2026 PCRS ANNUAL MEETING

REPRODUCTIVE FRONTIERS: BRIDGING BIOLOGY, PRACTICE, AND POSSIBILITY

MARCH 18-22 | RANCHO MIRAGE, CA

PACIFIC COAST
REPRODUCTIVE
SOCIETY

Mastering the Management of PCOS

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Disclosure

- Neither I nor members of my immediate family have any actual or potential financial interests to disclose relating to the content of this presentation.

Needs Assessment Statement and Expected Learning Outcomes

Past and present diagnostic criteria

Management when not trying to conceive

Fertility treatment

Boots' Secret Objectives

Communication/ Patient Education Tips

Gentle empathy reminder

Be Creative

"…PCOS is not a discrete or specific endocrine disorder having a unique cause or pathophysiology. Instead, the condition is best viewed as a final common pathway in the chronic anovulatory state."

- Marc Fritz, MD

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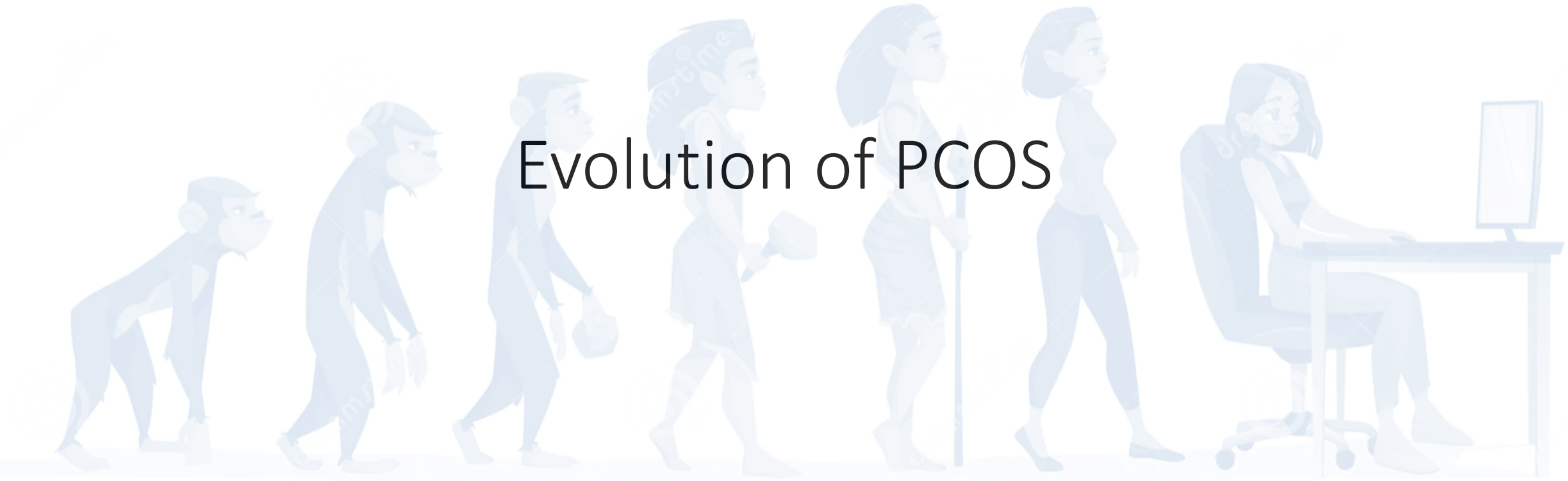
Evolution of PCOS



1935- Stein & Leventhal

- **7 women**
  - Enlarged, polycystic ovaries
  - Hirsutism
  - Oligo-/amenorrhea

→ Wedge resection

→ Resumption of menses within few months

→ 2 spontaneously conceived

TABLE I. BILATERAL POLYCYSTIC OVARIES WITH AMENORRHEA. HISTOPATHOLOGIC FINDINGS

	TUNICA	STROMA	BLOOD VESSELS	NORMAL FOLLICLES	FOLLICLE CYSTS		CORPORA LUTEA	CORPORA ALBICANTIA	TUMOR
					THECA	GRANULOSA			
1	Thick		Tortuous Distended	Few	Many		Recent	Many, old	
2	Thick				Many			Few, old, small	
3	Thick		Tortuous Thick		Many	Few		Few, small	
4	Normal to moderately thick		Tortuous Distended	One in section	Many			Many, small	Early papillary cystadenoma
5	Moderately thick	Edematous Vascular	Hyperplasia		Many	Few	Recent with c.l. cyst	Large, recent	
6	Normal to moderately thick			Few	Many	Few	One, recent	Many	
7	Moderately thick	Vascular	Dilated		Many		c.l. cyst	Many	

Stein & Leventhal. AJOG, 1935

50+ years later…

- **1990 NIH criteria**
  - Hyperandrogenism/ hyperandrogenemia
  - Menstrual dysfunction
- **2003 ESHRE/ASRM Rotterdam**
  - Oligo/anovulation
  - Clinical &/or biochemical signs of hyperandrogenism
  - Polycystic ovaries on u/s
- **2006 Androgen Excess Society**
  - Hyperandrogenism
  - Ovarian dysfunction (oligo/anovulation &/or polycystic ovaries)

TABLE 1. Diagnostic criteria for PCOS

Criteria	NIH 1990 "classic"	Rotterdam 2003	AE-PCOS
Oligomenorrhea[a]	+	+/–	+/–
Clinical or biochemical hyperandrogenism[b]	+	+/–	+
Polycystic ovaries on ultrasound[c]	+/–	+/–	+/–

NIH, Presence of both oligomenorrhea and clinical/biochemical hyperandrogenism; Rotterdam, any two of the above criteria; AE-PCOS, presence of clinical/biochemical hyperandrogenism and one other criterion.

[a] Eight or less menses per year.

[b] Acne or hirsutism or androgenic alopecia.

[c] Ovarian volume >10 ml and/or >12 follicles less than 9 mm in size in at least one ovary.

Wild, et al. JCEM, 2010; PMID: 20375205

ENDOCRINE SOCIETY JCEM THE JOURNAL OF CLINICAL ENDOCRINOLOGY & METABOLISM

J Clin Endocrinol Metab. 2023 Oct; 108(10): 2447–2469.
Published online 2023 Aug 15. doi: 10.1210/clinem/dgad463: 10.1210/clinem/dgad463

PMCID: PMC10505534
PMID: 37580314

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

Helena J Teede,[✉] Chau Thien Tay, Joop J E Laven, Anuja Dokras, Lisa J Moran, Terhi T Piltonen, Michael F Costello, Jacky Boivin, Leanne M Redman, Jacqueline A Boyle, Robert J Norman, Aya Mousa, and Anju E Joham, on behalf of the International PCOS Network

https://www.monash.edu/medicine/mchri/pcos

The Guideline

- Technical evidence report and analyses (~6000 pages) underpins 77 evidence-based and 54 consensus recommendations, with 123 practice points.
- Key updates include:
  - i) further refinement of **individual diagnostic criteria,** a **simplified diagnostic algorithm** and **inclusion of AMH** levels as an alternative to ultrasound in adults only
  - ii) strengthening **recognition of broader features of PCOS** including metabolic risk factors, cardiovascular disease, sleep apnea, very high prevalence of psychological features, and high risk status for adverse outcomes during pregnancy
  - iii) emphasizing the poorly recognized, diverse burden of disease and the need for greater healthcare professional education, evidence-based patient info, improved models of care and shared decision making to improve patient experience, alongside greater research
  - iv) maintained emphasis on **healthy lifestyle, emotional wellbeing and quality of life,** with awareness and consideration of weight stigma
  - v) emphasizing evidence-based medical therapy and cheaper and safer fertility management.

