The Intersection of Obesity and Obesity-Related Conditions

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ZEALAND PHARMA MEDICAL AFFAIRS TRAINING PROGRAM

MODULE 3

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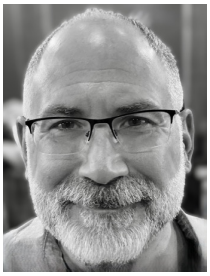
Your Speaker



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Disclosures:
Currax Pharmaceuticals, Speaker (obesity symposium)
Epitomee Medical, Speaker, outside consultant
The Obesity Society, Advisory board meetings with Kailera, Metsera, Zealand

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Learning Objectives

- Define obesity as a chronic, relapsing disease with dual pathogenic mechanisms (fat-mass and sick-fat/adiposopathy)
- Summarize most common obesity-related complications and comorbidities
- Map obesity's mechanisms to organ-system complications (CKM, MASLD, OSA/OHS, endocrine, cancer, diabetes)
- Describe how current evidence leads to individualized, mechanism-based interventions

CKM, cardiovascular-kidney-metabolic; MASLD, metabolic dysfunction-associated steatotic liver disease; OSA, obstructive sleep apnea; OHS, obesity hypoventilation syndrome.

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Obesity Is a Disease

- **Chief clinical sign:** Excess adiposity
- **Two mechanisms:**
  - Fat-mass disease (biomechanical)
  - Sick-fat disease (adiposopathy)

Significant overlap: Most patients have both (70% in CKM)

CKM, cardiovascular-kidney-metabolic.

Bays HE, et al. *Endocr Pract.* 2023;29(7):498-513. Mechanick JI, Hurley DL. *J Am Coll Cardiol.* 2019;75(5):525-538.

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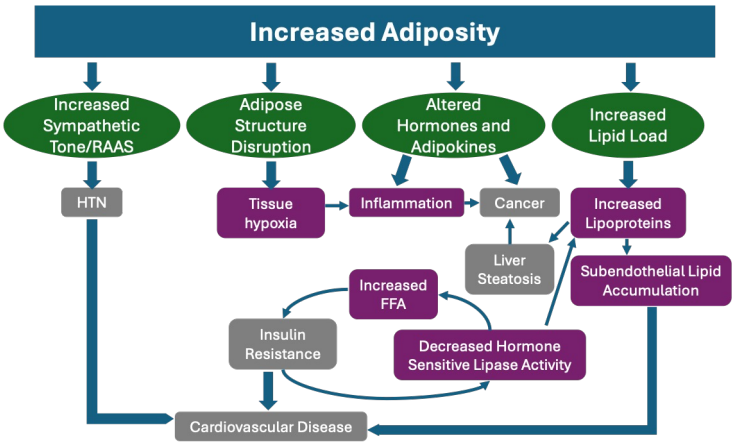
Pathology of Comorbidities: Physiological (Sick-Fat Disease)[1-3]

- Excessive secretion of proinflammatory adipokines leads to low-grade, systemic inflammation
- Chronic overactivity of the sympathetic nervous system may account for multiple pathophysiological processes
  - CVD
  - Insulin resistance
  - MASH/MASLD
  - Dyslipidemia
  - T2D



CVD, cardiovascular disease; FFA, free fatty acid; HTN, hypertension; MASH, metabolic-associated steatohepatitis, MASLD, metabolic dysfunction-associated steatotic liver disease; RAAS, renin-angiotensin-aldosterone system; T2D, type 2 diabetes.

1. Busebee B, et al. *Mayo Clin Proc.* 2023;98(12):1842-1857. 2. Kloock S, et al. *Pharmacol Ther.* 2023;251:108549. 3. Lingvay I, et al. *Lancet.* 2024;404(10456):972-987.

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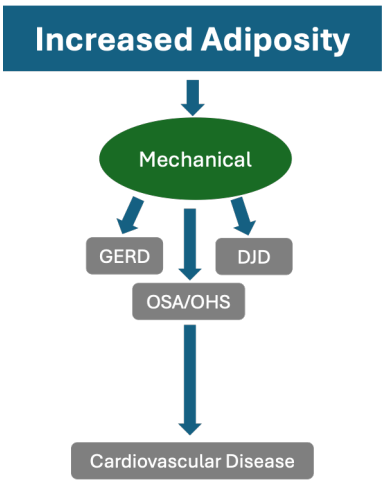
Pathology of Comorbidities: Biomechanical[1-3]



Mechanical stress of obesity and overweight also leads to pathophysiology

- Obstructive sleep apnea
- Osteoarthritis
- Hypoventilation
- Gastroesophageal reflux disease
- Internal organ compression
  - Kidneys
  - Lungs

DJD, degenerative joint disease; GERD, gastroesophageal reflux disease; OHS, obesity hypoventilation syndrome; OSA, obstructive sleep apnea.

1. Busebee B, et al. *Mayo Clin Proc.* 2023;98(12):1842-1857. 2. Kloock S, et al. *Pharmacol Ther.* 2023;251:108549. 3. Lingvay I, et al. *Lancet.* 2024;404(10456):972-987.

