

Women and MASLD:

Influence of Sex Hormones on Liver Disease

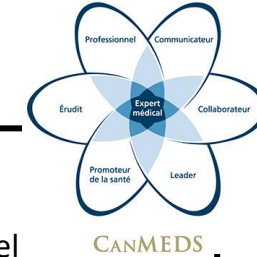
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DISCLOSURES

Grant funding and consulting for Gilead Sciences

Compétences CanMEDS



X	Expert médical (En tant qu'experts médicaux, les médecins assument tous les rôles CanMEDS et s'appuient sur leur savoir médical, leurs compétences cliniques et leurs attitudes professionnelles pour dispenser des soins de grande qualité et sécuritaires centrés sur les besoins du patient. Pivot du référentiel CanMEDS, le rôle d'expert médical définit le champ de pratique clinique des médecins .)
X	Communicateur (En tant que communicateurs, les médecins développent des relations professionnelles avec le patient et ses proches ce qui permet l'échange d'informations essentielles à la prestation de soins de qualité.)
X	Collaborateur (En tant que collaborateurs, les médecins travaillent efficacement avec d'autres professionnels de la santé pour prodiguer des soins sécuritaires et de grande qualité centrés sur les besoins du patient.)
X	Leader (En tant que leaders, les médecins veillent à assurer l'excellence des soins, à titre de cliniciens, d'administrateurs, d'érudits ou d'enseignants et contribuent ainsi, avec d'autres intervenants, à l'évolution d'un système de santé de grande qualité.)
X	Promoteur de santé (En tant que promoteurs de la santé, les médecins mettent à profit leur expertise et leur influence en oeuvrant avec des collectivités ou des populations de patients en vue d'améliorer la santé. Ils collaborent avec ceux qu'ils servent afin d'établir et de comprendre leurs besoins, d'être si nécessaire leur porte-parole, et de soutenir l'allocation des ressources permettant de procéder à un changement.)
X	Érudit (En tant qu'érudits, les médecins font preuve d'un engagement constant envers l'excellence dans la pratique médicale par un processus de formation continue, en enseignant à des tiers, en évaluant les données probantes et en contribuant à l'avancement de la science.)
X	Professionnel (En tant que professionnels, les médecins ont le devoir de promouvoir et de protéger la santé et le bien-être d'autrui, tant sur le plan individuel que collectif. Ils doivent exercer leur profession selon les normes médicales actuelles, en respectant les codes de conduite quant aux comportements qui sont exigés d'eux, tout en étant responsables envers la profession et la société. De plus, les médecins contribuent à l'autoréglementation de la profession et voient au maintien de leur santé.)

Overview

1

PMOS and the Liver

What is PMOS? Role of androgens in MASLD

2

Premature Ovarian Insufficiency

Early estrogen loss and MASLD risk

3

MASLD Across Reproductive Life

Considerations for MASLD prevalence and severity

4

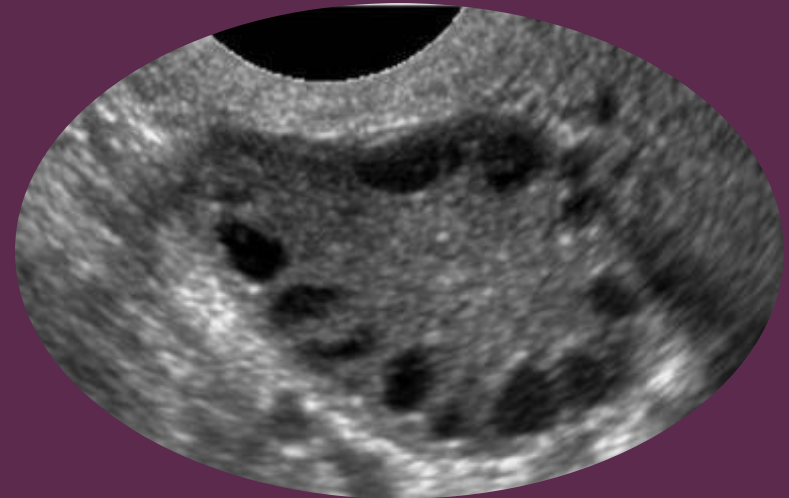
The Changing MHT Landscape

FDA-removal of black box warning – what this means for patients with liver disease

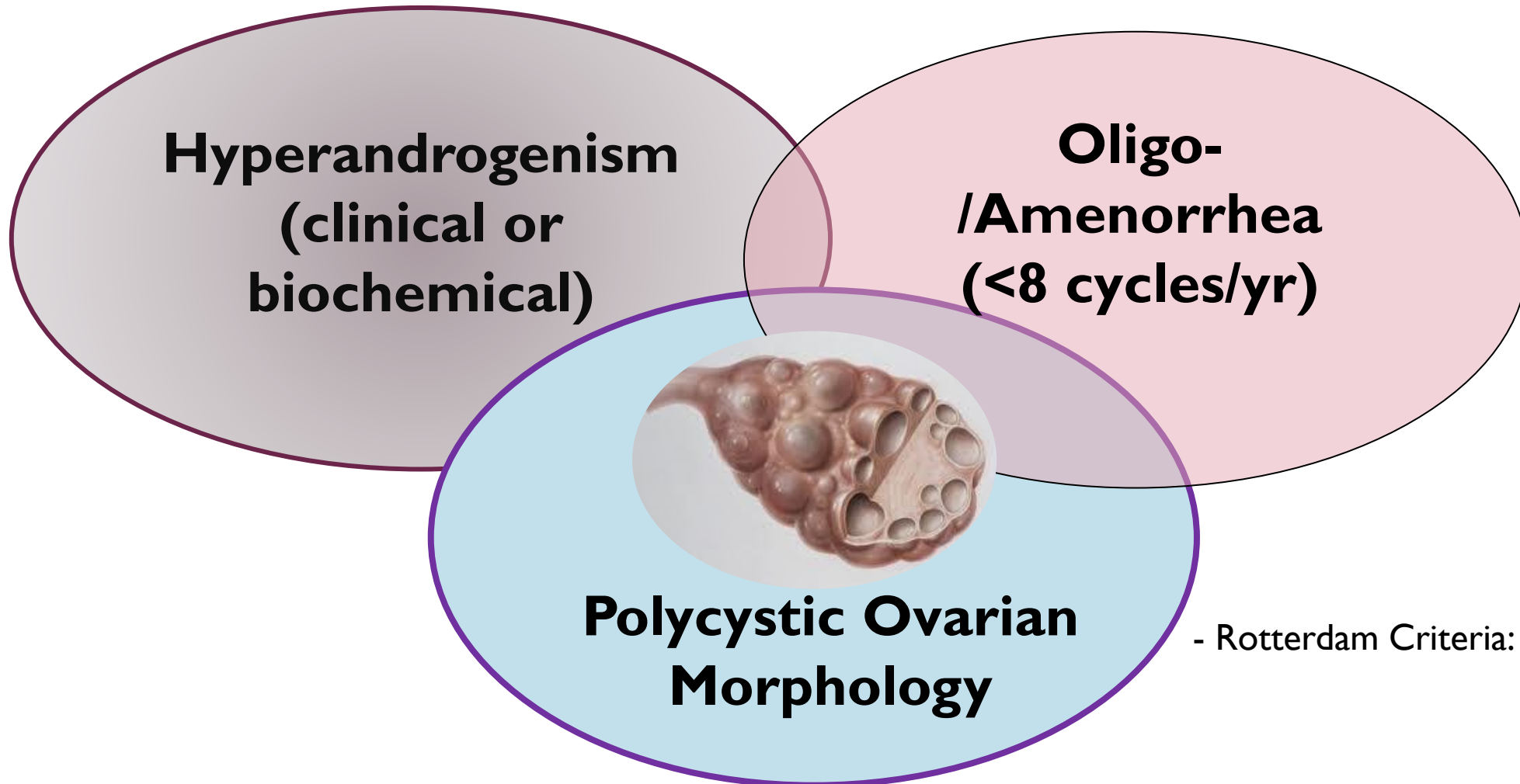
POLYCYSTIC OVARY SYNDROME (PCOS)

2026....“Polyendocrine Metabolic Ovarian Syndrome (PMOS)”

MOST COMMON endocrinopathy
in reproductive-aged women
(prevalence 10-15%)



