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Professional Education Series

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PCOS Nutrition: A Sustainable & Science-Backed Approach

TODAY'S AGENDA:

- Welcome
- Speaker Introduction
- Presentation
- Q&A
- Closing



WEBINAR HOST:

Keith Hine, MS, RD

VP of Healthcare, Sports & Professional Education
Orgain, LLC



WEBINAR PRESENTER:

Melissa Groves Azzaro, RDN, LD

Award-Winning Integrative Registered Dietitian



Learning Objectives

Understand that polycystic ovary syndrome (PCOS) is a complex, multi-system condition

Learn about the underlying root causes driving PCOS symptoms

Understand how nutrition and lifestyle changes can improve underlying root causes to improve symptoms and lower long-term risks of PCOS

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What IS PCOS?

- Polycystic Ovary Syndrome (PCOS) is a complex hormonal and metabolic syndrome
- First identified by Stein & Leventhal in 1935
- Affects anywhere from 5-20% of reproductive-aged people AFAB (depending on criteria used)

Teede H. *BMC Medicine*. 2010;8:41.
Stein IF, Leventhal ML. *AJOG*. 1935;29(2):181-191.
Dumesic DA. *Endocr Rev*. 2015;36(5):487-525.

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marked photophobia. Nuchal rigidity was slight and Kernig's sign was negative. There was no muscle weakness and all reflexes were present and normal. Fundoscopic examination revealed no abnormality. Lumbar puncture produced an opalescent fluid with a high protein as shown by a strong positive Pandy's test, cell count was 320/c.mm., protein 300 mg./100 ml., sugar 18 mg./100 ml., and chlorides 630 mg./100 ml. Scanty acid/alcohol-fast bacilli were seen on direct microscopy. Her white blood-cell count was 18,400/c.mm. Intradermal Mantoux test 1/1,000 was strongly positive.

Antituberculous therapy was commenced on the day of admission. During the next three weeks her condition did not improve and on April 3 choroidal tubercles were seen in the left eye, and x-ray examination on March 30 showed military tuberculosis throughout both lung fields. In view of the lack of progress and in the absence of hydrocephalus steroid therapy was added to the chemotherapy. From this stage improvement was marked and she became afebrile on April 17.

On April 9 her cerebrospinal fluid was xanthochromic with the protein content almost 400 mg./100 ml., suggesting a block. There was, however, no papilloedema. Response to Queckenstedt's test was very slow, but the C.S.F. pressure was normal (70-120 ml. water on the manometer), and it was thought that the block was spinal.

On September 14 it was noticed that her fluid intake/output, which had been gradually increasing, amounted to some 7 pints (4 litres) a day, with a maximum urinary S.G. of 1010. Vasopressin tannate in oil, 0.5 ml., was exhibited subcutaneously. This resulted in only a slight fall in her fluid balance level. The effect of withholding fluid was tried and this resulted in the typical symptoms of dehydration, i.e., irritability, headache, and muscular pains. The vasopressin tannate in oil was continued for a month and the patient was then changed to vasopressin snuff thrice daily, which resulted in a considerable reduction in intake/output. The child, however, disliked the snuff and after six days refused to take it. It was therefore necessary to re-exhibit the vasopressin tannate subcutaneously, which, while reducing the level of the fluid balance, was not entirely satisfactory.

She was, however, discharged quite well on November 4. When seen as an out-patient on December 1 she was still passing large amounts of urine, but continued to have the vasopressin, which her father had been taught to administer. Reviewed on March 24, 1960, lumbar puncture showed a normal fluid, chest x-ray showed old military tuberculosis almost completely resolved, and the child had gained weight. Although she complained of occasional headaches, she had no sickness. She frequently wet her bed (2-3 times a week). She drinks large amounts of fluid and passes urine frequently, but still refuses to take vasopressin snuff, which makes her sick. Her appetite is poor and her bowels regular. She is back at school and leading a normal life. An attempt is now being made to stabilize her with varying amounts of vasopressin tannate in oil.

CASE 2

The second patient was admitted on July 5, 1953, a diagnosis of tuberculous meningitis was made, and treatment given successfully. She was discharged quite well in April, 1954. She was readmitted on October 25, 1954, with a story of polydipsia, polyuria, and enuresis. X-ray examination of the skull showed calcification in the region of the pituitary. The diagnosis of diabetes insipidus was confirmed by the response to vasopressin snuff.

The difference in these two cases was in the time lapse before the onset of the symptoms of diabetes insipidus. In the first case the symptoms gradually appeared from about 3-4 weeks after the diagnosis of tuberculous meningitis was made. In the second case there was a lapse of some 15 months. The second patient had radiographic calcification of her pituitary, whereas the

It is known that tuberculous meningitis exerts its most severe effects on the basal cisterns. It is interesting to note that in the first case the child was thought to have had a block, and it was more or less from this date that her fluid balance became disturbed and she exhibited all the symptoms of diabetes insipidus, probably due to mechanical damage to the pituitary by the tuberculous process itself without calcification, whereas the second case was probably caused by damage to the pituitary when calcification of the tuberculous exudate occurred.—I am, etc.,

JEROME P. WALL.

Gateside Hospital,
Greenock.

REFERENCES

- ¹ Lorber, J., *Arch. Dis. Childh.*, 1958, 33, 315.
- ² Acheson, R. M., and Smith, H. V., *Quart. J. Med.*, 1958, 27, 83.
- ³ Levin, A. R., *Brit. med. J.*, 1959, 2, 1061.
- ⁴ Hay, D. R., *ibid.*, 1960, 1, 707.

Stein-Leventhal Syndrome

Sir.—The article by Mr. D. W. T. Roberts and Dr. Magnus Haines (June 4, p. 1709) on "Is there a Stein-Leventhal Syndrome?" raises points of interest. I would like to draw attention to the fact that the so-called Stein-Leventhal syndrome, described by them in 1935,¹ was actually described many years before, by Bartel and Herrmann in 1911,² in which amenorrhoea, menstrual irregularities, and sterility were stated as the clinical symptoms.

Rubin and Novak³ stated that the French term *ovaires sclerokystiques* was the proper designation for this condition. The latter authors pointed out that in this condition one was dealing with the congenital type of polycystic ovaries, characterized by larger size, smooth or only slightly uneven surfaces, thick tunica albuginea, dense fibrous stroma, and a low total amount of follicles. The cortex is studded with small cystic follicles, many of them atretic. This congenital type of condition must be differentiated from another type of polycystic ovaries, which, owing to an abnormal activity of the hypophysis, have become polycystic. The stroma is normal, the tunica thin, and there is a normal content of the follicles. The increased size of these ovaries results only from their content of cystic follicles and recedes to normal when normal function is restored.—I am, etc.,

ALBERT SHARMAN.

Royal Samaritan Hospital
for Women,
Glasgow, S.2.

REFERENCES

- ¹ Stein, I. F., and Leventhal, M. L., *Amer. J. Obstet. Gynec.*, 1935, 29, 181.
- ² Bartel, J., and Herrmann, E., *Mschr. Geburtsh. Gynäk.*, 1911, 33, 125.
- ³ Rubin, I. C., and Novak, J., *Integrated Gynecology*, 1956, McGraw-Hill, New York.