Diagnosis: 2 of the following

- i) clinical/biochemical hyperandrogenism
- ii) ovulatory dysfunction
- iii) polycystic ovaries on ultrasound; and here in 2023, alternatively AMH can now be used instead of ultrasound.

- Exclusion of other etiologies.
- Adolescents: both hyperandrogenism and ovulatory dysfunction are required, with ultrasound and AMH not recommended due to poor specificity.

Algorithm 1: Diagnostic algorithm for polycystic ovary syndrome (PCOS)

Step 1: Irregular cycles + clinical hyperandrogenism
(exclude other causes)* = diagnosis
Step 2: If no clinical hyperandrogenism
Test for biochemical hyperandrogenism (exclude other causes)* = diagnosis
Step 3: If ONLY irregular cycles OR hyperandrogenism
Adolescents ultrasound is not indicated = consider at risk of PCOS and reassess later Adults - **request ultrasound for PCOM***, if positive (exclude other causes)* = diagnosis

Irregular cycles and ovulatory dysfunction

Irregular cycles and ovulatory dysfunction

Irregular menstrual cycles are defined as follows:

- Normal in the first year post menarche as part of the pubertal transition.
- 1 to <3 years post menarche: <21 or >45 days.
- **3 years post menarche to perimenopause: <21 or >35 days or <8 cycles per year.**
- 1 year post menarche >90 days for any 1 cycle.
- Primary amenorrhea by age 15 or >3 years post thelarche (breast development).

When irregular menstrual cycles are present, a diagnosis of PCOS should be considered and assessed according to these PCOS Guidelines.

Check out Table 4- Full summary!

Androgen Excess

Appreciate that unwanted excess hair growth has a high degree of validity and merits close evaluation, even if overt clinical signs of hyperandrogenism are not readily evident on examination

Androgen Excess

- Total and free testosterone
- Validated, highly accurate tandem mass spectrometry (LC-MS/MS) assays
- Not on hormones or discontinued >3mo
- Androgen levels reach adult ranges at 12-15yo
- If super high, eval for other stuff
- Hirsutism alone is predictive of biochemical hyperandrogenism and PCOS
- Recognize that female pattern hair loss and acne in isolation (without hirsutism) are relatively weak predictors of biochemical hyperandrogenism
- Consider ethnicity, time course, self treatment

Polycystic ovarian morphology

Polycystic ovarian morphology

- **Original criteria:**
  - >12 follicles 2-9 mm per whole ovary
- **Current approach:**
  - Follicle number per ovary ≥ 20
    - If good u/s
    - If not, use ovarian volume >10mL

- PCO in regularly cycling women without hyperandrogenism?
  - Confirm ovulation, but usually not significant

Ultrasound Requirements

- Last menstrual period (or stage of cycle).
- Transducer bandwidth frequency.
- Approach/route assessed.
- Total number of 2-9 mm follicles per ovary.
- Measurements in 3 dimensions (in cm) or volume of each ovary.
- Other ovarian features and/or pathology including ovarian cysts, corpus lutea, dominant follicles (≥10 mm) (which should not be included in ovarian volume calculations).
- Reliance on the contralateral ovary FNPO for diagnosis of PCOM, where a dominant follicle is noted.
- Uterine features and/or pathology including endometrial thickness and pattern.

AMH!!!!!

- Polypeptide of TGF-β family
- Solely secreted by granulosa cells of the preantral & small antral follicles
- Correlates with AFC: But……. U/S will give you more info?

Excludes diminished ovarian reserve

No specific value for criteria

Depends on assay

Depends on age

Depends on BMI

Depends on race

Depends on ovarian suppression (on OCP's for last 10yrs?)

PCOS Phenotypes

Complex!
No capture of the metabolic aspect

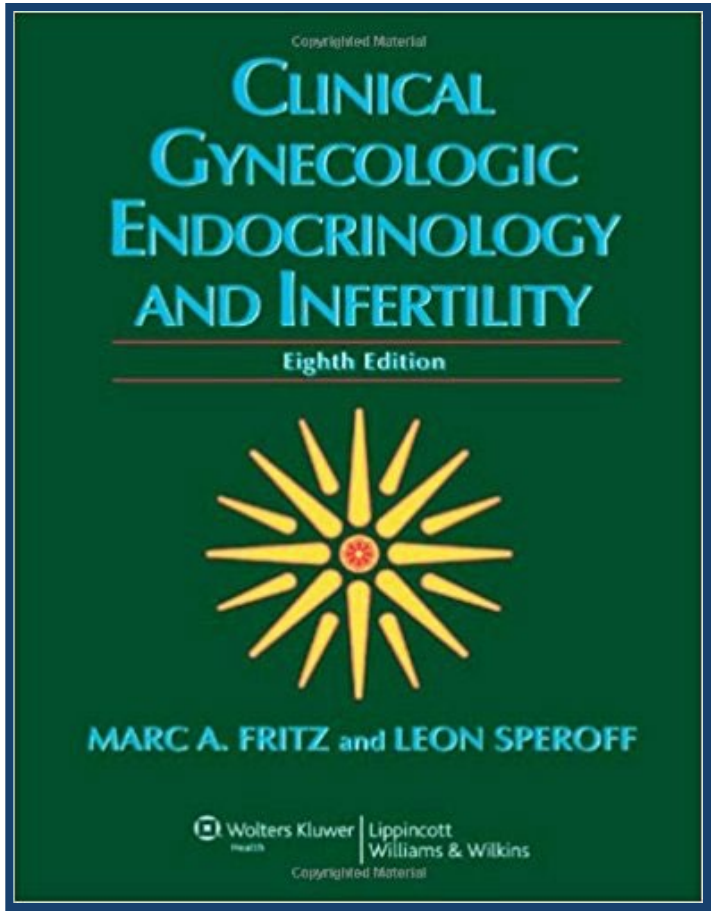
1990 US NIH criteria
2006 AE-PCOS criteria
2003 Rotterdam criteria

	Phenotype A	Phenotype B	Phenotype C	Phenotype D
Hyperandrogenism and hirsutism	**Present**	**Present**	**Present**	Absent
Ovulatory dysfunction	**Present**	**Present**	Absent	**Present**
Polycystic ovarian morphology	**Present**	Absent	**Present**	**Present**

PCOS simplified?

-In our view, the primary advantage to having specific diagnostic criteria for PCOS relates to research, because varying criteria cloud the conclusions and questions the generalizability of results from studies involving women with "PCOS".

-In clinical medicine, simply knowing & understanding the health implications & consequences of chronic anovulation and methods for their effective management are far more important than assigning a specific diagnosis of PCOS…



Managing PCOS today…

- Diagnostic criteria are not critical for clinical care
  - What to focus on?
    - Symptoms drive women to seek care:
      - Oligomenorrhea/amenorrhea, infertility
      - Hyperandrogenism: hirsutism, acne
      - Metabolic health/ weight
    - Opportunity for ***PREVENTION***…

