PMOS

previously named

PCOS

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Michelle Mildenberg New York Times 9/4/2020

Disclosure

I have no actual or potential conflict of interest in relation to this program/presentation.

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Polyendocrine metabolic ovarian syndrome, the new name for polycystic ovary syndrome: a multistep global consensus process

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Panel 1: Context and the case for a new name

The term polycystic ovary syndrome (PCOS) has long been recognised as inaccurate and potentially harmful. The following evidence-based considerations informed the need for a new name:

- The term polycystic ovary implies the presence of pathological ovarian cysts, which are not a feature of the condition. This misnomer contributes to misunderstandings among patients, clinicians, policy makers, and the public.
- PCOS encompasses diverse endocrine, metabolic, reproductive, psychological, and dermatological features. The current name reflects only one organ and fails to capture the disorder's multisystem nature.
- Confusion arising from the current name can delay diagnosis and hinder effective communication between patients and health professionals, contributing to patient dissatisfaction with care.
- The reproductive focus of the name can reinforce stigma, particularly in sociocultural contexts where fertility carries high value. Many individuals report distress associated with the name itself.
- The misnomer complicates epidemiological classification, research comparability, and health system coding. A more accurate name is expected to improve scientific coherence, research funding, and policy alignment.
- International guidelines, expert groups, and patient organisations have repeatedly called for renaming, with serial surveys and workshops culminating in a mandate to change the name through a rigorous, global consensus process.
- A new name must support long-term clinical care, research, and global adoption, and enable a smooth transition from existing terminology.

Teede H, Khomami M, Morman R et al.
Polyendocrine metabolic ovarian syndrome, the new name for polycystic ovary syndrome: a multistep global consensus process
The Lancet, 2026; 407, 2329-2339

Agenda for today..

- Prevalence, significance
- Diagnostic definition of PMOS
- Pathophysiology of PMOS
- 4 phenotypes of PCOS
- Evaluation of PMOS
  - History
  - Exam
  - Differential diagnosis
  - Diagnostic testing
- Management



PMOS prevalence

The most common endocrine disorder in reproductive age women. Affects 10-13% of women globally

- 1 in 8 women or more than 170 million reproductive aged women worldwide

Rotterdam criteria:

- Eastern Mediterranean (15.1%)
- South-East Asian (14.3%) regions,
- Europe (11.7%),
- Americas (10.5%),
- Western Pacific (9.1%);
- no data were available from Africa.

Accounts for about 20% of oligmenorrhea/amenorrhea

Nature Reviews. Disease Primers. 2024. Stener-Victorin E, Teede H, Norman RJ, et al] lancet

PMOS Genetics

Familial clustering and twin studies strongly support an underlying genetic basis for PMOS.

- Having mother or sister with PMOS confers a 30-50% risk

* differentially expressed in normal and neoplastic cells domain-containing protein 1
** thyroid adenoma-associated protein
† Azziz R, et al. Polycystic ovary syndrome. Nature Reviews Disease Primers. 2016
‡ Yildiz, B, et al. Impact of obesity on the risk for polycystic ovary syndrome. JCEM. 2008

PMOS diagnostic criteria

NIH Consensus criteria (1990)

Must have both:

a. Androgen excess (clinical or biochemical)

b. Oligo-anovulation

Other causes must be excluded

Rotterdam criteria (2003)

Must have 2 or more:

a. Increased Androgens (clinical or biochemical)

b. Oligo-anovulation

c. Polycystic ovaries on TVUS or high AMH (>12)

Other causes must be excluded

Androgen Excess Society criteria (2006)

Must have both:

a. Androgen excess (clinical or biochemical)