Herpes Zoster with Chicken-pox

Sir.—Dr. D. Taylor-Robinson (June 4, p. 1713) describes a case of herpes zoster occurring in a patient with chicken-pox, and suggests that this is rare.

We had an outbreak of chicken-pox in the village here five years ago. A 70-years-old man developed typical chicken-pox on October 18, 1955, and two days later began to show fairly numerous lesions of herpes zoster on the left buttock and scrotum. He had been in previous good health, made a satisfactory recovery, and is still alive and well. There is some residual scarring of the affected area, but I doubt if the patient would agree to any attempt at emulating Dr. Taylor-Robinson's excellent photograph.—I am, etc.,

FRANK E. HARRIS.

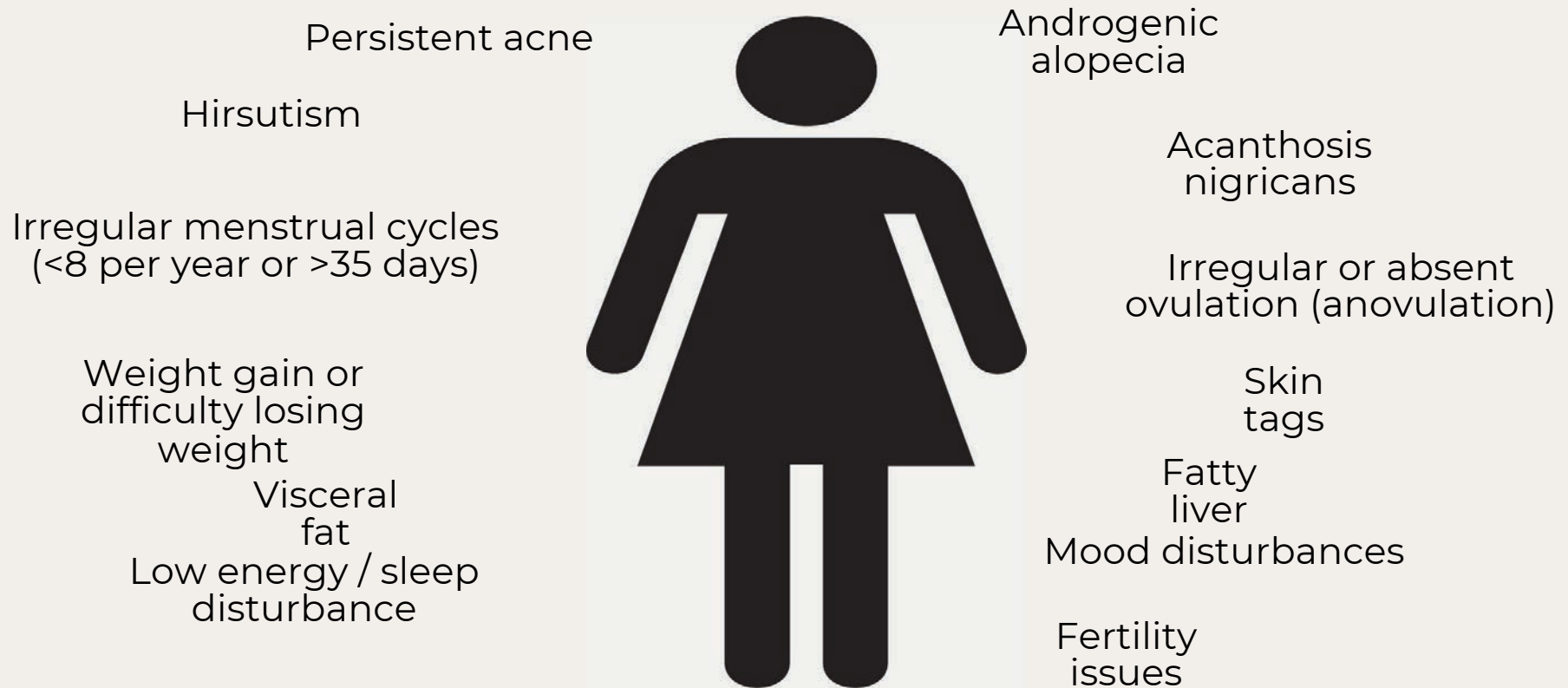


Disclosures

- Dietitians in Integrative and Functional Medicine - Social Media Chair (2018-2020), Chair-Elect (2020-2021), Chair (2021-2022), Past-Chair (2022-2023), Communications Chair (2023-present)
- Owner and Founder of The Hormone Dietitian LLC
- Author of A Balanced Approach to PCOS, Victory Belt 2020
- This presentation is provided with support by Orgain

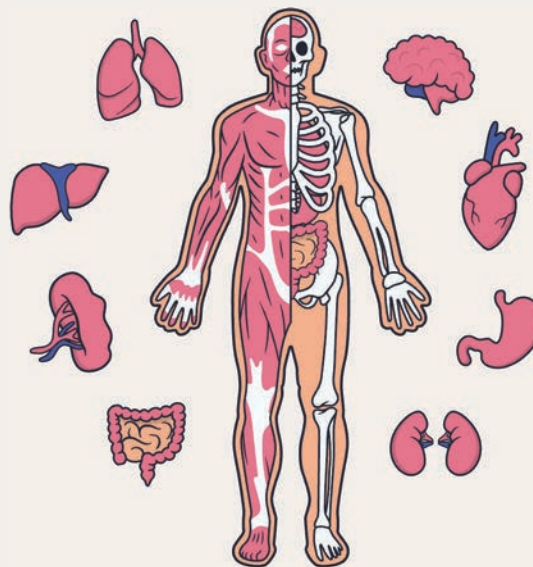


Not Just a “Reproductive” Condition



Long-Term Risks of PCOS

- Type 2 diabetes (50% prediabetic by age 40)
- Gestational diabetes
- Heart disease (4-7x higher risk)
- Dyslipidemia ($\uparrow$ LDL, $\downarrow$ HDL, $\uparrow$ TGL)
- Hypertension
- Obesity (30-75%)
- Metabolic syndrome (1 in 3)



- Infertility
- Miscarriage
- Eating disorders (esp BED) 4x $\uparrow$ risk
- Endometrial / uterine cancer 3x $\uparrow$ risk
- Sleep apnea and other sleep disorders

For the first time, the 2023 International Expert Guidelines have included cardiovascular & diabetes risks, sleep apnea, endometrial hyperplasia & cancer, and familial risks.

CDC.gov. PCOS and Diabetes.
Torchen LC. *Curr Diab Rep.* 2017;17(12):137.
Diamani-Kandarakis E. *Endocr Rev.* 2012;33(6):981-1030
Teede HJ, Tay CT, Laven JJ et al. *J Clin Endocrinol Metab.* 2023;108(10):2447-2469.

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PCOS Diagnosis

Rotterdam Criteria (Modified)

Must have 2 out of the 3 following:

- Irregular or absent ovulation
- Hyperandrogenism as determined by lab testing or symptoms
- Polycystic ovaries seen on transvaginal ultrasound (>20 in at least 1 ovary)

**Exclusion of other relevant disorders.*

Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group 2004 Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertil Steril* 81:19–25

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UPDATED Diagnostic Criteria

AMH levels can now be used instead
of ultrasound in adults

Teede HJ, Tay CT, Laven JJ et al. *J Clin Endocrinol Metab.*
2023;108(10):2447-2469.