Pathophys…

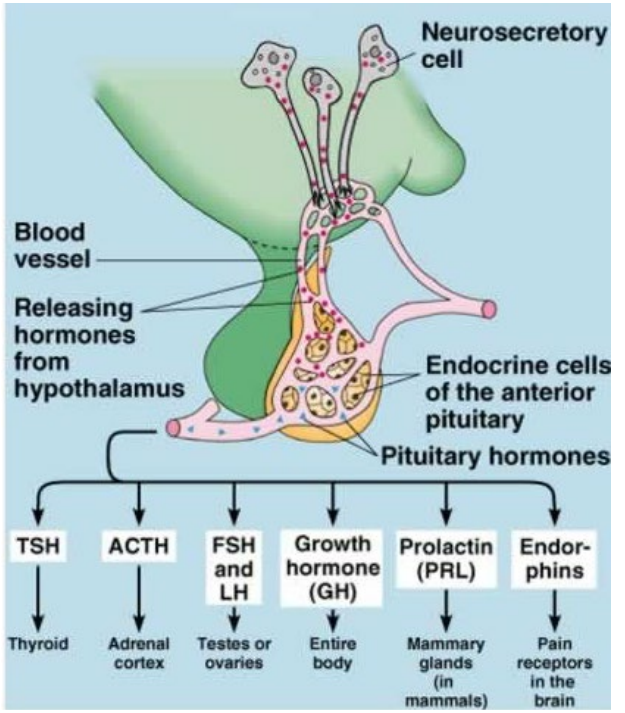
It’s Genetic

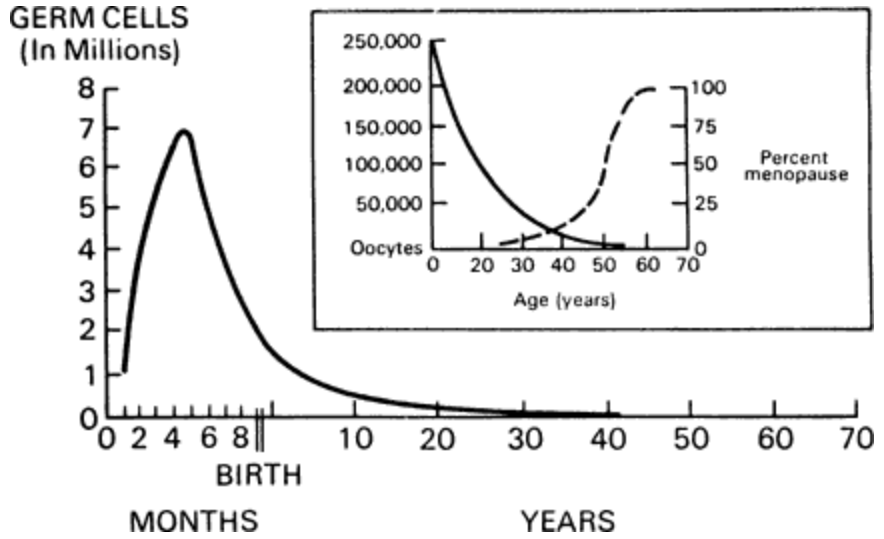
- And also environmental

Ovulatory Dysfunction

HPO Axis

- **Hypothalamic**
  - Structural
  - Functional
  - PCOS
- **Pituitary**
  - Thyroid stimulating hormone
  - Prolactin
  - PCOS
- **Ovarian**
  - Primary ovarian insufficiency
  - PCOS





What causes anovulation?
Why are androgen levels high?
Why are insulin levels high?

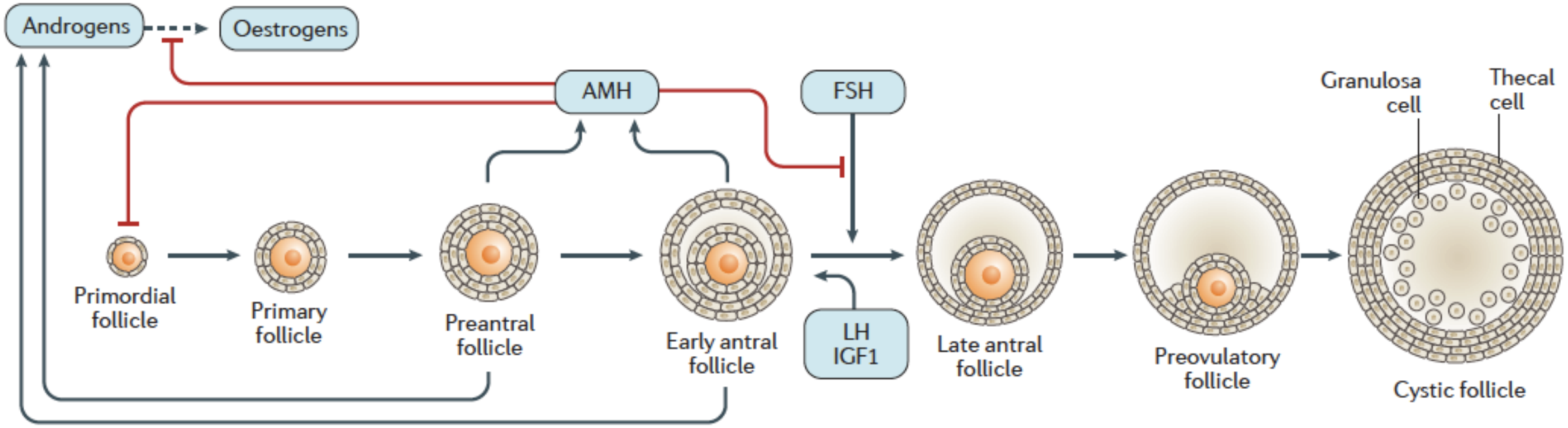


Figure 3 | **Ovarian follicular maturation arrest in PCOS.** Normal ovulation is the result of synchronized signalling between centrally released gonadotropins and factors produced in the developing follicle of the ovary. Anovulation in women with polycystic ovary syndrome (PCOS) is characterized by arrested follicle growth at the early antral stage. Hypersecretion of luteinizing hormone (LH) and insulin-like growth factor 1 (IGF1) leads to hyperandrogenism, which results in follicular maturation arrest[93]. In addition, high levels of anti-Müllerian hormone (AMH) in PCOS block follicle-stimulating hormone (FSH) action, contribute to hyperandrogenism and inhibit the recruitment of further primordial follicles. Dashed line indicates androgen to oestrogen conversion.

Azziz 2016

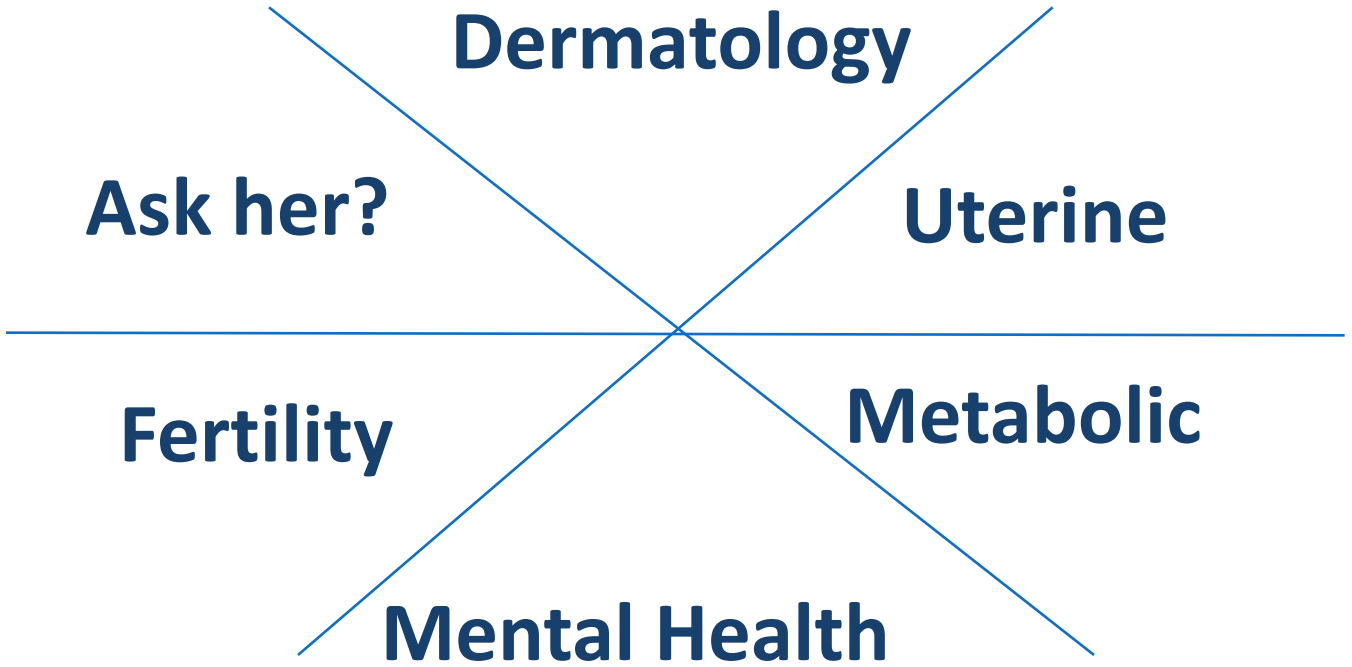
What causes anovulation?
Why are androgen levels high?
Why are insulin levels high?