PMOS DIAGNOSIS



- Rotterdam Criteria: 2/3 for diagnosis

PMOS DIAGNOSIS

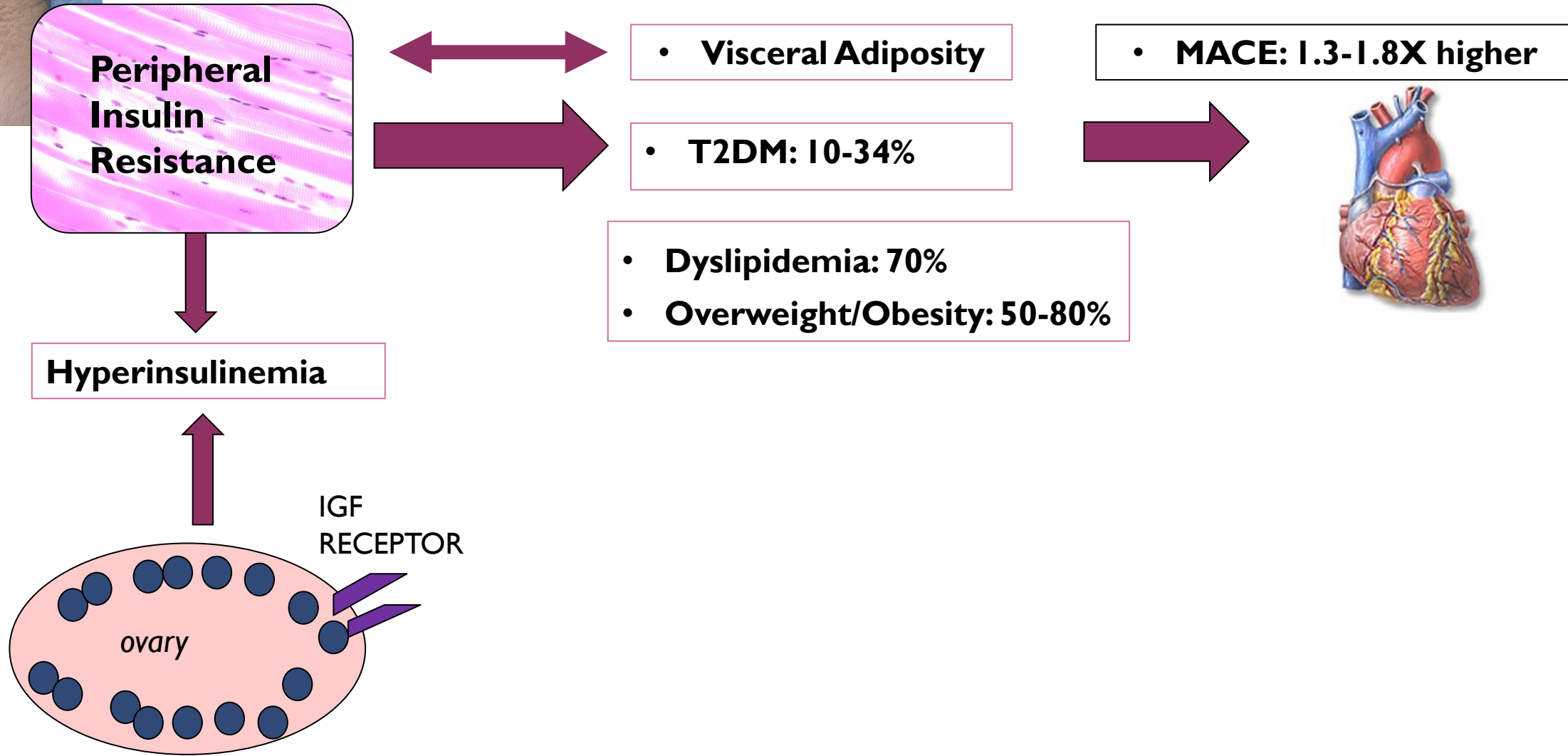
**Hyperandrogenism
(clinical or
biochemical)**

ELEVATED ANDROGENS:

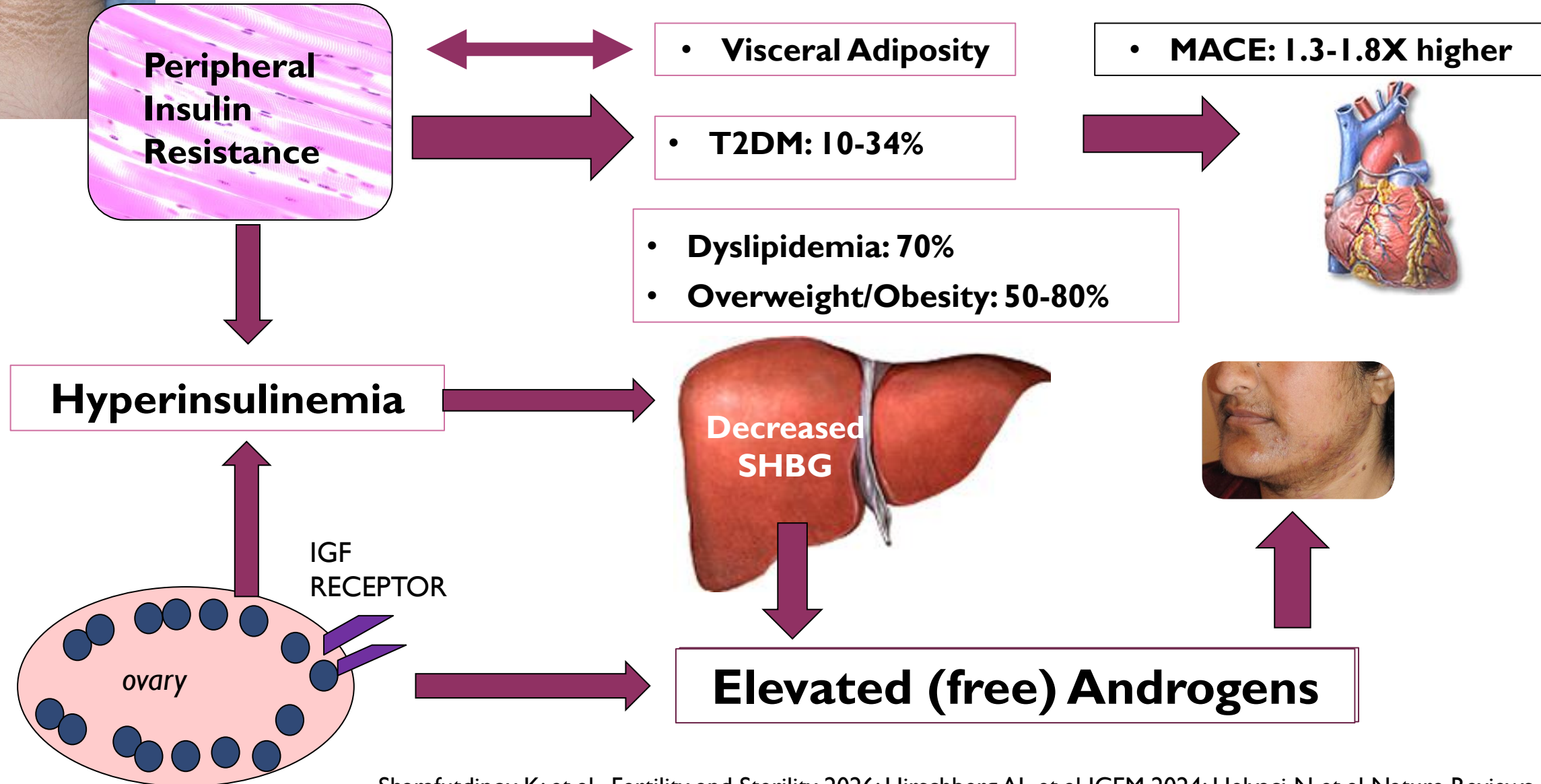
- Testosterone
- DHEAS
- Androstendione



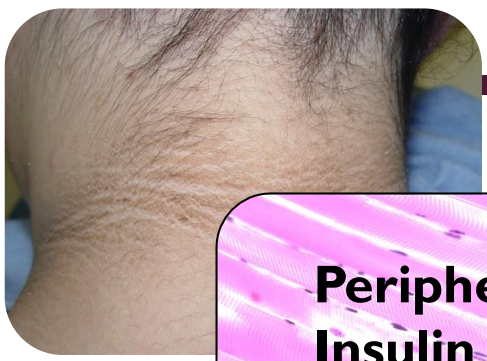
METABOLIC INSULTS OF PMOS



METABOLIC INSULTS OF PMOS



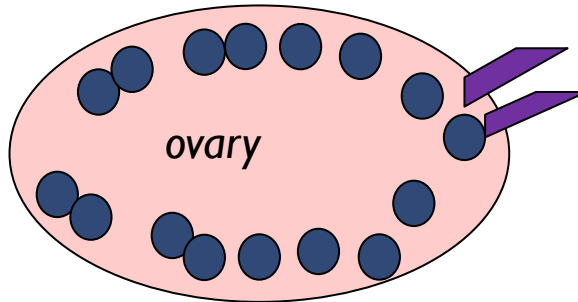
...MASLD IS COMMON IN PMOS



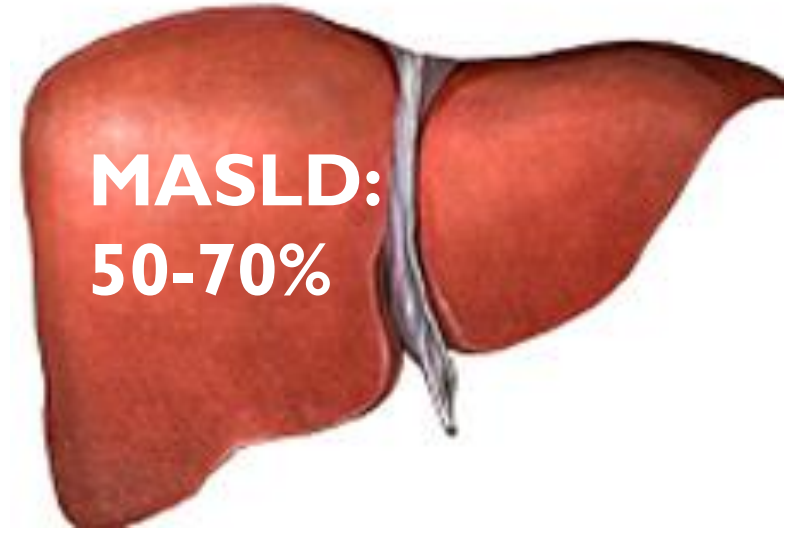
Peripheral
Insulin
Resistance

Hyperinsulinemia

IGF
RECEPTOR



- Visceral Adiposity
- IR/T2DM
- Dyslipidemia
- Overweight/Obesity



MASLD:
50-70%

MASLD SEVERITY IN PMOS

- Reproductive-aged women at UCSF; biopsied performed for suspected MASH
- 37/102 (36%) had PMOS
- MASH: 76% PMOS vs 66% non-PMOS

Table 2. Association of PCOS with NASH Severity (n=102)		
Outcome	Unadjusted OR (95 % CI), p value	Adjusted OR (95 % CI), p value
Severe hepatocyte ballooning	3.4 (1.2-9.2) p=0.02	3.4 (1.1-10.6) p=0.03
Advanced fibrosis	3.0 (0.8-11.2) p=0.02	7.1 (1.3-39.1) p=0.02