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What Causes PCOS? Theories:

- Hypothalamic-pituitary dysfunction
- Ovarian & adrenal hyperandrogenism
- Disorder of peripheral insulin resistance
- In utero exposure to high AMH and/or androgen levels
- Environmental exposures
- Gene SNP mutations
- Gut microbiota

Legro RS. Evaluation and Treatment of Polycystic Ovary Syndrome. [Updated 2017 Jan 11]. In: Feingold KR, Anawalt B, Blackman MR, et al., editors. *Endotext* [Internet].
Goodarzi M, Dumesic, D, Chazenbalk, G. et al. *Nat Rev Endocrinol.* 2011;7:219-231.
Khan MJ, Ullah A, Basit S. *Appl Clin Genet.* 2019;12:249-260.
Guo H, Luo J, Lin H. *Medicine.* 2023;102(50):e36075.



PCOS “Types” (NIH)

Type A

- High androgens / androgenic signs
- Irregular periods / delayed ovulation
- Polycystic ovaries

Type B

- High androgens / androgenic signs
- Irregular periods / delayed ovulation
- Normal ovaries

Type C

- High androgens / androgenic signs
- Regular periods (<35 d)
- Polycystic ovaries

Type D

- Normal androgens
- Irregular periods / delayed ovulation
- Polycystic ovaries

Conventional Medical Treatment



Symptom-based:

- Hormonal birth control (OCP, IUD) - withdrawal bleeds, endometrial thickness management
- Anti-androgen medications (spironolactone, finasteride)
- Fertility medications (clomiphene, letrozole, adjunct / off-label metformin)
- Anti-obesity medications (sibutramine, orlistat, other GLP-1 agonists)

Newer treatments - based on root causes:

- Insulin-sensitizing medications (metformin)
- Diet, lifestyle, supplements
- 5-10% reduction in weight

Every Case of PCOS is DIFFERENT



- Symptoms and presentation
- Root cause drivers and severities
- Nutritional needs
- Cultural backgrounds, preferences, likes and dislikes
- Concomitant medical conditions
- Access to care, budgets
- Skill sets
- Goals

To truly provide the best care for people with PCOS, nutrition interventions should be tailored to food preferences, flexible, individual, and CO-developed

Evidence-Based Medicine



A Root Cause Approach

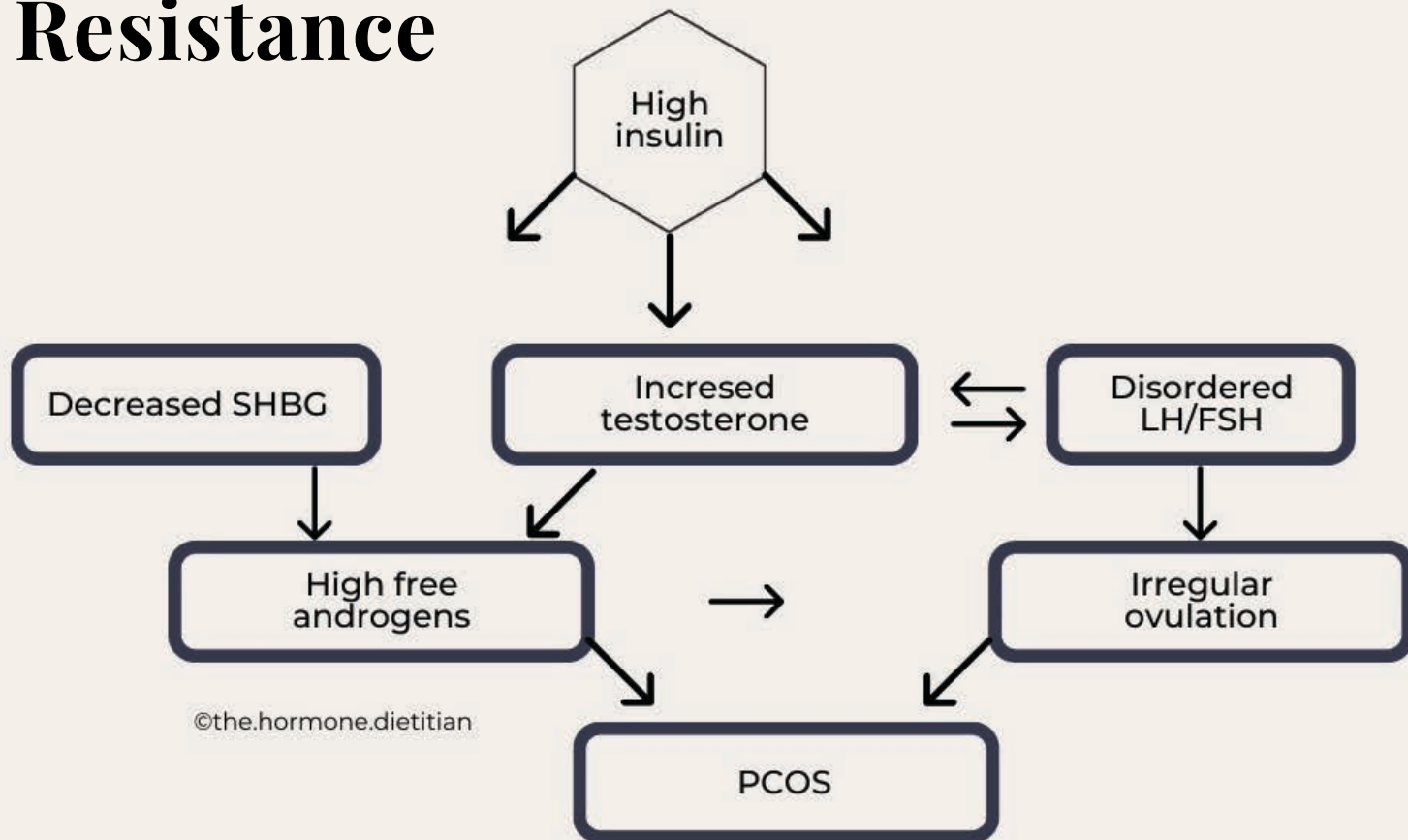
**Insulin
Resistance**

Inflammation

**Gut imbalances /
dysfunction**

**Hormone
imbalances**

Insulin Resistance



©the.hormone.dietitian



PCOS & Insulin Resistance

- Cells do not adequately respond to insulin, so blood sugar remains high and cells don't get enough energy
- Insulin levels continue to rise in the presence of high FBG
- Excessive for degree of adiposity & found in lean PCOS
- Increases with age (esp menopause) & worsened by obesity, puberty & pregnancy
- Increases LH & free T levels
- Increased testosterone leads to typical PCOS symptoms (acne, hirsutism, hair loss)



