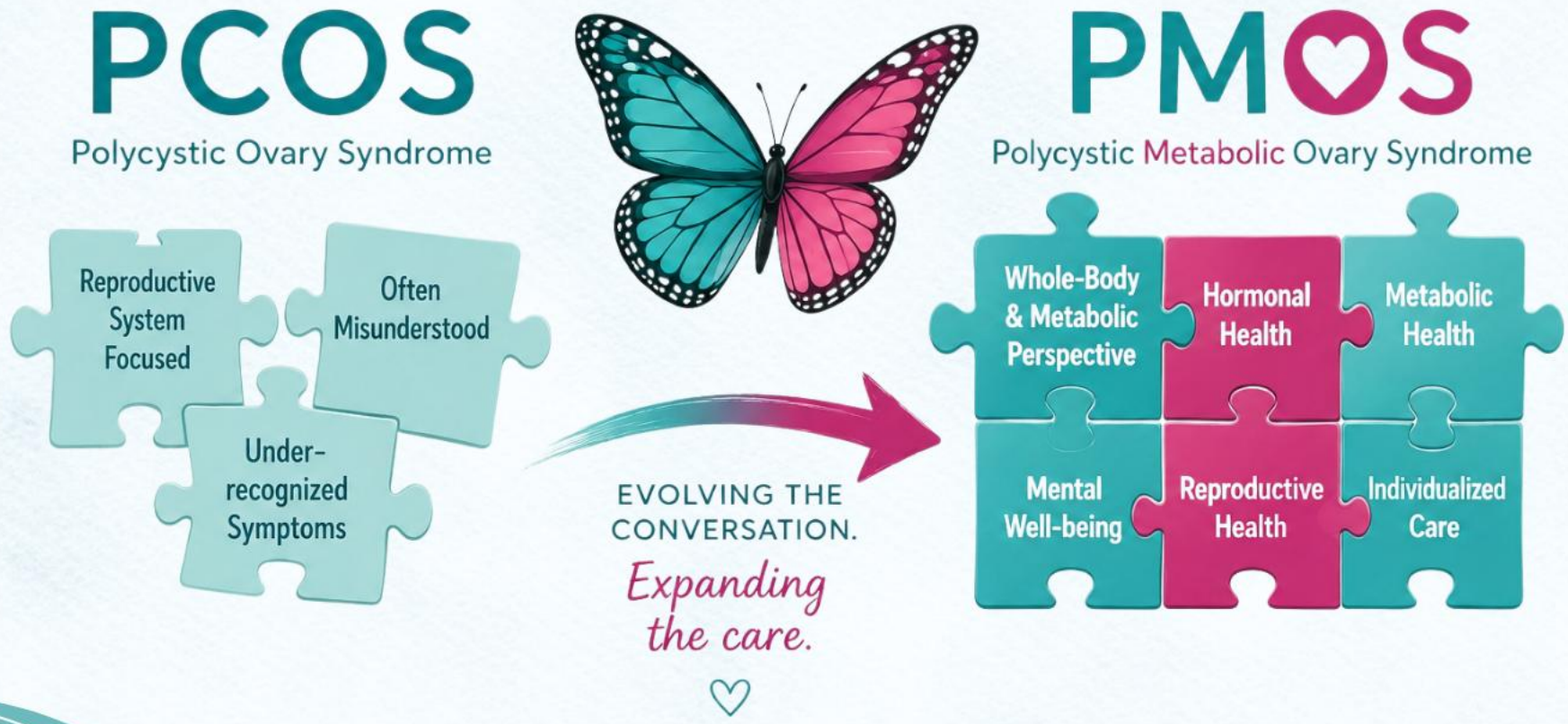
New Frontiers in Polyendocrine Metabolic Ovarian Syndrome (PMOS) Care: Evidence-based Diagnosis, Assessment, and Treatment utilizing the Latest Guideline Updates

Dr. Lynsey Johnson, DNP, APRN, FNP-C



Speaker Introduction & Disclosures

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The views expressed in this webinar are solely those of the presenter and do not necessarily reflect the official policies, positions, or endorsement of Emory University or the Nell Hodgson Woodruff School of Nursing.

Objectives

1. Apply updated evidence-based guidelines to diagnose and assess Polyendocrine Metabolic Ovarian Syndrome (PMOS) across the lifespan.
2. Differentiate common PMOS presentations and comorbidities, including infertility, obesity, insulin resistance, and diabetes risk, to improve early identification.
3. Develop individualized evidence-based treatment plans for PMOS, including lifestyle changes, hormonal and non-hormonal therapies, and GLP-1 receptor agonists.

Background

320M - 520M Women worldwide have PMOS/6M in USA

10-20% of all women globally

34% of women survey reported >2 years for a diagnosis
47% saw 3+ health professionals before being diagnosed
Only 5.6% were satisfied with the information they received

PCOS is the **#1** cause of infertility in reproductive aged women

>50% of PMOS women develop diabetes by age 40.

70% remain undiagnosed or misdiagnosed.

19% increased risk of cardiovascular event such as stroke or heart attack

80% are obese or overweight

Gibson-Helm et. al, 2017
CDC, 2024
Wang et. al, 2025

Why the Name Change to PMOS?