b. Oligo-anovulation **or** Polycystic ovaries on TVUS

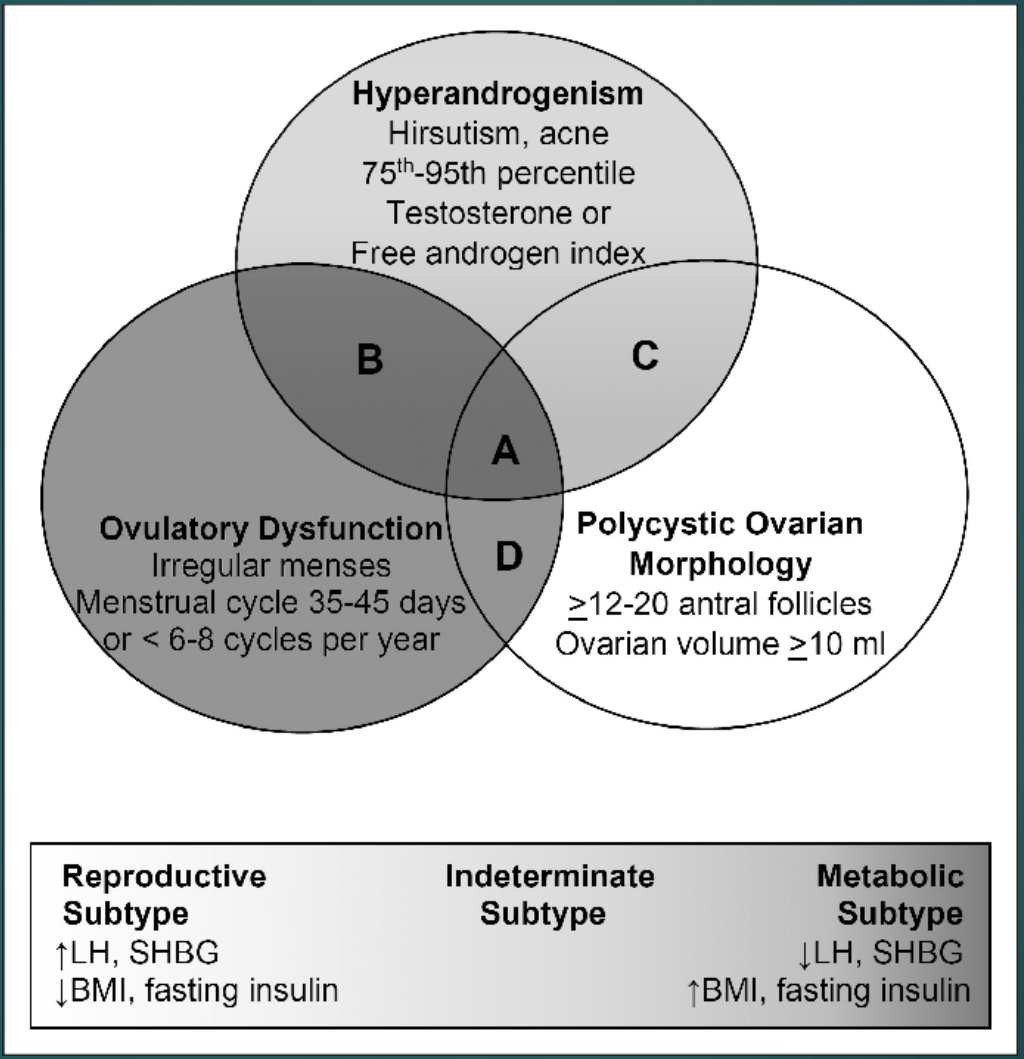
Other causes must be excluded

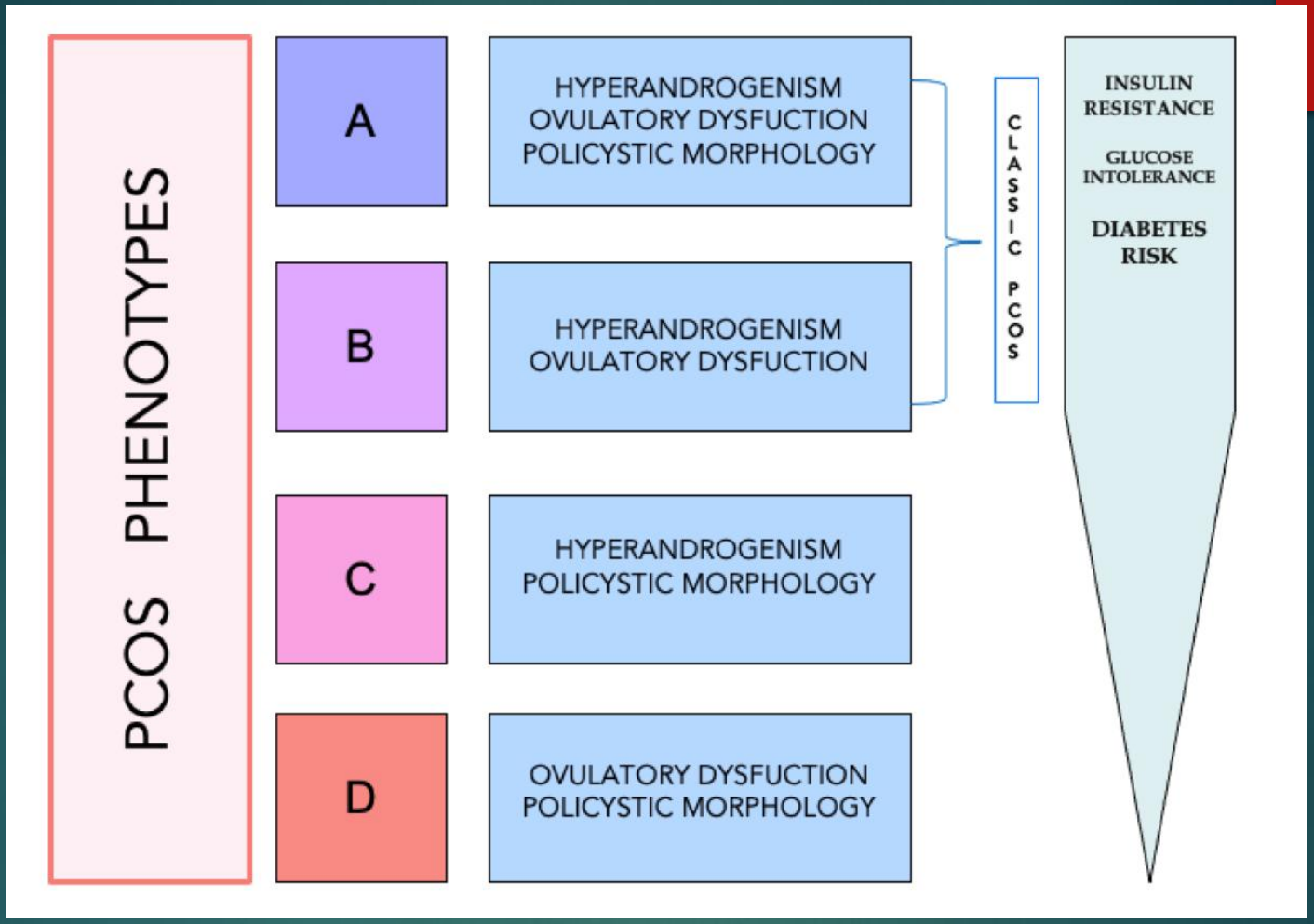
Polycystic ovarian morphologic features are defined as 12 or more antral follicles (2 to 9 mm in diameter) in either ovary, an ovarian volume that is greater than 10 ml in either ovary, or both.



Menstrual cycle

- Normal menstrual cycle is 21-35 (28 days most common)
- By convention, the 1st day of menstruation is "day 1" of a cycle.
- Menstrual cycles have 3 phases
  - Follicular phase
  - Ovulatory phase
  - Luteal phase


Hyperandrogenism
Hirsutism, acne
75th-95th percentile
Testosterone or
Free androgen index
B
C
A
D
Ovulatory Dysfunction
Irregular menses
Menstrual cycle 35-45 days
or < 6-8 cycles per year
Polycystic Ovarian Morphology
≥12-20 antral follicles
Ovarian volume ≥10 ml
Reproductive Subtype
↑LH, SHBG
↓BMI, fasting insulin
Indeterminate Subtype
Metabolic Subtype
↓LH, SHBG
↑BMI, fasting insulin



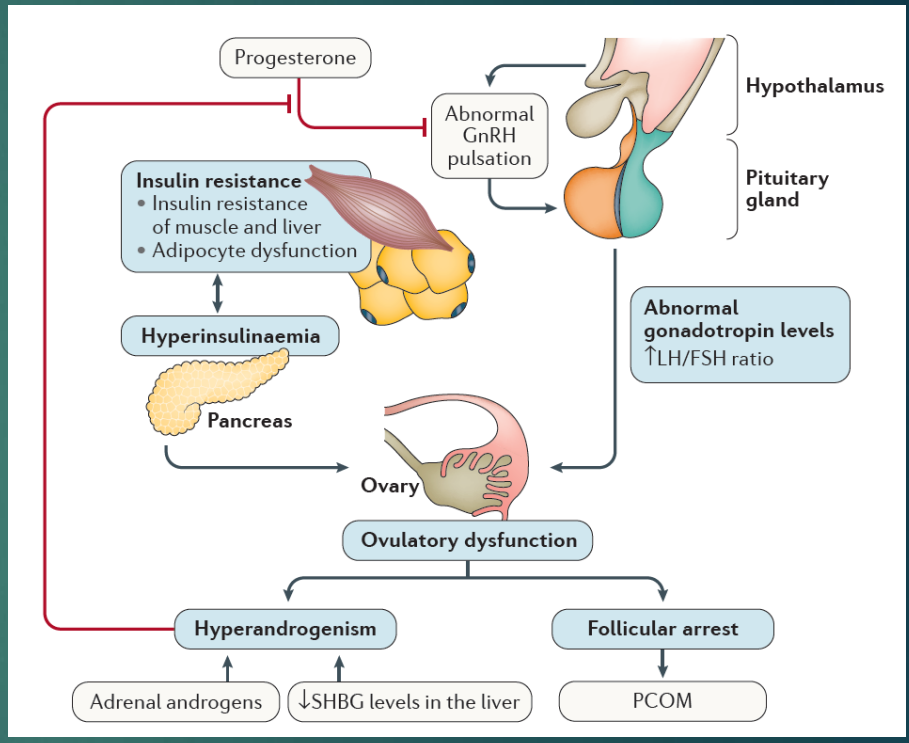
Mancini A, Bruno C, Vergani E, d'Abate C, Giacchi E, Silvestrini A. Oxidative stress and low-grade inflammation in polycystic ovary syndrome: controversies and new insights. *Int J Mol Sci.* 2021;22(4):1667. doi:10.3390/ijms22041667

PMOS pathophysiology

- Poly-metabolic ovary syndrome develops when a chronic anovulatory state persists for a sufficient length of time
- There are currently 3 mechanisms thought to be involved, to different degrees depending on the patient's phenotype

1: Increased GnRH pulse frequency

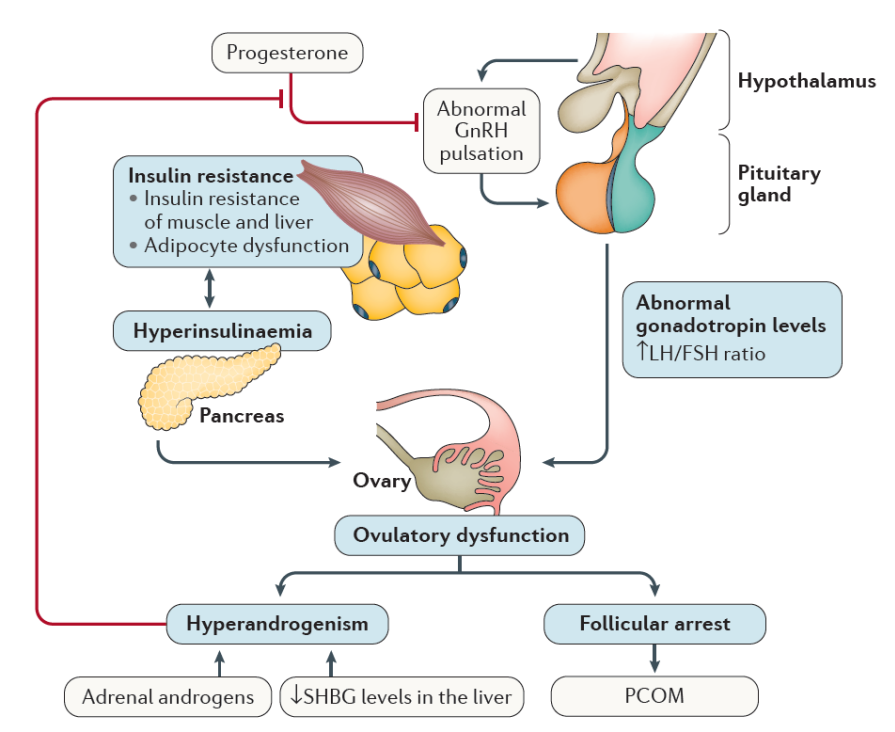
- Increased GnRH pulse frequency results in increased LH secretion and decreased FSH secretion.
- ↑LH ↓FSH results in:
  - increased androgen production
  - interference with normal follicular development
- ↑ androgens further exacerbate the problem by inhibiting progesterone-mediated inhibition of GnRH pulses.



† Azziz R, et al. Polycystic ovary syndrome. Nature Reviews Disease Primers. 2016

2. Inherent abnormalities of ovarian (and adrenal) steroidogenesis.

- Theca cells have increased sensitivity to LH and secrete more androgens.
- Adrenal androgen production (DHEA-S) is increased in at least 30% of women with PCOS, but the mechanism is unknown.