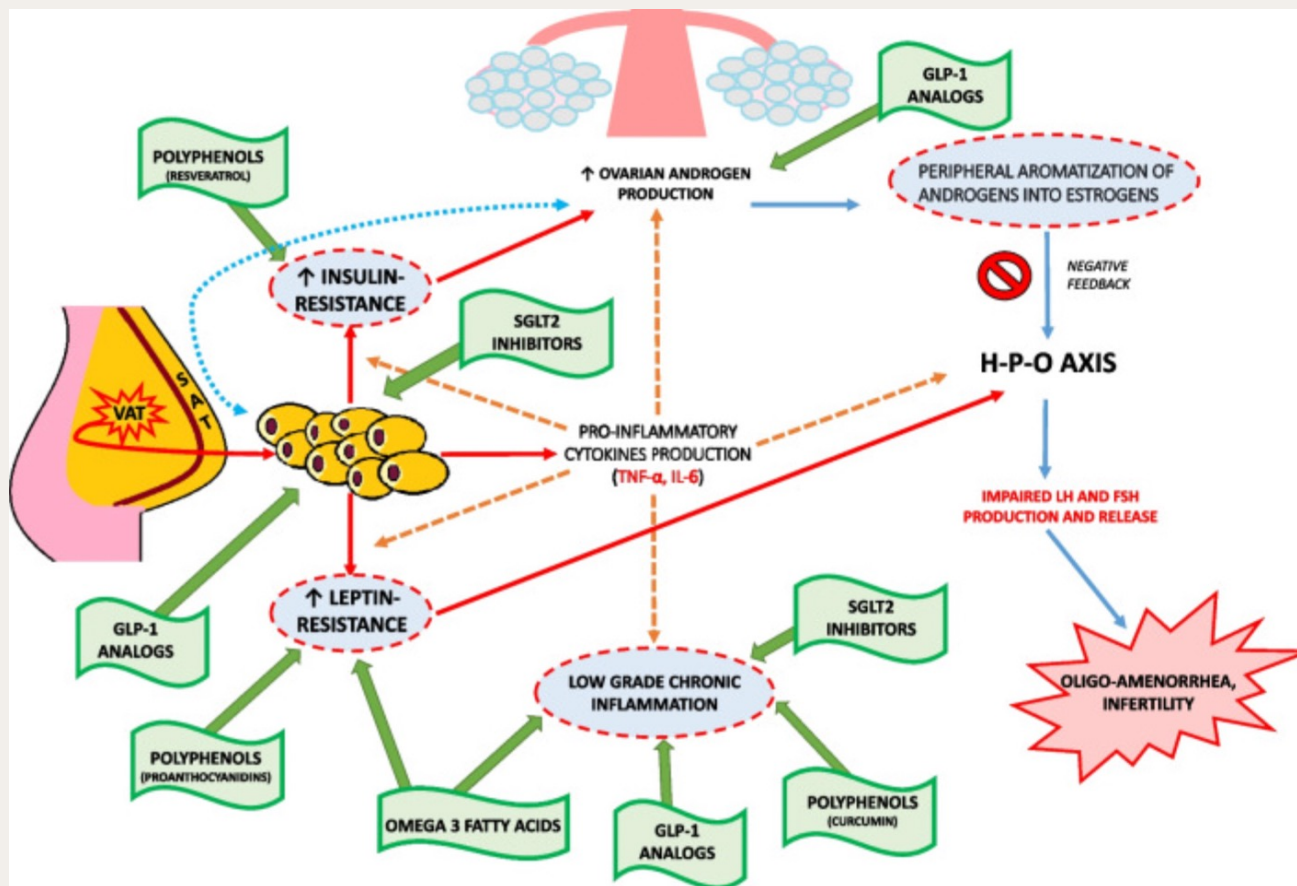


Prevalence & Risks of IR

- Seen in 75% of “lean” and 95% of overweight people with PCOS
- Increases cravings (esp for carbs, sugar)
- Linked to weight gain, increased visceral fat, difficulty losing weight
- linked to MASLD (formerly NAFLD)
- Increased inflammation (CRP) & other inflammatory markers
- Linked to dyslipidemia (LDL, VLDL, TGL, APOB)
- Can lead to impaired fasting glucose, impaired glucose tolerance & T2DM



Inflammation



PCOS & Inflammation

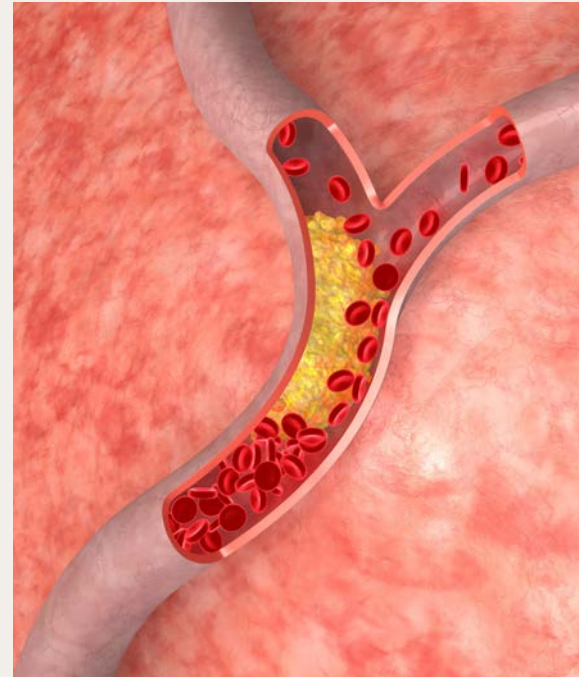


- Multiple signaling pathway abnormalities in PCOS lead to oxidative stress and inflammatory responses
- Adipose tissue, esp. visceral adipose tissue, releases pro-inflammatory cytokines
- In PCOS, fat tissue acts metabolically different from that of those without the condition and appears to be hyperresponsive to glucose & saturated fats
- The link between obesity and inflammation is well established
 - leptin, IL-6, TNF- α

Rosenfield RL, Ehrmann DA. *Endocr Rev.* 2016;37(5):467-520.
Ellulu MS, Patimah I, Khaza'ai H, et al. *Arch Med Sci.* 2017;13(4):851-863.

