
- OCP use before diagnosis is common — consider AMH if USS unreliable
- Psychological impact of diagnosis at this age requires sensitive counselling

Source: Peña AS et al. BMC Med. 2025;23:151; Teede HJ et al. Eur J Endocrinol. 2023

Laboratory Investigations: First-Line Workup for PMOS

CONFIRM HYPERANDROGENISM	**Total testosterone**	**SHBG**	**FAI (Free Androgen Index)**	**DHEA-S**
	Morning sample; calculate free T	Suppressed in IR — calculate FAI	FAI = Total T×100/SHBG; >3.5 abnormal	Adrenal androgen source
	mU/L or nmol/L	nmol/L	Ratio	µmol/L
ASSESS METABOLIC RISK	**Fasting glucose + insulin**	**75GOGT/HbA1c**	**Fasting lipids**	**Liver function**
	Calculate HOMA-IR (×0.05 = normal <1.8)	Screen for prediabetes/T2DM every 1–3 yr	TG, HDL, LDL, Total Chol	Screen for MASLD
	mmol/L	mmol/mol	mmol/L	IU/L
EXCLUDE DIFFERENTIALS	**TSH**	**Prolactin**	**17-OHP (basal + ACTH stimulated)**	**24hr urinary cortisol/DST**
	Thyroid dysfunction	Hyperprolactinaemia	Non-classical CAH	Cushing syndrome if suspected
	mIU/L	mIU/L	nmol/L	Various

Source: Bizuneh AD et al. Hum Reprod Update. 2025;31:48–63; Teede HJ et al. Eur J Endocrinol. 2023

Ultrasound in PMOS: 2023 Updated Criteria

Updated 2023 Ultrasound Criteria

≥20 follicles per ovary (2–9 mm)

Updated from ≥12; higher threshold for modern high-frequency transducers

Ovarian volume ≥10 mL

Either one or both ovaries; exclude dominant follicle/cyst

Either criterion sufficient

Follicle count OR volume; not both required

Transvaginal preferred

Higher resolution; transabdominal acceptable if vaginal not feasible

Not diagnostic in adolescents

Polycystic morphology common in normal puberty; avoid overdiagnosis

Practical Points

Perform in early follicular phase (D2–5)

Luteal phase and mid-cycle images may miss PCOS morphology

On OCP

AMH preferred — pill suppresses follicle count

AMH as alternative

AMH ≥3.4 ng/mL substitutes for ultrasound in adults

Report both criteria

Follicle count per ovary (not total) + ovarian volume

Source: Pea J et al. Hum Reprod Update. 2024;30:109–130; Teede HJ et al. Eur J Endocrinol. 2023

Differential Diagnosis: What to Exclude Before Diagnosing PMOS

Non-Classical CAH

Test: Basal 17-OHP + ACTH stimulation test

↑ 17-OHP >6 nmol/L basal; ACTH stimulated >30 nmol/L

Thyroid Dysfunction

Test: TSH (reflex fT4 if abnormal)

Hypothyroidism → irregular cycles; hyperthyroidism → amenorrhoea

Hyperprolactinaemia

Test: Prolactin (fasting, repeat if elevated)

Prolactin suppresses GnRH → oligomenorrhoea; galactorrhoea

Cushing Syndrome

Test: 24hr urine free cortisol, 1mg overnight DST

Cortisol excess → central obesity, hirsutism, irregular cycles

Androgen-Secreting Tumour

Test: Total T >5 nmol/L → pelvic USS + adrenal CT

Rapid-onset severe virilisation; very high testosterone

Primary Ovarian Insufficiency

Test: FSH, LH, oestradiol, AMH (very low)

↑ FSH, ↓ oestrogen, hot flushes; age <40

Source: Teede HJ et al. Eur J Endocrinol. 2023; Stener-Victorin E et al. Nat Rev Dis Primers. 2024

SECTION 6 · MANAGEMENT

Comprehensive Management of PMOS

Lifestyle · Hormonal · Metabolic · Fertility · Psychological

Treatment Framework: Personalised, Holistic, Lifelong

Management is driven by the patient's primary concerns, comorbidities, and reproductive wishes:

1 Lifestyle

- 5–10% weight loss improves all features
- Diet + exercise: 150min moderate/week
- Mediterranean or low-GI diet preferred
- Even 5% loss restores ovulation in many

2 Hormonal

- OCP: first-line menstrual regulation
- Anti-androgens: spironolactone, cyproterone
- Inositol (myoinositol): emerging evidence
- Progestin therapy: endometrial protection

3 Metabolic

- Metformin: insulin sensitiser + metabolic
- GLP-1 agonists: weight + metabolic benefit
- Bariatric surgery: severe obesity (BAMBINI)
- Screen T2DM, CVD, MASLD annually

4 Fertility

- Letrozole: first-line ovulation induction
- Clomiphene: alternative (inferior to letrozole)
- Gonadotrophins: second-line
- IVF/ICSI: third-line; antagonist protocol

