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CLINICAL WEBINAR

Applied Weight Loss Nutrition in Clinical Practice: From Physiology to Protocol

Presented by:
Kira Sutherland, Naturopathic Sports Nutritionist

Presenter | Kira Sutherland

BHSc, Grad Dip Sports Nut, Adv Dip Nat, Adv Dip Nut, Adv Dip Herbal Med

Kira Sutherland is an award-winning Australian naturopath, herbalist, and sports nutritionist with over 30 years of clinical experience. She divides her time between clinical practice, undergraduate teaching, and mentoring health practitioners in holistic sports nutrition.

Known for her vibrant and straightforward teaching style, Kira is passionate about empowering her clients and students to perform at their best – whether as athletes or healthcare professionals. A lifelong endurance athlete, she also serves as the performance nutritionist for the TCS Sydney Marathon. Kira's journey reflects a deep commitment to wellness and a belief in inspiring others to follow their own path toward optimal health and performance.

Kira divides her time between clinical practice, lecturing at the undergraduate level, and mentoring complementary medicine practitioners in applying holistic sports nutrition. In her free time, she competes in endurance sports and skis with her family as much as possible.

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Host | Linda Dal Molin

Linda Dal Molin is the Director of Sales and Education for Designs for Health Australia.

Linda has a Masters in Human Nutrition, Bachelor of Health Science (Complementary Medicine), Advanced Diploma Naturopathy. She has been a practitioner for over 26 years and worked in the natural health space for 30 years having owned and managed health food stores and a large multi-modality allied health clinic. Linda practices on Saturday mornings in a busy clinic in Sydney's inner city with a focus on women's and gut health.

Linda has developed a strong relationship with the Designs for Health practitioner community. She will moderate the Q&A discussion with Tracee in this webinar and engage our live Designs for Health practitioner community to bring insight and practical clinical pearls for all.

What we will cover

1. **Weight Loss Resistance –** Physiological drivers beyond calorie balance, including gender-specific metabolic differences
2. **Body-Composition-First Protocols** – Metabolic phenotyping and protein-forward macronutrient strategies
3. **Evidence-Based Supplementation –** Clinical mechanisms for OEA, Magnesium, Protein, Collagen, and Creatine
4. **GLP-1 Integration –** Structured support and exit protocols to prevent rebound hyperphagia
5. **Overlooked Fat Loss Drivers –** Sleep, circadian rhythm, cortisol, appetite hormones, and NEAT
6. Summary and Questions

Main pathways of energy metabolism

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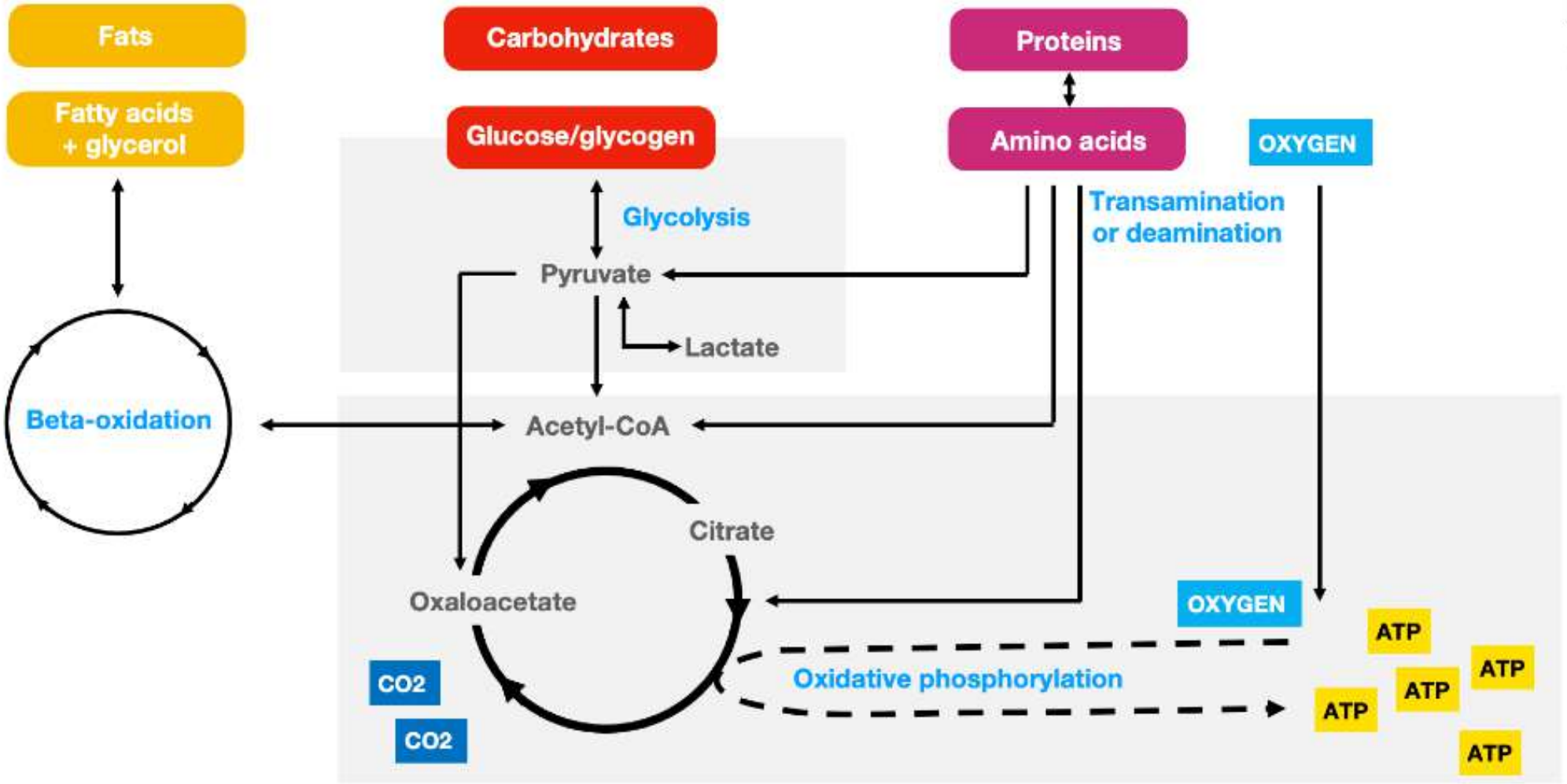