"The renaming to Polyendocrine Metabolic Ovarian Syndrome acknowledges that this condition extends far beyond the ovaries, encompassing complex polyendocrine and metabolic dysfunction that affects multiple body systems."
— Teede et al., 2026

Also the name is a misnomer! That's correct....there have never been "cysts." The physician who discovered PCOS mistook excess immature follicles for cysts.

What is PMOS?

Definition

Polyendocrine Metabolic Ovarian Syndrome (PMOS), formerly known as PCOS, was officially renamed in May 2026 to better reflect the syndrome's complexity.

The name change acknowledges that this condition extends far beyond ovarian cysts alone.

Core Features

PMOS encompasses three interconnected manifestations: polyendocrine dysfunction, metabolic disturbances, and ovarian abnormalities.

These features interact synergistically, creating a complex clinical presentation requiring comprehensive care.

Pathophysiology

The syndrome is characterized by hormonal imbalance, hyperandrogenism, and insulin resistance affecting multiple body systems.

Understanding these mechanisms is essential for effective diagnosis and treatment planning.

Clinical Manifestations & Comorbidities

Hyperandrogenism

Hirsutism: excess facial and body hair growth
Acne: persistent, often severe
Alopecia: male-pattern hair thinning

Clinical signs often first noticed by patients, prompting medical evaluation

Ovulatory Dysfunction

Irregular or absent menstrual cycles
Chronic anovulation
#1 cause of infertility in reproductive-aged women

Hormonal imbalance disrupts normal egg development and release

Metabolic Risks

Insulin resistance affecting 70-80%
50% develop type 2 diabetes by age 40
70-80% overweight or obese

Elevated cardiovascular risk factors require ongoing monitoring

Faces of PMOS

PCOS (PMOS) – PATHOPHYSIOLOGY

Interconnected Mechanisms and Feedback Loops

Stimulatory / Increase
Inhibitory / Decrease
Flow / Effect
Feedback Loop

1 INSULIN RESISTANCE (Core Defect)

LIVER
↑ Gluconeogenesis
↑ VLDL production

SKELETAL MUSCLE
↓ Glucose uptake
(GLUT4 resistance)

INSULIN RESISTANCE

ADIPOSE TISSUE
↑ Lipolysis
↑ Free fatty acids
↑ Pro-inflammatory adipokines (TNF-α, IL-6)
↓ Adiponectin

COMPENSATORY HYPERINSULINEMIA

EFFECTS OF HYPERINSULINEMIA

- ↑ Ovarian theca cell androgen production (synergizes with LH)
- ↓ SHBG production in liver → ↑ Free testosterone
- ↑ IGF-1 activity (↓ IGFBP-1) → ↑ Androgen production
- ↑ Appetite & weight gain → worsens insulin resistance

2 VICIOUS CYCLE (Feed-forward Loop)

Hyperinsulinemia → Insulin Resistance → ↑ Androgens → Weight Gain (especially central obesity) → Chronic Inflammation → Hyperinsulinemia

HYPOTHALAMUS

↑ GnRH pulse frequency
(↑ amplitude)

- Influenced by genetic, epigenetic & environmental factors (obesity, insulin resistance, stress, sleep disturbance, endocrine disruptors)

ANTERIOR PITUITARY

↑ LH secretion
(relative ↑ over FSH)

- Increased pulse frequency favors LH synthesis
- LH:FSH ratio > 1.5–2 : 1

↓ FSH secretion

- Reduced FSH pulse frequency/amplitude
- Activin B ↓
  Inhibin B ↓

LH

FSH

THECA CELL
(LH responsive)

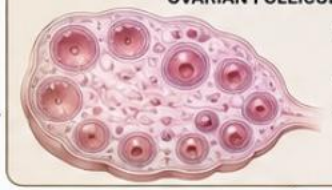
Cholesterol
↓ (via StAR, CYP11A1)
Androstenedione
↓ (CYP17A1)
Testosterone (T)

TESTOSTERONE

GRANULOSA CELL
(FSH responsive)

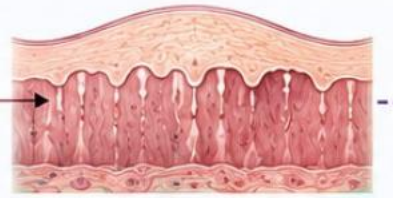
↓ Aromatase (CYP19A1)
↓ Conversion of T → E_2
↓ Estradiol (E_2)
↓ Inhibin B
↑ Activin A

OVARIAN FOLLICULAR ARREST



- Early antral follicles (2–9 mm) accumulate and fail to mature
- Lack of dominant follicle
- No ovulation → Corpus luteum deficiency → ↓ Progesterone

↓ Progesterone (anovulation)
Unopposed estrogen (from peripheral aromatization of androgens in adipose tissue) → Endometrial proliferation → Irregular shedding / Infertility
Risk of endometrial hyperplasia (long term)



ENDOMETRIUM

↑ ANDROGENS (T, Δ4A)
(from theca & adrenal)

Peripheral effects
- Hirsutism, acne, alopecia
- Sebaceous gland activity
- Skin changes

↓ SHBG (liver)
(Decreased by insulin)

ADRENAL CONTRIBUTION
(Subset of patients)
- ↑ Adrenal androgens (DHEA, DHEAS)
- Due to ACTH hyper-responsiveness or intrinsic enzyme activity

↑ FREE TESTOSTERONE
(Bioavailable)