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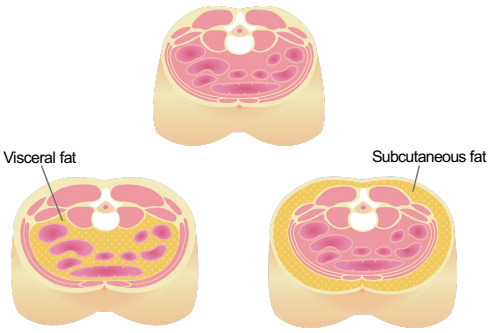
Biological Threads of Adiposopathy

- **Visceral Adipose Tissue (VAT):** More metabolically active:
  - More prone to lipolysis
  - More prone to hypertrophy

Visceral Fat and Subcutaneous Fat



Bays HE, et al. *Endocr Pract.* 2023;29(7):498-513. American Diabetes Association Professional Practice Committee. *Diabetes Care.* 2025;48(suppl 1):S1-S5. Sacks J, et al. *J Clin Endocrinol Metab.* 2025;110(3):456-472.

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Biological Threads of Adiposopathy

- **Visceral Adipose Tissue (VAT):** More metabolically active:
  - More prone to lipolysis
  - More prone to hypertrophy
- **Ectopic Adipose Tissue (EAT):** Local lipotoxicity
  - Ceramides → organ-specific inflammation/fibrosis

Result: Hormonal dysregulation
- ↓ adiponectin, ↑ leptin resistance; dysregulated cortisol/HPA axis
- Insulin resistance, dyslipidemia, endothelial dysfunction—diabetes hub

HPA, hypothalamus-pituitary-adrenal axis.

Bays HE, et al. *Endocr Pract.* 2023;29(7):498-513. American Diabetes Association Professional Practice Committee. *Diabetes Care.* 2025;48(suppl 1):S1-S5. Sacks J, et al. *J Clin Endocrinol Metab.* 2025;110(3):456-472.

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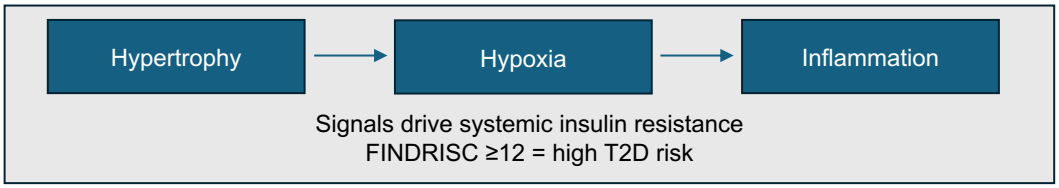
Mechanistic Flow: Adipocyte Hypertrophy → Organ Damage



FINDRISC, Finnish Diabetes Risk Score; T2D, type 2 diabetes.

Rubino F, et al. *Lancet Diabetes Endocrinol*. 2025;13(3):221-262. Wilding JPH, et al. *N Engl J Med*. 2021;384(11):989-1002. Nadolsky K, et al. *AACE Clinical Guidance*. 2025;31(11):1351-1394. Sacks J, et al. *J Clin Endocrinol Metab*. 2025;110(3):456-472.

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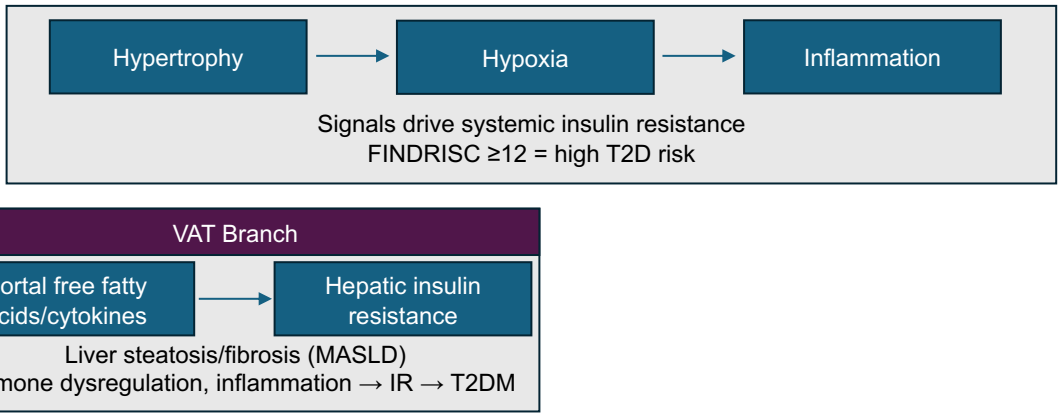
Mechanistic Flow: Adipocyte Hypertrophy → Organ Damage



FINDRISC, Finnish Diabetes Risk Score; IR, insulin resistance; MASLD, metabolic dysfunction-associated steatotic liver disease; T2D, type 2 diabetes; VAT, visceral adipose tissue.

Rubino F, et al. *Lancet Diabetes Endocrinol*. 2025;13(3):221-262. Wilding JPH, et al. *N Engl J Med*. 2021;384(11):989-1002. Nadolsky K, et al. *AACE Clinical Guidance*. 2025;31(11):1351-1394. Sacks J, et al. *J Clin Endocrinol Metab*. 2025;110(3):456-472.