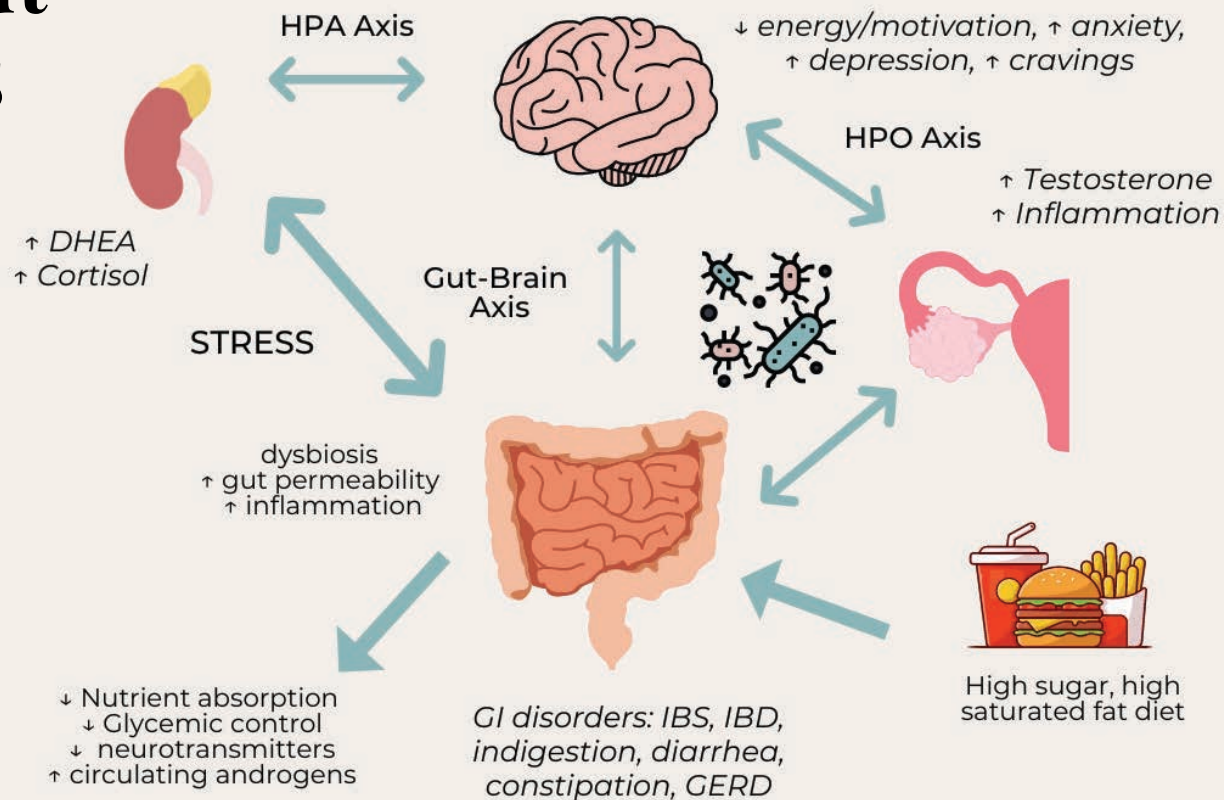
Prevalence & Risks of Inflammation

- Prevalence of inflammation in PCOS is unknown
- Women with PCOS with higher BMIs have been shown to have higher levels of hs-CRP vs women with lower BMIs
- Inflammation also found in women with lean PCOS
- Increased risk for miscarriage & pregnancy complications
- Increased risk for cardiovascular conditions / events
- Increased incidence of obstructive sleep apnea



Oh JY, Lee H, Oh JY, et al. *Korean J Intern Med.* 2009;24(4):350-355.
Gonzalez F, Kirwan JP, Rote NS, et al. *Am J Physiol Endocrinol Metab.* 2013;304(4):E375-383.
Alvarez YR, Pico M, Ashokprabhu N, et al. *Curr Atheroscler Rep.* 2023;25(12):1003-1011.
Teede HJ, Tay CT, Laven JJ et al. *J Clin Endocrinol Metab.* 2023;108(10):2447-2469.

PCOS & Gut Imbalances



PCOS & Gut Dysbiosis



- Numerous studies have shown a link between gut dysbiosis & PCOS
- Studies have shown lower gut microbiome diversity and altered composition vs women without PCOS
- Alterations of markers of gut barrier function & endotoxemia have also been seen
 - Changes in Bacteroidetes & Firmicutes, low levels of Akkermansia linked to metabolic dysfunction
- In rodent models, fecal microbiota transplant from women with PCOS has led to development of PCOS-like symptoms
- Studies on probiotics in PCOS show improvements in weight, BMI, insulin, and HOMA-IR

Sun J, Wang M, Kan Z. *Front Endocrinol*. 2024;15:1280983
Lindheim L, Bashir M, Munzker J, et al. *PLoS One*. 2017;12(1): e0168390.
Thackray VG. *Trends Endocrinol Metab*. 2019;30(1):54-65.
Calcaterra V, Rossi V, Massini G, et al, Fabiano V. *Nutrients*. 2023;15(14):3144.

PCOS & Gut Dysfunction



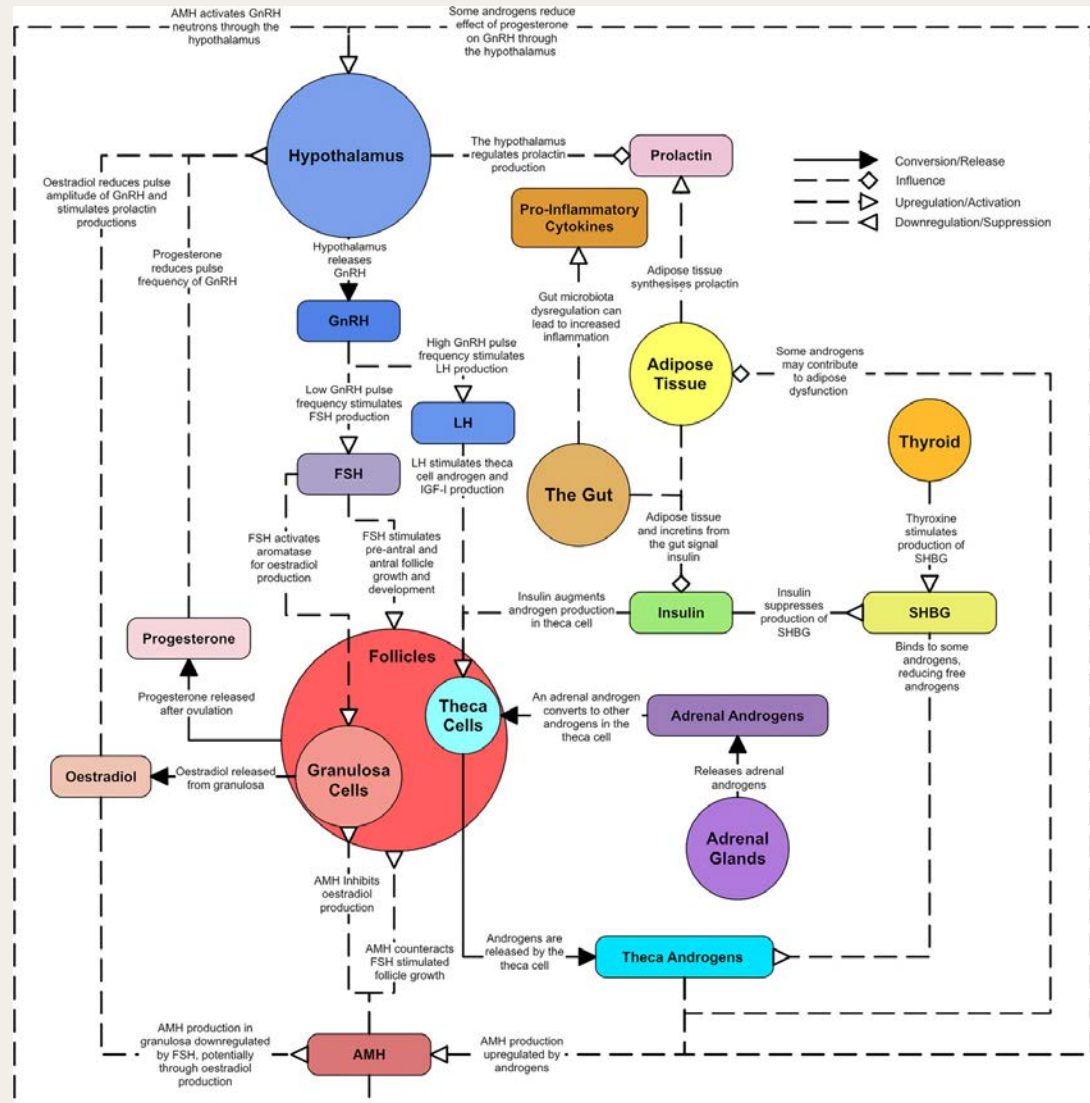
- Link between PCOS and digestive issues
 - 4x more likely to have IBS (42% vs 10%)
- A recent meta-analysis showed a nearly 2x higher risk for IBS with PCOS
- Dysbiosis has been associated with higher risk for inflammatory bowel diseases including Crohn's & ulcerative colitis
- Other digestive conditions that have been observed in PCOS include GERD, steatosis