Hyperandrogenemia

- At low levels, androgens serve as substrate for FSH-induced aromatization and estrogen production.
- At higher concentrations, androgens are converted alternatively to more potent 5α-reduced androgens, which cannot be converted to estrogen and also inhibit aromatase activity and FSH induction of LH receptors on granulosa cells.
- Consequently, abnormally high local androgen concentrations, from any cause, impede follicular maturation, promote atresia, and predispose to a chronic anovulatory state.

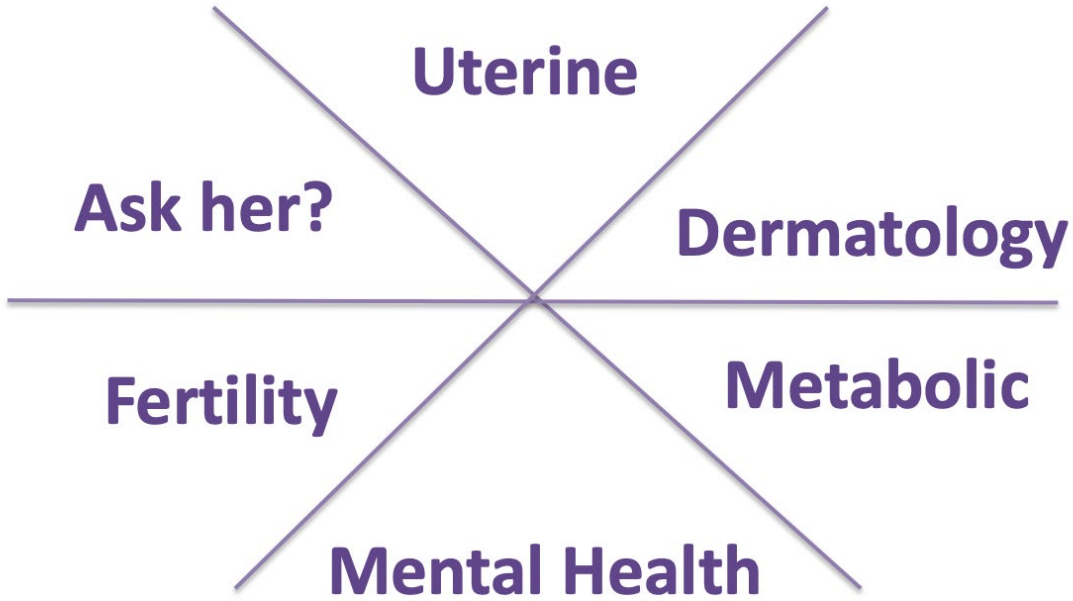
Approach to your patient with PCOS

Approach


Dermatology
Ask her?
Uterine
Fertility
Metabolic
Mental Health

My PCOS dotphrase ;)

Discussed PCOS pathophysiology, etiology, evaluation and treatment options in detail:
1) Endometrium/ menstrual cyclicity:
- Discussed importance of regular cycles if not on meds or taking meds to protect endometrium from hyperplasia/ cancer
- Discussed use of OCPs, nuvaring, IUD, etc
2) Hyperandrogenism: Acne/Hirsutism/Alopecia:
- Discussed symptoms and need for lab evaluation of testosterone
- Discussed use of combined contraceptives and spironolactone
- Discussed comanagement with dermatology
- Discussed expectations of treatment and that terminal hairs will not reve permanent hair removal solutions can do this (ie laser), but that pills can he new terminal hairs
3) Metabolic:
- Discussed risk of CVD, DM, obesity, etc.
- Discussed screening for above
- Discussed importance of lifestyle: diet/nutrition, exercise, weight manag
- Discussed potential benefit of metformin
4) Psychology:
- Discussed risk of depression, anxiety, body image
- Aware of psychology service offered in our office and elsewhere
5) Fertility:
- Discussed causes of infertility, typically anovulation
- Explained treatment options are available if/when needed
- *** egg freezing



Menstrual cycles

- Risk of **endometrial hyperplasia** → Cancer
- Chronic unopposed estrogen
  - Obesity
  - Time
- Uterine protection:
  - Combined estrogen/progesterone
    - Pill, patch, ring
  - Progesterone alone
    - Cyclic oral (Provera or Prometrium)
    - Systemic (Depo Provera or Nexplanon)
    - Intrauterine (IUD- Mirena, liletta, Skyla, etc.)

Hyperandrogenism

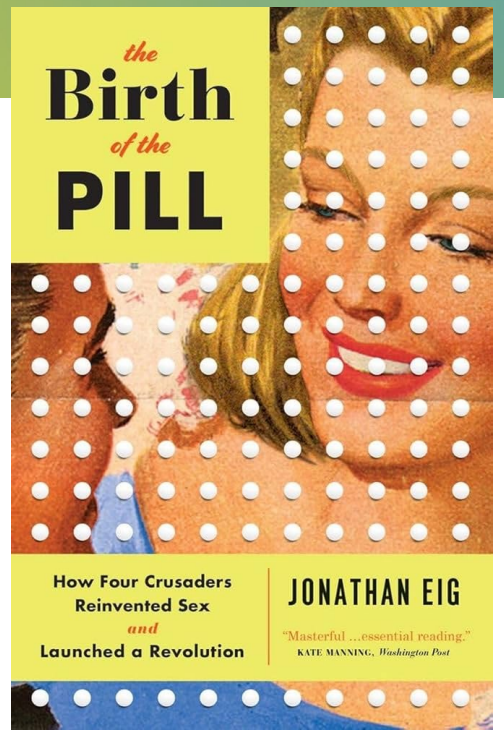
- Hirsutism *(65-75% of women with PCOS)*
  - OCPs → *Which one?*
  - Spironolactone
  - Hair removal
- Acne (15-25%)
  - OCP's
  - Spironolactone
- Alopecia
  - Same

→ *Derm consult?!*

Birth Control aka PCOS Pill

Birth Control aka PCOS Pill

- Ethinyl estradiol
  - 35mcg
  - 30mcg (desogen/ Apri)
  - 20mcg (Yaz, Loestrin, Junel)
  - 10mcg (only one and no generic, Loloestrin)
- Progestins



First generation

- Norethindrone acetate
- Ethynodiol diacetate
- Lynestrenol
- Norethynodrel

Second generation

- dl-Norgestrel
- Levonorgestrel

Third generation

- Desogestrel
- Gestodene
- Norgestimate

Unclassified

- Drospirenone
- Cyproterone acetate

Level of activity	Generic name(s)
High	Norgestrel
	Levonorgestrel
Middle	Norethindrone
	Norethindrone acetate
Low	Ethynodiol
	Norgestimate
	Desogestrel
	Drospirenone
	Dienogest