Adjusted for Age and BMI

- Among women with advanced fibrosis, median age 5 years younger in PMOS (40 vs 45 years, p=0.02)

MASLD SEVERITY IN PMOS

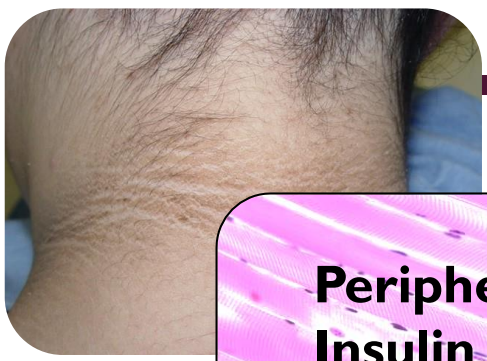


- Retrospective study of U.S.-wide hospital discharge data in reproductive-aged women with MASLD
- Evaluated association of PMOS with dedicated ICD code for “NASH”
- 9,415 PMOS vs 180,025 without PMOS

Characteristic	AOR (95% CI)	p-value
PMOS	1.2 (1.1-1.4)	0.008

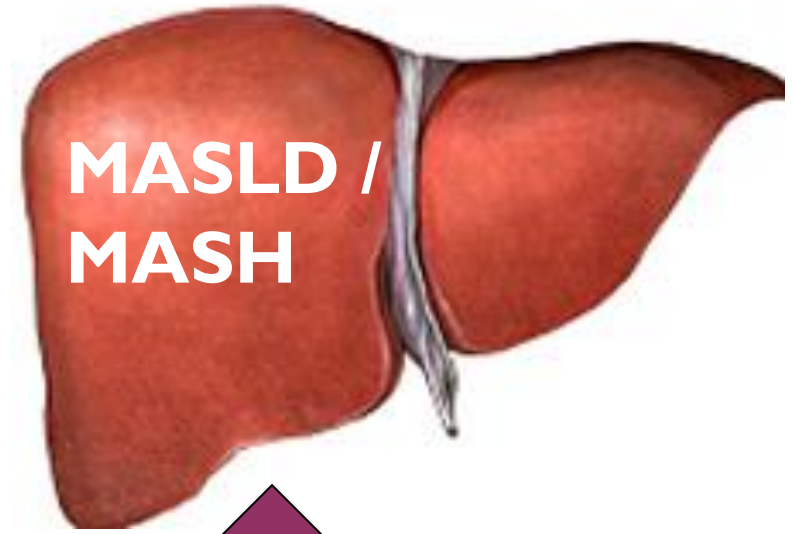
Adjusted for age, race, insurance type, rural vs urban location, DM, obesity, dyslipidemia, HTN

...DRIVERS OF MASLD / MASH IN PMOS



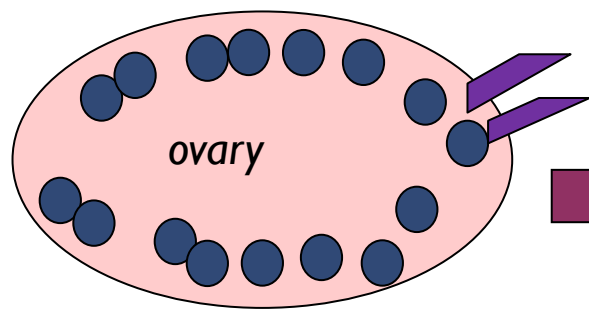
**Peripheral
Insulin
Resistance**

- Visceral Adiposity
- IR/T2DM
- Dyslipidemia
- Overweight/Obesity



Hyperinsulinemia

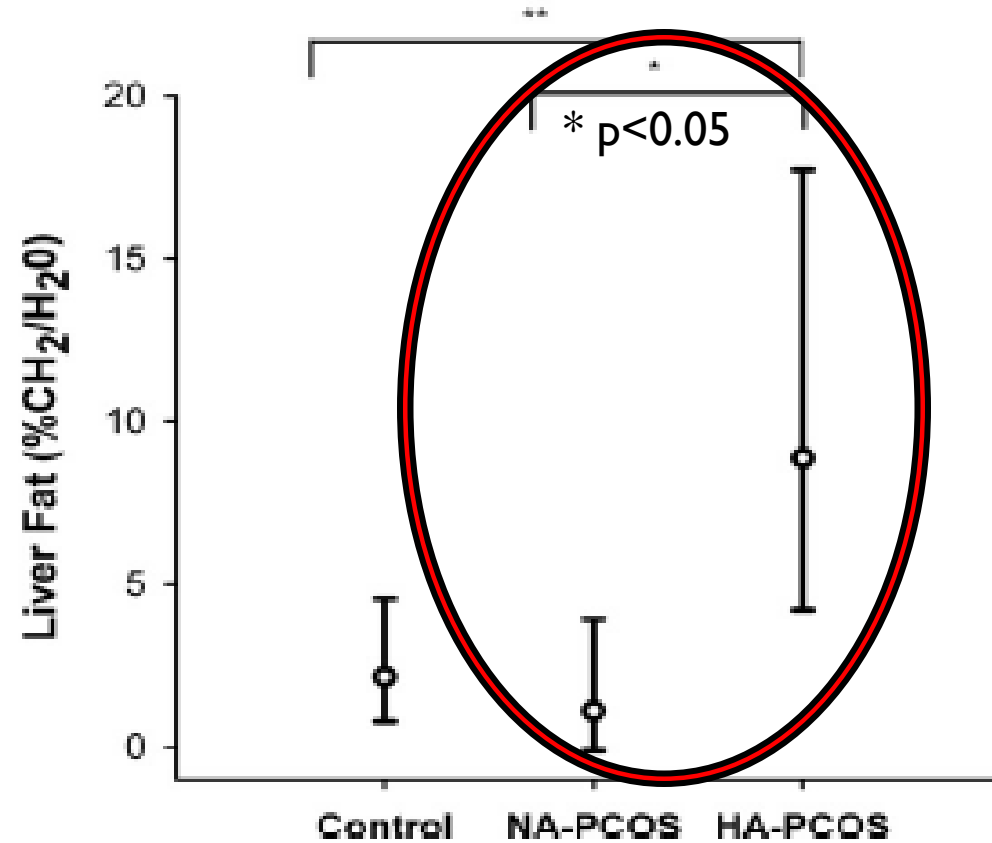
IGF
RECEPTOR



Elevated Androgens

ANDROGENS AND MASLD IN PMOS

- Case control study: 29 PMOS, 22 controls
- Hepatic steatosis assessed by MRI spectroscopy
- Adjusted for HOMA-IR and visceral adipose tissue volume



ANDROGENS AND MASLD IN WOMEN

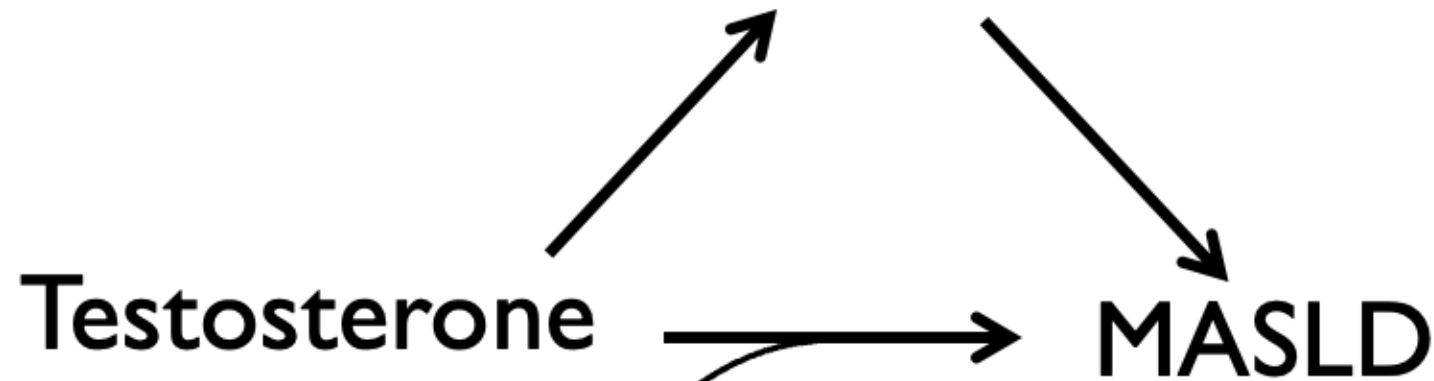
- 1052 healthy young women in CARDIA, followed for 25 years
- Association of testosterone levels in young adulthood with MASLD in midlife
- MASLD defined by CT scan
- Adjusted for baseline and change in metabolic comorbidities



Characteristic*	Univariate Analysis		Multivariate Analysis**	
	OR (95% CI)	p value	AOR (95% CI)	p value
Free testosterone at Year 2 (quintiles)	1.29 (1.10-1.51)	0.001	1.25 (1.04-1.50)	0.015
Age, per 5 years	1.01 (0.75-1.37)	0.933		
White vs black race	1.23 (0.79-1.90)	0.357		
CARDIA center	0.98 (0.81-1.20)	0.843		
Hormonal birth control use	0.63 (0.39-1.03)	0.066		
BMI (per 5 kg/m ²)	1.36 (1.14-1.62)	<0.001	-----	-----
Δ BMI (per 5 kg/m ²)	2.01 (1.69-2.40)	<0.001	-----	-----
Waist circumference (per 1cm)	1.04 (1.03-1.06)	<0.001	1.05 (1.02-1.08)	<0.001
Δ Waist circumference (per 1cm)	1.08 (1.06-1.10)	<0.001	1.08 (1.05-1.10)	<0.001
Triglycerides (per 20 mg/dL)	1.15 (1.06-1.26)	0.002	1.23 (1.09-1.39)	0.001
Δ Triglycerides (per 20 mg/dL)	1.14 (1.08-1.20)	<0.001	1.09 (1.03-1.15)	0.002
Total cholesterol (per 10mg/dL)	1.01 (0.18-5.64)	0.989		
Δ Total cholesterol (per 10mg/dL)	2.15 (0.68-6.85)	0.193		
LDL (per 10 mg/dL)	1.02 (0.95-1.09)	0.659		
Δ LDL (per 10 mg/dL)	1.02 (0.95-1.09)	0.651		
HDL (per 5mg/dL)	0.93 (0.84-1.01)	0.095		
Δ HDL (per 5mg/dL)	0.81 (0.74-0.88)	<0.001	-----	-----
HOMA-IR (per unit)	1.26 (1.04-1.55)	0.018	0.81 (0.56-1.16)	0.243
Δ HOMA-IR (per unit)	1.32 (1.23-1.42)	<0.001	1.11 (1.04-1.18)	0.002

WHY DO ANDROGENS INCREASE STEATOSIS?