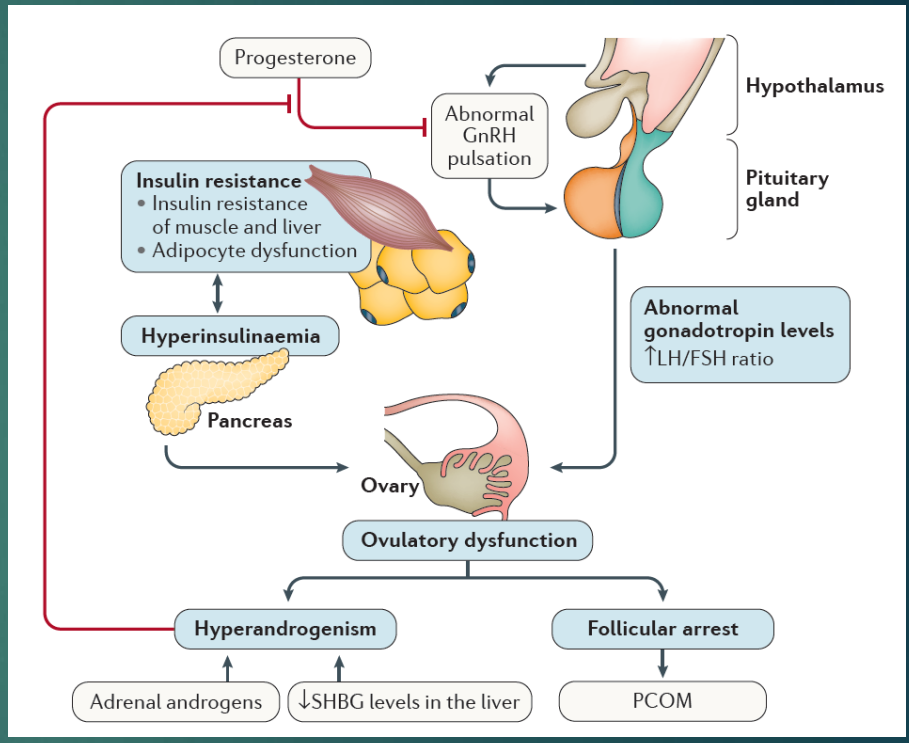


† Azziz R, et al. Polycystic ovary syndrome. Nature Reviews Disease Primers. 2016

3. Insulin resistance and hyperinsulinemia

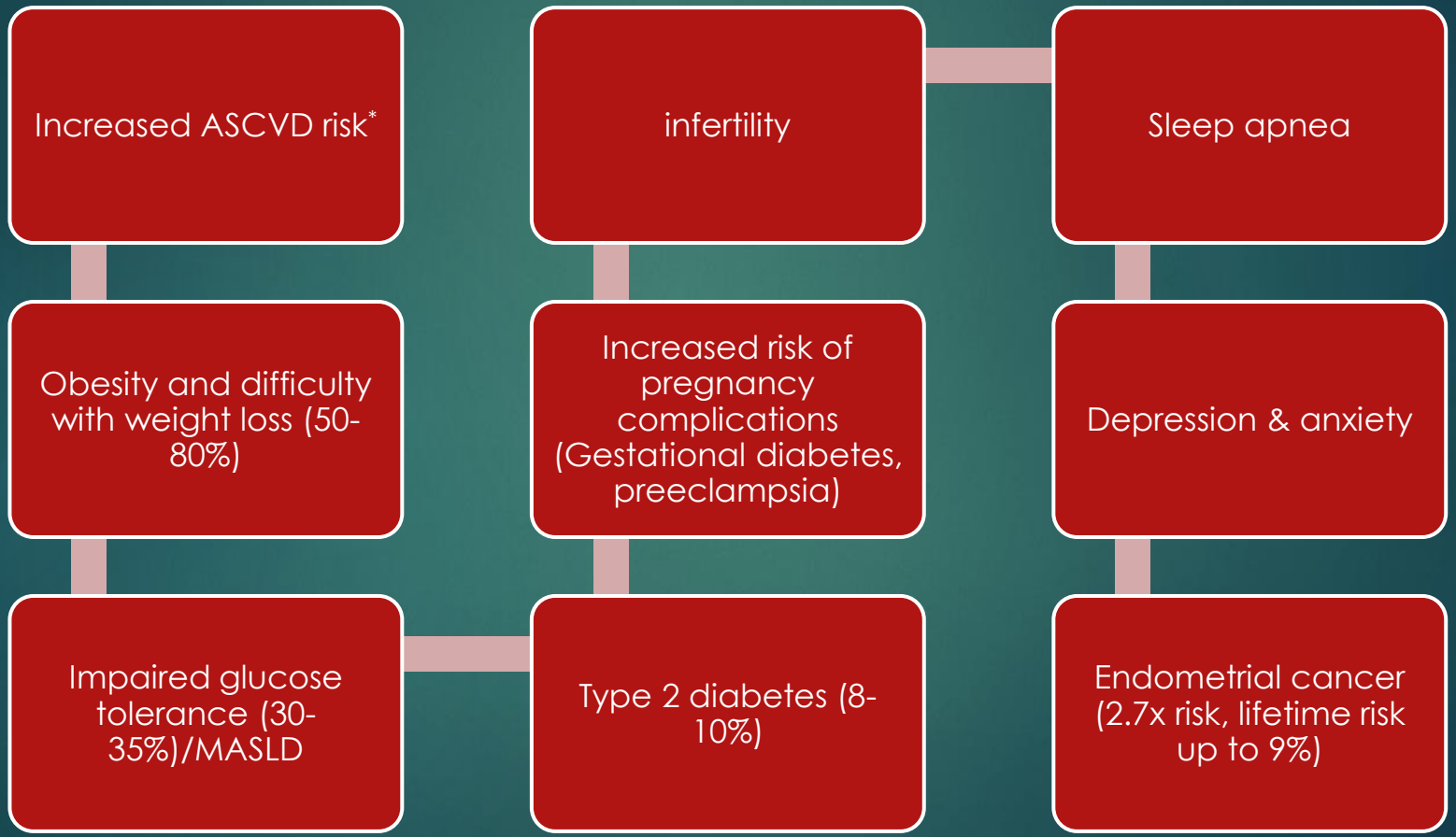
Insulin resistance:

- ↑ LH-stimulated androgen production by ovarian theca cells
- potentiates corticotropin-mediated adrenal androgen production
- inhibits hepatic synthesis of sex hormone–binding globulin (SHBG), which increases free testosterone levels.



† Azziz R, et al. Polycystic ovary syndrome. Nature Reviews Disease Primers. 2016

PMOS comorbidities and sequelae



- Wild RA, et al. Assessment of cardiovascular risk and prevention of cardiovascular disease in women with the polycystic ovary syndrome: a consensus statement by the Androgen Excess and Polycystic Ovary Syndrome (AE-PCOS) Society. JCEM 2010
- Dumesic DA, Lobo RA. Cancer risk and PCOS. Steroids 2013



History and Exam

Patient history: Irregular menstrual frequency

Ovulatory dysfunction is presumed if there is irregular menstrual cycle frequency, definition

- From 3 years post-menarche to menopause: < 21 days or > 35 days or < 8 cycles/yr
- In adolescents, the following are also irregular:
  - From one year post-menarche: > 90 days from prior cycle
  - Primary amenorrhea 3 years post-thelarche or after age 15

Ovulatory dysfunction can also be present in women with regular menses

- 15-40% of hyper-androgenic patients have regular menses†.
- Serum progesterone during midluteal phase (days 21-22) < 7 c/w anovulation*.