Adapted from Jeukendrup and Gleeson Sport Nutrition 2024

Fuel use during exercise at different intensities

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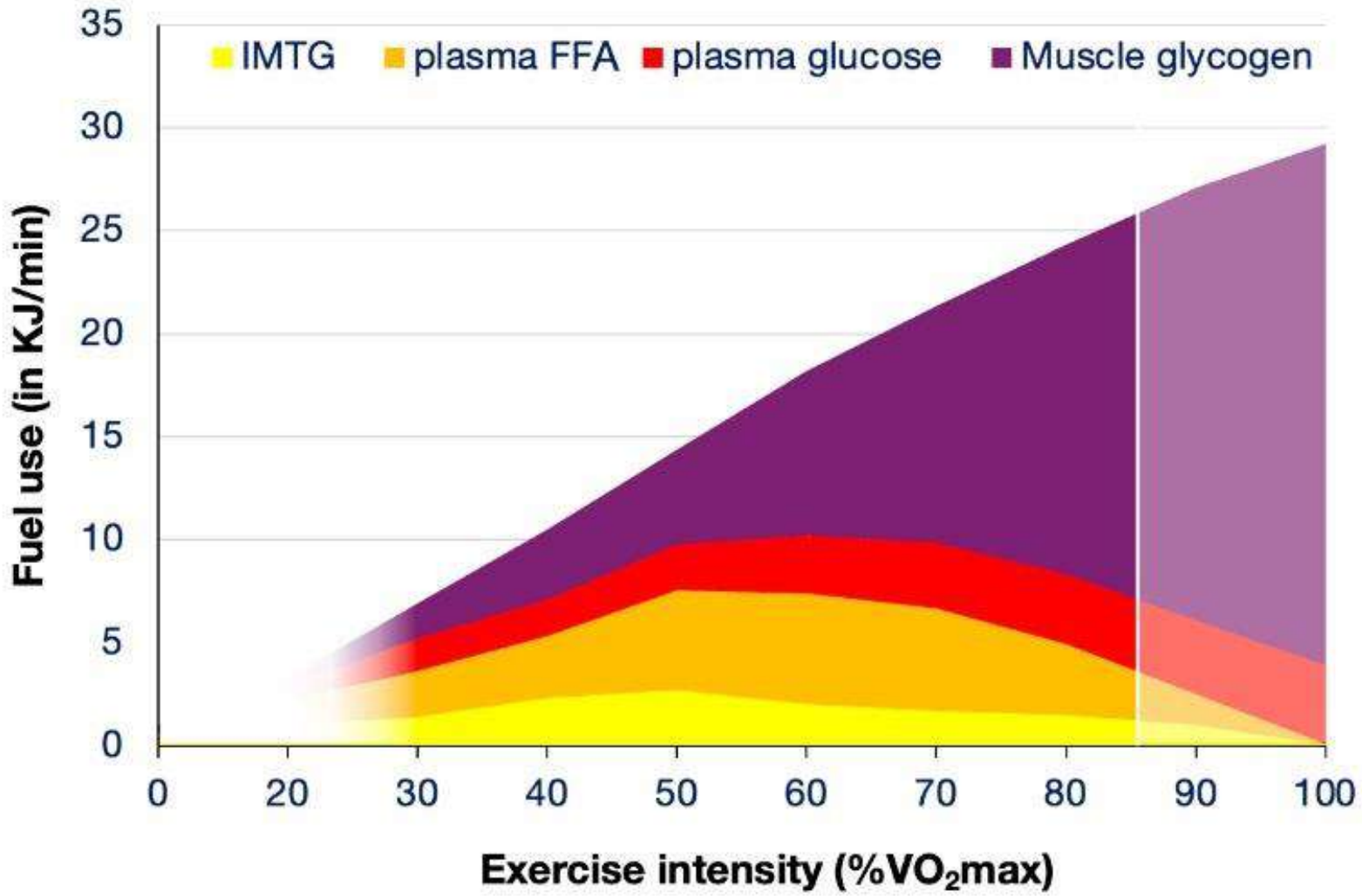


IMTG is intramuscular fat (fat stored in the muscle)

Plasma FFA are free fatty acids derived from adipose tissue

Plasma glucose is glucose coming from glycogen in the liver

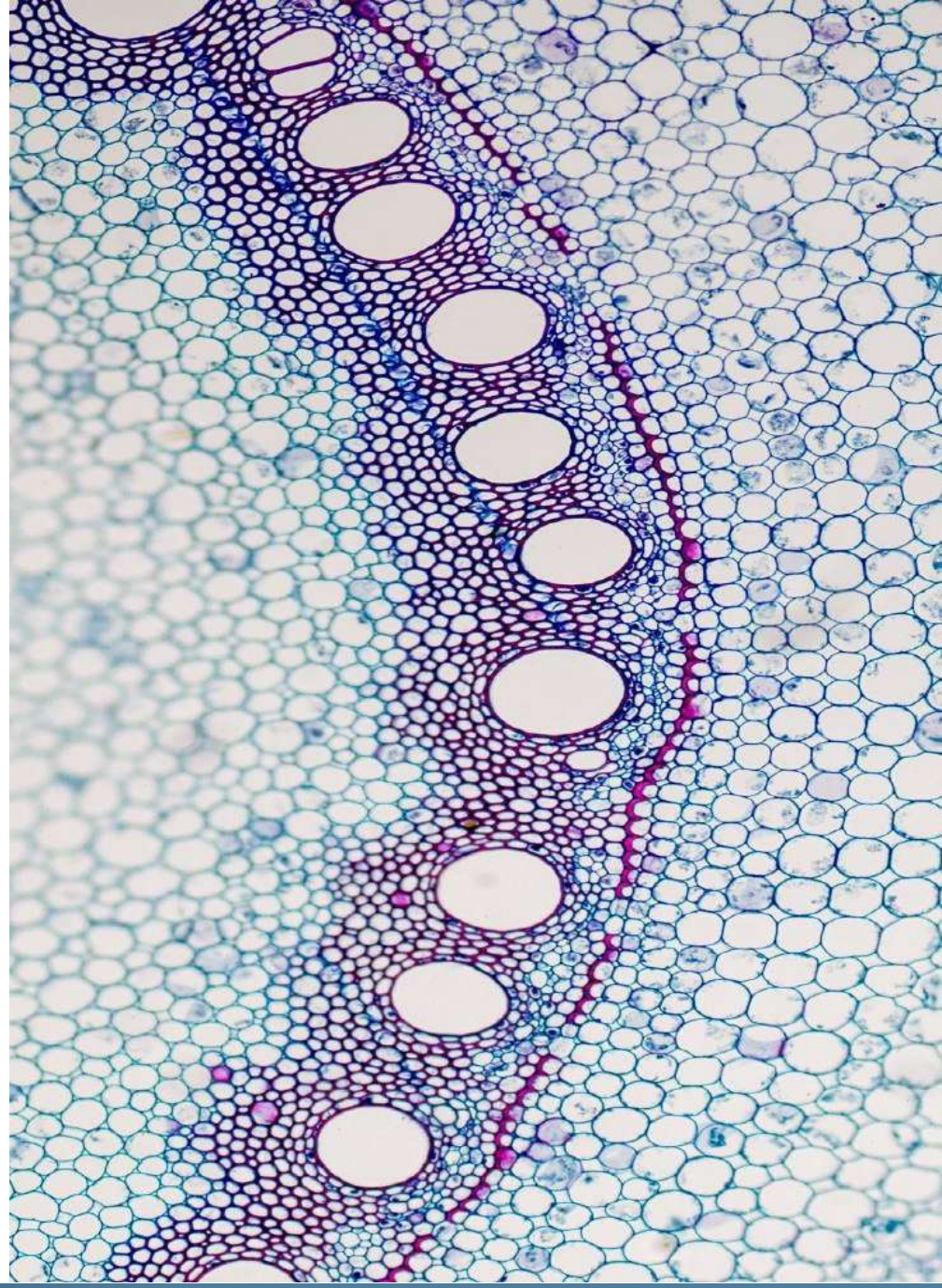
A mix of fuels is used at all intensities