Visceral Adiposity, Dyslipidemia, Insulin Resistance?



**41% of estimated
effect via VAT**
12% via Triglycerides



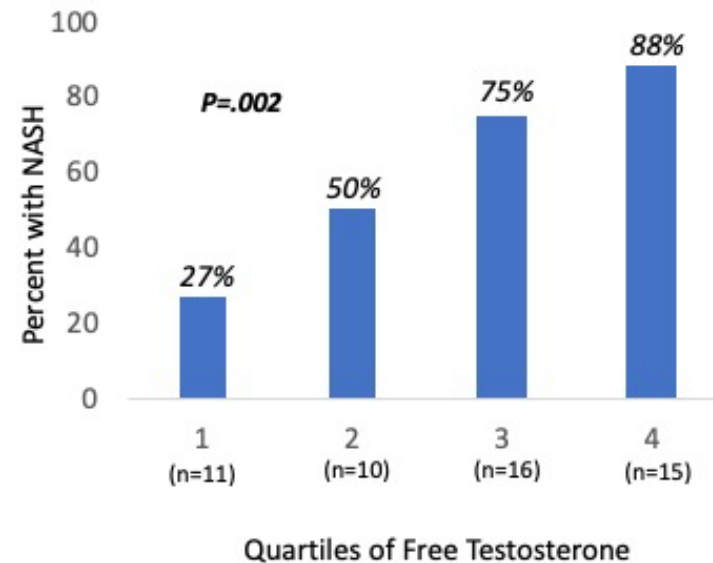
DO ANDROGENS PROMOTE LIVER INJURY?

- NASH CRN- NIDDK funded prospective cohort, comprehensive serologic, histologic data
- Predictor: Free T within 6 mo of liver biopsy
- Outcome: MASLD histology



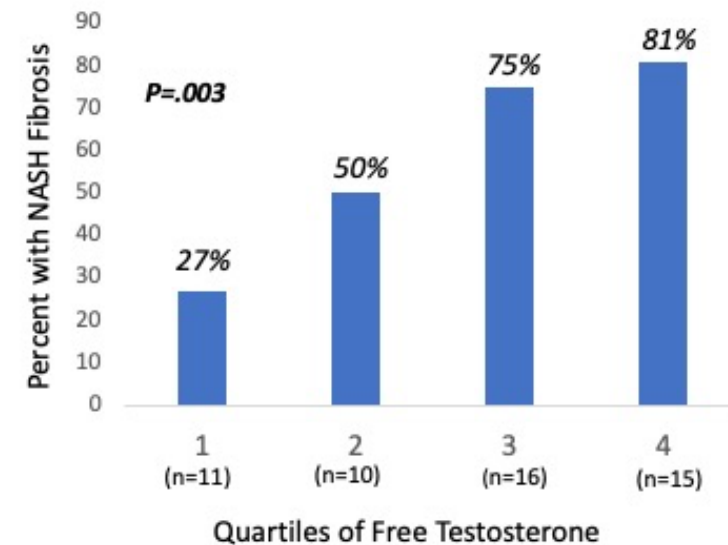
MASH

Figure 1a



MASH Fibrosis

Figure 1b



ANDROGENS AND MASH IN PRE-MENOPAUSAL WOMEN

MASLD Comparisons (n=207)	Adjusted OR (95% CI), p-value
MASH (vs simple steatosis)	2.3 (1.2-4.4), p=0.01
Any fibrosis	2.1 (1.1-3.8), p=0.02
Higher fibrosis stage	1.9 (1.1-3.4), p=0.02



Adjusted for DM2, abdominal adiposity, and dyslipidemia

FREE TESTOSTERONE & VISCERAL ADIPOSITY

Outcomes (n=207)	Unadjusted OR (95% CI), p-value	Adjusted••
T2DM	1.0 (0.78-1.3), p=0.89	---
Dyslipidemia	1.0 (0.53-1.8), p=0.73	---
Abdominal adiposity	2.2 (1.2-3.8), p<0.01	2.2 (1.2-3.8), p<0.01

••Adjusted for Age, T2DM, dyslipidemia

ROLE FOR AR ANTAGONISM?



- Reproductive-aged women with biopsy-confirmed MASH
- 2:1 double-blind, pilot RCT
- 100mg spironolactone (n=11) vs placebo (n=6)
- 6-month duration, optional 12-month extension

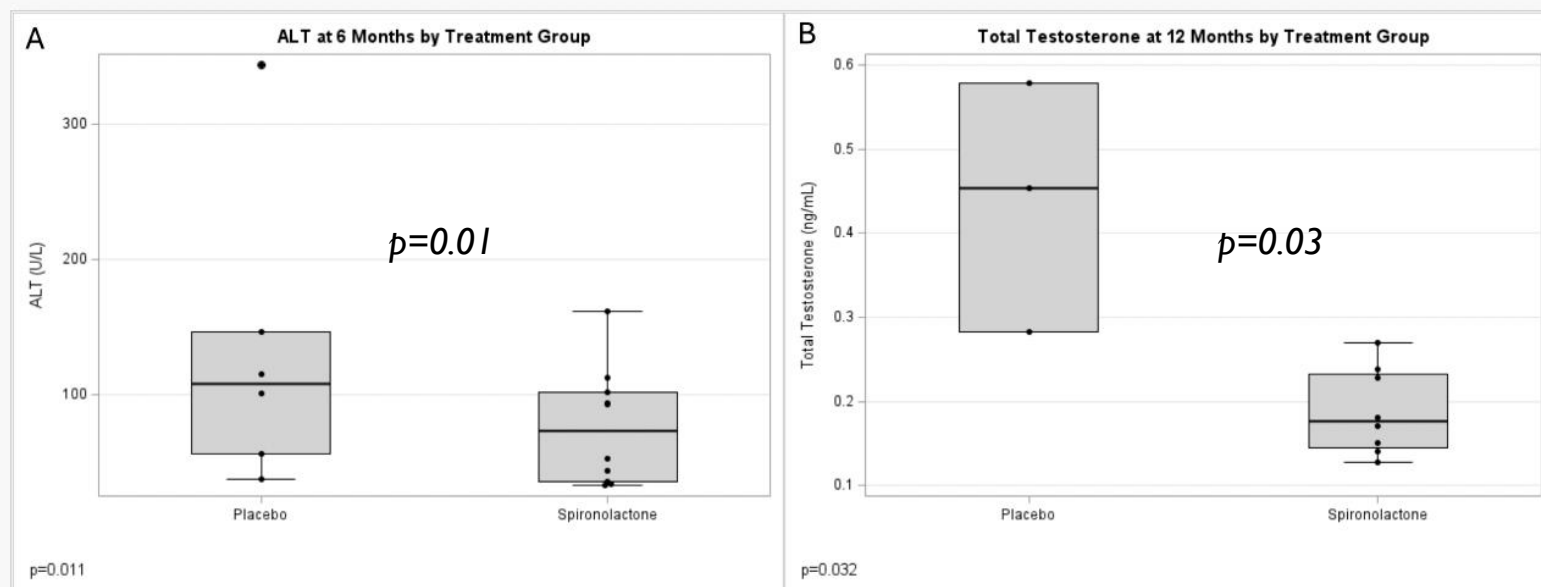


Figure 1: Change in ALT and Total Testosterone After Treatment with Spironolactone

(A) After 6 months, participants who received spironolactone had significantly lower ALT levels compared with those who received the placebo ($p=0.011$).

(B) After 12 months, participants who received spironolactone had significantly lower total testosterone levels compared with those who received the placebo ($p=0.032$).

ROLE FOR ANDROGEN ANTAGONISM?