5 CLINICAL MANIFESTATIONS

HYPERANDROGENISM
- Hirsutism
- Acne
- Androgenic alopecia

OVULATORY DYSFUNCTION
- Oligo/anovulation
- Irregular menses
- Infertility

POLYCYSTIC OVARIES
- ↑ Antral follicle number (≥20)
- Follicles 2–9 mm
- Ovarian volume >10 mL

METABOLIC FEATURES
- Central obesity
- Dyslipidemia
- Hypertension
- Insulin resistance / T2DM risk

PSYCHOLOGICAL IMPACT
- Anxiety, depression
- Body image concerns
- Reduced quality of life

6 LONG-TERM RISKS

- Type 2 Diabetes Mellitus
- Cardiovascular Disease
- Non-alcoholic Fatty Liver Disease
- Endometrial Hyperplasia / Carcinoma
- Obstructive Sleep Apnea
- Mood Disorders

KEY PATHWAYS SUMMARY

1. **INSULIN RESISTANCE** (driven by genetics, obesity, low-grade inflammation) → Hyperinsulinemia
2. **HYPERINSULINEMIA** + ↑ LH → + ↑ Theca androgen production + ↓ SHBG
3. **↑ ANDROGENS** (from ovary & adrenal) + ↓ SHBG → ↑ Free testosterone
4. **FOLLICULAR ARREST** ↓ FSH action + ↑ Androgens → Impaired follicular development → Anovulation
5. **CLINICAL & METABOLIC MANIFESTATIONS** (heterogeneous expression)
6. **LONG-TERM RISKS** (increased cardiovascular, metabolic & endometrial risk)

NOTE: PCOS is a heterogeneous disorder with multiple interacting genetic, environmental and metabolic factors. Not all pathways are abnormal in every patient.

Why the Name Changed: PCOS → PMOS

A more accurate name for a more inclusive, evidence-based understanding.

WHAT WAS "PCOS"?

PCOS = Polycystic Ovary Syndrome

Coined in the 1990s when the presence of "polycystic ovaries" on ultrasound was considered central to diagnosis.

- Focused on ovarian morphology
- Required ultrasound "cysts" for many diagnoses
- Missed many individuals without polycystic ovarian morphology who still had the condition
- Contributed to misunderstanding of disease mechanisms

WHAT IS "PMOS"?

PMOS = Polycystic Ovary Syndrome

The 2023 International Evidence-based Guideline recommends using "PMOS" as the preferred term.

- ✓ **Reflects that polycystic ovarian morphology (PCOM) is NOT required for diagnosis**
- ✓ **Centers the diagnosis on what matters most: ovarian dysfunction + hyperandrogenism**
- ✓ **More inclusive:** recognizes the diversity of phenotypes and presentations
- ✓ **Reduces stigma and visual bias** associated with the word "cystic"
- ✓ **Aligns the name with the true nature of the disorder: a heterogeneous, systemic condition**

KEY CHANGE

PCOS → PMOS

The change from PCOS to PMOS is a scientific and patient-centered evolution in terminology that improves accuracy, inclusivity, and understanding.

THE BOTTOM LINE

PMOS better reflects the complexity of the condition and ensures no one is left undiagnosed or misunderstood.

EVIDENCE BEHIND THE CHANGE

Many individuals with PMOS do not have polycystic ovarian morphology (PCOM) on ultrasound but still meet criteria for the condition.

Diagnosis is based on the presence of both:

- ✓ Ovarian dysfunction
- ✓ Clinical and/or biochemical hyperandrogenism

PMOS is a heterogeneous disorder with variable presentation, mechanisms, and metabolic, reproductive, and psychological impacts.

The name change is supported by the 2023 International Evidence-based Guideline for the Assessment and Management of PMOS.

WHAT DOESN'T CHANGE

- ✓ The diagnostic criteria
- ✓ The seriousness and impact of the condition
- ✓ The need for early recognition and appropriate management
- ✓ Our commitment to improving the lives of all individuals with PMOS

A NEW NAME. SAME RECOGNITION. BETTER UNDERSTANDING. STRONGER SUPPORT.

***PMOS is not* just a name change—it's a step forward in science, inclusivity, and care.**

PMOS DIAGNOSIS

The Latest (2023) Evidence-Based Criteria

International evidence-based guideline for the assessment and management of PMOS

PMOS CAN BE DIAGNOSED WHEN BOTH OF THE FOLLOWING ARE PRESENT

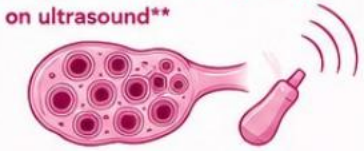
1. Evidence of **OVARIAN DYSFUNCTION**
+
2. Evidence of **CLINICAL AND/OR BIOCHEMICAL HYPERANDROGENISM**

After exclusion of other specific etiologies that can mimic PMOS

1 OVARIAN DYSFUNCTION

One or more of the following is present:

- **Irregular menstrual cycles (varies by age)***
  - Adolescents: beyond 2 years post-menarche
  - Adults: cycle length <21 days or >35 days, or <8 cycles/year
- **Anovulation** (absence of ovulation)
- **Polycystic ovarian morphology on ultrasound****



AND

2 CLINICAL AND/OR BIOCHEMICAL HYPERANDROGENISM

One or more of the following is present:

A. Clinical hyperandrogenism

- Hirsutism
- Moderate to severe acne
- Androgenic alopecia

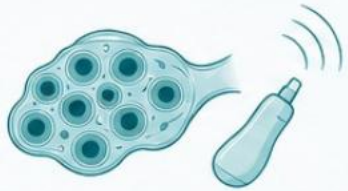
B. Biochemical hyperandrogenism

- Elevated total and/or free testosterone (by reliable assay)
- Elevated calculated free testosterone
- Elevated bioavailable testosterone
- Elevated androstenedione (if testosterone not elevated)

POLYCYSTIC OVARIAN MORPHOLOGY (PCOM)

PCOM is NOT required for diagnosis (should not be used as a diagnostic criterion)

Ultrasound should be used only for other indications and not solely for diagnosing PMOS



EXCLUDE OTHER SPECIFIC ETIOLOGIES

Before confirming PMOS, exclude conditions that can mimic PMOS:

- ✓ Pregnancy
- ✓ Thyroid disorders
- ✓ Hyperprolactinemia
- ✓ Non-classical congential adrenal hyperplasia (CAH)
- ✓ Cushing syndrome
- ✓ Androgen-secreting tumors
- ✓ Other causes of hyperandrogenism or ovulatory dysfunction

KEY POINTS

- PMOS is a heterogeneous disorder with variable presentation.
- Diagnosis is clinical and based on the presence of both ovarian dysfunction and hyperandrogenism.
- PCOM on ultrasound is not required for diagnosis.
- Criteria apply to all ages, including adolescents, with age-appropriate assessment.
- Co-existing metabolic, psychological and reproductive issues should be assessed in all individuals.

ASSESS THE WHOLE PERSON

- **Metabolic health** (Weight, glucose metabolism, lipids, blood pressure)
- **Mental health** (Screen for anxiety, depression, eating disorders)
- **Reproductive goals** (Fertility desires, contraception, menstrual goals)
- **Long-term risks** (CVD, type 2 diabetes, endometrial cancer, sleep apnea)

IMPORTANT NOTES

- Two criteria (1 and 2) must be present.
- PMOS remains a diagnosis of exclusion.
- Use a patient-centered, individualized approach.

REFERENCES

International Evidence-based Guideline for the Assessment and Management of Polycystic Ovary Syndrome 2023

Teede HJ, Tay CT, Laven JSE, et al. Hum Reprod Update. 2023;29(5):e28.

* Menstrual cycle irregularity definitions:
- Adolescents: irregular cycles within 2 years post-menarche are considered normal.
- Adults: <21 days or >35 days, or <8 cycles/year.

** PCOM (if assessed for other reasons):
≥20 follicles per ovary (2–9 mm in diameter) and/or ovarian volume >10 mL in at least one ovary.

Evolution of PCOS Diagnostic Criteria

U.S. National Institutes of Health (NIH) Criteria
1990

PCOS is diagnosed if both of the following criteria are present:

- Oligo-anovulation
- Clinical and/or biochemical signs of hyperandrogenism

Rotterdam Criteria
2003

PCOS is diagnosed if 2 out of 3 of the following criteria are present:

- Oligo-anovulation
- Clinical and/or biochemical signs of hyperandrogenism
- Polycystic ovaries on ultrasound

Androgen Excess and PCOS Society (AE-PCOS) Criteria
2006

PCOS is diagnosed if all of the following criteria are present:

- Clinical and/or biochemical hyperandrogenism
- Ovarian dysfunction (oligo-anovulation and/or polycystic ovarian morphology)
- Exclusion of other androgen excess or related disorders

1990 2003 2006 2023 2028

Updated Diagnostic Criteria

1. Hyperandrogenism (clinical or biochemical): Includes hirsutism, acne, alopecia, or elevated testosterone/DHEAS levels.

2. Ovulatory Dysfunction: Irregular or absent menstrual cycles indicating anovulation or oligo-ovulation (Periods that occur fewer than 21 days or more than 35 days apart)

OR

Polycystic Ovarian Morphology: ⩾20 follicles per ovary or ovarian volume >10mL on ultrasound imaging

OR

Anti-Mullerian Hormone (AMH) >4-5

Required
Exclusion of other androgen excess or related disorders

Teede et. al, 2023

When do you complete a an Ultrasound?

Prolonged Amenorrhea: to determine risk for uterine Cancer. Induce withdrawal bleed with progesterone.

Pain: PMOS does not directly cause pain. If pain occurs, it is likely secondary to another condition such as a true ovarian cyst, endometriosis, or heavy periods

Laboratory Evaluation &Differential Diagnosis

Hormone panels: Testosterone (free, total, bioavailable), AMH (if regular menstrual cycles)

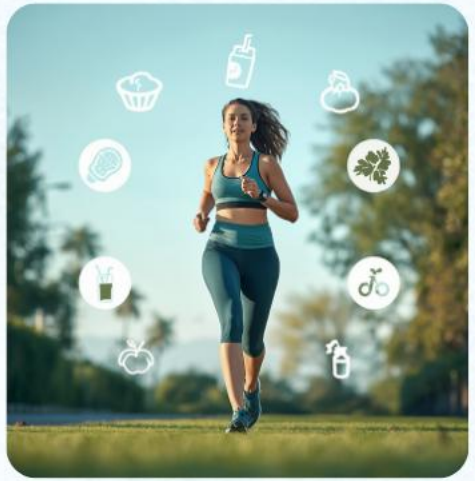
Exclude other endocrine disorders: Hypothyroidism (TSH), congenital adrenal hyperplasia (17-hydroxyprogesterone), prolactinoma (prolactin), Adrenal tumor (DHEA)