* Hull MG, et al. The value of a single serum progesterone measurement in the midluteal phase as a criterion of a potentially fertile cycle ("ovulation") derived from treated and untreated conception cycles. Fertil Steril. 1982

† Azziz R, Carmina E, Dewailly D, et al. The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report. Fertil Steril 2009

Patient history

- Hirsutism – excessive terminal hair
  - Less prominent in women of eastern Asian descent
  - More prominent in Hispanic, Mediterranean, and Middle Eastern women.
  - Below umbilicus, upper shoulder, inner thigh, chin, upper lip
- Acne
- Excess hair loss – hair thinning
- Acanthosis nigricans and skin tags
- Weight gain – abdominal fat
- Clitoromegaly (>10mm in length)
- Lack of pregnancy with repeated unprotected intercourse

Physical exam: Acanthosis nigricans & skin tags










Differential diagnosis

Differential diagnosis of PMOS

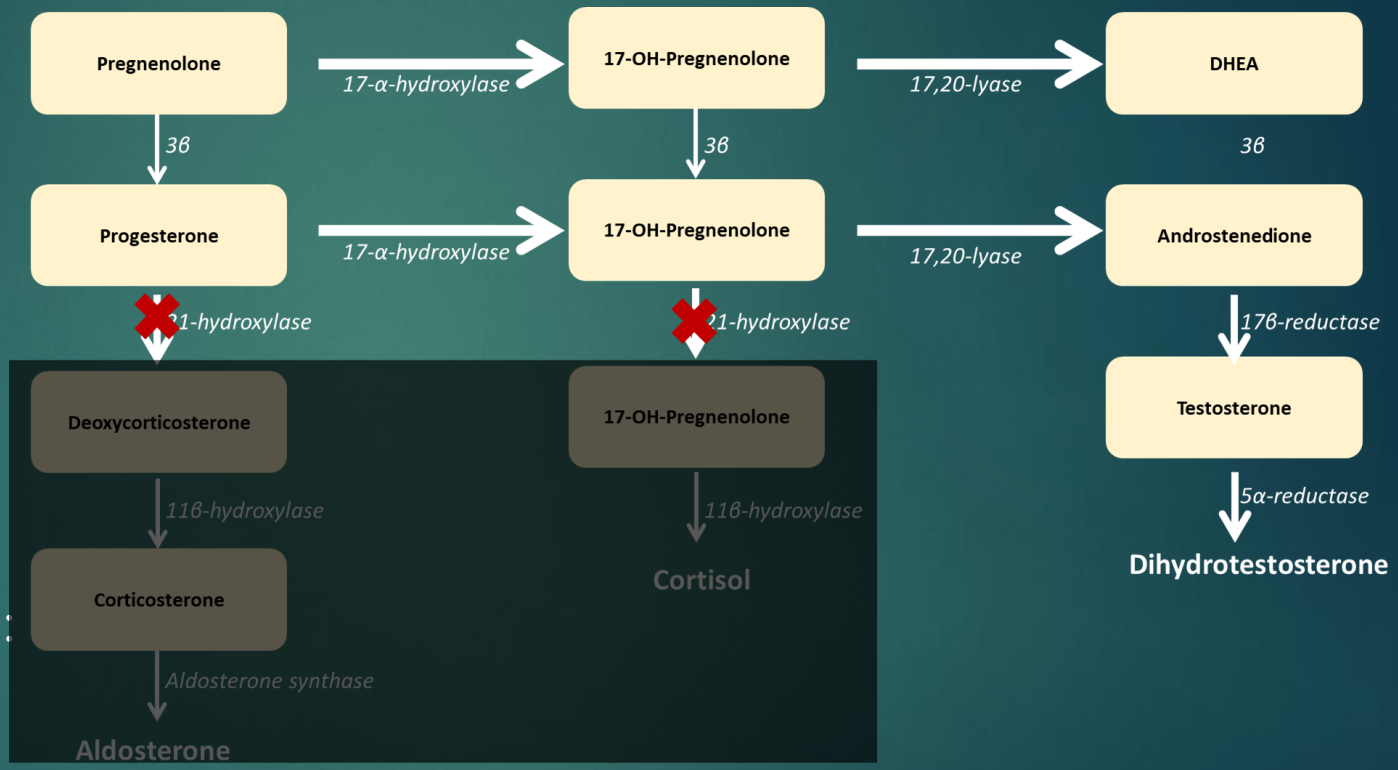
- Idiopathic hirsutism
- Non-classical CAH
- Virilizing tumors of the adrenal or ovary – typically later in life
- Cushing's syndrome
- Hypothyroidism
- Acromegaly
- Prolactinoma
- Premature ovarian insufficiency - AMH
- Functional hypothalamic amenorrhea
- Medication-induced hyperandrogenism
  - androgens, danazol, glucocorticoids, Dilantin, diazoxide, minoxidil
- Partial androgen insensitivity
- 5a-reductase deficiency

Differential diagnosis of PMOS: Nonclassical ("late onset") CAH

Caused by autosomal recessive defect of 21-hydroxylase

1-4% prevalence in American white and Hispanic women.

Highest prevalence: Mediterranean, Hispanic, Slavic, Ashkenazi Jewish





Diagnostic Testing

Lab evaluation: assessing for hyperandrogenemia

- Androstendione/DHEA-s
- Total testosterone (ONLY USE LC-MS)
  - Elevated in most, but not all, pts with PCOS
  - Immunoassays on the scale of levels present in women are inaccurate. For testosterone levels below 100ng/dL, 56–90% were ± 20% of those measured by the gold standard*†.
- **Free testosterone (by equilibrium dialysis)**
- SHBG (typically low)
  - Allows you to calculate free testosterone, but this is typically unnecessary.

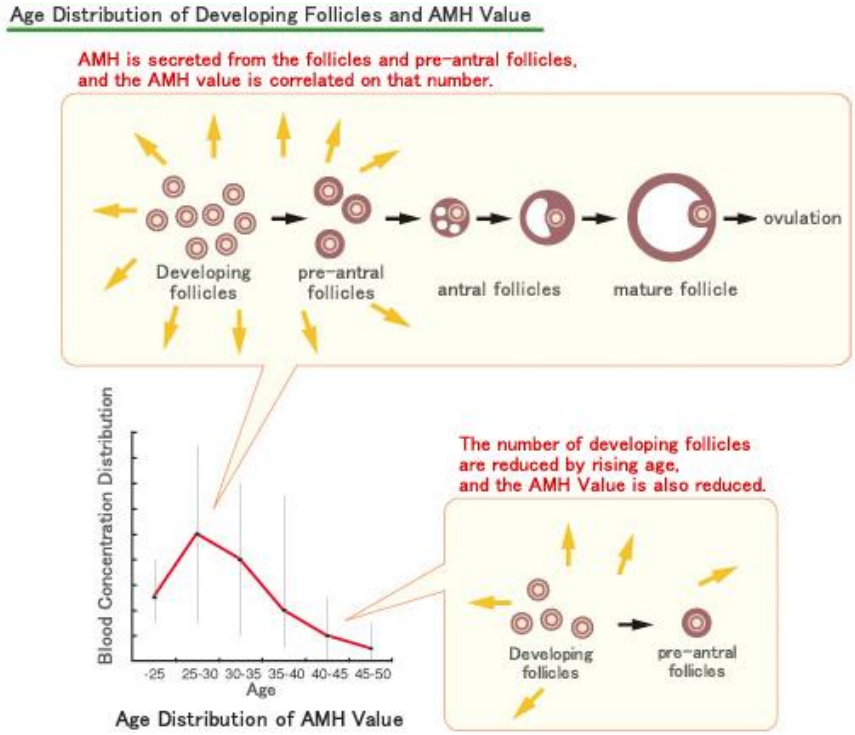
* Wang C, et al. Measurement of total serum testosterone in adult men: comparison of current laboratory methods versus liquid chromatography-tandem mass spectrometry. JCEM 2004.
† Miller KK, et al. Measurement of free testosterone in normal women and women with androgen deficiency: comparison of methods JCEM. 2004.
‡ Carmina E. Ovarian and adrenal hyperandrogenism. Ann N Y Acad Sci. 2006