Metabolism and reasons for weight gain

- Gut dysbiosis
- Chronic inflammation
- Thyroid and hormone issues
- Nervous system dysregulation
- Insulin resistance, fatty liver
- Medications (OCP, antidepressants etc.)
- Loss of muscle/sarcopenia
- Lack of exercise, injury, sedentary lifestyle
- Mitochondrial dysfunction/loss
- Genetic predisposition
- Excess of calories eaten vs expenditure

Mitochondria

- Mitochondrial biogenesis via exercise
- The only place we burn fat as a fuel
- Also burns carbohydrates and proteins (aerobic respiration)
- Fat adaptation (aerobic exercise)
- More muscle = more mitochondria (strength training)
- Weight loss and sarcopenia can = loss of mitochondria



Mitochondria

Supporting mitochondria

- Healthy insulin levels
- Oestrogen and progesterone
- Adequate melatonin production
- Healthy thyroid function
- Exercise/movement
- Intermittent fasting
- Circadian rhythm alignment
- Adequate sleep/rest

Damage to mitochondria

- Alcohol, smoking
- Pesticides, antibiotics
- Statins, paracetamol
- Lack of exercise, loss of muscle
- Vegetable oils (diet high in Omega 6)
- Overeating, high fructose corn syrup consumption

Sarcopenia and mitochondria

- Muscle means loss of mitochondrial capacity
- Maintain muscle mass = maintain mitochondrial numbers and function, slows the aging process
- Periodised aerobic training in a low-carb state (1-2 times per week) can activate mitochondrial biogenesis pathways (Bartlett et al., 2015)
- Strength training 2-3 times a week to maintain muscle
- Adequate protein intake to maintain muscle mass

Exercise benefits glucose and lipid metabolism, skeletal muscle function and growth, maintains bone density and assists insulin sensitivity in adipose tissue

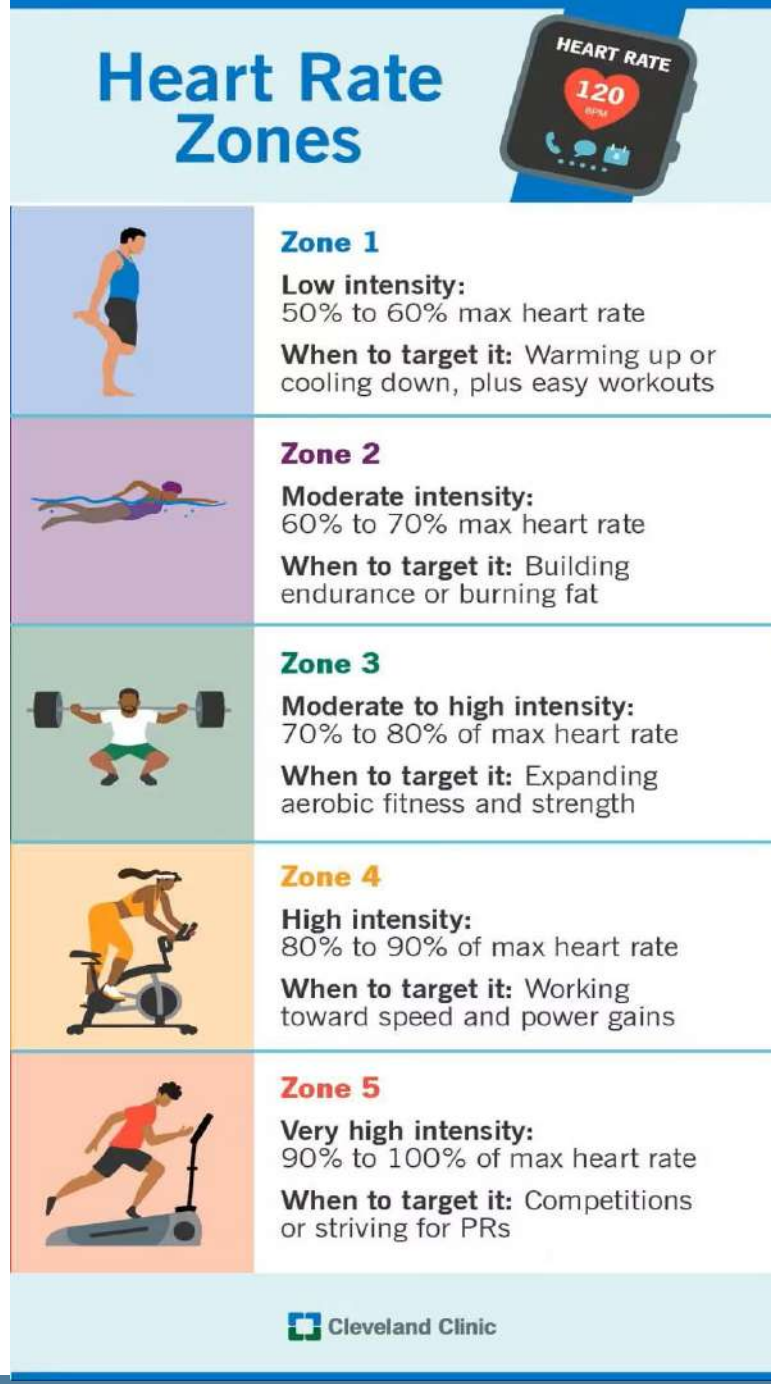
Exercise

- **Aerobic** for cardiovascular health and mitochondrial biogenesis, reduces insulin resistance and elevates mood.
- **Strength/lifting heavy** – maintain and build muscle, bone density, increase metabolism, reduce insulin resistance and improve body composition
- **HIIT/sprint training** – stimulates MPS, metabolic enhancement, increases cardiovascular health, decreases waist circumference and improves BMI
- **Stabilising/Plyometric** exercise (jump, skip, side to side) supports bone, muscle and connective tissue
- **Walking, yoga, Pilates, stretching and core work** increase flexibility, limit cortisol, improve posture and aid in injury prevention