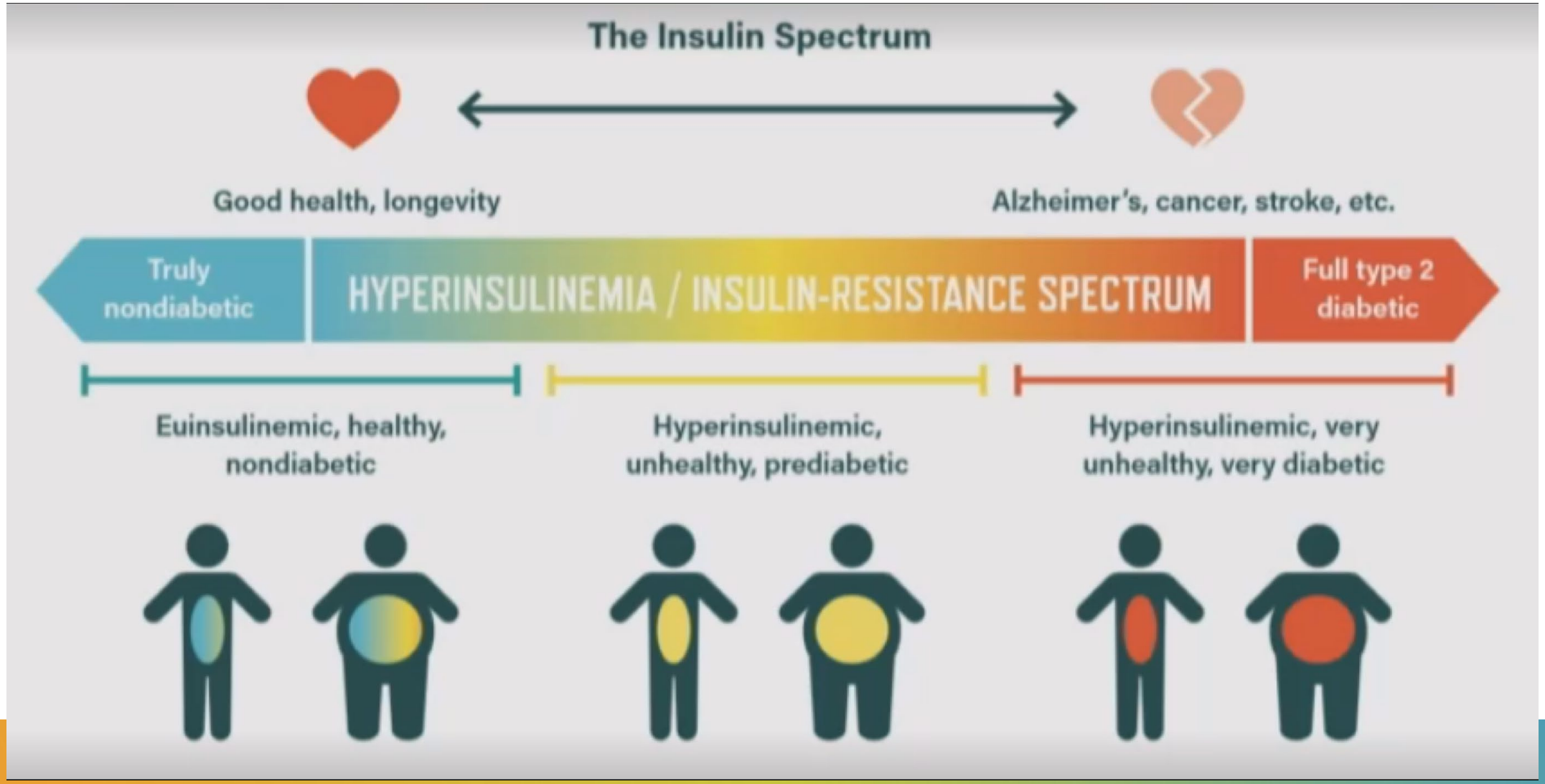
Mental Health in PCOS

- Depression
- Anxiety
- Disordered eating
- Self esteem/ Confidence
- Overwhelmed with diagnosis and multi-disciplinary management

Metabolic aspects of PCOS

- Women with PCOS are at increased risk for impaired glucose tolerance (~30%) & diabetes (~10%)
- Optimal screening method?
  - 2h 75g oral glucose tolerance test (OGTT)
  - Hemoglobin A1c
  - Fasting glucose
  - Continuous glucose monitoring
- Follow up:
  - For women with normal results:
    - Rescreen at least every 2 years or more frequently if additional risk factors
  - For women with IGT:
    - screen annually to r/o progression to DM

Salley, et al. JCEM, 2007


The Insulin Spectrum
Good health, longevity
Alzheimer's, cancer, stroke, etc.
Truly nondiabetic
HYPERINSULINEMIA / INSULIN-RESISTANCE SPECTRUM
Full type 2 diabetic
Euinsulinemic, healthy, nondiabetic
Hyperinsulinemic, unhealthy, prediabetic
Hyperinsulinemic, very unhealthy, very diabetic

What Insulin does

- Sugar into cells (muscles and liver)
- Converts carbs to fat (in liver)
- Disrupts brain pleasure centre (dopamine receptors)
- Dampens sympathetic activity (lethargy, lazy)
- Disrupts hunger and movement center
- Turns off fat burning

Inositol

- Myo-2000mg + Chiro-50mg twice daily
- Inositols naturally occur as five stereoisomers, with Myo-inositol and D-chiro-inositol as the most abundant
  - Present in almost all forms of life
  - Precursors of InsP3 (second messenger in insulin signaling)
- Many PCOS studies, heterogenous phenotypes, duration of treatment, outcomes measured
- 2018 Meta-analysis of RCT's
  - 10 trials, 362 women with inositol, 179 controls
  - Regulate cycles, improve ovulation, induce metabolic changes
  - Evidence is lacking for pregnancy, miscarriage or live birth.

Metformin 101

- Improves insulin sensivity
  - Prevents diabetes
    - Weight loss
  - Improves ovulation
  - Decreases androgens

- Indication:
  - Documented impaired glucose tolerance (or all PCOS?)
- Objective:
  - Reduces hepatic glucose production→ Lowers insulin
  - Reduces mitochondrial energy generation→ lower gluconeogenesis→ Improved insulin sensitivity
  - Increases GDF 15 serum peptide 15→ Anorexia→ Weight loss
  - Enhanced GLP-1 production
  - Suppression of inflammatory cytokines
  - Stimulation of adipogenesis, Etc.
  - Lowers androgen levels (~20%)
  - Improved lipid profile
- Contraindications:
  - Renal insufficiency
  - Elevated liver enzymes
- Use:
  - Start at 500 mg daily
  - Titrate up weekly to 1500 to 2000 mg/day
  - Extended release
  - Evening, postprandial

TABLE 1
Side effects of metformin

Gastrointestinal side effects	Diarrhea Nausea Vomiting Bloating Abdominal discomfort Flatulence Indigestion Constipation Heartburn Unpleasant metallic taste in mouth
Other side effects	Sneezing Cough Runny nose Flushing of skin Nail changes
Rare side effects	Lactic acidosis Chest pain Rash