UCSF Clinical Trials

Search Browse ▾ Equity in Research About the site ▾

Home > Nonalcoholic Fatty Liver Disease Studies > This trial

Search UCSF clinical trials

Bicalutamide Therapy in Young Women With NAFLD and PCOS

I'm interested!



a study on [Nonalcoholic Fatty Liver Disease](#),
[Polycystic Ovary Syndrome](#), and [Liver Disease](#)



for females ages 18-42 ([full criteria](#))



at [San Francisco, California](#)



study started February 2024
study ends around August 2028

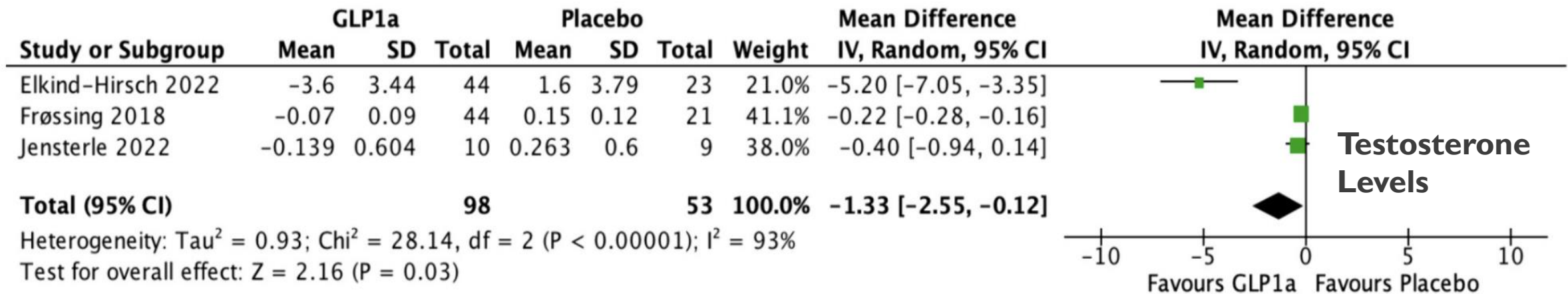


by [Monika Sarkar](#)

Monika
Sarkar

GLPI-RECEPTOR AGONISTS IN PMOS

- Meta-analysis of RCTs using GLPIs vs placebo in PMOS patients



-- Also evident for BMI and triglycerides



2021 AASLD Reproductive Health Guidance:

In reproductive-aged adolescents and women with NAFLD-

- *Evaluation for symptoms associated with PCOS is recommended.*



In clinic....:

“Have you ever been told that you have a condition called PCOS/PMOS?”

“When you were in your 20s, did you get your periods most months?”

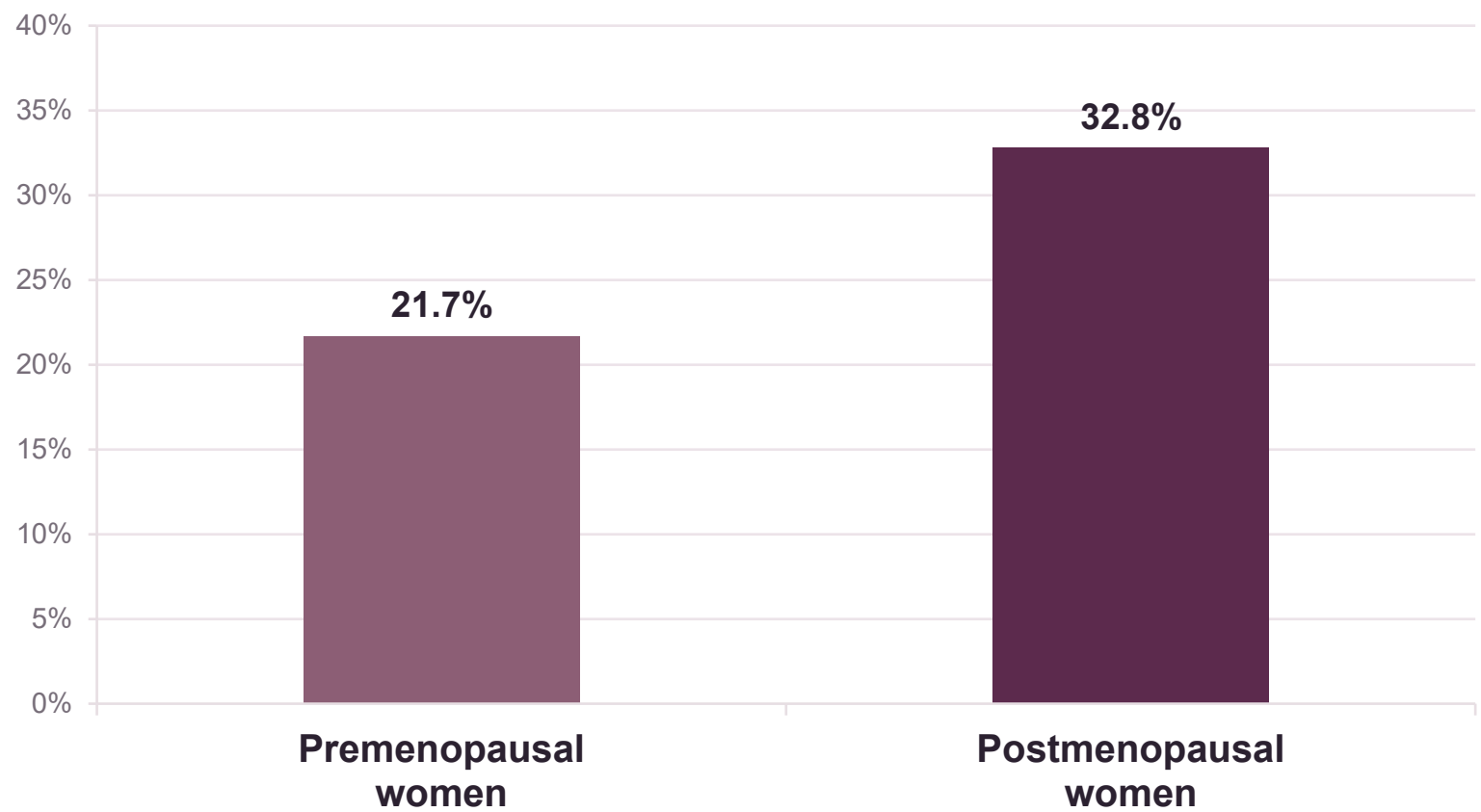
“Have you noticed excess hair growth in places like your chin, chest, or back?”

...refer to reproductive endocrinology if PMOS suspected



....Transition to Menopause

MASLD Prevalence Across Reproductive Age



Cross-sectional analysis, NHANES 2017–2020 (US adults ≥ 18y); MASLD by CAP/VCTE, per consensus definitions

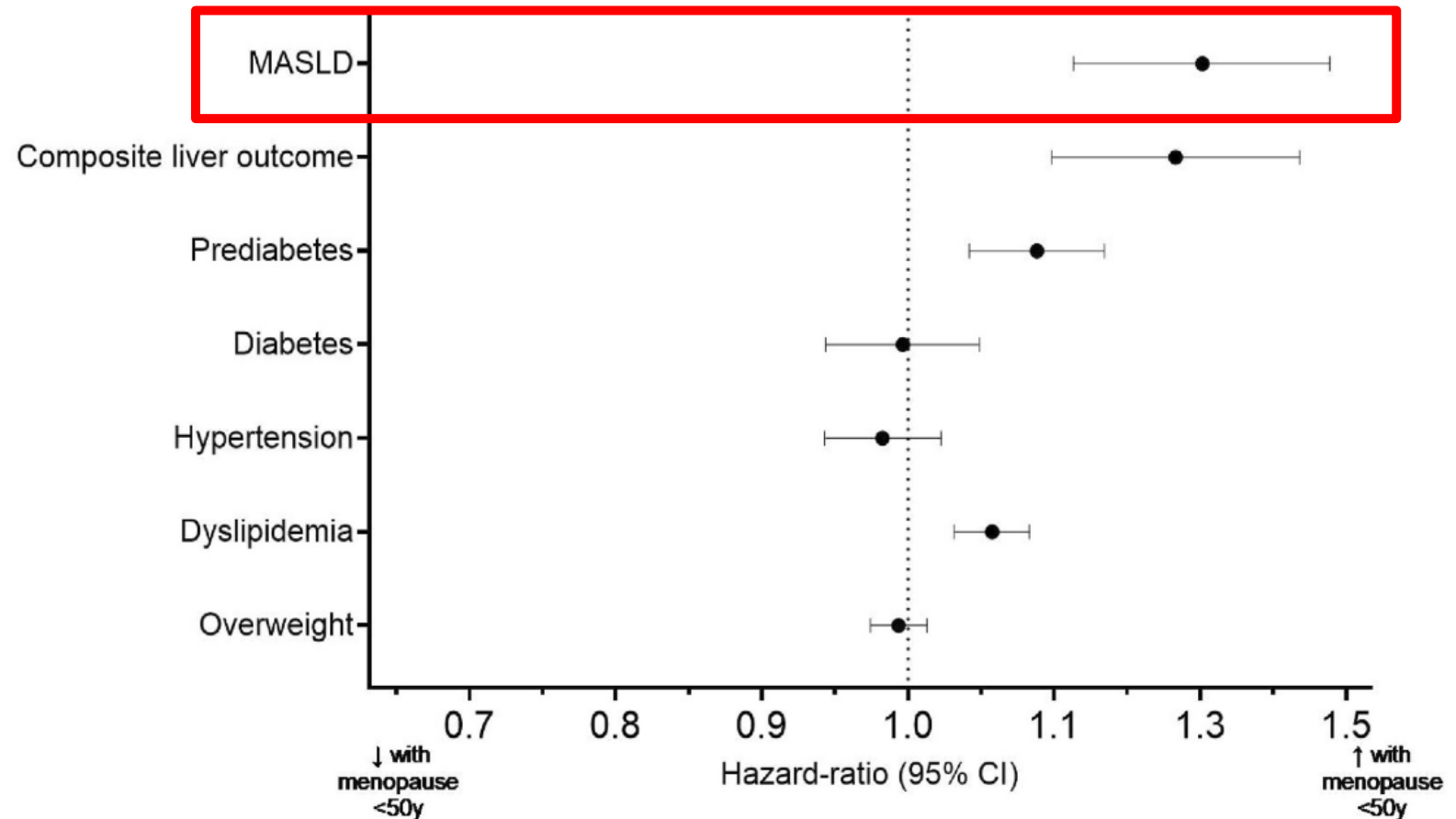
Greater MASLD Risk With Earlier Age of Menopause

- ~ 270K women without MASLD recruited from 2006-2010 in UK Biobank
- Followed through 2021 to assess incident MASLD via ICD codes