Heart rate exercise zones

BEATS PER MINUTE	EXERCISE ZONES									
	AGE									
	20	25	30	35	40	45	50	55	65	70
100%	200	195	190	185	180	175	170	165	155	150
	VO_2 Max (Maximum effort)									
90%	180	176	171	167	162	158	153	149	140	135
	Anaerobic (Hardcore training)									
80%	160	156	152	148	144	140	136	132	124	126
	Aerobix (Cardio training / Endurance)									
70%	140	137	133	130	126	123	119	116	109	105
	Weight control (Fitness / Fat burn)									
60%	120	117	114	111	108	105	102	99	93	90
	Moderate activity (Maintenance / Warm up)									
50%	100	98	95	93	90	88	85	83	78	75

https://iantaylortrekking.com/blog/understanding-heart-rate-zones-for-your-trekking-training/



Macronutrients

Protein requirements

examine.com 2022

Daily protein requirements are expressed in grams, either per kilogram of body weight (g/kg) or per pound of body weight (g/lb). Ranges in the table below reflect known individual variances.

Optimal daily protein intake for adults (g/kg*)

	Of healthy weight			Overweight	Pregnant	Lactating
Sedentary	1.2–1.8			1.2–1.5	≥1.8	≥1.5
Active	1.4–2.0	1.6–2.4			unknown	
Goal	Maintenance	Muscle gain	Fat loss			

Maintenance: eucaloric diet | *Muscle gain:* eucaloric diet (if sedentary) or hypercaloric diet (if active) | *Fat loss:* hypocaloric diet | *

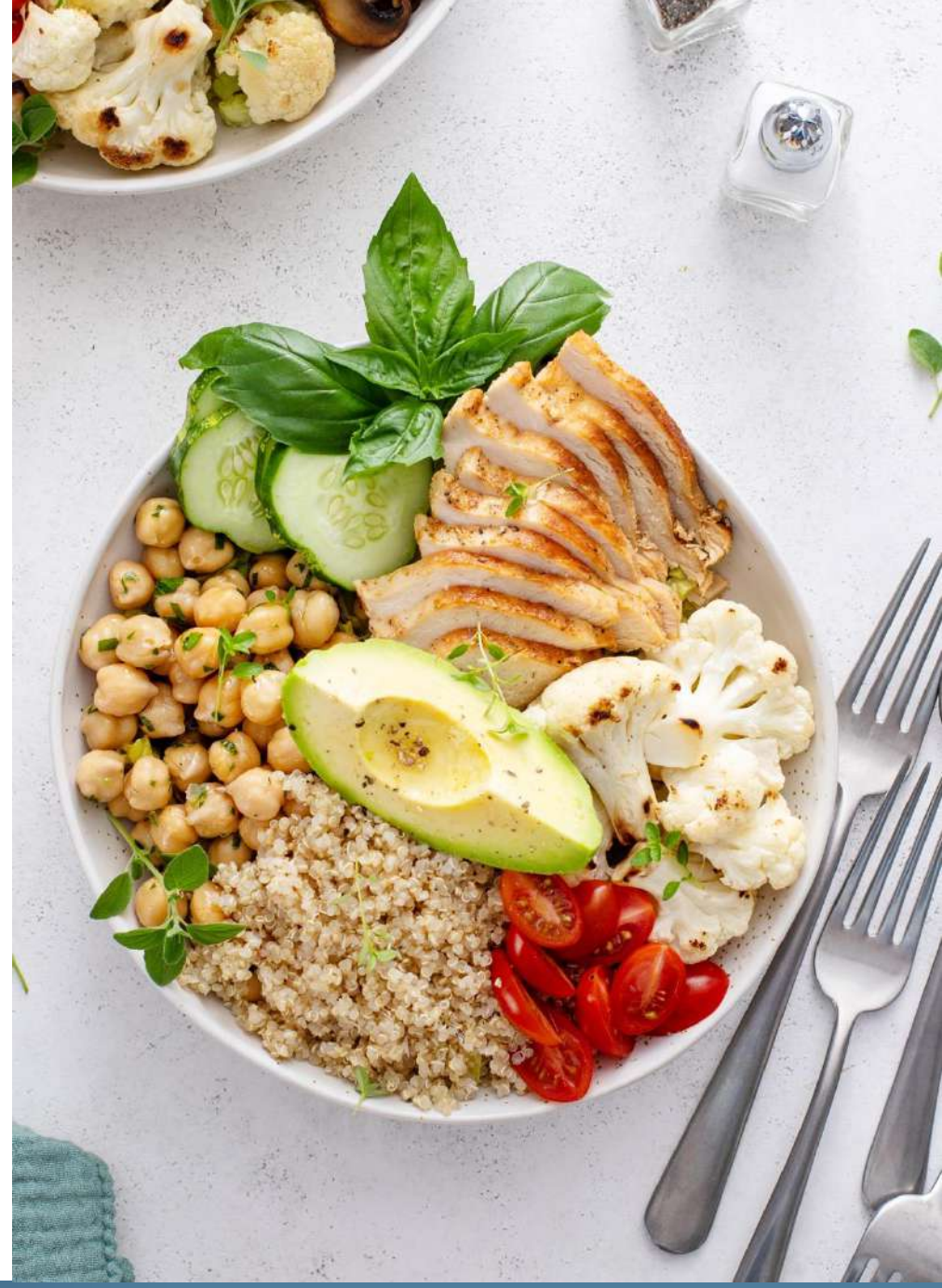
Grams per kilogram of body weight

The protein-forward paradigm

It preserves lean mass during energy restriction, increases satiety by releasing PYY and GLP-1, and raises the thermic effect of feeding.