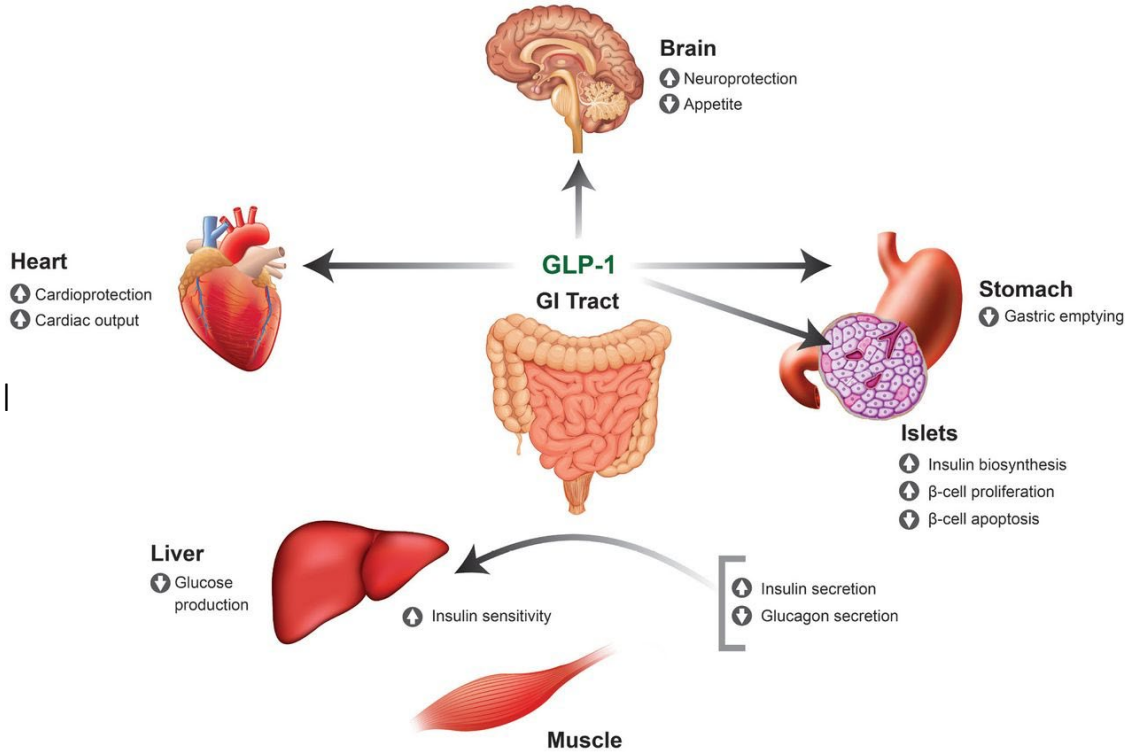
Mathur. Use of metformin in polycystic ovary syndrome. Am J Obstet Gynecol 2008.

Azziz. AJOG, 2008; Nestler. NEJM, 2008

GLP-1 Agonist

- Glucose-Like Peptide-1:
  - GI peptide released in setting of meal
  - Stimulates insulin synthesis and secretion
  - GLP-1 receptors are expressed in various tissues
    - Pancreatic beta cells → Insulin release, inhibit glucagon, beta cell regeneration?
    - Gastric mucosa → Slows gastric emptying
    - Hypothalamus → Decreased appetite
    - Kidney/ Lung/ Heart/ Immune cells/ Skin/ etc.

- GLP-1 Receptor Agonists:
  - Improve glycemic control and weight loss
  - Longer acting→ more effective
    - Administration: subcutaneous weekly or oral daily
    - Long terms studies on PCOS and fertility still needed
  - Side effects: GI n/v, constipation



Lamos et al, Expert Review 2017; Han et al., Reprod Biomed, 2019;
Figure: arab-diabetes.com/blog/glp-1-agonists/

GLP- 1 Agonist in Reproduction

- Weight loss → Decreased obesity-induced GnRH suppression
- Direct stimulation on GnRH and LH release (via GABA and kisspetpin neurons)
- Anti-inflammatory effects on ovaries and endometrium
- Possible decrease in androgens
- Increase in SHBG

- Compared to metformin
  - GLP-1 Agonists: more effective reduction of insulin resistance and BMI
- Synergistic with metformin

Jensterle et al., HR Update, 2019; Han et al., Reprod Biomed, 2019

Metabolic aspect of PCOS

Obesity

- Complex interaction between **obesity** and PCOS
- Many studies show increased risk and earlier onset of being overweight/ obese
  - Exacerbates reproductive phenotype
- However, limited studies using unselected population
  - Unselected studies show more similar BMI between women with/without PCOS
  - Uniform prevalence worldwide, despite varying prevalence of obesity
  - Suggests obesity is a driver for referral
- Obesity does not drive development of PCOS
- Obesity is independent and additive risk factor for metabolic disease

Legro et al, JCEM 2005; Norman RJ, et al, HR 2011; Hudecova M et al, HR 2011; Gambineri et al, Diab 2012; Schmidt J, et al. JCEM 2011

Metabolic aspect of PCOS

Dysfunctional Adipose

- Regardless of actual BMI
  - Abdominal/visceral fat distribution
  - Favor IR and subclinical inflammation
  - Larger adipocytes
  - Lower lipase activity
  - Impaired lipolysis
  - Greater adiponectin suppression

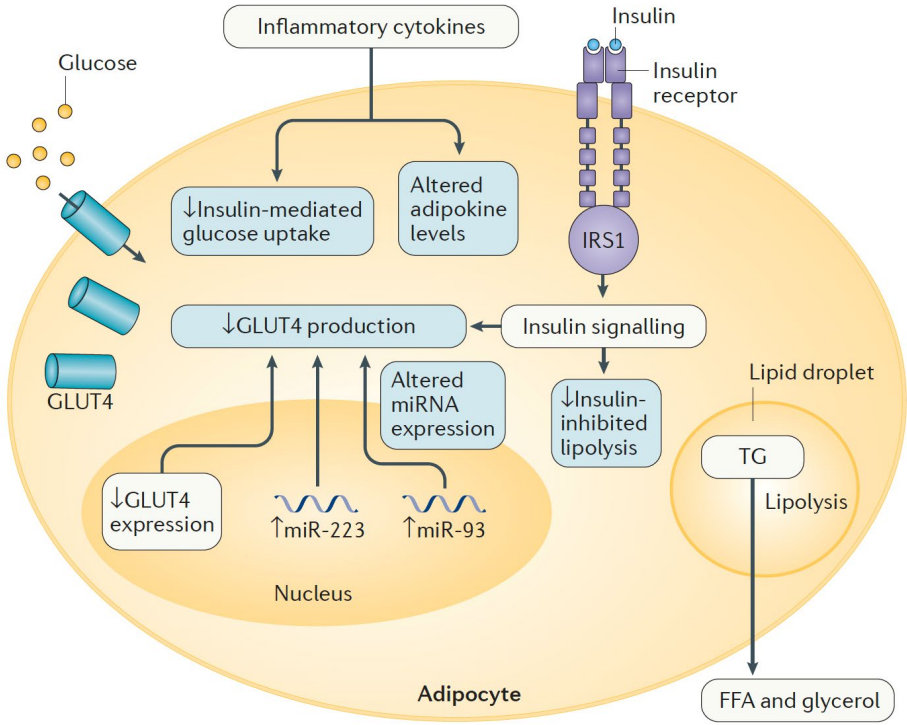
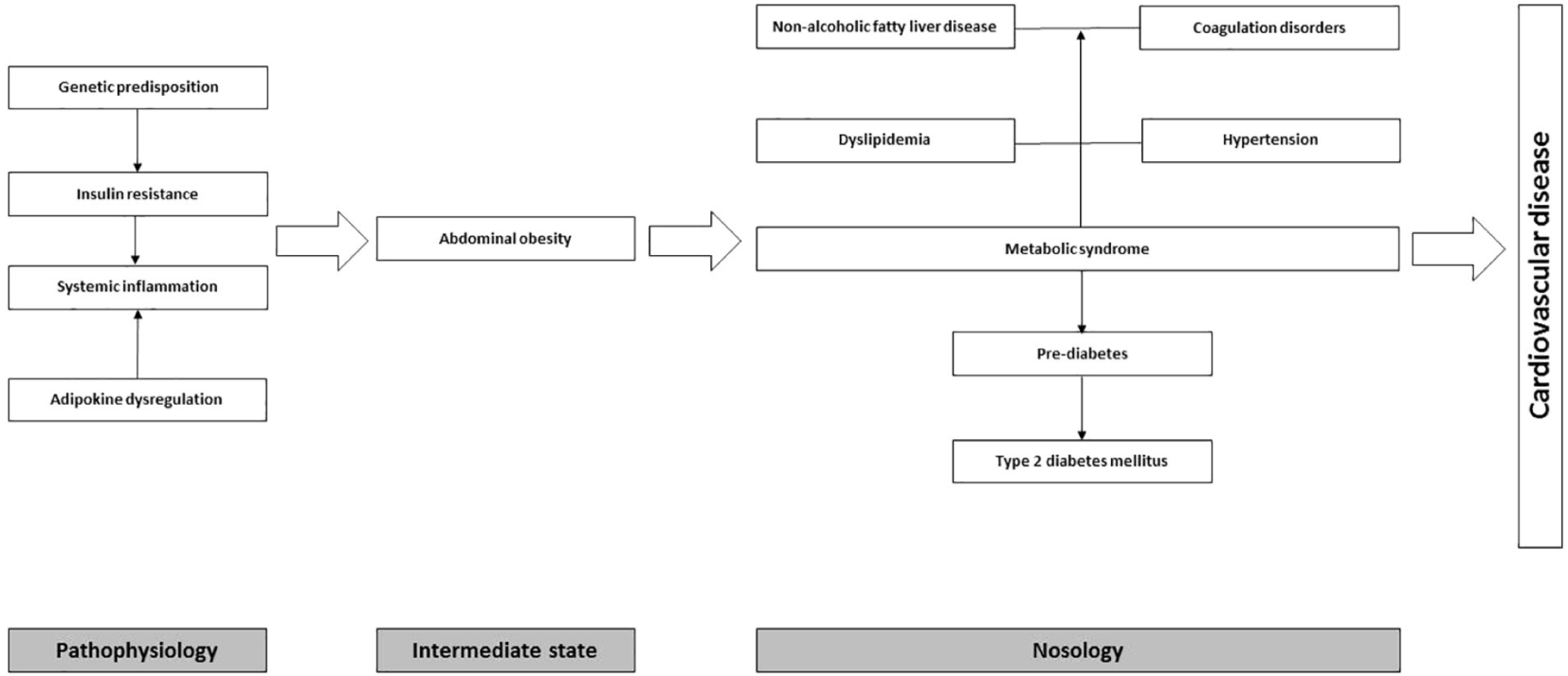