Lab evaluation: assessing for ovulatory dysfunction

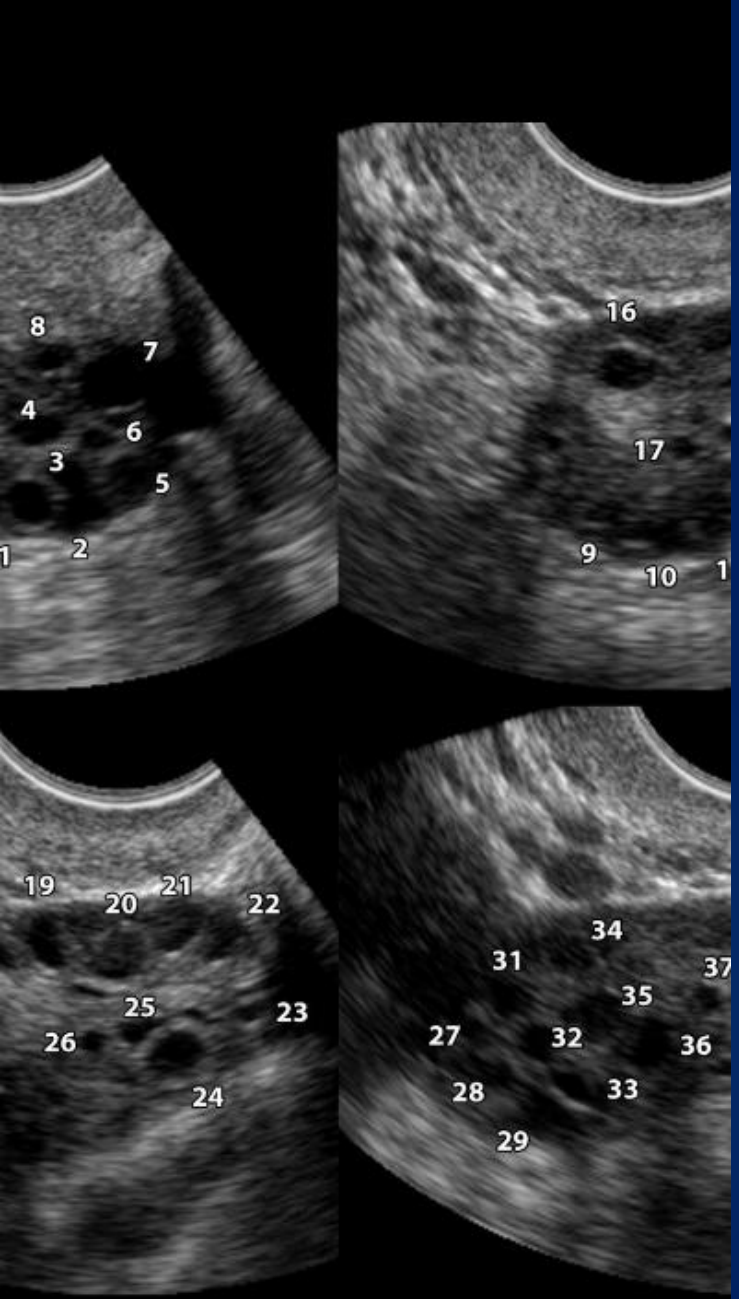
Anti-Mullerian Hormone

Elevated levels (>4.7ng/mL) can be a useful substitute for ovarian morphology

Should not be used for adolescents



van der Ham K, Laven J, Tay C ...**Anti-müllerian hormone as a diagnostic biomarker for polycystic ovary syndrome and polycystic ovarian morphology: a systematic review and meta-analysis,** Fertility and Sterility, 2024; 122, 727-739



Imaging evaluation of PMOS

PCO Morphology*

- ≥ 20 follicles (2-9mm) on either ovary and/or Increased volume (≥ 10 ml) on either ovary
- These are updated based on newer US technology

Very Important notes

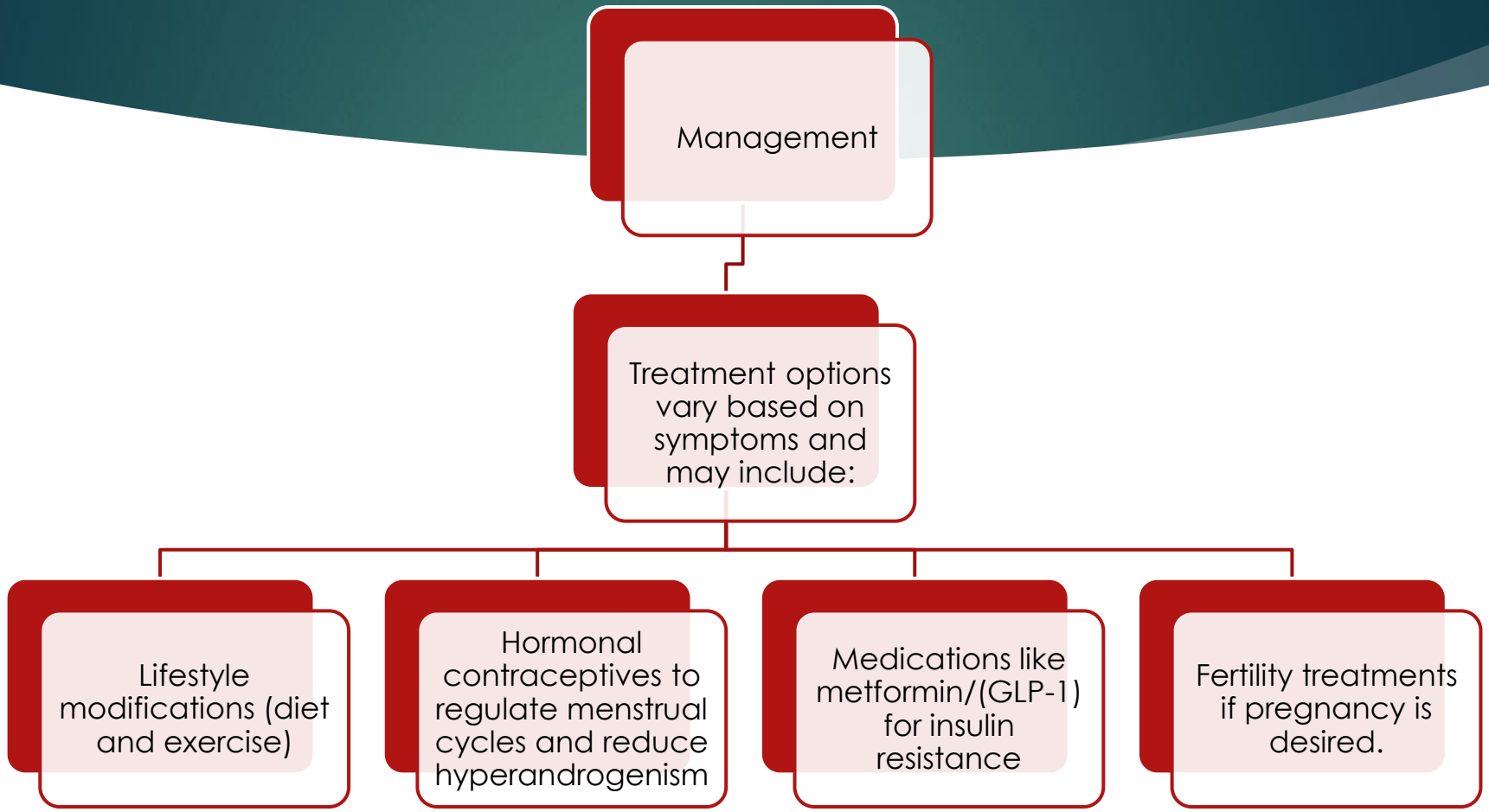
- Up to 30% of normal women can have PCOM*. (that is not a typo)
- PCOM can be seen in patients with functional hypothalamic anovulation and hyperprolactinemia.
- Do not use these criteria in women on OCPs (OCPs may cause false negative findings)

Polycystic ovaries do not establish and are not required for diagnosis of PCOS

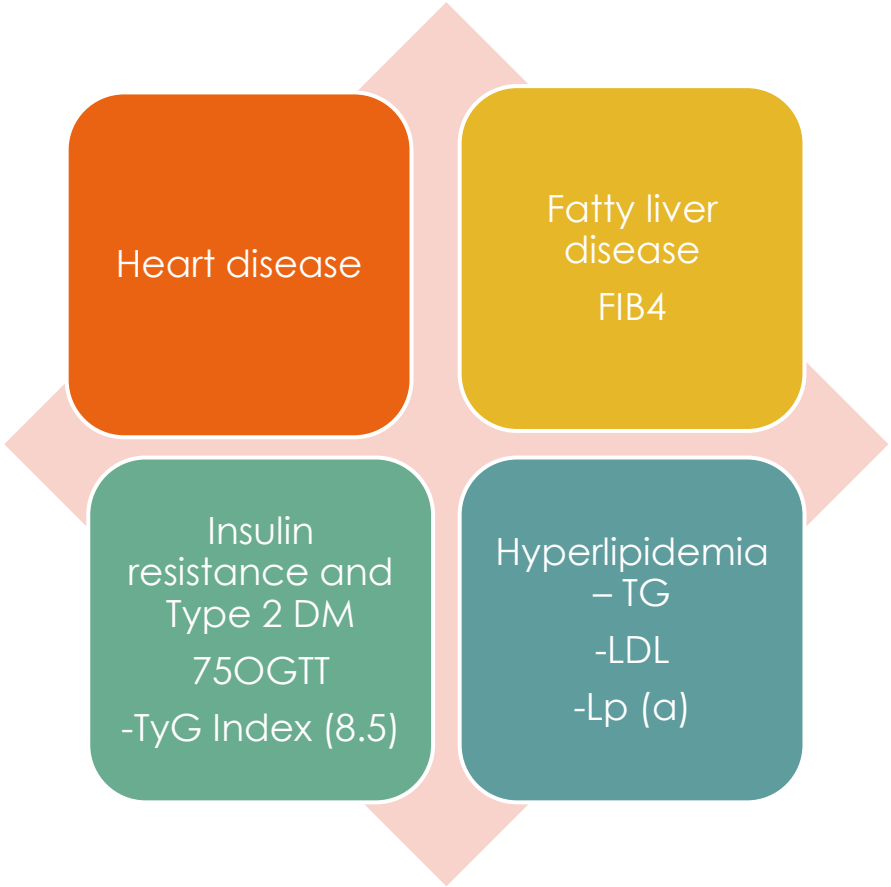
* Dewailly D, et al. Definition and significance of polycystic ovarian morphology: a task force report from the Androgen Excess and Polycystic Ovary Syndrome Society. Hum Reprod Update. 2014



Management of PMOS


Management
Treatment options vary based on symptoms and may include:
Lifestyle modifications (diet and exercise)
Hormonal contraceptives to regulate menstrual cycles and reduce hyperandrogenism
Medications like metformin/(GLP-1) for insulin resistance
Fertility treatments if pregnancy is desired.