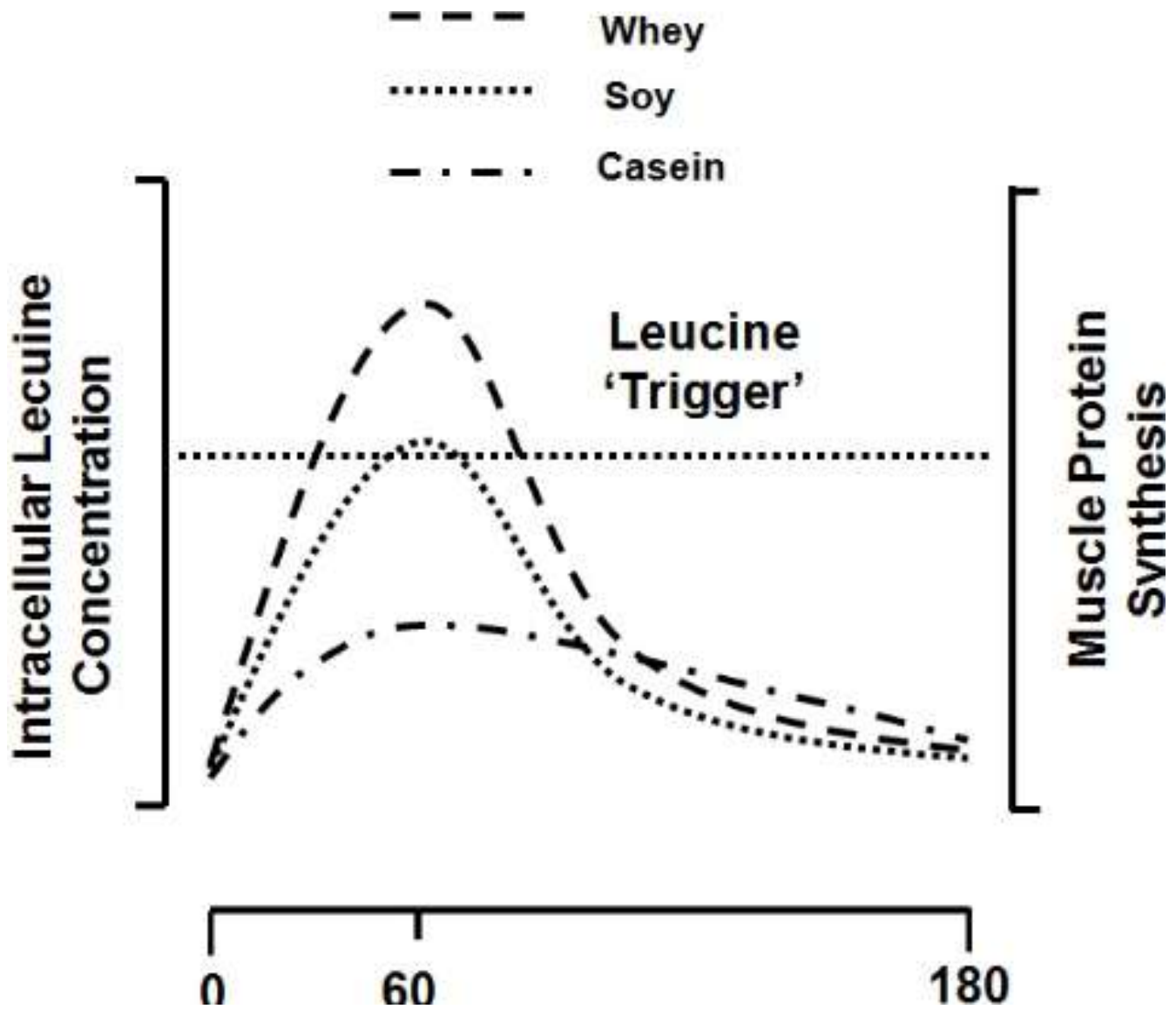
- **1.6–2.2g protein per kg/day**
- To preserve lean mass during a caloric deficit
- **30% Thermic Effect expended in the digestive process**
- Approximately 20-40 g per meal of pure protein
- 2.5-3gm leucine per meal

(Jäger et al, 2017)(Belski et al, 2019)
(Morton and Phillips, 2015)



Protein sources for hypertrophy

(Belski et al, 2019)
(Morton and Phillips, 2015)



Carbohydrates

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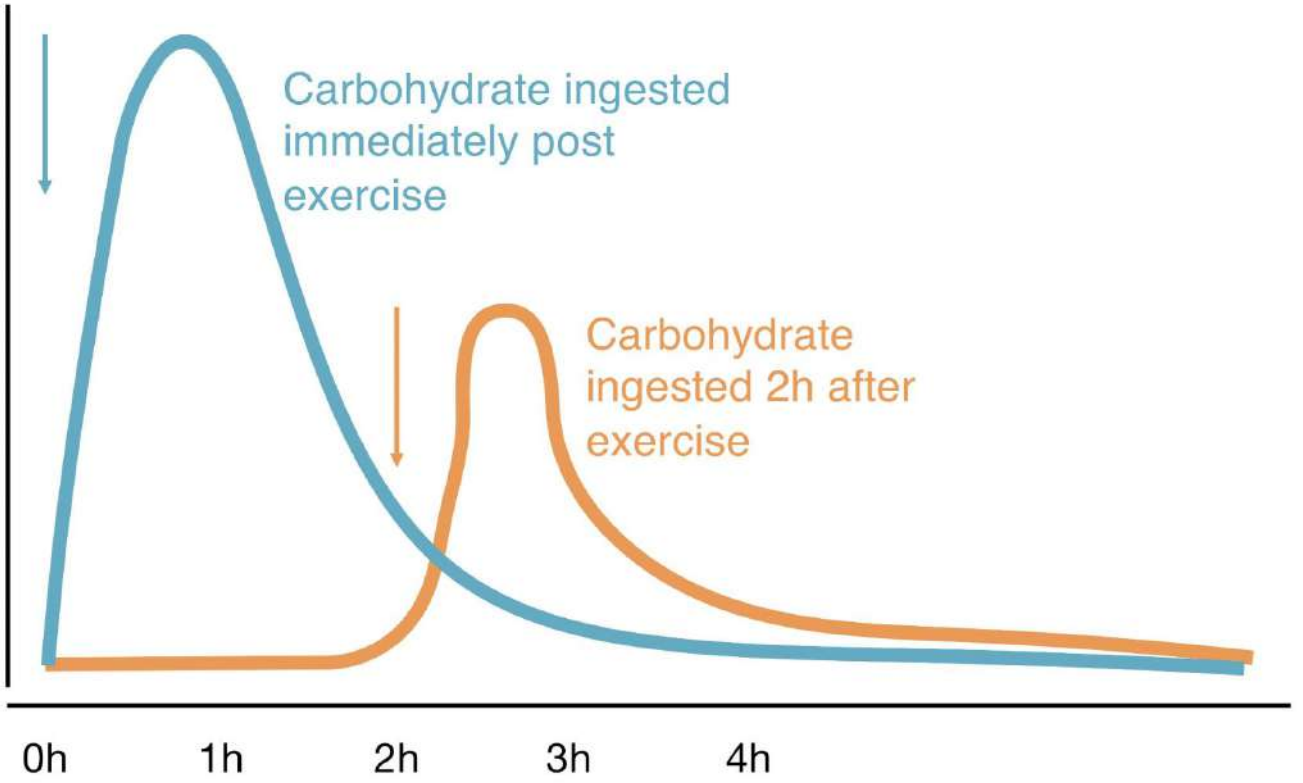
Depends on many factors