Mathur R, Ko A, Hwang LG, et al. *Dig Dis Sci*. 2010;55:1085-1089.
Wei Z, Chen Z, Xiao W, et al. *Gynecol Endocrinol*. 2023;39(1):2239933.
Carding S, Verbeke K, Vipond DT, et al. *Microb Ecol Health Dis*. 2015;;26:26191.
Cowan S, Grassi A, Monahan Couch L, et al. *Nutrients*. 2023;15(3):589..

PCOS & Hormone Imbalances: More than just androgens

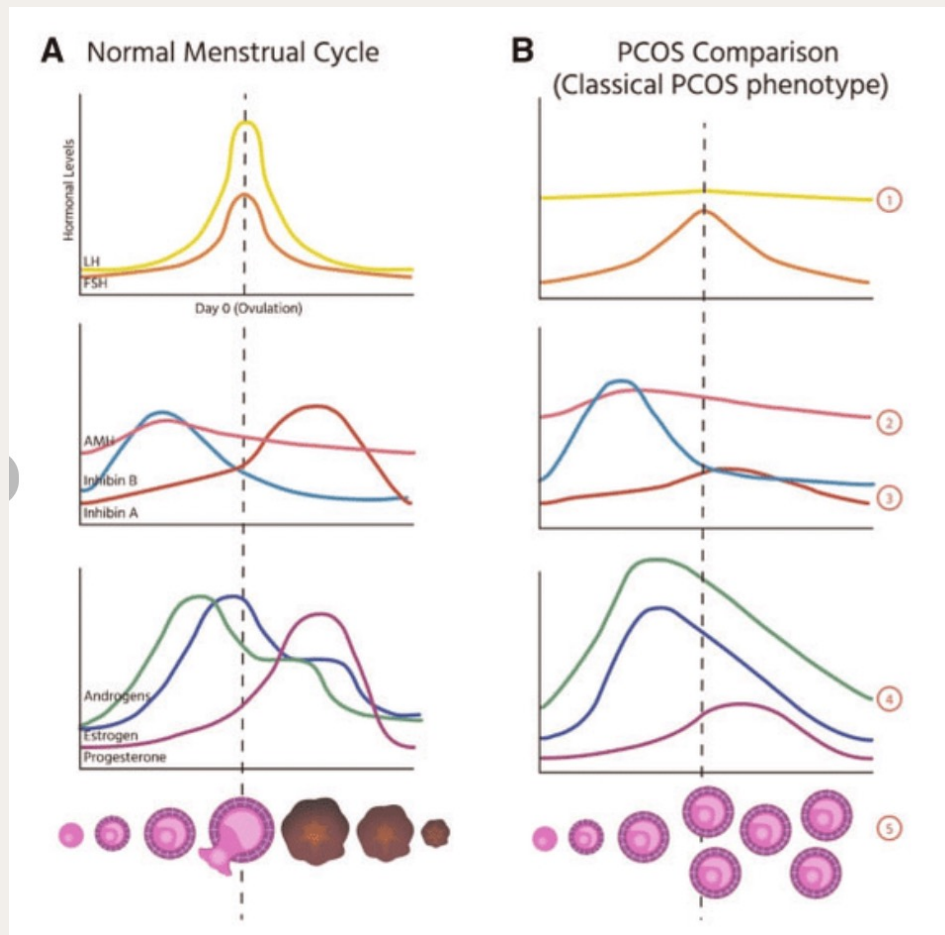
Emmanuel RHK, Roberts J, Docherty PD, et al.
Front Endocrinol. 2022;13:1017468.

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PCOS Menstrual Cycle Hormones

Charifson M, Trumble B. *Evolution med pub health*. 2019:50-63.



PCOS & Hormones



- While excess testosterone is the hormone imbalance most commonly associated with PCOS, the condition features imbalances in many of the hormones produced in the pituitary, ovaries, and adrenals
 - GnRH, LH, FSH, AMH, testosterone, DHEA/DHEA-S, DHT, estrogens, growth hormones, estrogen/progesterone balance
- In a normal menstrual cycle, estrogen is the dominant hormone for the first half of the cycle, then progesterone is dominant the second half
- 40-60% of women with PCOS have increased adrenal androgens (DHEA)
- Hypothyroidism is also more common in PCOS (11-14% vs 1-2%)

Dietary Approaches for PCOS



Should be individualized and account for:

- Food preferences / culture / accessibility
- Lifestyle (time available, culinary skill)
- Concurrent medical issues
- Goals
- Sustainability

“there is no evidence to support any one type of diet composition over another for anthropometric, metabolic, hormonal, reproductive or psychological outcomes”



PCOS Diet - Guidelines

3.3	Dietary interventions		
3.3.1	EBR	Healthcare professionals and women should consider that there is no evidence to support any one type of diet composition over another for anthropometric, metabolic, hormonal, reproductive or psychological outcomes.	❖❖❖ ⊕○○○
3.3.2	CR	Any diet composition consistent with population guidelines for healthy eating will have health benefits, and within this, healthcare professionals should advise sustainable healthy eating tailored to individual preferences and goals.	❖❖❖❖
3.3.3	PP	Tailoring of dietary changes to food preferences, allowing for a flexible, individual and co-developed approach to achieving nutritional goals and avoiding unduly restrictive and nutritionally unbalanced diets, are important, as per general population guidelines.	
3.3.4	PP	Barriers and facilitators to optimise engagement and adherence to dietary change should be discussed, including psychological factors, physical limitations, socioeconomic and sociocultural factors, as well as personal motivators for change. The value of broader family engagement should be considered. Referral to suitably trained allied healthcare professionals needs to be considered when women with PCOS need support with optimising their diet.	