Characteristics	Cases/Total	Model 3
		HR (95% CI)
Menopause status		
No (ref)	538/63,403	1.00 (ref)
Yes	737/163,012	1.11 (0.96–1.29)
Type of menopause		
Nonmenopausal (ref)	538/63,403	1.00 (ref)
Natural menopause	1519/151,051	1.05 (0.91–1.22)
Surgical menopause	83/4596	1.51 (1.17–1.95)
Age at natural menopause, y		
Nonmenopausal (ref)	538/63,403	1.00 (ref)
<45	296/24,382	1.12 (0.93–1.34)
45–49	345/33,512	1.05 (0.88–1.24)
>50	878/93,157	0.99 (0.84–1.16)
Age at surgical menopause, y		
Nonmenopausal (ref)	538/63,403	1.00 (ref)
<45	41/1590	1.64 (1.14–2.36)
45–49	21/1498	1.15 (0.71–1.88)
≥50	21/1508	1.31 (0.79–2.18)
Hysterectomy		
None (ref)	2063/218,386	1.00 (ref)
Yes	348/19,577	1.49 (1.32–1.68)
Yes, after menopause	117/7406	1.32 (1.09–1.59)
Hysterectomy without bilateral oophorectomy		
None (ref)	2044/216,977	1.00 (ref)
Yes	151/9884	1.35 (1.14–1.60)
Yes, after menopause	45/3269	1.30 (0.96–1.75)
Bilateral oophorectomy		
None (ref)	2537/243,681	1.00 (ref)
Yes	447/21,404	1.60 (1.45–1.78)
Yes, after menopause	348/16,224	1.65 (1.48–1.85)

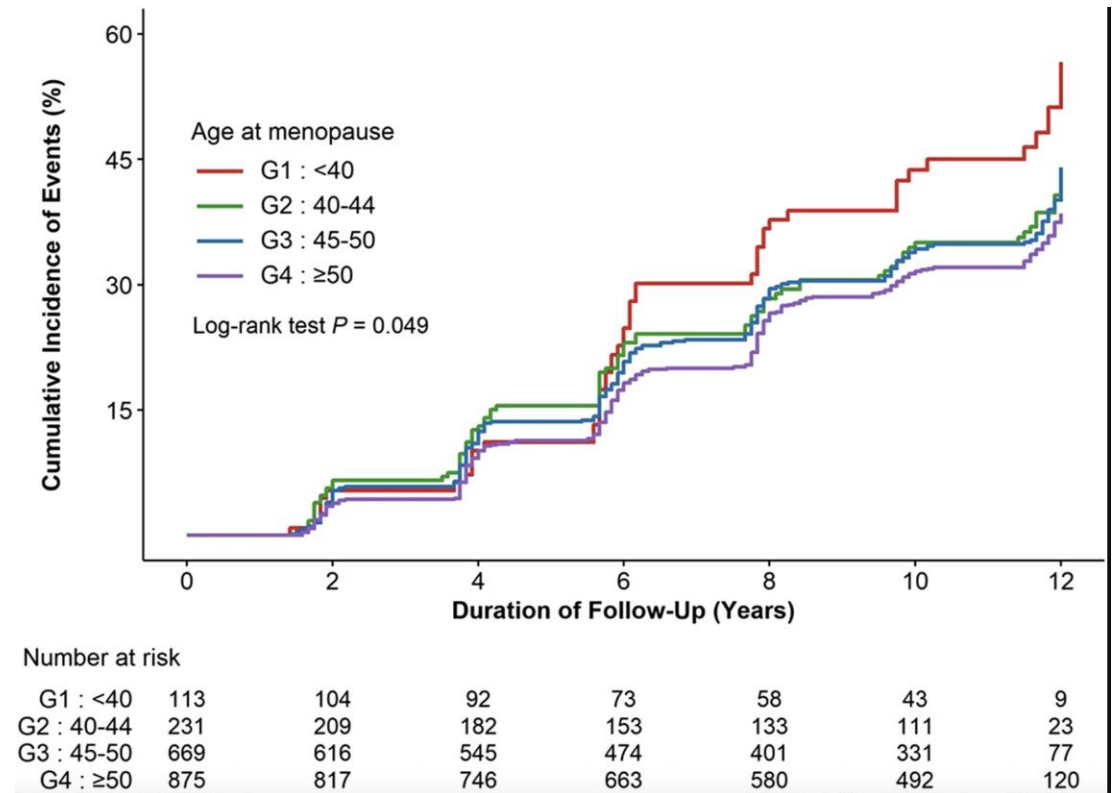
Greater MASLD Risk With Earlier Age of Menopause

- TriNetX study comparing outcomes with earlier menopause (< 50 years) vs age-matched pre-menopausal women (n=20K group)
- Excluded POI, surgical menopause
- Propensity-matched, adjusted for metabolic risk factors



Greater MASLD Incidence With Premature Ovarian Insufficiency (POI)

- **POI**= menopause < age 40 yrs
- Prospective study of 1,888 Korean women, median f/u 12.3 yrs
- Assessed association of menopausal age with incident MASLD (via NAFLD-liver fat score)
- Incident MASLD in <40 vs ≥ 50 yrs: **HR 1.38 (95% CI 1.01–1.88)**, Adjusted for Age, BMI, alcohol, tobacco, physical activity, HTN, dyslipidemia, menarche age, parity, MHT



MASH Fibrosis Greater With Premature Ovarian Insufficiency (POI)

- NASH CRN Cohort: 488 post-menopausal women
- Biopsy-confirmed MASLD
- Assessed association of POI with fibrosis severity

Premature menopause, Time from menopause and risk of Fibrosis

	Univariate COR(95% CI), p-value	Model 1 COR(95% CI), p-value	Model 2 COR(95% CI), p-value
Premature menopause			
No	-	-	-
Yes	1.5 [1.0–2.1], p=0.03	1.9 [1.3–2.7], p=0.001	1.5 [1.0–2.3], p=0.03
Time from menopause, for 5 years	1.2 [1.1–1.3], p<0.0001	1.2 [1.1–1.3], p=0.002	1.1 [1.0–1.2], p=0.06

Model 1: Adjusted for age at enrollment, race, waist circumference/BMI, current smoking/alcohol use, hypertension, diabetes/IFG, HOMA-IR, and HRT.

Model 2: Adjusted for the above covariates, hepatocyte ballooning grades, and portal inflammation grades.

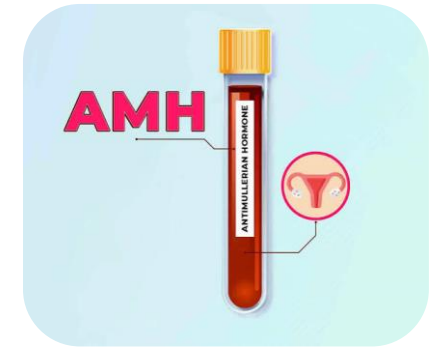
Anti-mullerian Hormone and MASLD

- CARDIA: Longitudinal, multicenter US study of healthy young adult, followed for 25 yrs
- Association of AMH levels in young adulthood with MASLD in midlife
- Adjusted for baseline and change in metabolic comorbidities

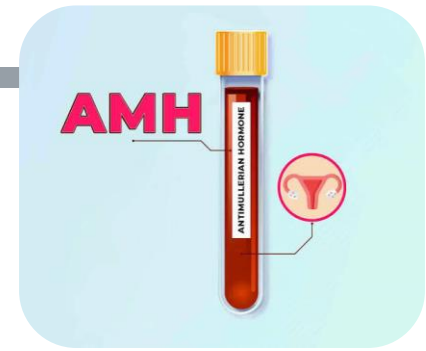
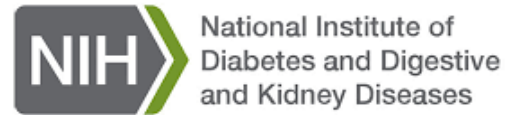
TABLE 3 Association of low AMH with prevalent MASLD, adjusted for baseline and change in covariates from baseline to end of study (n = 585)

	aOR (95% CI)	p
Low AMH (reference: non-low AMH)	2.36 (1.05–5.27)	0.04
Age > 42 y (reference: ≤ 42)	0.72 (0.35–1.50)	0.39
Hysterectomy (reference: no hysterectomy)	2.53 (1.16–5.49)	0.02
BMI (per kg/m ²)	1.10 (1.04–1.15)	< 0.01
ΔBMI (per Δ5 kg/m ²)	2.03 (1.31–3.15)	< 0.01
Triglycerides (per mg/dL)	1.01 (1.00–1.01)	< 0.01
ΔTriglycerides (per Δ10 mg/dL)	1.09 (1.02–1.15)	< 0.01
HOMA-IR (per 1 unit)	1.04 (0.91–1.19)	0.58
ΔHOMA-IR (per 1 unit)	1.07 (1.00–1.15)	0.04

Abbreviations: AMH, anti-Mullerian hormone; BMI, body mass index; HOMA-IR, homeostatic model assessment of insulin resistance; Δ, delta; MASLD, metabolic dysfunction–associated steatotic liver disease; PCOS, polycystic ovary syndrome.