- Weight loss goals
- Type of diet selected
- Exercise volume
- Insulin sensitivity
- Phenotype and more
- During weight loss, common suggestions are **2-3 g/kg/day,** increasing with volume of exercise

Glycogen resynthesis can be higher in the first 2 hours after exercise but only with carbohydrate feeding



Post exercise fuelling window

- 1-2 hours, optimum is **30ish minutes**
- Glycogen replacement, glucose control
- Muscle recovery and synthesis (MPS)
- Catabolic to anabolic
- Decreases cortisol
- Supports microbiome
- Reduces risk of low energy availability (LEA)
- Supports the immune system
- Eat a meal, not a snack
- **Carb/Protein ratio 3-4:1 ratio as soon as possible (aerobic exercise)**
- **A 2:1 ratio may be more appropriate for strength sports**

The effect of exercise on glucose responses lasts many days

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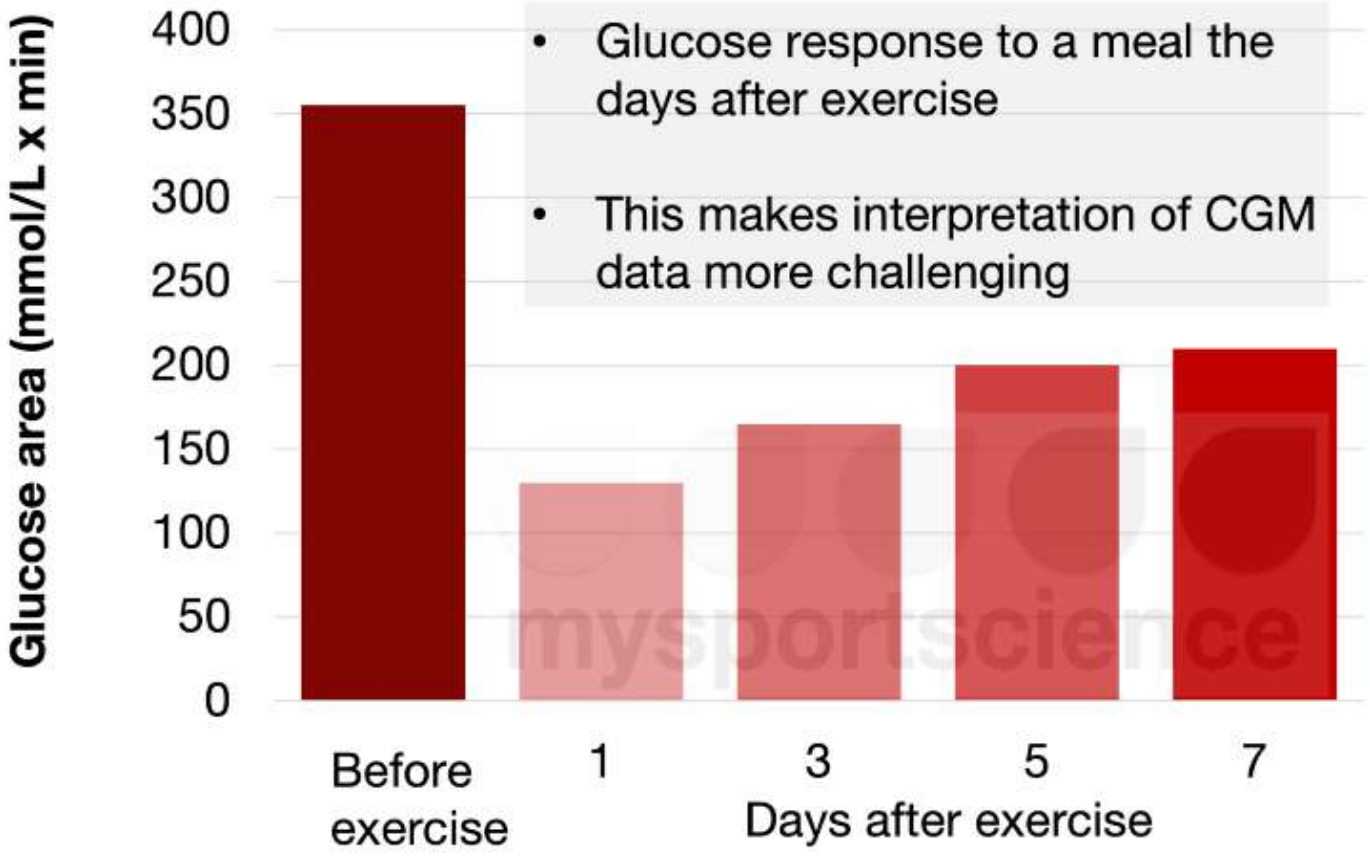


Macronutrient estimates

Carbohydrates

- Rest days/light exercise 2-2.5 grams/kg/day
- Exercise of 1-2 hours, 2.5-4 grams/kg/day
- Exercise of 2+ hours 3-5 grams/kg/day
- Focus on intake pre- and post-exercise

Protein

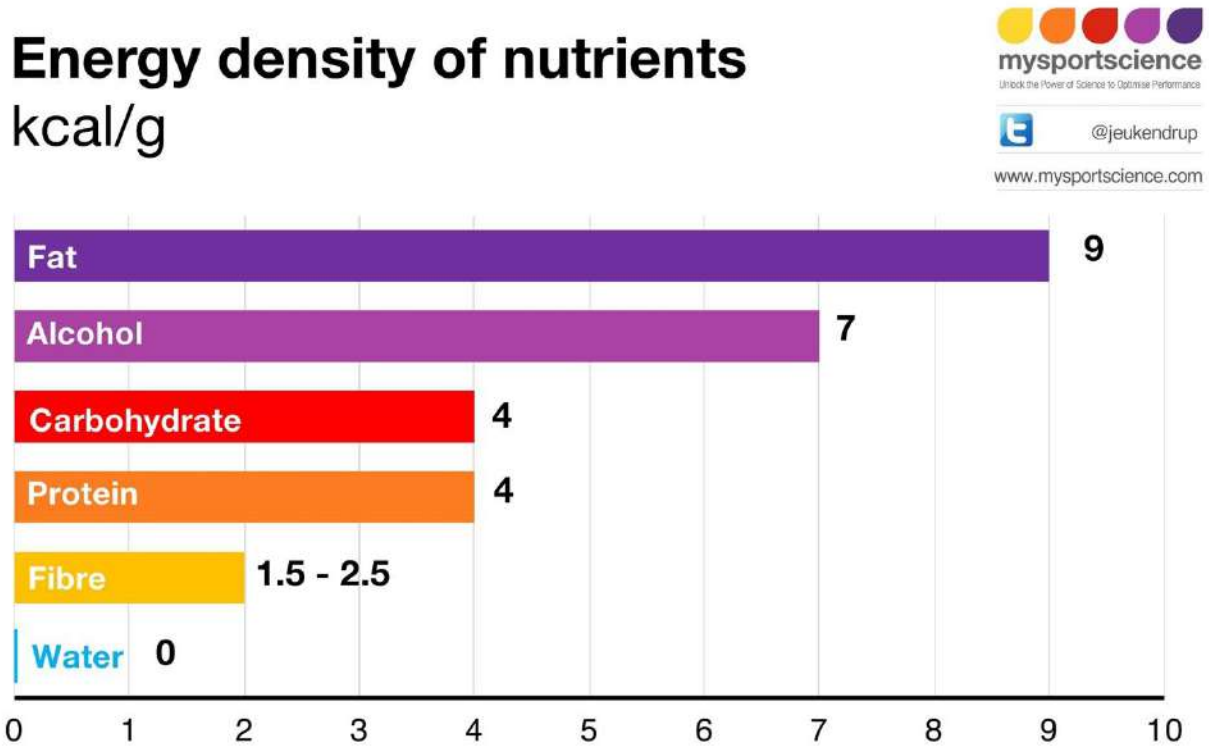
- 1.5-2.2 grams/kg/day
- Higher amounts on strength days or weight loss
- Lower on endurance or light exercise days
- 20-40grams within 30-45 minutes post-training
- With every snack