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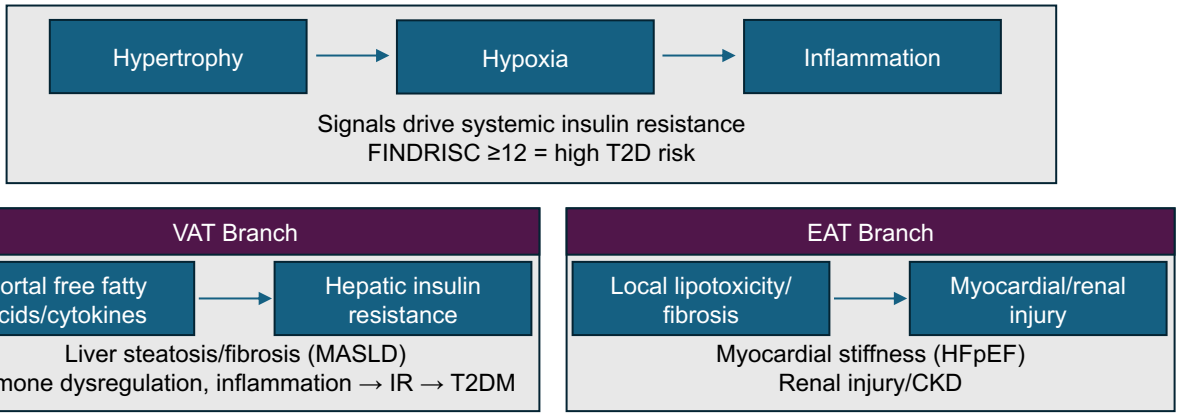

CKD, chronic kidney disease; EAT, epicardial adipose tissue; FINDRISC, Finnish Diabetes Risk Score; HFpEF, heart failure with preserved ejection fraction; IR, insulin resistance; MASLD, metabolic dysfunction-associated steatotic liver disease; T2D, type 2 diabetes; VAT, visceral adipose tissue.

Rubino F, et al. *Lancet Diabetes Endocrinol*. 2025;13(3):221-262. Wilding JPH, et al. *N Engl J Med*. 2021;384(11):989-1002. Nadolsky K, et al. *AACE Clinical Guidance*. 2025;31(11):1351-1394. Sacks J, et al. *J Clin Endocrinol Metab*. 2025;110(3):456-472.

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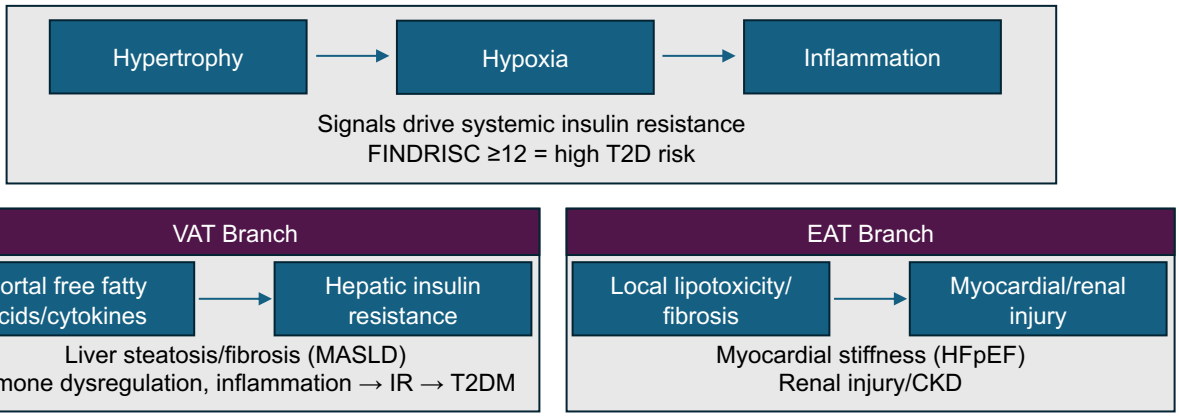
Mechanistic Flow: Adipocyte Hypertrophy → Organ Damage



Treating obesity modifies this cascade.

Rubino F, et al. *Lancet Diabetes Endocrinol*. 2025;13(3):221-262. Wilding JPH, et al. *N Engl J Med*. 2021;384(11):989-1002. Nadolsky K, et al. *AACE Clinical Guidance*. 2025;31(11):1351-1394. Sacks J, et al. *J Clin Endocrinol Metab*. 2025;110(3):456-472.

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From Adipose Dysfunction to Systemic Disease: Pathophysiologic Continuum

Pathophysiologic Stage	Primary Mechanism	Key Mediators	Clinical Expression
Adipose Expansion	Adipocyte hypertrophy and hypoxia	↑ TNF-α, IL-6, MCP-1	Local inflammation, macrophage infiltration
Adiposopathy (Sick Fat)	Altered adipokine secretion, lipolysis dysregulation	↓ Adiponectin, ↑ leptin resistance	Insulin resistance, dyslipidemia
Systemic Insulin Resistance	Ectopic lipid accumulation (liver, muscle)	Diacylglycerol, ceramides	Hyperinsulinemia, hepatic steatosis (MASLD)
β-Cell Stress and Failure	Glucolipotoxicity, oxidative stress	ER stress markers, impaired GLP-1 secretion/response	Type 2 diabetes onset
Systemic Sequelae	Chronic inflammation, endothelial dysfunction	IL-1β, CRP	CKM disease, HFpEF, microvascular injury

CKM, cardiovascular-kidney-metabolic; CRP, C-reactive protein; ER, endoplasmic reticulum; GLP-1, glucagon-like peptide-1; HFpEF, heart failure with preserved ejection fraction; IL-1β, interleukin-1 beta, also known as leukocytic pyrogen, leukocytic endogenous mediator, mononuclear cell factor, lymphocyte activating factor; IL-6, interleukin 6; MASLD, metabolic dysfunction-associated steatotic liver disease; MCP-1, monocyte chemoattractant protein-1; TNF-α, tumor necrosis factor alpha.