5 Psychological

- Screen ALL: PHQ-9, GAD-7
- CBT: strong evidence for anxiety/depression
- Mindfulness, peer support, body positivity
- Address name-related distress explicitly

Source: Teede HJ et al. Eur J Endocrinol. 2023;189:G43–G64 — 2023 International Evidence-Based Guidelines

Lifestyle Interventions: The Foundation of PMOS Management

Evidence Base

5–10% weight loss

Restores menstrual regularity in 50–70%; reduces androgen levels; improves IR

Exercise (150 min/wk)

Moderate-intensity cardio + resistance training; improves metabolic AND psychological outcomes

Diet quality

Mediterranean, low-GI, or low-carbohydrate diets — all improve insulin sensitivity and reduce androgens

Mindfulness

Reduces cortisol, improves stress and depression; complements dietary and exercise intervention

Practical Prescription

Weight Goal:	5–10% body weight reduction; even <5% shows benefit
Exercise:	150 min/wk moderate OR 75 min/wk vigorous; 2× resistance/wk
Diet:	Mediterranean or low-GI; reduce ultra-processed foods; no rigid calorie counting
Sleep:	Treat sleep apnoea; 7–9 hrs/night; disrupted sleep worsens IR
Referral:	Dietitian + exercise physiologist MDT; behavioural support
Monitoring:	Weight, menstrual pattern, lipids, HOMA-IR every 6–12 months

Source: Dobbie LJ et al. Hum Reprod. 2023;38:1168–1182; Teede HJ et al. Eur J Endocrinol. 2023

Hormonal Management: OCP, Anti-Androgens & Beyond

Combined OCP	**Indication:**	Menstrual irregularity, hirsutism, acne
	Dose:	Lowest effective oestrogen dose; progestin with anti-androgenic properties
	Note:	Not for women seeking pregnancy; annual BP review; VTE risk counselling
Spironolactone	**Indication:**	Hirsutism, acne (when OCP not suitable or adjunct)
	Dose:	50–200 mg/day; effective contraception required (teratogenic)
	Note:	Monitor K^+; can cause menstrual irregularity; monitor BP in older patients
Cyproterone Acetate	**Indication:**	Severe hirsutism, severe androgenic alopecia
	Dose:	2 mg in combined pill; 50–100 mg higher dose for alopecia
	Note:	Not available in all countries; VTE risk higher than standard OCP; avoid >6 months high-dose
METFORMIN/ Myoinositol	**Indication:**	Ovulation induction; metabolic improvement; androgen reduction
	Dose:	Metformin: 500-2500mg/day; MI: 2–4 g/day with folic acid; particularly in lean PMOS
	Note:	Well-tolerated; emerging evidence; not yet in all guidelines as first-line

Source: Teede HJ et al. Eur J Endocrinol. 2023;189:G43–G64; Goldberg A et al. Obes Rev. 2024;25:e13704

Metabolic Management: Insulin Sensitisers & Weight Interventions

Metformin Biguanide — Insulin Sensitiser	Use: Metabolic features, irregular cycles, prevention T2DM, adjunct ovulation induction Evidence: RCTs: ↓ fasting glucose, ↓ insulin, modest ↓ androgens, modest weight loss; superior to placebo **Dosing: 500–2500 mg/day with food; start low, titrate over 4–8 weeks**
GLP-1 Receptor Agonists Semaglutide, Liraglutide, Dulaglutide	Use: Overweight/obese PMOS; weight loss + metabolic benefit; ± restore ovulation Evidence: Systematic review (2024): significant weight loss 5–15%; ↓ androgens; ↓ IR; improving evidence **Dosing: Semaglutide 0.25–2.4 mg/wk SC; liraglutide 0.6–3.0 mg/day SC**
Bariatric Surgery Metabolic Surgery — Severe Obesity	Use: BMI ≥35 (or ≥30 with comorbidities) — BAMBINI trial: spontaneous ovulation restored Evidence: BAMBINI RCT (Lancet 2024): 86% ovulation restoration post-sleeve gastrectomy vs 57% lifestyle **Dosing: MDT decision; ensure nutritional prep; delay pregnancy 12–18 months post-op**

Source: Goldberg A et al. Obes Rev. 2024;25:e13704; Samarasinghe SNS et al. Lancet. 2024;403:2489–2503

Fertility Management: Evidence-Based Ovulation Induction

Management pathway for PMOS-related anovulatory infertility:

FIRST LINE	**Letrozole:**	Aromatase inhibitor; superior to clomiphene in 2023 guidelines • Live birth rate: 27.5% vs 19.1% clomiphene (PCOSII RCT) • 2.5–7.5 mg D3–7; lower OHSS risk than gonadotrophins
SECOND LINE	**Clomiphene Citrate:**	Selective oestrogen receptor modulator; historical first-line • 50–150 mg D2–6; anti-oestrogenic effects on endometrium • + Metformin: improves ovulation and live birth rates
THIRD LINE	**Gonadotrophins:**	FSH injections (± LH); requires careful monitoring • Low-dose step-up protocol preferred; ↑ OHSS risk • Laparoscopic ovarian drilling: alternative (reduces gonadotrophin need)
FOURTH LINE	**IVF / ICSI:**	For failed ovulation induction or additional infertility factors • GnRH antagonist protocol preferred in PMOS (↓ OHSS risk) • Segmented cycles: freeze all strategy reduces OHSS • Avoid trigger with hCG if E2 >15,000 pmol/L

Source: Teede HJ et al. Eur J Endocrinol. 2023; Samarasinghe SNS et al. Lancet. 2024;403:2489–2503

Psychological Management: Essential, Not Optional

Screening Protocol

Depression:

PHQ-9 at every visit; PHQ-2 as rapid screen

Anxiety:

GAD-7; consider Beck Anxiety Inventory

Eating Disorders:

EDE-Q; ask about binge eating, restriction

Quality of Life:

PCOSQ or SF-36 (PMOS-specific QoL tool)

Body Image:

Body Image Disturbance Questionnaire

Evidence-Based Interventions

Cognitive Behavioural Therapy (CBT)

Strong RCT evidence for depression + anxiety in PMOS; address body image, disordered eating, health anxiety

Mindfulness-Based Therapy

MBSR + MBCT: ↓ cortisol, ↓ anxiety, ↑ self-compassion; complements lifestyle intervention

Motivational Interviewing

Lifestyle behaviour change; reduce shame and stigma around weight; patient-centred approach

Patient Support Communities

Verity UK, PCOS Challenge (USA), POSAA (Australia); peer support ↓ isolation and ↑ self-management

Source: Teede HJ et al. Eur J Endocrinol. 2023; Stener-Victorin E et al. Nat Rev Dis Primers. 2024;10:27

SECTION 7 · ROAD AHEAD

PMOS: The Global Implementation

8-Stage Roadmap · Transition Period · Future Research

8-Stage Global Implementation Strategy

1 — Publication & Academic Dissemination

The Lancet publication + commentaries, clinical reviews, textbook updates

2 — Resource Development

Co-designed multilingual patient and clinician resources across platforms

3 — Global Communication

Society toolkits, multimedia campaigns, professional education, patient events worldwide

4 — Health Information Systems

EHR integration, SNOMED-CT, major EMR vendors, university curricula

5 — Policy & Research Alignment

Research funders, journal editors, regulators, pharmaceutical industry

6 — International Classification

WHO engagement → ICD integration (ICD-11 and future revisions)

7 — 3-Year Managed Transition

Monitoring, evaluation, subtype refinement as science evolves

8 — Guideline Integration

2028 International Guideline update (195 countries); NICE guideline revision

Source: Teede HJ et al. The Lancet. 2026 — Panel 4: Eight stages for global PMOS implementation

What Changes with PMOS: Benefits of the New Name

	Before	After
For Patients:	Before: 'I have polycystic ovary syndrome' — confusion, fear of cysts	After: 'I have PMOS' — accurate framing; less stigma; better understanding
For Clinicians:	Before: Narrow reproductive/gynaecological focus; metabolic risk underrecognised	After: Systemic framing prompts CV, metabolic, psychological screening
For Researchers:	Before: Heterogeneous definitions; limited funding for non-reproductive aspects	After: Aligned nomenclature; broader funding opportunities; clearer GWAS groupings
For Policy:	Before: Coded as reproductive condition; limited health system priority	After: ICD reclassification; metabolic disease recognition; wider health system engagement
For Education:	Before: Taught as 'ovarian cyst syndrome'; narrow curriculum focus	After: Multi-system disorder in all health professional training programmes

Source: Teede HJ et al. The Lancet. 2026. DOI: 10.1016/S0140-6736(26)00717-8

KEY TAKEAWAYS

Polyendocrine Metabolic Ovarian Syndrome (PMOS)

1. PMOS is the new, accurate name for what was PCOS — reflecting its polyendocrine, metabolic, and ovarian nature
2. Pathophysiology involves GnRH–LH axis dysregulation, hyperandrogenism, insulin resistance, and ovarian dysfunction — all interacting
3. Diagnosis requires 2 of 3 criteria (adults): oligo-anovulation, hyperandrogenism, polycystic morphology / elevated AMH — after excluding other causes
4. Management is multimodal: lifestyle is first-line for ALL; OCP for hormonal symptoms; metformin/GLP-1 for metabolic; letrozole for fertility
5. Screen every patient for depression, anxiety, and reduced QoL — psychological management is non-negotiable
6. 70% remain undiagnosed — clinical awareness, accurate terminology, and multidisciplinary care are essential for global health impact

Teede HJ et al. The Lancet. 2026. DOI: 10.1016/S0140-6736(26)00717-8 · Teede HJ et al. Eur J Endocrinol. 2023;189:G43–G64