Fat

- 0.75-1.25 grams/kg/day
- It depends on exercise volume, body fat goals, and body type

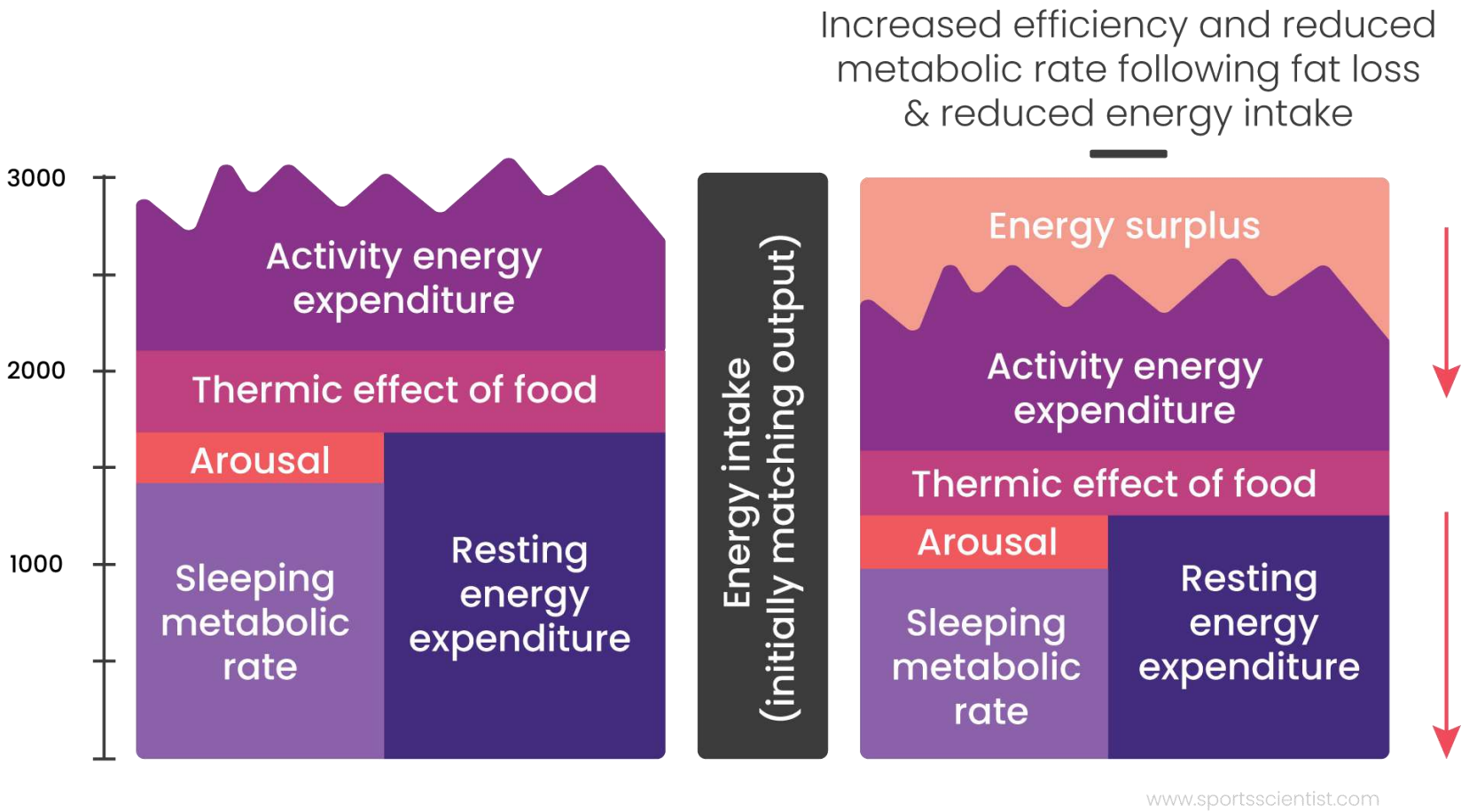
Issues when dieting

- Calories in, calories out theory
- Protein takes 30% of calories to digest (TEF)
- Carbohydrates take 6-10% to digest
- Fat takes 1-2% of calories to digest
- Labels can be out by 20%
- We underestimate our intake by up to 30%



Metabolism

- Thermic effect of food 10%
- Basal metabolic rate 70%
- Exercise and movement 20% (most variable)
- Set point theory



Adaptive Thermogenesis (AT)

- The metabolic alteration to minimise the degree of energy deficit created by continual energy restriction (CER)
- One study found a decrease in total daily energy expenditure (TDEE) by 6-18% due to AT
- Decrease thyroid hormones (T3 and T4)
- Change in appetite-regulating hormones (ghrelin/leptin)
- Potential increase in cortisol

Methods for estimating energy expenditure

Total daily energy expenditure (TDEE) is the energy used by an individual during a 24-hour period

TDEE can be partitioned into three main components:

- Basal Metabolic Rate (BMR)
- The Thermic Effect of Food (TEF)
- Physical Activity (PA) Associated Energy Expenditure (PAEE)

(Tanhoffer et al., 2012)

New calculations for RMR in adults

New predictive RMR equations were developed using age, body weight, height, and sex parameters (doi: 10.3390)

Kilograms:

- RMR males: (9.65 × weight in kg) + (573 × height in m) − (5.08 × age in years) + 260
- RMR females: (7.38 × weight in kg) + (607 × height in m) − (2.31 × age in years) + 43

Pounds:

- RMR males: (4.38 × weight in pounds) + (14.55 × height in inches) − (5.08 × age in years) + 260
- RMR females: (3.35 × weight in pounds) + (15.42 × height in inches) − (2.31 × age in years) + 43

Body weight planner

https://www.niddk.nih.gov/bwp

To lose 1 kilo = 7500 calories (approx.)