Figure: Azziz, et al. Nature Rev 2016. Legro et al, JCEM 2005; Norman RJ, et al, HR 2011; Hudecova M et al, HR 2011; Gambineri et al, Diab 2012; Schmidt J, et al. JCEM 2011

Metabolic aspect of PCOS

- Dyslipidemia
- Disordered sleep breathing/ OSA
- MASLD
- Cardiovascular disease



Legro et al, JCEM 2005; Norman RJ, et al, HR 2011; Hudecova M et al, HR 2011; Gambineri et al, Diab 2012; Schmidt J, et al. JCEM 2011; Figure: Adaped from: Anagnostis P et al. Metabolism 2017.

Weight Management in PCOS

- Diet
  - Sustainability
- Exercise
  - Sustainability
- Medical/surgical
  - Pharmacotherapy
  - Bariatric surgery
- Resources
  - Dietician/nutrition consult, Weight Watchers, trainer, Fitbit

EN

!!

PCOS & Fertility

2026 approach to ovulation induction in PCOS

- R/O other factors that contribute to infertility?
  - Semen analysis
  - Tubal and cavity evaluation
- Lifestyle modifications?
  - If room for improvement and willing and young…
    - Losing 5-10% body weight may restore ovulation
    - Morbidly obese women may require more weight loss

Table 1

Screening test results in potential PPCOS subjects (n=1313)

Criteria for exclusion	Abnormal test/total subjects tested	Prevalence
Normal serum androgen level	249/1280	19.5%
Oligospermia	95/881	10.1%
Bilateral tubal blockage	35/839	4.2%
Currently pregnant	20/968	2.0%
Diabetes	14/902	1.5%
Congenital adrenal hyperplasia	10/937	1.1%
Hyperprolactinemia	10/993	1.0%
Liver disease	8/822	0.9%
Uncontrolled thyroid disease	9/985	0.9%
Premature menopause	5/985	0.5%
Androgen-secreting tumors	1/1038	0.1%
Cushing's syndrome	1/1032	0.1%

PCOS & Pregnancy– PRECONCEPTION COUNSELING

What's known
• Increased risk of adverse pregnancy outcomes; these risks may be exacerbated by obesity and/or insulin resistance   • GDM, HTN
• Health should be optimized before conception
• Miscarriage rates?
• No evidence that metformin improves live-birth rates or decreases pregnancy complications
Knowledge gaps
• Specific periconceptional diets?
• Increased monitoring?
• Long-term outcome of children?
• Long-term outcome of mothers who develop complications?

Fauser, et al. Fertility and Sterility, 2012

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 FEBRUARY 8, 2007 VOL. 356 NO. 6

Clomiphene, Metformin, or Both for Infertility in the Polycystic Ovary Syndrome

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Letrozole versus Clomiphene for Infertility in the Polycystic Ovary Syndrome

ORIGINAL ARTICLES: REPRODUCTIVE ENDOCRINOLOGY

A randomized controlled trial of combination letrozole and clomiphene citrate or letrozole alone for ovulation induction in women with polycystic ovary syndrome

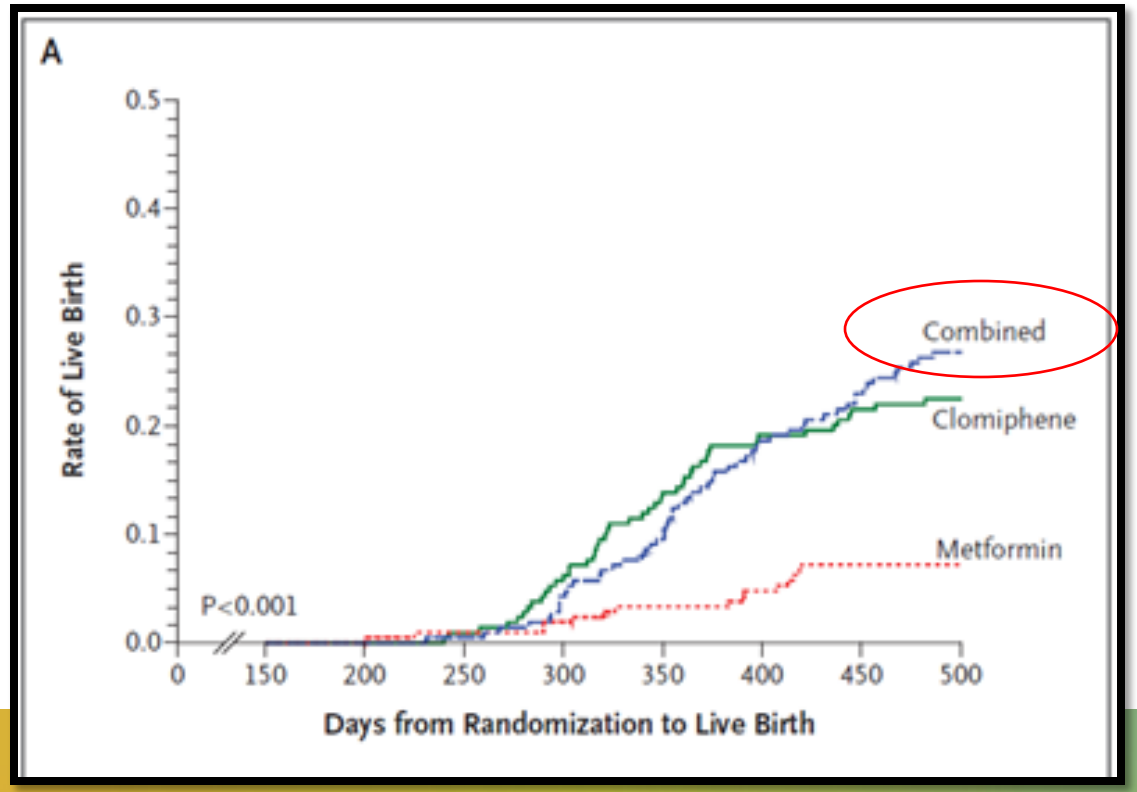
Rachel B. Mejia, D.O., Karen M. Summers, M.P.H., C.H.E.S., Jessica D. Kresowik, M.D., and Bradley J. Van Voorhis, M.D.