Adapted from Bays HE, et al. *Endocr Pract*. 2023;29(7):498-513. Mechanick JI, Hurley DL. *J Am Coll Cardiol*. 2019;75(5):525-538. American Diabetes Association Professional Practice Committee. *Diabetes Care*. 2025;48(suppl 1):S1-S5. Nadolsky K, et al. *AACE Clinical Guidance*. 2025;31(11):1351-1394.

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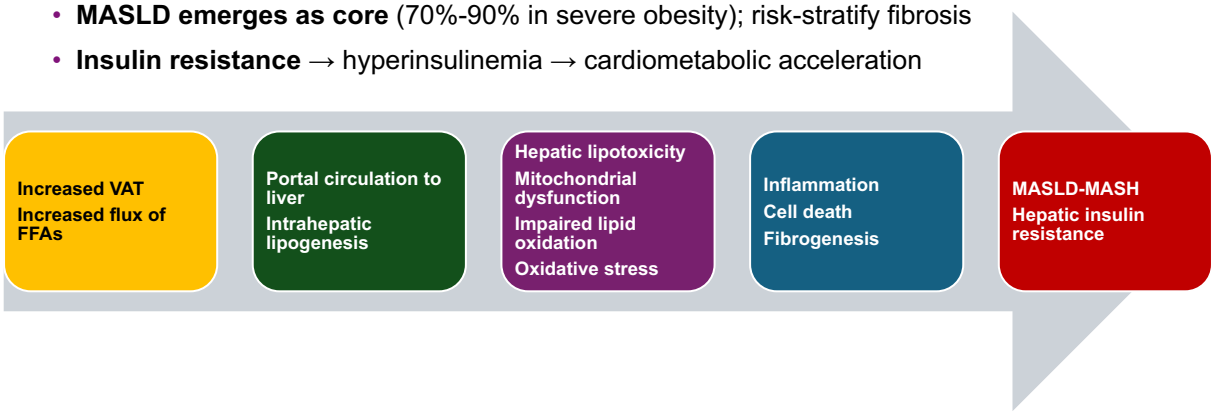
Metabolic-Inflammatory Pathway (Adiposopathy/Sick-Fat Prototype)

- **Visceral adiposity** secretes pro-inflammatory cytokines → hepatic insulin resistance
- **MASLD emerges as core** (70%-90% in severe obesity); risk-stratify fibrosis
- **Insulin resistance** → hyperinsulinemia → cardiometabolic acceleration



FFA, free fatty acid; MASH, metabolic dysfunction associated steatohepatitis; MASLD, metabolic dysfunction-associated steatotic liver disease, VAT, visceral adipose tissue.

Obesity Medicine Association. Obesity Algorithm. Accessed March 4, 2026. https://obesitymedicine.org/resources/obesity-algorithm/
European Association for the Study of the Liver, European Association for the Study of Diabetes, & European Association for the Study of Obesity. *J Hepatol*. 2025;82(4): 567-598.

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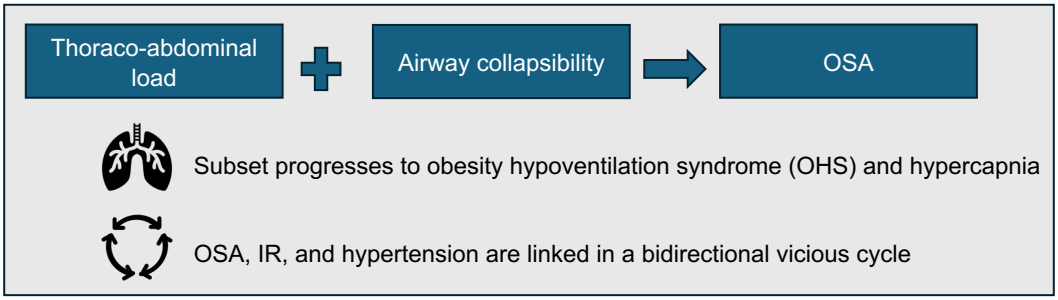
Mechanical Pathway (Biomechanical/Fat-Mass Prototype)



IR, insulin resistance; OSA, obstructive sleep apnea.

Masa JF, et al. *Lancet Resp Med*. 2019;7(8):731-742. Mokhlesi B, et al. *Am J Respir Crit Care Med*. 2019;200(3):e6-e24.