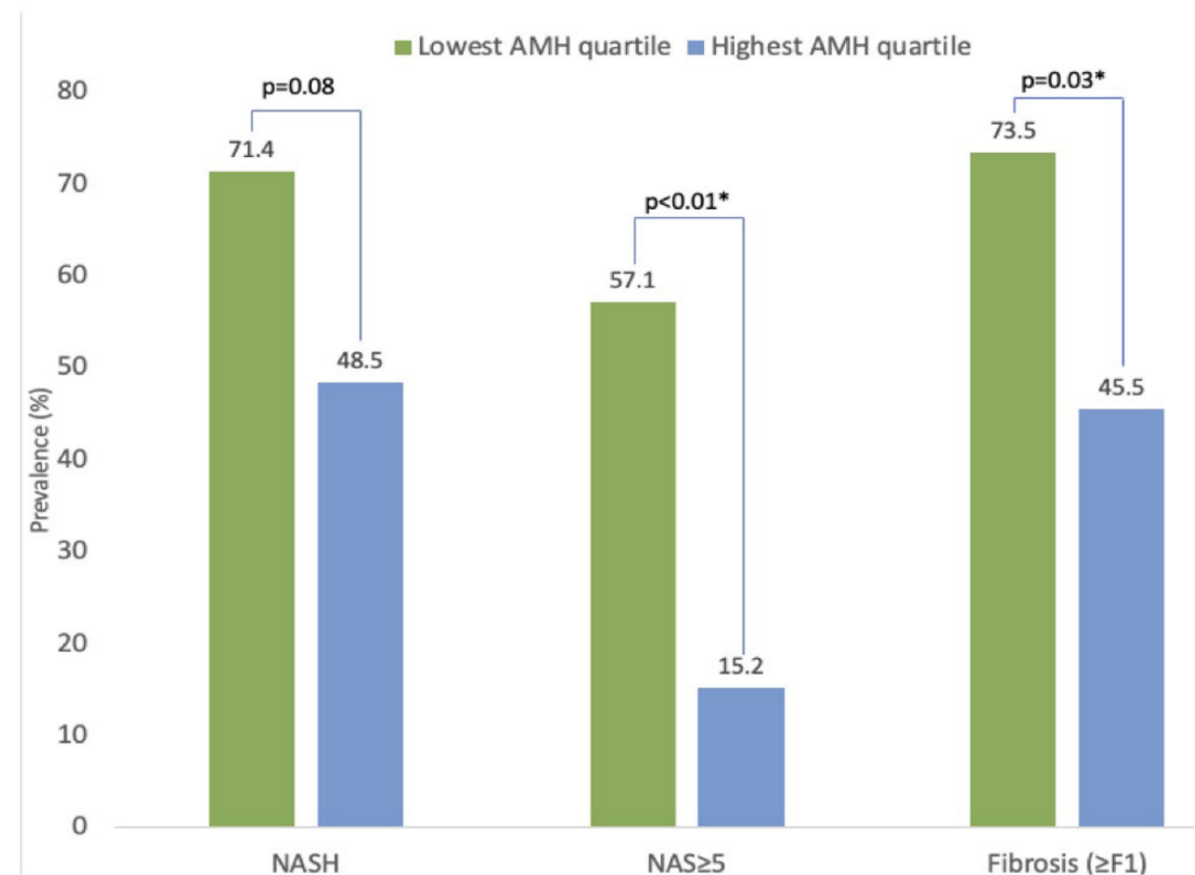


AMH and MASH

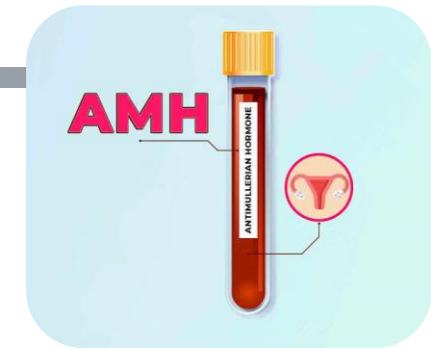


- NASH CRN- NIDDK funded prospective cohort, comprehensive serologic, histologic data
- Association of AMH levels with biopsy features of MASH (n=164)

b: Measures of NAFLD Severity by AMH Quartile in Women Without PCOS (n=164)



AMH and MASH



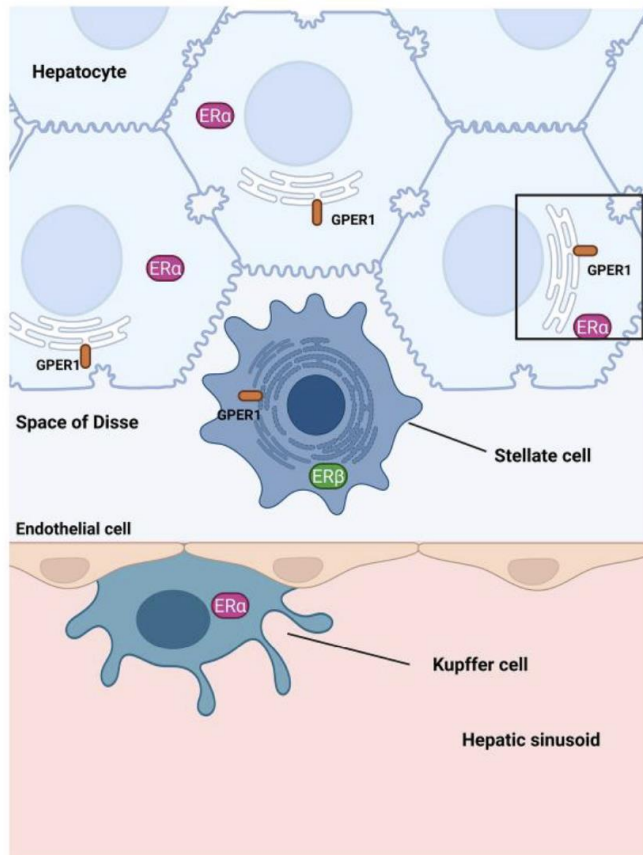
- NASH CRN: NIDDK-funded prospective cohort, comprehensive serologic, histologic data
- Association of AMH levels with MASH histology (n=164)



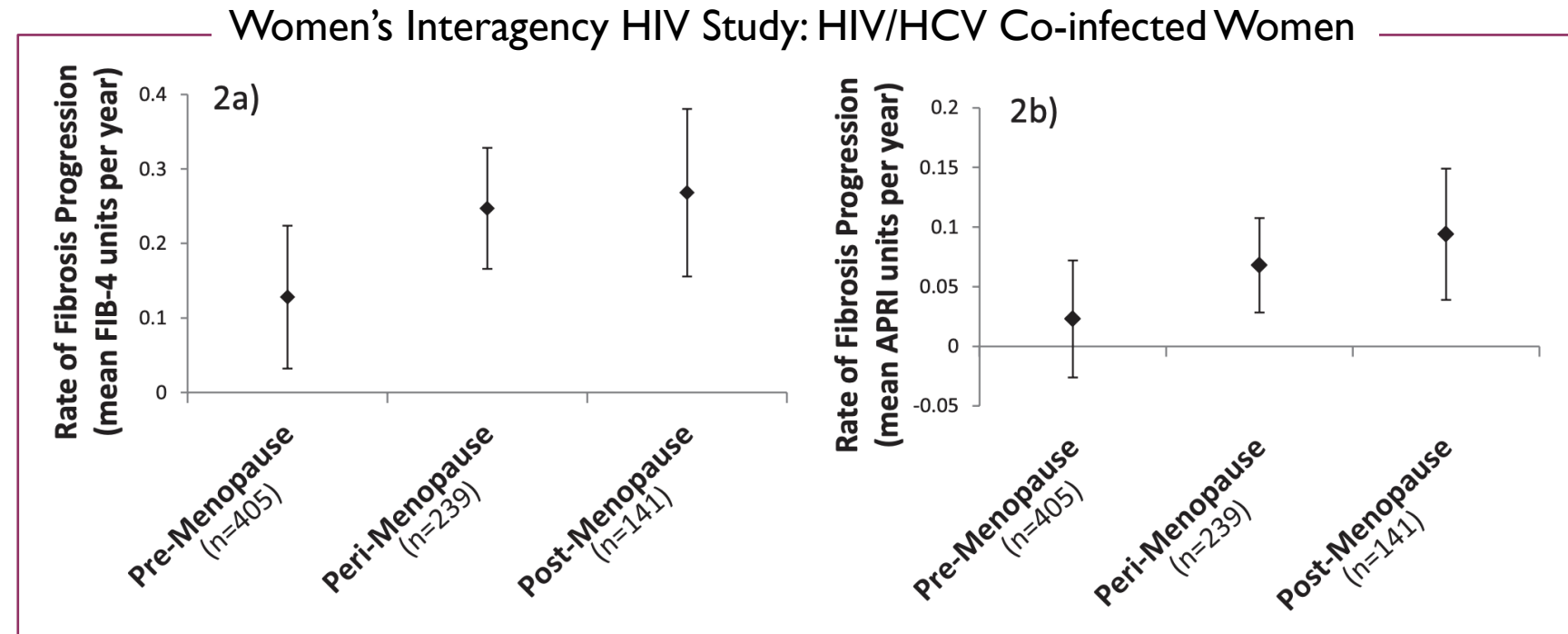
Association of AMH with NAFLD Histology in Women without PCOS (n=164)

Characteristic	Univariate Analysis		Multivariate Analysis *	
	OR (95% CI)	P value	AOR (95% CI)	P value
NASH (definite or borderline)	0.76 (0.57 – 1.03)	.075	0.53 (0.32 – 0.90)	.019
NAS≥5	0.62 (0.46 – 0.83)	.001	0.46 (0.29 – 0.75)	.002
Any steatosis (≥5%) **	-	-	-	-
Severe steatosis (>66%)	1.06 (0.79 – 1.42)	.701	0.92 (0.60 – 1.43)	.717
Severe lobular inflammation (>4 foci/200x)	0.76 (0.52 – 1.13)	.173	0.54 (0.28 – 1.04)	.066
Any portal inflammation	0.93 (0.63 – 1.37)	.719	1.06 (0.57 – 1.98)	.854
More than mild portal inflammation (>mild)	0.51 (0.34 – 0.80)	.003	0.40 (0.21 – 0.78)	.007
Any ballooning	0.68 (0.51 – 0.91)	.010	0.61 (0.39 – 0.96)	.031
Many/prominent ballooning	0.69 (0.51 – 0.93)	.016	0.51 (0.32 – 0.83)	.006
Mallory’s hyaline	0.70 (0.51 – 0.96)	.027	0.53 (0.32 – 0.88)	.013
Any fibrosis (>stage 1)	0.68 (0.50 – 0.92)	.012	0.54 (0.32 – 0.93)	.026
Advanced fibrosis (stage 3 or 4)	0.65 (0.42 – 0.99)	.046	0.56 (0.28 – 1.09)	.086
Higher fibrosis stage ***	0.62 (0.47 – 0.81)	.000	0.59 (0.40 – 0.88)	.009