Department of Obstetrics and Gynecology, University of Iowa Carver College of Medicine, Iowa City, Iowa

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 FEBRUARY 8, 2007 VOL. 356 NO. 6

Clomiphene, Metformin, or Both for Infertility in the Polycystic Ovary Syndrome



Legro, et al. 2007

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Letrozole versus Clomiphene for Infertility in the Polycystic Ovary Syndrome

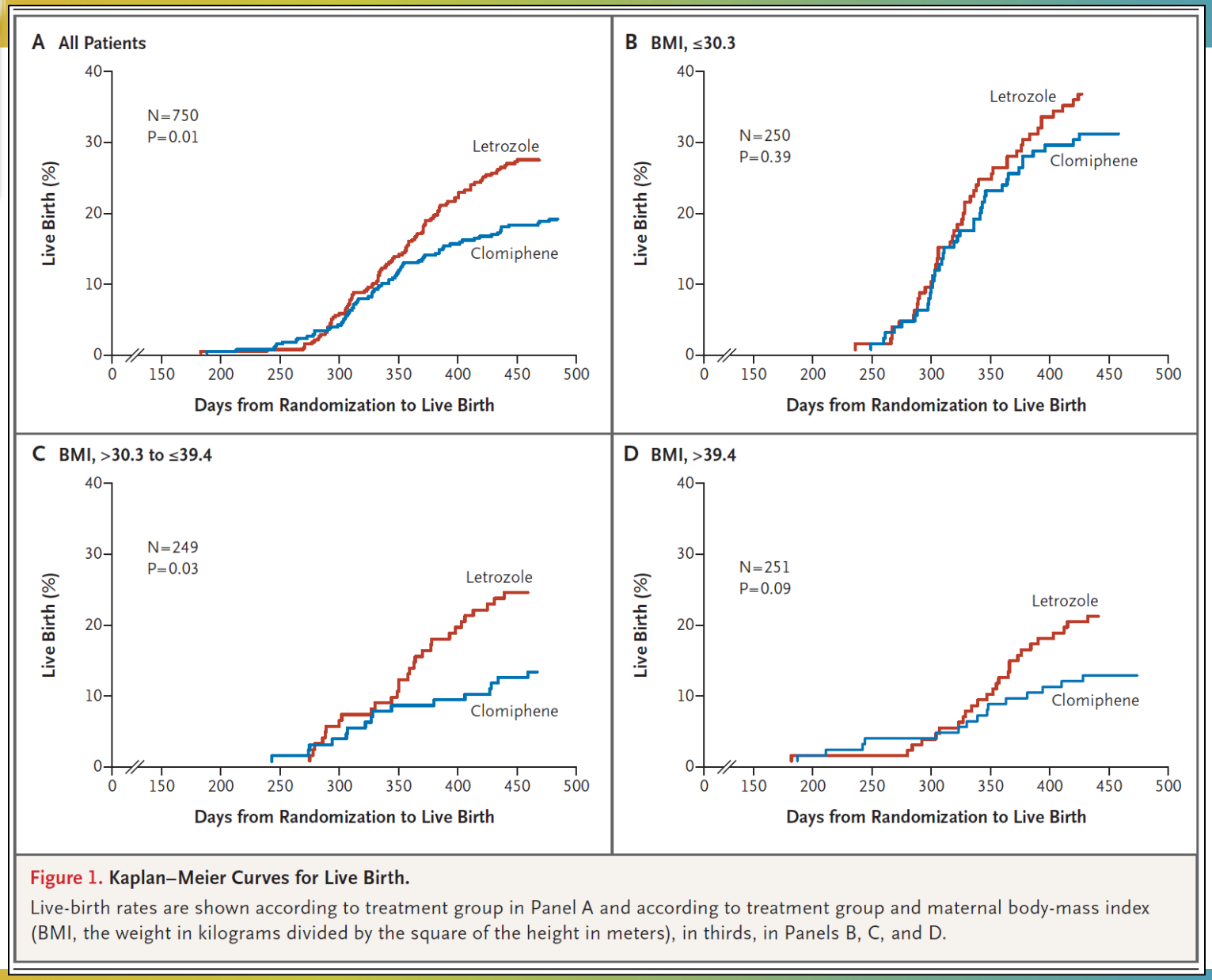


Figure 1. Kaplan–Meier Curves for Live Birth.

Live-birth rates are shown according to treatment group in Panel A and according to treatment group and maternal body-mass index (BMI, the weight in kilograms divided by the square of the height in meters), in thirds, in Panels B, C, and D.

Legro, et al. 2014

ORIGINAL ARTICLES: REPRODUCTIVE ENDOCRINOLOGY

A randomized controlled trial of combination letrozole and clomiphene citrate or letrozole alone for ovulation induction in women with polycystic ovary syndrome

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Ovulation Induction

Ovulation

Letrozole

3 7 14 28

Decreased Estradiol

Increased Estradiol

Increased FSH

To bleed or not to bleed?

Table 1. Pregnancy Outcomes in the Pregnancy in Polycystic Ovary Syndrome Trial as a Function of the Menstrual Status of the Preceding Cycle*

		Ovulation		Conception			Live Birth			
Menstrual Status Group	Cycles (n)	n	Ovulation per Cycle	n	Conception per Cycle	Conception per Ovulation	n	Live Birth per Cycle	Live Birth per Ovulation	Live Birth per Conception
Spontaneous menses	1,185	853	72.0	39	3.3	4.5	26	2.2	3.0	66.7
Anovulatory with progestin withdrawal	551	166	30.1	11	2.0	6.6	9	1.6	5.4	81.8
Anovulatory without progestin withdrawal	1,073	289	26.9	81	7.5	27.7	57	5.3	19.7	70.4
Total	2,809	1,308	46.6	131	4.7	9.9	92	3.3	7.0	70.1
P			<.001		<.001	<.001		<.001	<.001	NS

NS, not significant.
Data are % unless otherwise specified.
* Spontaneous menses, anovulation with progestin induced withdrawal bleed, and anovulation without progestin induced withdrawal bleed.

Diamond, et al. Obstetrics and Gynecology, 2012

Fertility & PCOS

- Reasonable approach
  1. Letrozole
  2. Clomiphene citrate
  3. Metformin if documented insulin resistance
     1. Not to be used as a sole agent!

- Next?
  - Ovarian drilling?
  - Injectable medications ± inseminations?
  - IVF?

Summary

- Think of diagnosis as a spectrum
- Treatment should focus on symptoms
- Lifestyle is the best medicine
- Letrozole for ovulation induction
- Metformin if IR
- Compassion

Thank you!!!

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Q&A