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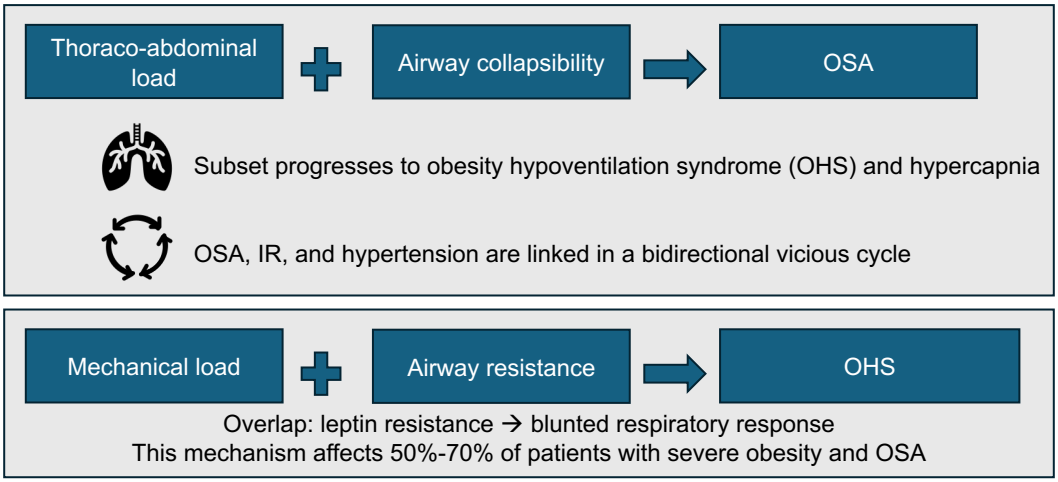
Mechanical Pathway (Biomechanical/Fat-Mass Prototype)



IR, insulin resistance; OSA, obstructive sleep apnea.

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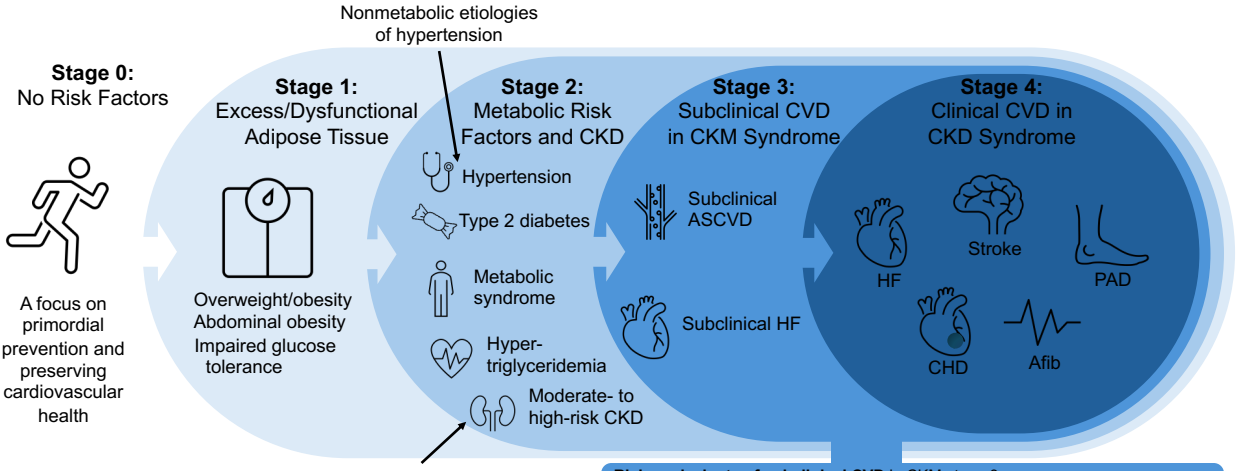
Stages of CKM Syndrome



Risk equivalents of subclinical CVD in CKM stage 3:
- Very high-risk CKD
- High predicted risk for CVD using risk calculator

Afib, atrial fibrillation; ASCVD, atherosclerotic cardiovascular disease; CHD, coronary heart disease; CKD, chronic kidney disease; CKM, cardiovascular-kidney-metabolic; CVD, cardiovascular disease; HF, heart failure; PAD, peripheral artery disease.

Ndumele CE, et al. *Circulation*. 2023;148(20):1606-1635.

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Endocrine & Reproductive Links

Women	Men
Hyperandrogenism/follicular dysfunction ↓ Polycystic ovarian syndrome (PCOS) *Insulin resistance (IR) is a major driver of PCOS—roughly 50%-70% of women with PCOS have IR*	Aromatization of androgens to estrogen ↓ Hypogonadism *Low testosterone increases visceral fat accumulation and sarcopenia creating a bidirectional loop*

Weight reduction restores balance/fertility

Marshall JC, Dunaif A. *Fertil Steril*. 2012;97(1):18-22. Amisi CA. *World J Diabetes*. 2022;13(3):129-149. Lundegaard Haase C, et al. *Hum Reprod*. 2023;38(3):471-481. Colleluori G, et al. *Front Endocrinol*. 2020:11;277.