PCOS Diet - Research



Various approaches have shown potential benefits:

- **Moderate carbohydrate** ($\leq 40\%$ carb) - decreased insulin, testosterone
- **High protein** (30% pro) - $\uparrow$ weight loss, $\downarrow$ FBG
- **Low-glycemic** - improved insulin sensitivity, $\uparrow$ ovulation
- **High fiber** - inverse relationship between fiber, HOMA-IR, body fat
- **DASH diet** - $\downarrow$ BMI, weight, AMH, insulin
- **Moderate fat** (30-40%) - mono/polyunsaturated

Gower A. *Clin Endocrinol*. 2013;79(4):550-557.
Sorensen LB. *Am J Clin Nutr*. 2012;95(1):39-48.
Wang F, Dou P, Wei W, Liu PJ. *Nutr Diabetes*. 2024;14(1):6
Barr S. *J Acad Nutr Diet*. 2013;113(11):1523-153.
Sordia-Hernandez LH. *Clin Exp Obstet Gynecol*. 2016;43(4):555-559
Cunha NBD. *Clin Nutr*. 2018;S0261-5614(18)32483-X.
Azadi-Yazdi M. *J Hum Nutr Diet*. 2017;30(3):275-283.
Foroozanfard F. *Clin Endocrinol*. 2017;87(1):51-58.

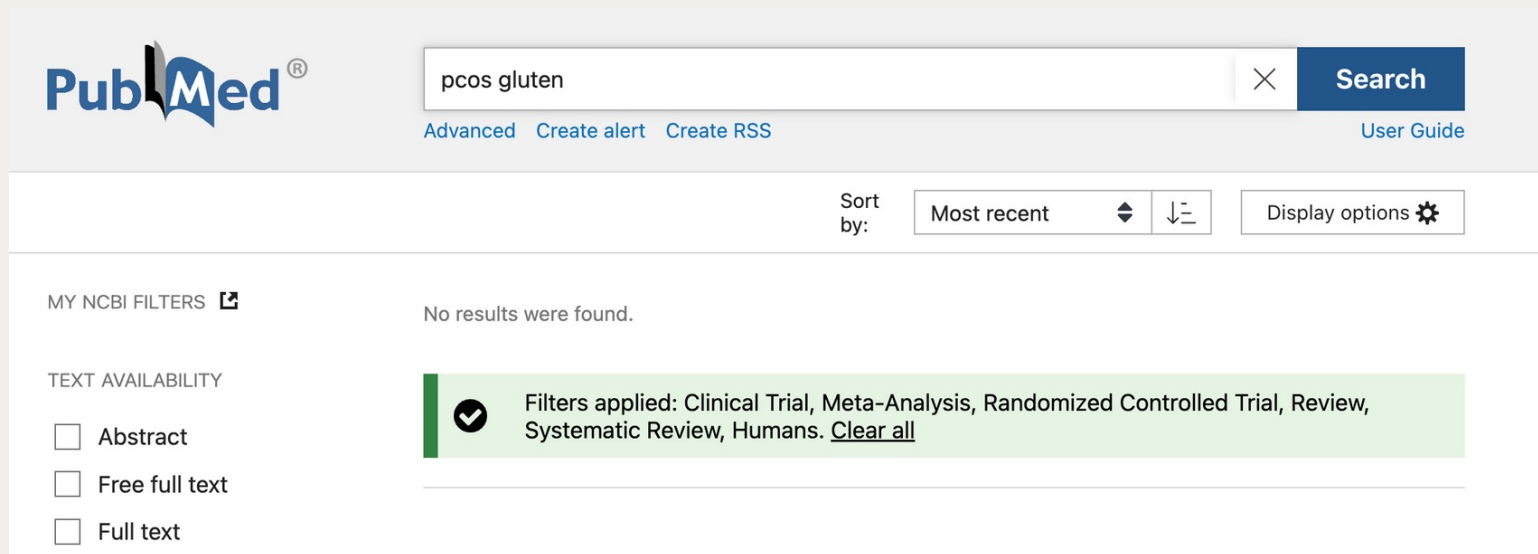
PCOS Diet - Research



Limited research on:

- **Vegan diet** - weight loss at 3 months, no change at 6 months - 67% attrition rate
- **Keto diet** - ↓ weight, insulin, testosterone; only 5/11 women completed the study
- **Intermittent fasting** - 8-hour eating window ↓ weight, BMI, testosterone, SHBG, insulin, HOMA-IR, improved menstrual cycle regularity (n=18)
- **Caloric timing** -
 - 980 kcal BF/640 kcal L / 190 kcal D vs 190 kcal BF / 640 kcal L / 980 kcal D (isocaloric 1810)
 - ↓ testosterone (50%), glucose (7%), insulin (54%)

PCOS Diet - NO Research



The screenshot shows the PubMed search interface. At the top left is the PubMed logo. To its right is a search bar containing the text "pcos gluten". Below the search bar are links for "Advanced", "Create alert", and "Create RSS". To the right of the search bar is a "Search" button. Below the search bar is a "User Guide" link. Below the search bar is a "Sort by:" dropdown menu set to "Most recent", and a "Display options" button with a gear icon. Below the search bar is a "MY NCBI FILTERS" link. Below the search bar is a "TEXT AVAILABILITY" section with three checkboxes: "Abstract", "Free full text", and "Full text". Below the search bar is a green box with a checkmark icon and the text "Filters applied: Clinical Trial, Meta-Analysis, Randomized Controlled Trial, Review, Systematic Review, Humans. [Clear all](#)". Below the search bar is a message "No results were found."

There is no evidence that eliminating ANY food is necessary or beneficial for PCOS.

Busting PCOS Myths



There is a LOT of misinformation about PCOS online, much from influencers who are not licensed, credentialed healthcare practitioners

- You have to eat a certain way for PCOS - keto, “clean”
- You have to cut out gluten, dairy, sugar, soy, fruit, seed oils, etc
- You have to only do low-intensity exercise to avoid “spiking” insulin

Every case of PCOS is different.

There is no “perfect” diet for all people with PCOS.

We have to consider: needs, goals, preferences, labs, culture, access, other medical conditions, etc.

PCOS Diet - In Practice



The guidelines recommend a personalized, tailored approach within general nutrition guidelines

From best available research, we can glean:

- Balanced macronutrients
 - **Low-to-moderate carbohydrate** (~40% carb) AMDR 45-60% - emphasis on high fiber, low-glycemic
 - **Moderate-to-high protein** (25-30%) AMDR 10-35%; RDA 0.8 g/kg, ISSN: 1.4 -2.0 g/kg emphasis on variety of protein sources
 - **Moderate fat** AMDR 20-35% - emphasis on healthy fats & Omega-3s
- 40% carb / 30% protein / 30% fat with optimal physical activity may improve PCOS symptoms

PCOS Diet - Root Cause Approach



Insulin resistance	Inflammation	Gut imbalances	Hormone imbalances
↑ Protein ↑ Fiber ↓ Refined carbs, Added sugars ($\leq 5\%$) ↓ Saturated fat ↑ Resistant starch ↓ Snacking/grazing → ACV, cinnamon	↑ F&V ↑ Omega-3s ↑ Herbs/spices (turmeric, garlic, ginger, fresh herbs) ↑ green tea, coffee, chocolate, etc ↓ Fried foods	↑ Fiber, prebiotics ↑ Probiotics / fermented foods ↑ Antioxidants / polyphenols → chewing, mindfulness, rest & digest mode	↑ nutrient density - B vitamins, magnesium ↑ Antioxidants - Adequate fat & cholesterol → flaxseed, spearmint tea, adaptogens (eg, reishi, tulsi) prn