Comprehensive metabolic screening essential for diabetes risk assessment: CMP, A1c, Lipid profile, CBC with heavy menstrual cycles

Fertility Testing if interested: Add on LH, FSH, Estradiol, Progesterone, AMH

*Hormones must be interpreted with caution if patient is already on PMOS treatment as results often appear normal. e.g. Natural estradiol could be zero or low if someone is taking birth control
*There is no lab standardization for these values, so you must understand what is high enough to be concerning for tumor vs. monitoring

Treatment Goals & Approaches included in 2023 Guideline updates



Lifestyle

DIET, EXERCISE, WEIGHT MANAGEMENT

Insulin Sensitizers

METFORMIN

GLP-1 Agonists

SEMAGLUTIDE
TIRZEPATIDE

Hormonal Therapy

HORMONAL BIRTH CONTROL AND ANTI-ANDROGENS E.G. SPIRONOLACTONE

GLP-1 vs. Metformin

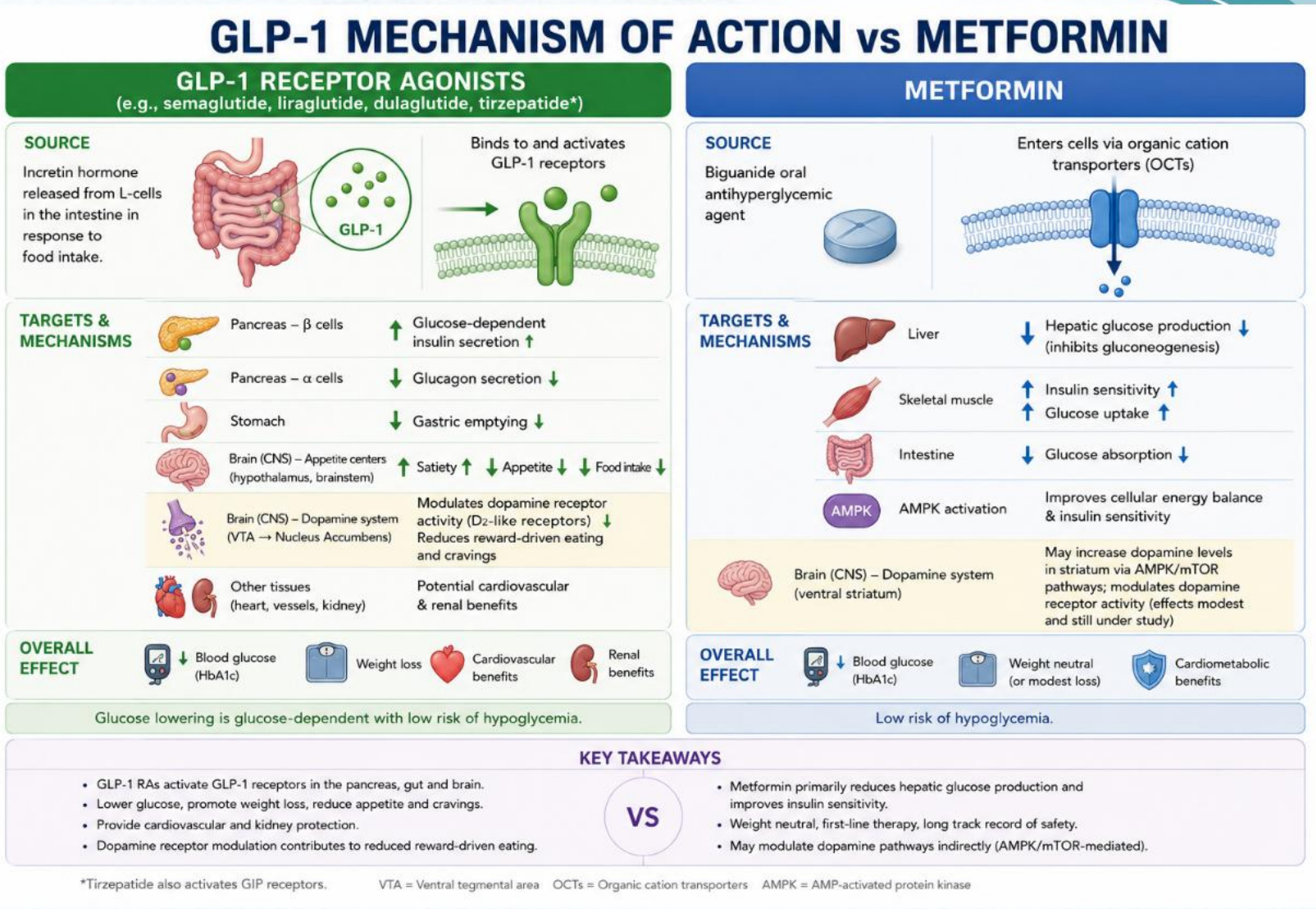
GLP-1 management (Semaglutide or tirzepatide): Titrate to less than 10lbs weight loss per month and insure patients eat at least 1200 calories per day to avoid rebound weight gain and side effects

*2 month wash out for GLP-1 required before pregnancy attempts. Rodent studies showed birth defects

Metformin extended release only please! Its now very affordable and make a tremendous difference in patient adherance.