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Endocrine & Reproductive Links

Sex/Life Stage	Endocrine/Hormonal Consequences	Reproductive Consequences
Women (Reproductive Age)	- Hyperinsulinemia, insulin resistance - Increased aromatase activity (↑ estrogen) - Decreased sex hormone–binding globulin (SHBG) - ↑ Androgens (PCOS) - Altered adipokines (↑ leptin, resistin, visfatin) - Chronic inflammation	- Menstrual irregularities - Anovulation, infertility - ↑ Risk of PCOS - Reduced assisted reproductive technology (ART) success - ↑ Miscarriage, gestational diabetes, preeclampsia
Men (Reproductive Age)	- Hypogonadotropic hypogonadism - ↓ Testosterone, ↑ estradiol (via aromatase) - Decreased SHBG - Insulin resistance - Altered adipokines	- Reduced sperm count/quality - Erectile dysfunction - Infertility - ↑ Risk of male obesity-associated secondary hypogonadism (MOSH)
Children/ Adolescents	- Insulin resistance, hyperinsulinemia - Thyroid dysfunction (possible hypothyroidism) - ↑ Leptin (leptin resistance) - ↑ Cortisol	- Precocious puberty (especially girls) - Early growth acceleration, possible short adult stature - Early hypothalamic-pituitary-gonadal (HPG) activation
Pregnancy	- Worsened insulin resistance - ↑ Estrogen, altered progesterone - ↑ Inflammatory cytokines	- ↑ Risk of gestational diabetes, preeclampsia - ↑ Miscarriage, congenital anomalies - Adverse perinatal outcomes

Malhotra R, et al. *Cureus*. 2025;17(7):e87283. Zheng L, et al. *Front Endocrinol*. 2024;14:1326546. Schon SB, et al. *Fertil Steril*. 2024:122(2):194-203. Katib A. *Cent European J Urol*. 2015;68(1):79-85. Service CA, et al. *Fertil Steril*. 2023;120(6):1098-1111.

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Neoplastic Consequences

- Hyperinsulinemia and inflammatory signaling promote tumorigenesis (≥13 cancers)
- Risk rises with BMI/duration (10%-25% increase per 5 kg/m²); obesity under-screened
- Sustained weight reduction lowers incidence/recurrence

Cancer type	Screening start age	Obesity impact
Colorectal	45 y	Increased risk, under-screening
Breast (postmenopausal)	45-50 y	Equipment adaptation
Endometrial	Symptom-driven	Abnormal bleeding vigilance
Pancreatic	Risk-based	MASLD link

BMI, body mass index; MASLD, metabolic dysfunction-associated steatotic liver disease.

Steele CB, et al. *MMWR Morb Mortal Wkly Rep*. 2017;66:1052-1058. Centers for Disease Control and Prevention. Obesity and Cancer. Updated June 11, 2025. Accessed March 4, 2025. https://www.cdc.gov/cancer/risk-factors/obesity.html

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Person-Centered and Complication-Centric Care

2025 AACE guidelines asked clinicians to:

1	Emphasize whole-person health and de-emphasize weight-centric approaches
2	Evaluate patients with empathy; include anthropometric and clinical components of diagnosis
3	Clinically confirm excess adiposity, waist circumference, waist to height ratio, and body composition when needed
4	Assess risk, presence, and severity of ORCD. Include internalized weight bias and stigma as part of clinical staging
5	Use shared decision-making to determine health goals while targeting weight reduction goals for health
6	Develop comprehensive treatment plan including lifestyle, pharmacotherapy, and surgical options with an individualized approach
7	Goals of therapy are to improve health and QoL by preventing or treating ORCD, not solely weight reduction
8	Therapy beyond lifestyle should be considered at all stages when needed to improve health
9	Implement a long-term follow-up plan and monitor for health goals, clinical outcomes, and treatment modifications

AACE, American Association of Clinical Endocrinologists; OCRD, obesity-related complications and disease; QoL, quality of life.

Nadolsky K, et al. *AACE Clinical Guidance*. 2025;31(11):1351-1394.

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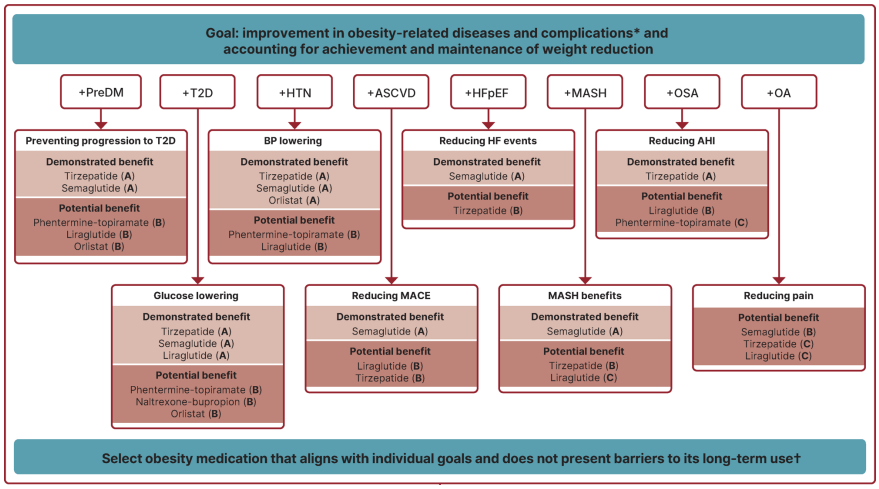
Individualized Therapy Mapping



ABCD, adiposity-based chronic disease; ASCVD, atherosclerotic cardiovascular disease; DM, diabetes mellitus; HFpEF, heart failure with preserved ejection fraction; HTN, hypertension; MASH, metabolic dysfunction-associated steatohepatitis; OA; osteoarthritis; T2D, type 2 diabetes.