PCOS Supplements



Possibly effective for PCOS

- inositol
- berberine

- inositol (71 studies)
- chromium (43 studies)
- resveratrol (36 studies)
- l-carnitine (27 studies)
- magnesium (32 studies)
- berberine (24 studies)

- CoQ10 (18 studies)
- fish oil (19 studies)
- NAC (9 studies)
- Resistant starch (6 studies)

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Natural Medicines Database: <https://naturalmedicines.therapeuticresearch.com/>

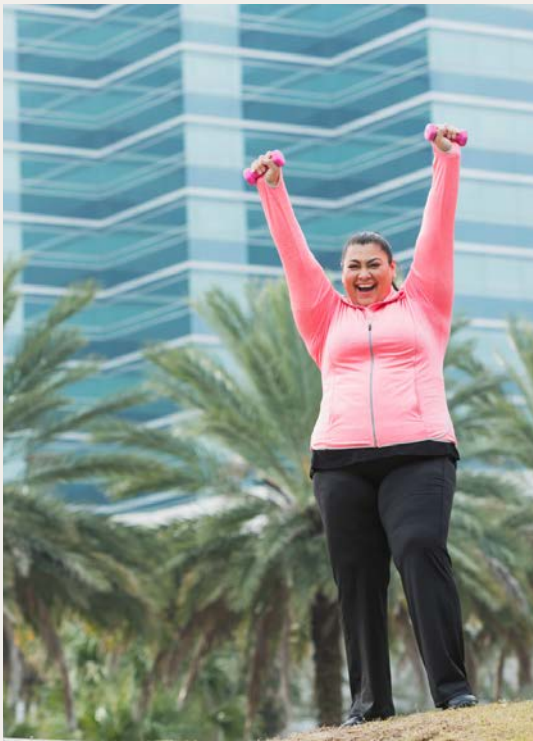
Examine Database:

<https://examine.com/conditions/polycystic-ovary-syndrome/>





Lifestyle Recommendations



Exercise - regular exercise may help improve IR & fertility, boost mood, and improve weight

Sleep - sleep disorders including insomnia, daytime sleepiness & obstructive sleep apnea common in PCOS; <6 hours a night increases fasting insulin & risk for IR

Stress - since adrenal hormones can play a major role, efforts should be taken to reduce stress & manage response to stress

Environment - multi-strain improved menstrual cycle regularity, decreased total testosterone, improved QoL

Fernandez RC, Moore VM, Van Ryswyk EM, et al. *Nat Sci Sleep*. 2018;10:45-64.

Lim AJ, Huang Z, Chua SE, et al. *PLoS One*. 2016;11(11):e0167048.

Siddiqui S, Mateen S, Ahmad R, et al. *J Assist Reprod Genet*. 2022;39(11):2439-2473

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PCOS Lifestyle - Guidelines

3.1	Effectiveness of lifestyle interventions		
3.1.1	EBR	Lifestyle intervention (exercise alone or multicomponent diet combined with exercise and behavioural strategies) should be recommended for all women with PCOS, for improving metabolic health including central adiposity and lipid profile.	◆◆◆◆ ⊕○○○
3.1.2	CR	Healthy lifestyle behaviours encompassing healthy eating and/or physical activity should be recommended in all women with PCOS to optimise general health, quality of life, body composition and weight management (maintaining weight, preventing weight gain and/or modest weight loss).	◆◆◆◆
3.1.3	PP	Healthcare professionals should be aware that lifestyle management is a core focus in PCOS management.	

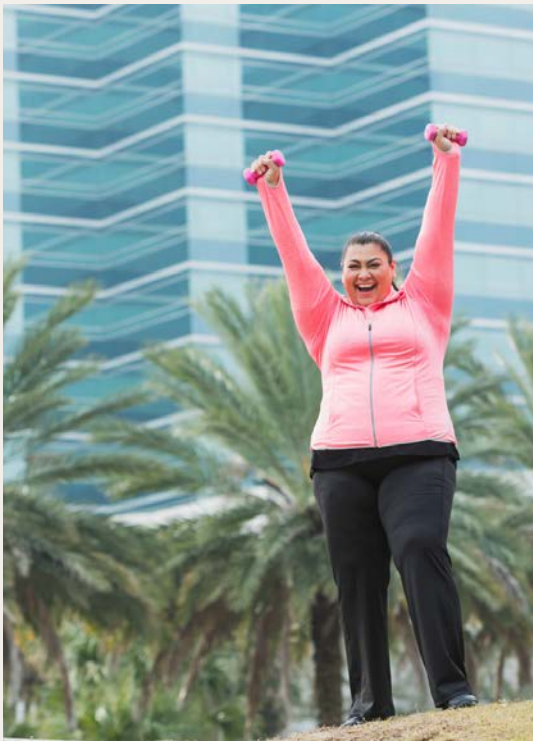


PCOS Exercise - Guidelines

3.4 Exercise interventions

3.4.1	EBR	Healthcare professionals and women could consider that there is a lack of evidence supporting any one type and intensity of exercise being better than another for anthropometric, metabolic, hormonal, reproductive or psychological outcomes.	◆◆◆ ⊕○○○
3.4.2	CR	Any physical activity consistent with population guidelines will have health benefits and within this, healthcare professionals should advise sustainable physical activity based on individual preferences and goals.	◆◆◆◆

PCOS Exercise - Research



Aerobic / cardio -

- 45-60 min 3x/week resulted in 2.3% loss in body fat & improved insulin sensitivity
- Walking after meals - even 2-5, 10 minutes, lowers BG

Weight / resistance training -

- Weight training 3x/week decreased visceral fat, increased lean body mass, decreased testosterone & FBG

Yoga-

- 60 min daily yoga significantly improved insulin, FBG, HOMA-IR vs 60 min conventional exercise ($p<.05$)

Scott D. *PLoS One*. 2017;12(8):e0182412.
Buffey AJ, Herring MP, Langley CK, et al. *Sports Med*. 2022;52(8):1765-1787.
Kogure GS. *Med Sci Sports Exerc*. 2016;48(4):589-598.
Nidhi R. *Int J Gynaecol Obstet*. 2012;118(1):37-41.

PCOS Recommendations - Summary



Nutrition

- increase protein, increase fiber
- low-to-moderate carbohydrate, emphasis on quality, personalized for goals
- moderate fat, emphasis on quality / Omega-3s
- increase antioxidants

Lifestyle

- Exercise: 150-300 min cardiovascular exercise, full resistance training 2-3x/week
- Adequate sleep (7-9 hr), manage stress
- Be mindful of endocrine disrupting chemicals

Testing

- Track cycles & ovulation
- Test insulin, A1c, glucose, hs-CRP
- Sex hormones, adrenal & thyroid hormones, gut testing as warranted



“An interdisciplinary care model involves “the collaboration between a [woman] with PCOS and a care team who have shared goals for total wellbeing”

Treatment team may be comprised of: primary care physician, gynecologist, endocrinologist, diabetes educator, infertility specialist, dietitian, dermatologist, psychologist and/or an exercise physiologist

Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group 2004 Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertil Steril* 81:19–25

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