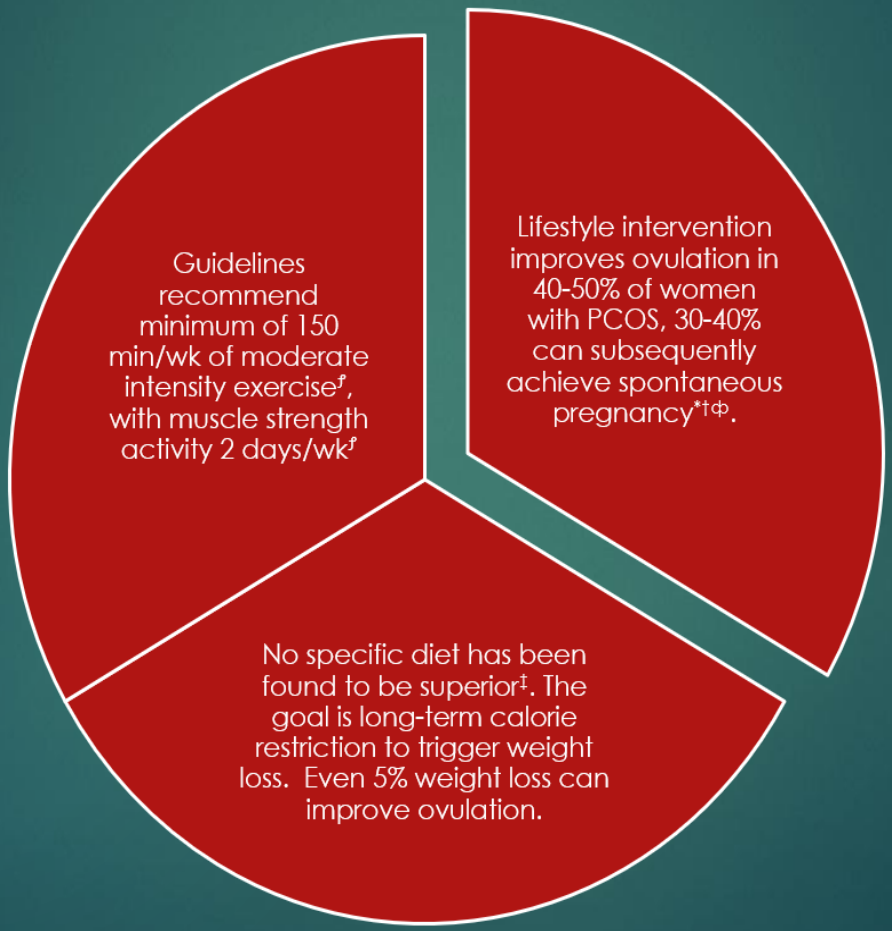
Poly-endocrine Cardio-metabolic syndrome



Workup after diagnosis of PMOS is made

2hr OGTT – 75 g • 35% have impaired glucose tolerance, 10% have diabetes	~~Don't check fasting insulin (this is Controversial)~~	Fasting lipid profile	Screening questions for sleep apnea
Screen for depression and anxiety	Check blood pressure	Check BMI	Fasting CMP (glucose and LFT'S)

Lifestyle changes – reduce the risk of CVD in women



Lipid goals – AHA

LDL-C <100 mg/dL is the general target for women

LDL-C <70 mg/dL for those who have additional cardiovascular risk factors.

Alvarez YR, Pico M, Ashokprabhu N, et al. Polycystic Ovarian Syndrome: a Risk Factor for Cardiovascular Disease. Current Atherosclerosis Reports. 2023.

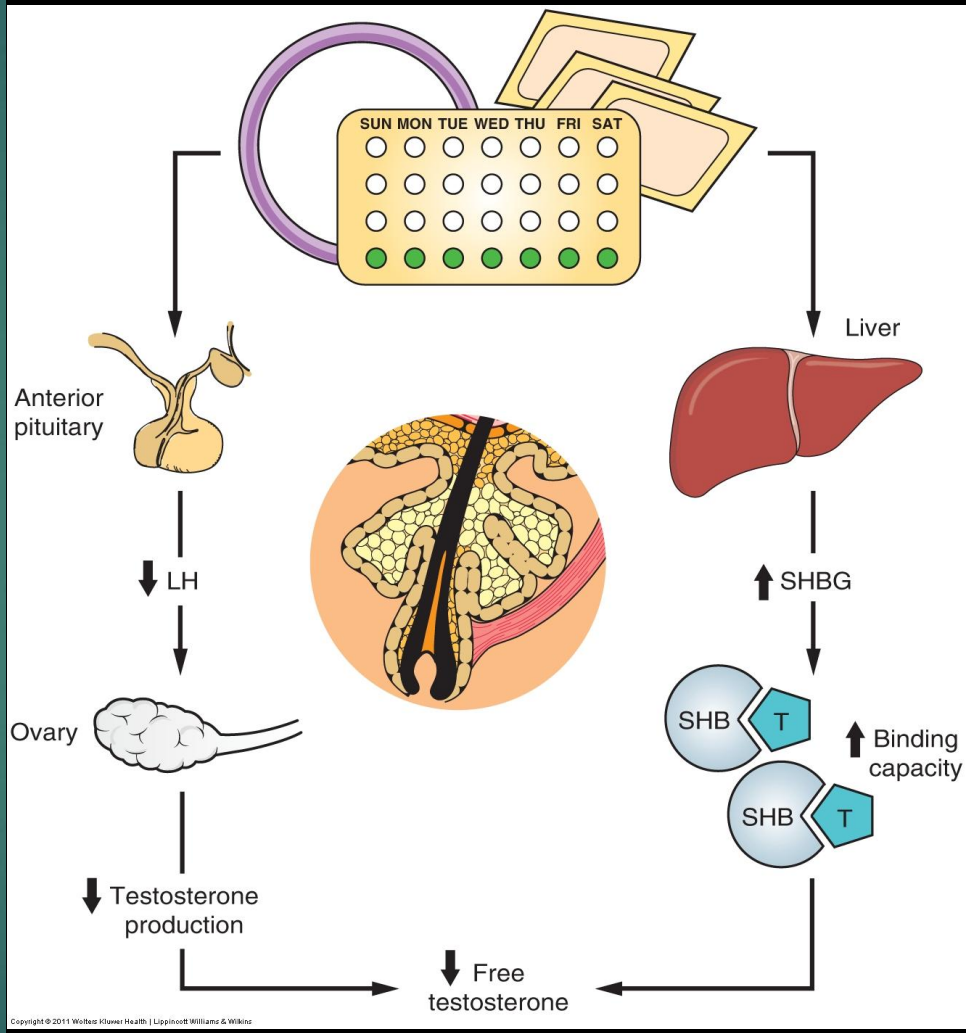
Combined OCP

Mechanism of Action

- Suppress of LH secretion and ovarian androgen production
- Stimulate SHBG, thereby lowering free testosterone levels

Benefits

- Reduces ovarian androgens
- Corrects irregular menstrual cycles
- Reduces risk of endometrial hyperplasia and cancer



*Fritz MA, Speroff L. Figure 13.11. Clinical Gynecology Endocrinology and Infertility, 8th Edition. Lipincott Williams & Wilkins. 2011.

Combined OCP

Estrogen (essentially all are ethinyl estradiol):

- Dose must be at least 20mg to suppress LH

Progestin

- Avoiding progestins with high androgenic effects. (Ex: norgestrel, levonorgestrel, norethindrone)
- Newer progestins have greater progestogenic and lower androgenic effect (Norgestimate, desogestrel, drospirenone)

Which COCP to choose?