- Neutral or modest weight loss
- Safe for conception and during pregnancy

*Immediate release often causes GI side effects



Spironalactone

- Anti-androgen, antihypertensive, diuretic:
- Great for skin and hair issues: Hair loss and Acne
- Causes birth defects if conception occurs while taking spironalactone
- Historically a blood pressure medication: higher doses can cause dizziness or heart palpitations
- Spironalactone is known to cause birth defects if pregnancy occurs while taking it (3 month washout needed)

Birth Control Selection & PMOS: They are Not All Created Equal

Birth controls have different androgen potencies! Do not give a high androgen potency progesterone to a patient with PMOS because some will convert back to testosterone potentially causing moodswings and other issues

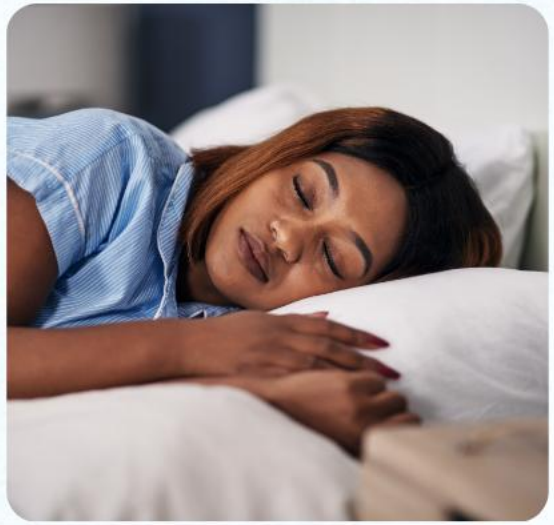
Correct birth control selection is Key to patient tolerance

Lifestyle changes for PMOS

Resistance Training is optimal since insulin receptors are most dominant on muscles

Intense cardio may increase cortisol levels, thereby increasing androgens, worsening PMOS

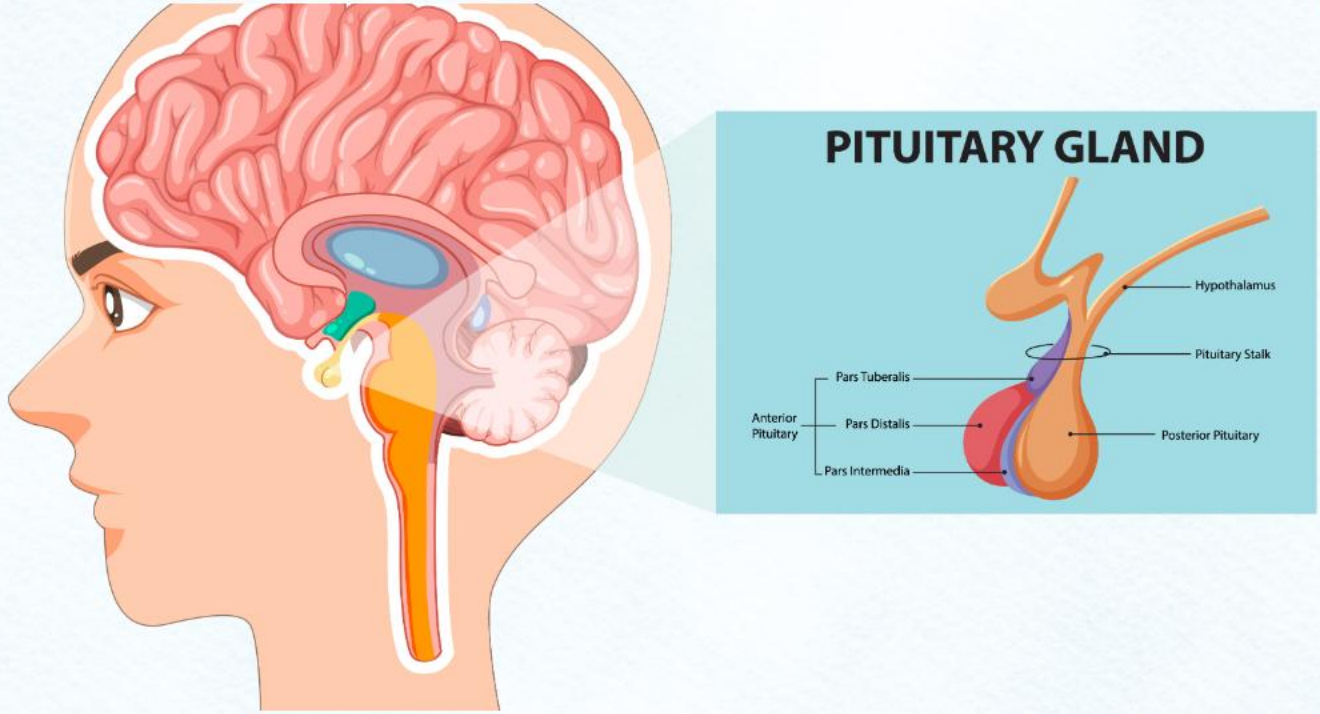
Mental health & Sleep hygiene: Increased stress may increase cortisol levels, thereby increasing androgens, worsening PMOS



How long do medications take to reach peak effect?