American Diabetes Association Professional Practice Committee for Obesity. *Diabetes, Obesity, and Cardiometabolic CARE*. 2026;1(1):5-36.

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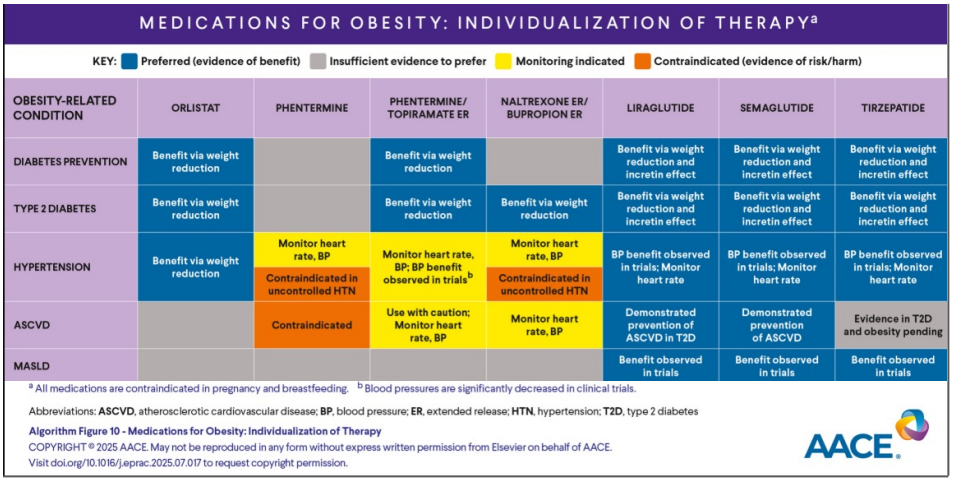
Individualized Therapy Mapping



MEDICATIONS FOR OBESITY: INDIVIDUALIZATION OF THERAPY[a]

KEY: Preferred (evidence of benefit) | Insufficient evidence to prefer | Monitoring indicated | Contraindicated (evidence of risk/harm)

OBESITY-RELATED CONDITION	ORLISTAT	PHENTERMINE	PHENTERMINE/ TOPIRAMATE ER	NALTREXONE ER/ BUPROPION ER	LIRAGLUTIDE	SEMAGLUTIDE	TIRZEPATIDE
DIABETES PREVENTION	Benefit via weight reduction		Benefit via weight reduction		Benefit via weight reduction and incretin effect	Benefit via weight reduction and incretin effect	Benefit via weight reduction and incretin effect
TYPE 2 DIABETES	Benefit via weight reduction		Benefit via weight reduction	Benefit via weight reduction	Benefit via weight reduction and incretin effect	Benefit via weight reduction and incretin effect	Benefit via weight reduction and incretin effect
HYPERTENSION	Benefit via weight reduction	Monitor heart rate, BP Contraindicated in uncontrolled HTN	Monitor heart rate, BP; BP benefit observed in trials[b]	Monitor heart rate, BP Contraindicated in uncontrolled HTN	BP benefit observed in trials; Monitor heart rate	BP benefit observed in trials; Monitor heart rate	BP benefit observed in trials; Monitor heart rate
ASCVD		Contraindicated	Use with caution; Monitor heart rate, BP	Monitor heart rate, BP	Demonstrated prevention of ASCVD in T2D	Demonstrated prevention of ASCVD	Evidence in T2D and obesity pending
MASLD					Benefit observed in trials	Benefit observed in trials	Benefit observed in trials

[a] All medications are contraindicated in pregnancy and breastfeeding. [b] Blood pressures are significantly decreased in clinical trials.

Abbreviations: **ASCVD**, atherosclerotic cardiovascular disease; **BP**, blood pressure; **ER**, extended release; **HTN**, hypertension; **T2D**, type 2 diabetes

Algorithm Figure 10 - Medications for Obesity: Individualization of Therapy

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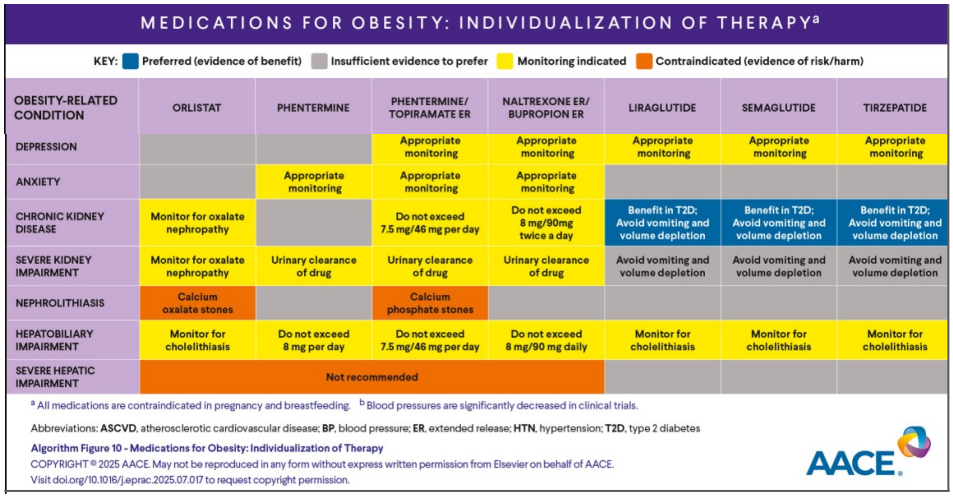
Individualized Therapy Mapping