- No one formulation is superior*
- Most OCPs have adequate ethinyl estradiol (20-35mg), so initial choice is more based on the progestin
- OCPs with Norgestimate or Desogestrel are a good initial choice

	Estrogen (mcg)	Progestin (mg)
MonoNessa	Ethinyl estradiol 35mg	Norgestimate 0.25mg
OrthoCyclen	Ethinyl estradiol 35mg	Norgestimate 0.25mg
Previfem	Ethinyl estradiol 35mg	Norgestimate 0.25mg
Sprintec	Ethinyl estradiol 35mg	Norgestimate 0.25mg
Apri	Ethinyl estradiol 30mg	Desogestrel 0.15mg
Desogen	Ethinyl estradiol 30mg	Desogestrel 0.15mg
OrthoCept	Ethinyl estradiol 30mg	Desogestrel 0.15mg
Reclipsen	Ethinyl estradiol 30mg	Desogestrel 0.15mg
Solia	Ethinyl estradiol 30mg	Desogestrel 0.15mg
Ocella	Ethinyl estradiol 30mg	Drospirenone 3mg
Safyral	Ethinyl estradiol 30mg	Drospirenone 3mg
Syeda	Ethinyl estradiol 30mg	Drospirenone 3mg
Yasmin	Ethinyl estradiol 30mg	Drospirenone 3mg
Zarah	Ethinyl estradiol 30mg	Drospirenone 3mg
Beyaz	Ethinyl estradiol 20mg	Drospirenone 3mg
Gianvi	Ethinyl estradiol 20mg	Drospirenone 3mg
Loryna	Ethinyl estradiol 20mg	Drospirenone 3mg
Vestura	Ethinyl estradiol 20mg	Drospirenone 3mg
Yaz	Ethinyl estradiol 20mg	Drospirenone 3mg

per FDA, OCPs with Drospirenone have increased risk of DVT compared to other OCPs

Legro, R. S. et al. Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline. J. Clin. Endocrinol. Metab

What if the patient can't/won't take an OCP?



If OCP contraindicated or declined, recommend progestin-only therapy to prevent endometrial hyperplasia.

- Cyclic progestin: Medroxyprogesterone 5-10mg 10-14 days /month
- continuous progestin (mini-pill)
- progestin IUD (Mirena)
- progestin depo injection (Depo provera)
- Progestin implant (Implanon)

Downsides of progestin-only

- Will not improve acne or hirsutism
- Cyclic progestin will not provide contraception



Metformin/ GLP-1*

Role in treating PCOS is debated.

Consider off-label use for patients with evidence of insulin resistance, for the following potential benefits:

- Reduced risk of diabetes
- Decrease in hyperinsulinemia→ lower androgens
- Can restore ovulatory cycles in ~50% of patients‡
- Mild weight loss

†N estler JE. Metformin for the treatment of the polycystic ovary syndrome. NEJM. 2008

‡ Moghetti P, et al. Metformin effects on clinical features, endocrine and metabolic profiles, and insulin sensitivity in polycystic ovary syndrome: a randomized, double-blind, placebo-controlled 6-month trial, followed by open, long-term clinical evaluation.

Treatment of hirsutism

- 1st line: OCP
- 2nd line: spironolactone 50-100mg BID
- 3rd line: Finasteride 5mg QD or
- Flutamide 150-250mg QD
- Improvement can take >6 months
- Anti-androgens must be used with contraception due to their teratogenic potential

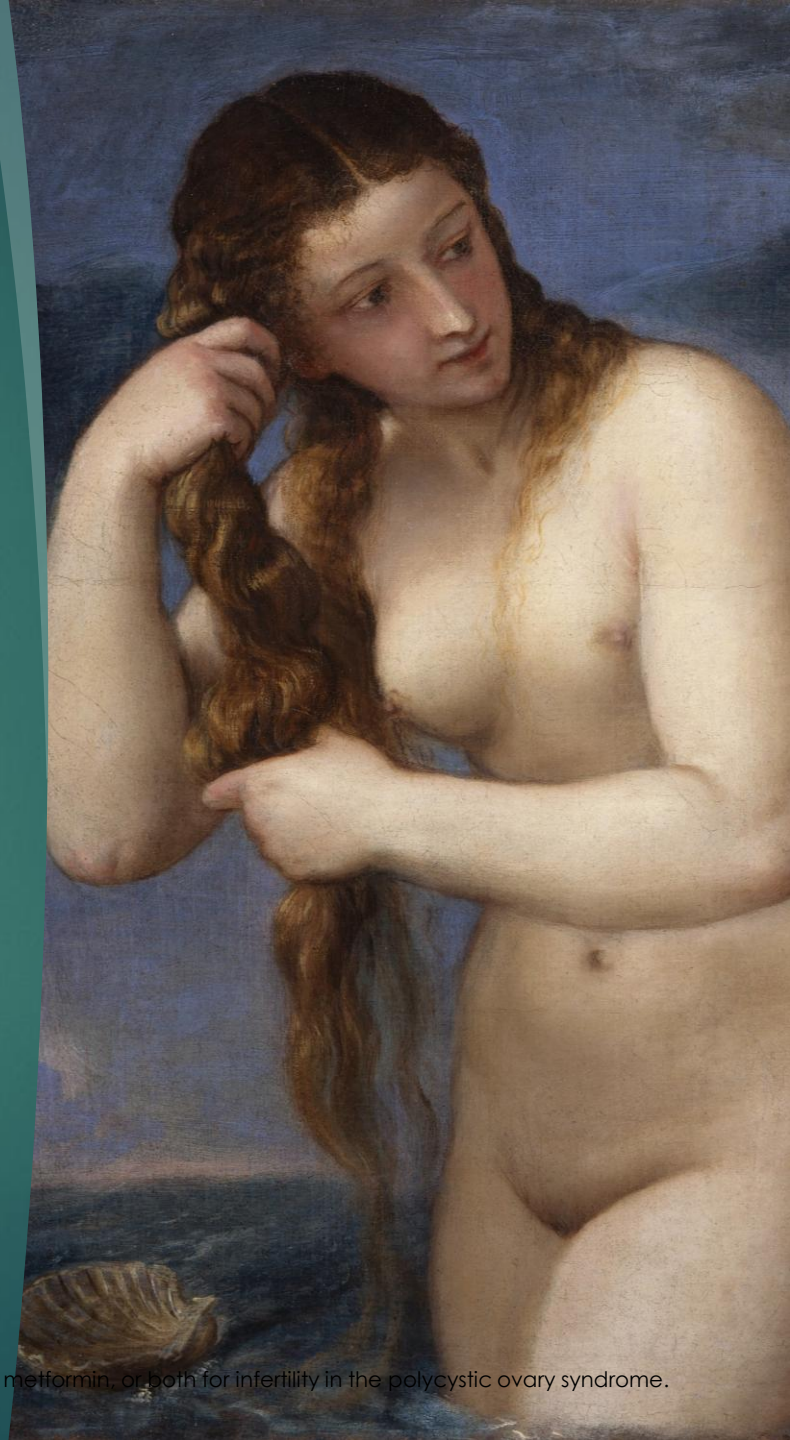


Swiglo, B. A. et al. Clinical review: antiandrogens for the treatment of hirsutism: a systematic review and metaanalyses of randomized controlled trials. JCEM. 2008