3 months for pituitary biofeedback to occur

Give all meds 3 months before major changes and keep in mind that hormonal methods such as birth control and spironalactone should be titrated knowing that each dose takes 3 months to be fully effective, start low and go slow to avoid side effects and overtreatment of PMOS



Where in the pathophysiology of PMOS does your patient experience symptoms?

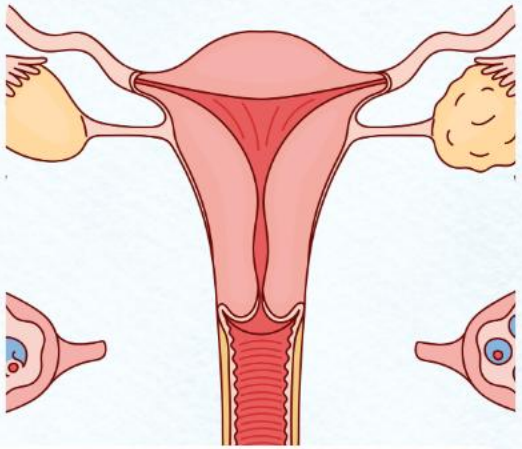
Weight loss: GLP-1 or metformin with life style changes. Adipose tissue is hormonally active.

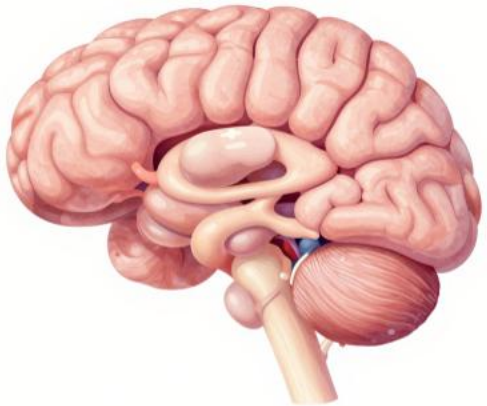
Hirsutism (unwanted hair): Estradiol (combination birth control) or androgen blockers (Spironalactone)

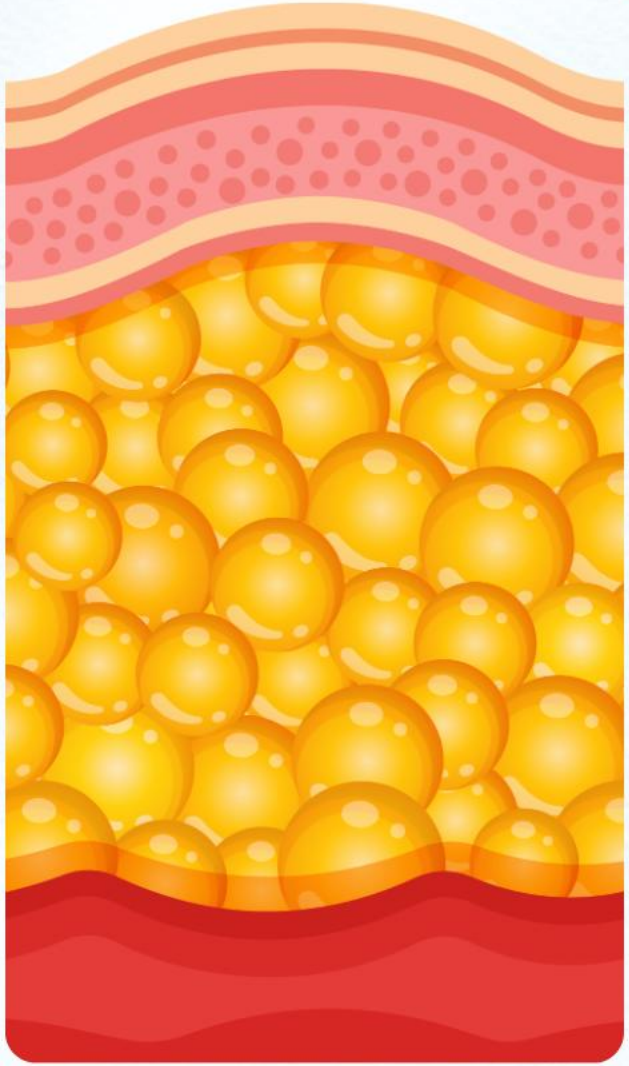
Cycle regulation: Metformin, GLP-1, hormonal birth control

Fertility: Metformin, GLP-1, Letrozole, Clomid

*Spironalactone is known to cause birth defects if pregnancy occurs while taking it, it's also a diuretic
***All meds, start low dosage and titrate up**
*2 month wash out for GLP-1 required. Rodent studies showed birth defects.