MEDICATIONS FOR OBESITY: INDIVIDUALIZATION OF THERAPY[a]

KEY: Preferred (evidence of benefit) | Insufficient evidence to prefer | Monitoring indicated | Contraindicated (evidence of risk/harm)

OBESITY-RELATED CONDITION	ORLISTAT	PHENTERMINE	PHENTERMINE/ TOPIRAMATE ER	NALTREXONE ER/ BUPROPION ER	LIRAGLUTIDE	SEMAGLUTIDE	TIRZEPATIDE
DEPRESSION			Appropriate monitoring	Appropriate monitoring	Appropriate monitoring	Appropriate monitoring	Appropriate monitoring
ANXIETY		Appropriate monitoring	Appropriate monitoring	Appropriate monitoring			
CHRONIC KIDNEY DISEASE	Monitor for oxalate nephropathy		Do not exceed 7.5 mg/46 mg per day	Do not exceed 8 mg/90mg twice a day	Benefit in T2D; Avoid vomiting and volume depletion	Benefit in T2D; Avoid vomiting and volume depletion	Benefit in T2D; Avoid vomiting and volume depletion
SEVERE KIDNEY IMPAIRMENT	Monitor for oxalate nephropathy	Urinary clearance of drug	Urinary clearance of drug	Urinary clearance of drug	Avoid vomiting and volume depletion	Avoid vomiting and volume depletion	Avoid vomiting and volume depletion
NEPHROLITHIASIS	Calcium oxalate stones		Calcium phosphate stones				
HEPATOBILIARY IMPAIRMENT	Monitor for cholelithiasis	Do not exceed 8 mg per day	Do not exceed 7.5 mg/46 mg per day	Do not exceed 8 mg/90 mg daily	Monitor for cholelithiasis	Monitor for cholelithiasis	Monitor for cholelithiasis
SEVERE HEPATIC IMPAIRMENT	Not recommended						

[a] All medications are contraindicated in pregnancy and breastfeeding. [b] Blood pressures are significantly decreased in clinical trials.

Abbreviations: **ASCVD**, atherosclerotic cardiovascular disease; **BP**, blood pressure; **ER**, extended release; **HTN**, hypertension; **T2D**, type 2 diabetes

Algorithm Figure 10 - Medications for Obesity: Individualization of Therapy

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Individualized Therapy Mapping (Emerging)

Phenotype	Clinical Pattern	Mechanistic Insight	Therapeutic Focus	Evidence Level
Hungry Brain	Large meals, delayed satiation	Impaired CNS satiety/reward	Phentermine/ topiramate (portion control)	B (Pragmatic RCTs, n>500)
Hungry Gut	Early hunger post meals	Rapid emptying, ↓ incretin	GLP-1/GIP RA (prolong satiety)	A (Phase 3 trials)
Emotional Eating	Craving with stress	Hedonic drive (dopamine)	Naltrexone/ bupropion + CBT	B (CORRONA registry)
Slow Burn	Fatigue, low energy	↓ Expenditure/mitochondria	High-protein + resistance; use medication as adjunct	C (Observational)

Rationale: AACE 2025 endorses phenotypes; levels per guideline grading; monitor 3-6 mo; sequence by dominant driver

Not guideline-mandated as of AACE 2025.

AACE, American Association of Clinical Endocrinologists; CBT, cognitive behavioral therapy; CNS, central nervous system; GIP RA, glucose-dependent insulinotropic polypeptide receptor agonist; GLP-1, glucagon-like peptide-1; RCT, randomized controlled trial.

Adapted from Acosta A, et al. *Obesity*. 2021;29(5):855-869.

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Summary

Obesity comorbidities are physiological or mechanical and may be both. Many of the mechanisms involved create bidirectional vicious cycles.

Adipose dysfunction contributes to a constellation of diseases, many of which can be alleviated to some degree by weight reduction.

Comprehensive obesity care should prioritize the underlying mechanisms driving each patient's disease, allowing interventions to be tailored for maximal clinical impact.

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Further Reading

- AACE Clinical Guidance—2025 (Obesity Algorithms)
- ADA Standards of Care—2025 (Obesity-T2D Section 8)
- EASL-EASD-EASO MASLD—2025
- TOS/OMA/OAC Expert Guidance Statement—2026
- AHA CKM Statement—2023/2025 Updates
- OMA Obesity Algorithm—2025 (MASLD Screening)
- Clinical Management of Obesity 3rd Ed 2025 (TOS; Apovian, et al)
- STEP/SURMOUNT Trials

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Thank you!

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