PHYSIOLOGIC BENEFITS OF ESTROGEN ON LIVER HEALTH



- Estrogen inhibits stellate cell activation^{1,2,3}

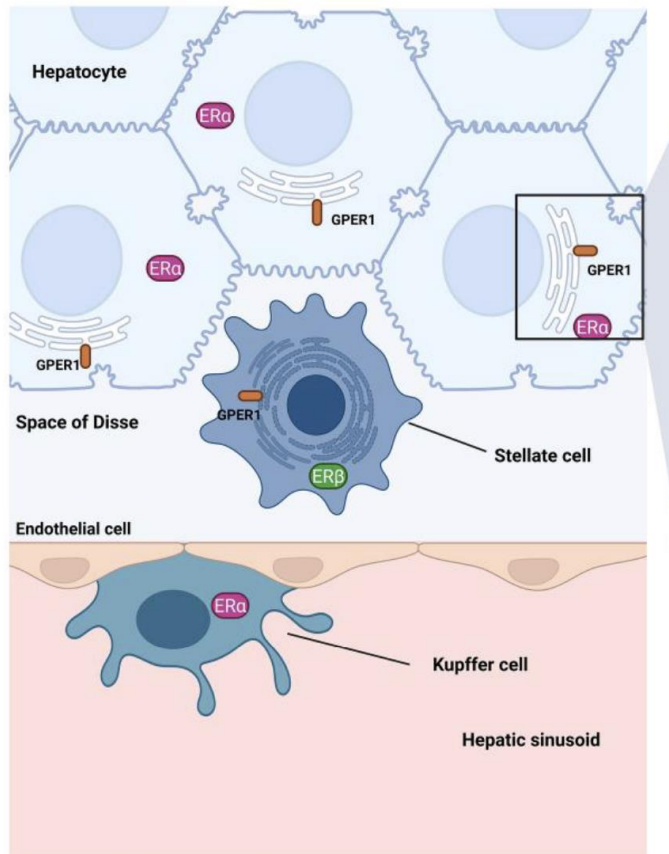


Sarkar et al, CID 2017

Dong J, et al Endocrine Reviews, 2025

1.Li et al, Cell Discovery 2025, 2.Yang et al, Nature Communications 2025, 3. Itagaki T, et al Gut 2005

ESTROGEN-MEDIATED EFFECTS WITHIN THE LIVER



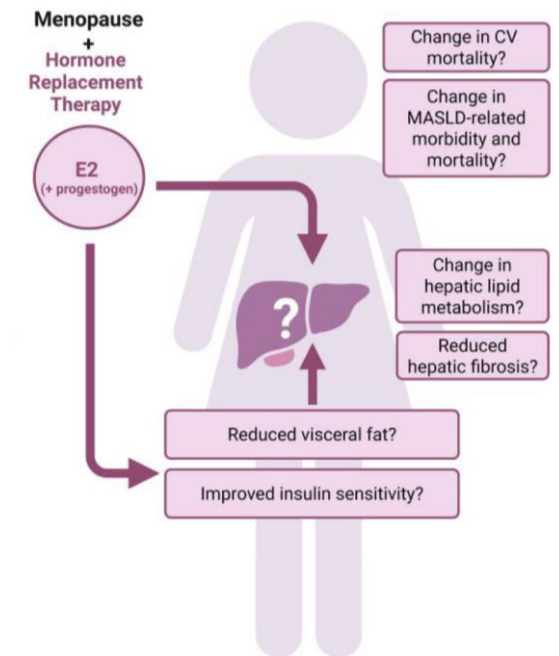
- Inhibits/decreases hepatic stellate cell activation
- Enhances hepatic lipid metabolism (fatty acid oxidation)
- Regulates insulin signaling, gluconeogenesis (enhanced insulin sensitivity within liver and peripherally)
- Protects against hepatic mitochondrial dysfunction

Dong J, et al *Endocrine Reviews*, 2025

Li et al, *Cell Discovery* 2025; Yang et al, *Nature Communications* 2025; Itagaki T et al, *Gut* 2005; Della Torre S et al, *Cell Rep* 2016; Besse-Patin A et al, *Gastroenterology* 2017; Galmes-Pascual BM et al, *J Endocrinol* 2017

MHT Benefits for MASLD?

Study	Design	Population	Intervention	Key Finding
McKenzie et al., Clin Endocrinol, 2006	Double-blind, placebo-controlled RCT	50 postmenopausal women with T2D	Oral 17 β -estradiol + norethisterone vs. placebo, 6 mo	Liver tests improved vs. placebo
Kim et al., Scientific Reports, 2023	Retrospective study	368 postmenopausal women	Transdermal vs oral MHT, 12 mo	Transdermal: Decrease in incident MASLD by imaging 24% $\rightarrow$ 17% Oral: Increase incident MASLD by imaging 25% $\rightarrow$ 29%
Henney et al., Liver International, 2026	Retrospective, propensity-matched cohort (TriNetX)	21,639 peri-menopausal women with pre-existing MASLD	MHT (estrogen $\pm$ progesterone) vs controls	MHT associated with lower 5 yr risk of MASLD-related outcomes, T2D, and MACE



Dong J et al, Endocrine Reviews 2025

- MHT not indicated to treat or reduce metabolic risk factors



But MHT Use is On the Rise...



Evolution of MHT Safety Messaging

2003

Black box warning following 2002 Women's Health Initiative Study (WHI)- citing breast cancer, stroke, VTE, MI, and dementia risk

JAMA

Original Contribution FREE

Risks and Benefits of Estrogen Plus Progestin in Healthy Postmenopausal Women
Principal Results From the Women's Health Initiative Randomized Controlled Trial

Writing Group for the Women's Health Initiative Investigators

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FDA convened expert panel to re-review data and messaging...

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F.D.A. Will Remove Black Box Warnings From Hormone Treatments for Menopause

The benefits of hormone replacement have been underappreciated, Dr. Marty Makary, the agency's commissioner, said on Monday. Critics described evidence for the change as insufficient.

▶ Listen · 5:11 min

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Estrogen pills used to treat menopause symptoms. Many hormone treatments have carried prominent warnings since 2003, when large trials found that oral combinations of estrogen and progestin raised the risks of blood clots and breast cancer. Photo Researchers, Inc./Science Source

 **By Roni Caryn Rabin**

Nov. 10, 2025

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The Food and Drug Administration will remove the so-called black box warning from all hormone replacement products containing estrogen, Dr. Marty Makary, the agency's commissioner, announced on Monday.

Nov 10, 2025

FDA removes black box warning for cardiovascular disease and breast cancer from all estrogen-containing MHT

FDA Removes MHT Black Box Warning for Estrogen-Containing Agents

2003

Black box warning following Women's Health Initiative Study (WHI)- citing breast cancer, stroke, VTE, MI, and dementia risk

Jul 2025

FDA convened expert panel to re-review data and messaging...

Nov 10, 2025

FDA removes black box warning for cardiovascular disease and breast cancer from all estrogen-containing MHT

2026

Labels rewritten with age-specific guidance
“Benefit greatest when MHT initiated within 10 years of menopausal onset, or before age 60”

FDA INDICATIONS FOR MHT

- Moderate-to-severe vasomotor symptoms (ie hot flashes, night sweats); occurs in 50-75% of women
- Moderate-to-severe vulvovaginal atrophy (ie genitourinary symptoms); occurs in > 50% of women
- Treatment of premature hypoestrogenism (ie premature ovarian insufficiency)
- Prevention of postmenopausal osteoporosis**



***U.S. Preventive Services Task Force and the American College of Obstetrics and Gynecology advises against MHT for this sole indication*

MHT AND CHRONIC LIVER DISEASE?

HEPATOLOGY



PRACTICE GUIDANCES | HEPATOLOGY, VOL. 73, NO. 1, 2021

Reproductive Health and Liver Disease: Practice Guidance by the American Association for the Study of Liver Diseases



Monika Sarkar,¹ Carla W. Brady ,² Jaquelyn Fleckenstein,³ Kimberly A. Forde,⁴ Vandana Khungar,⁴ Jean P. Mollenstien,⁵
Yalda Afshar,⁶ and Norah A. Terrault ⁷

- Avoid if: 1) Budd Chiari Syndrome 2) Decompensated cirrhosis, 3) Hepatocellular adenomas
- ...extrapolated from estrogen-containing hormonal contraception
- My practice: **risk-benefit discussion for everyone**; favor non-oral route (lower VTE/CV risks)
- White Paper on behalf of the AASLD Women's Health Initiative forthcoming ...

TAKEAWAYS: SEX HORMONES AND MASLD

TESTOSTERONE:

- Higher testosterone levels in young female patients promotes visceral adiposity, hepatic steatosis, and liver injury/fibrosis
- PMOS is common AND accelerates MASLD progression
 - Ask about PMOS symptoms (hirsutism and irregular menses) in all young female patients with liver disease

ESTROGEN:

- Consider premature ovarian insufficiency and the menopausal transition in MASLD risk assessment
- MASLD and related metabolic risk factors are not indications for MHT
- BUT...MHT use is rising in general population (your patients with liver disease will ask for it!)
- MHT is NOT contraindicated with most liver conditions; non-oral formulations favored

Merci beaucoup!



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