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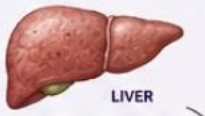

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SKELETAL MUSCLE
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(GLUT4 resistance)

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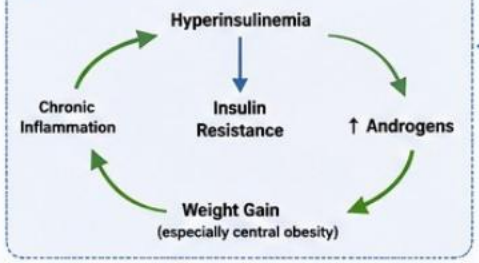

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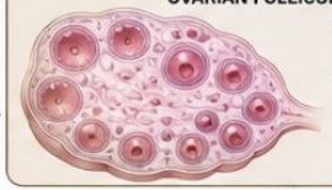

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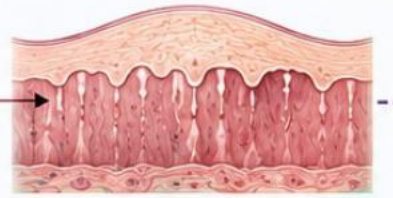

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2. HYPERINSULINEMIA + ↑ LH → ↑↑ Theca androgen production + ↓ SHBG
3. ↑ ANDROGENS (from ovary & adrenal) + ↓ SHBG → ↑ Free testosterone
4. FOLLICULAR ARREST ↓ FSH action + ↑ Androgens → Impaired follicular development → Anovulation
5. CLINICAL & METABOLIC MANIFESTATIONS (heterogeneous expression)
6. LONG-TERM RISKS (increased cardiovascular, metabolic & endometrial risk)

NOTE: PCOS is a heterogeneous disorder with multiple interacting genetic, environmental and metabolic factors. Not all pathways are abnormal in every patient.

Case Studies: Question

35 year old patient is 282 lbs, has acne, scalp hair thinning, no period for a year. Wants to get pregnant soon.

How do we manage this patient?

Discuss risks of pregnancy such as miscarriage if hormones are not stablized first. Recommend lowering testosterone through weight loss to reduce risk. GLP-1 ideal or metformin. Acne and scalp hair loss will likely improve over 3 months, but we can also add a hormonal birth control or spironalactone. Make sure she is aware that spironalactone causes birth defects (3 months washout). Semaglutide requires a 2 month washout before trying to conceive as well. She has not had a period for a year. Will need TVUS and induce menses with progesterone.

Case Studies: Answer

35 year old patient is 282 lbs, has acne, scalp hair thinning, no period for a year. Wants to get pregnant soon.

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Fertility Management Options

Letrozole vs. Clomiphene

Letrozole is now first-line for ovulation induction with higher live birth rates and lower multiple pregnancy risk compared to clomiphene citrate.

Clomiphene remains an option but with higher rates of ovarian hyperstimulation and twin pregnancies.

Lifestyle & Medical

Weight management and insulin sensitizers improve ovulation rates. Metformin may enhance response to ovulation induction therapies.

Lifestyle modifications should be initiated early as foundational therapy for all patients.

ART Referrals

Refer for assisted reproductive technologies (IVF/ICSI) when first-line treatments fail or additional fertility factors are present.

Early REI (Reproductive endocrinology & Infertility) referral to optimizes outcomes for complex cases.

***Severely obese patients have increased risk for miscarriage and are often high risk pregnancies. Discuss these risks with patient before initiating ovulation induction or referral.**

Heart Health & PMOS

Important to monitor blood pressure esp. while taking estradiol or hormonal birth control

Screen for Sleep apnea

Monitor for insulin resisitance and high cholesterol

Mental Health & PMOS

Women with PCOS are 77% more likely to experience anxiety, 53% more likely to have an eating disorder, and over twice as likely to suffer from depression compared to those without the condition

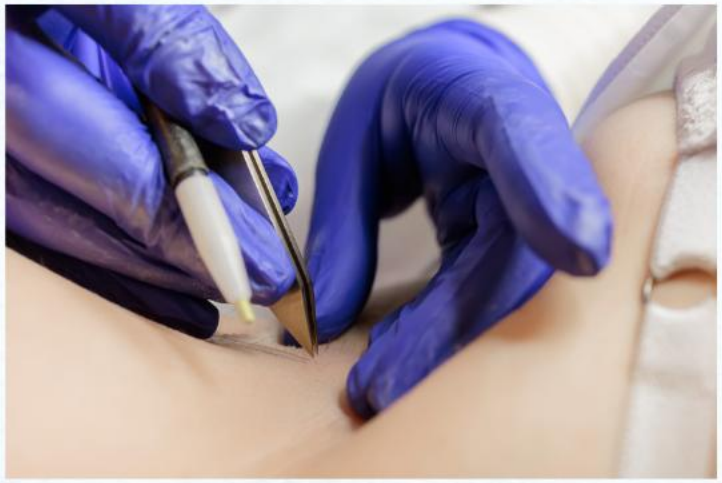
Chen et. al found that women with PCOS who were treated with metformin had a significantly reduced risk of developing bipolar disorder when compared to those who didn't take it (HR: 0.36, 95% CI: 0.16–0.81). Those on hormone therapy also saw a reduced risk, though not as significantly (HR: 0.68, 95% CI: 0.35–1.32). (2020).

This supports the concept that insulin resistance and inflammation—not just hormone levels—play a major role in PCOS and mental health .

Avoid antidepressants and antipsychotics that worsen weight gain and insulin resistance

Hirsutism: Ideal treatment is PMOS medication plus electrolysis

Electrolysis: The only FDA approved permanent hair removal method



Studies have shown laser induced paradoxical hypertrichosis has a prevalence of 16.2% of patients with nearly the double the risk for PMOS patients vs. non PMOS patients (Qeyam et al. 2025)

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Future Directions & Questions

Our team is continuing research into PMOS Screening Tool frameworks and clinical intake methodology. If you are an academic or clinical researcher interested in future scientific collaboration, protocol sharing, or piloting validation frameworks, please contact me at **lynsey@pcossisters.com**