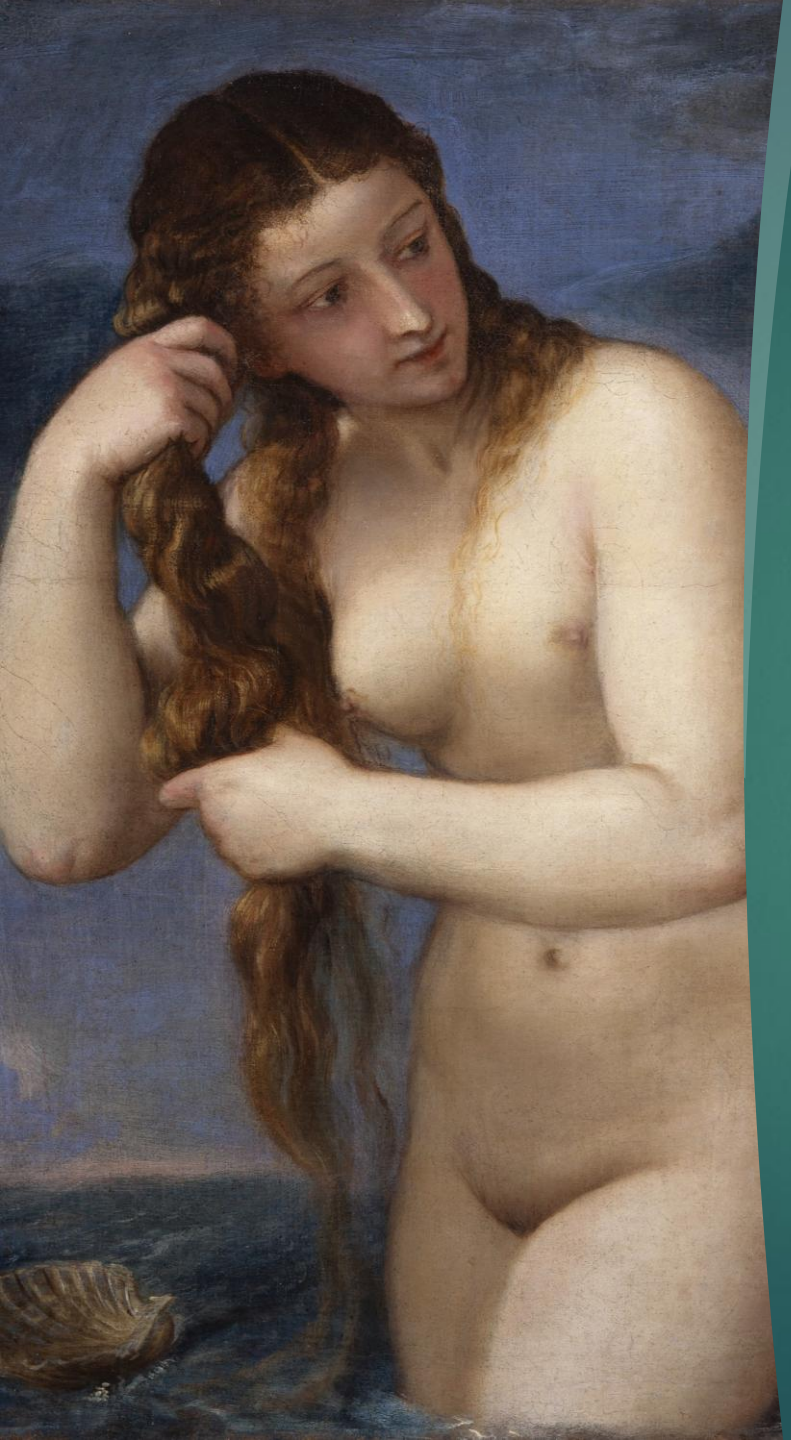
Treatment of Infertility

Goal is to restore ovulatory cycles with lifestyle interventions +/- medical therapy.

Set appropriate expectations. Odds of woman with PCOS conceiving is 5–10% per ovulatory cycle, compared to 10–15% per cycle in women without PCOS*.



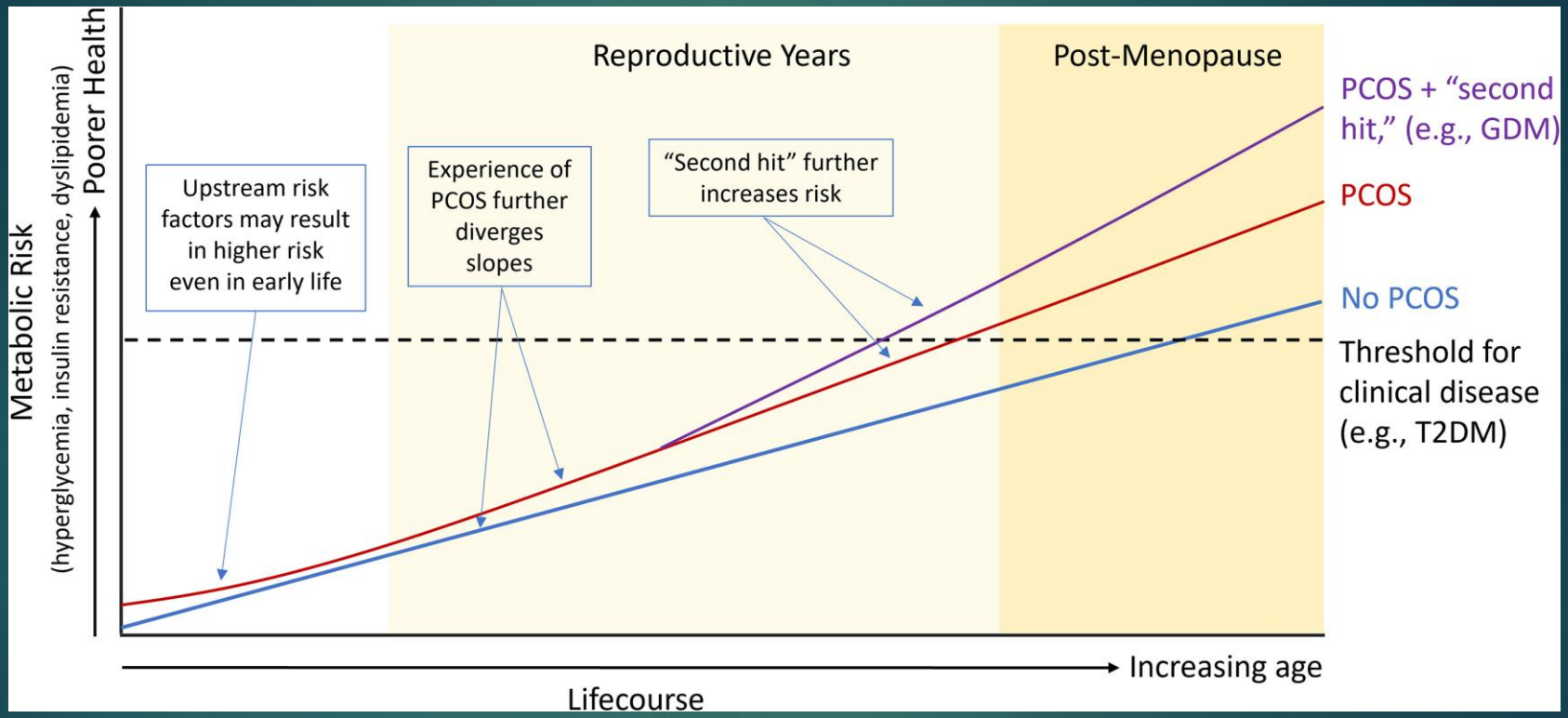
* Legro, R. S. et al. Clomiphene, metformin, or both for infertility in the polycystic ovary syndrome. NEJM. 2008



First line therapy is either Letrozole or Clomiphene citrate

- Letrozole
  - Mechanism: aromatase inhibitor, decreases estrogen production
  - Superior to Clomiphene in achieving pregnancy, but increased rate of multiple pregnancies (3-8%)
- Clomiphene citrate
  - Mechanism: SERM, decreases inappropriate estrogen feedback to CNS to restore ovulation

Into menopause…



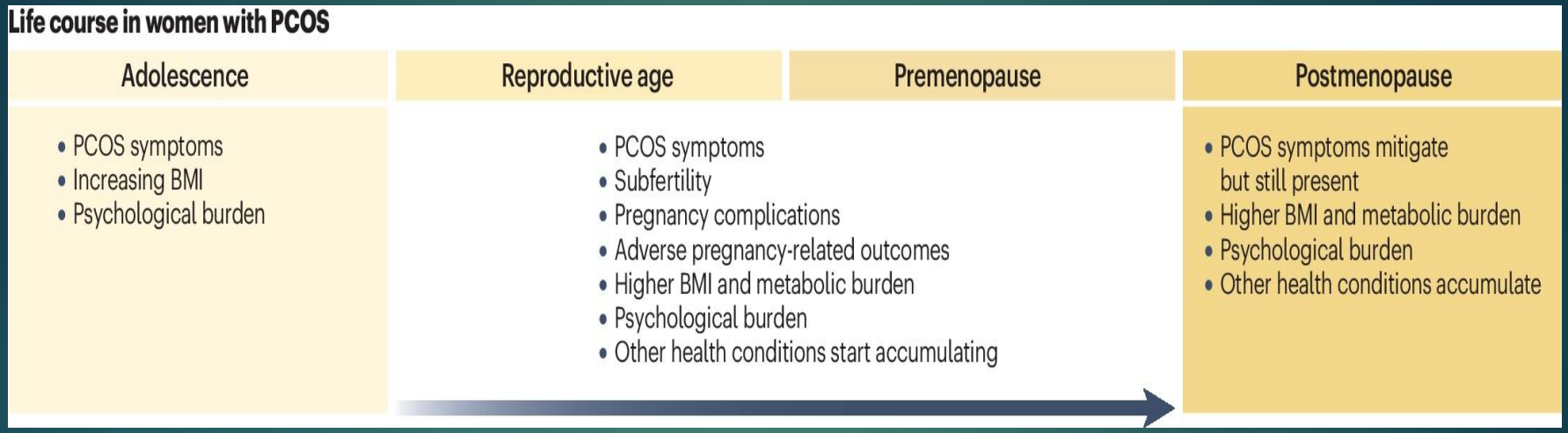
As estrogen levels drop…cardiometabolic features persist

Lipids get worse

More visceral fat – higher BMI

Increased insulin resistance – leads diabetes

Worsening lipid dysfunction


Life course in women with PCOS
Adolescence
Reproductive age
Premenopause
Postmenopause
• PCOS symptoms
• Increasing BMI
• Psychological burden
• PCOS symptoms
• Subfertility
• Pregnancy complications
• Adverse pregnancy-related outcomes
• Higher BMI and metabolic burden
• Psychological burden
• Other health conditions start accumulating
• PCOS symptoms mitigate but still present
• Higher BMI and metabolic burden
• Psychological burden
• Other health conditions accumulate

Thank you