300-calorie deficit per day

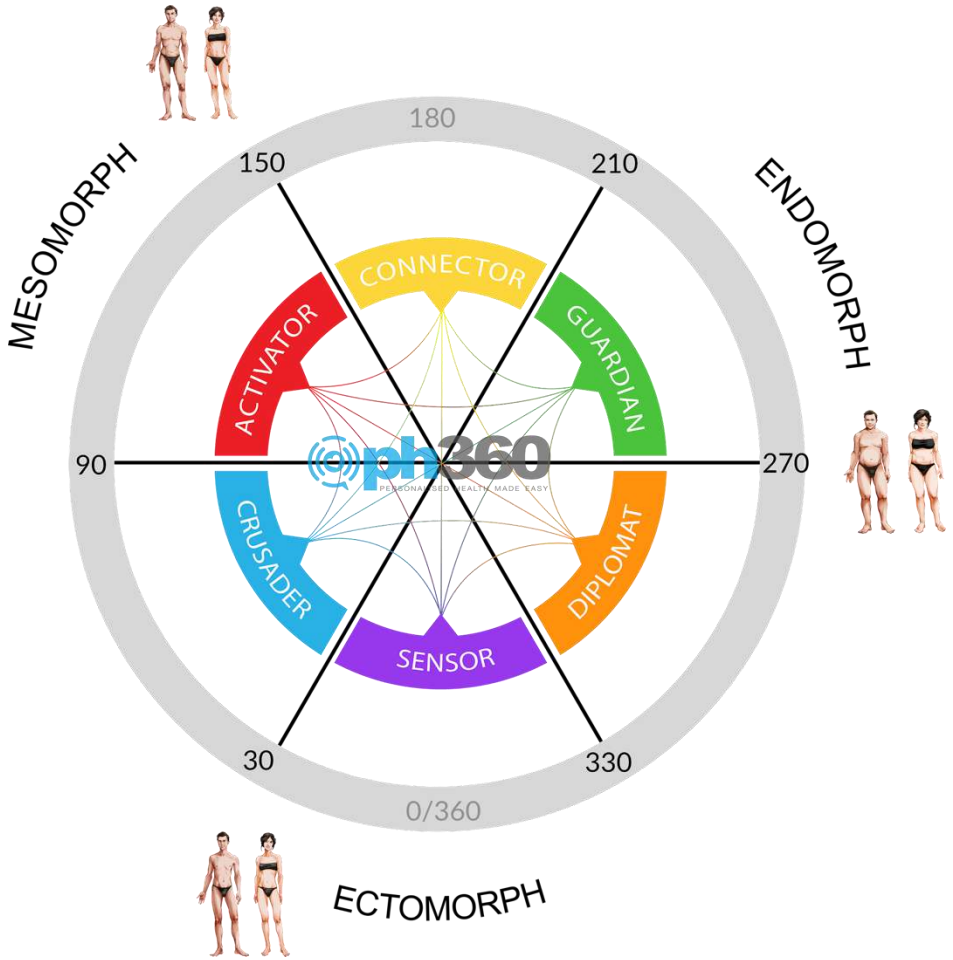
- 300 x 7 = 2100 per week
- = more than three weeks to lose a kilo

500-calorie deficit per day

- 500 x 7 = 3500
- = around two weeks to lose 1 kilo

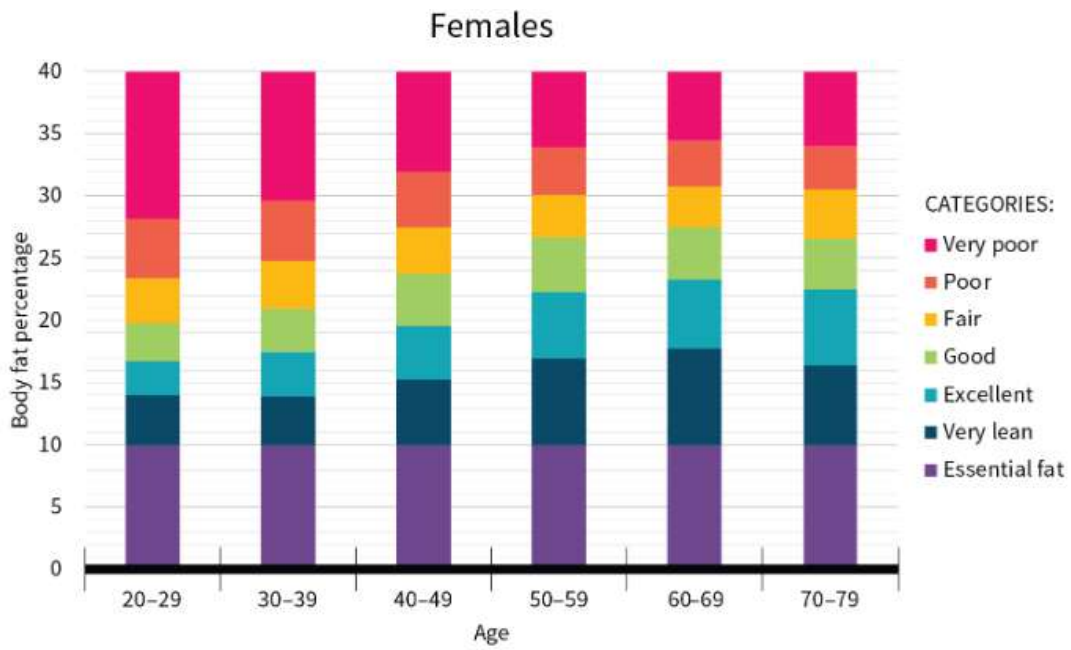
Parameters to measure

- Get a baseline of your measurements just like we do with hormones
- Weight (best in kilos)
- Body measurements (chest, waist, bum/hips)
- Lean body mass/muscle mass
- Body fat (19-32% considered healthy)
- DEXA, bioimpedance or bodpod
- Learn your body type/somatotype (ph360/precision health)



Male vs female body composition

Female body-composition categories based on age and percentage of body fat.



Adapted from American College of Sports Medicine. Body Composition (chapter 5 in *ACSM's Health-Related Physical Fitness Assessment Manual, 4th ed.* Lippincott Williams & Wilkins. 2013. ISBN:978-1451115680)

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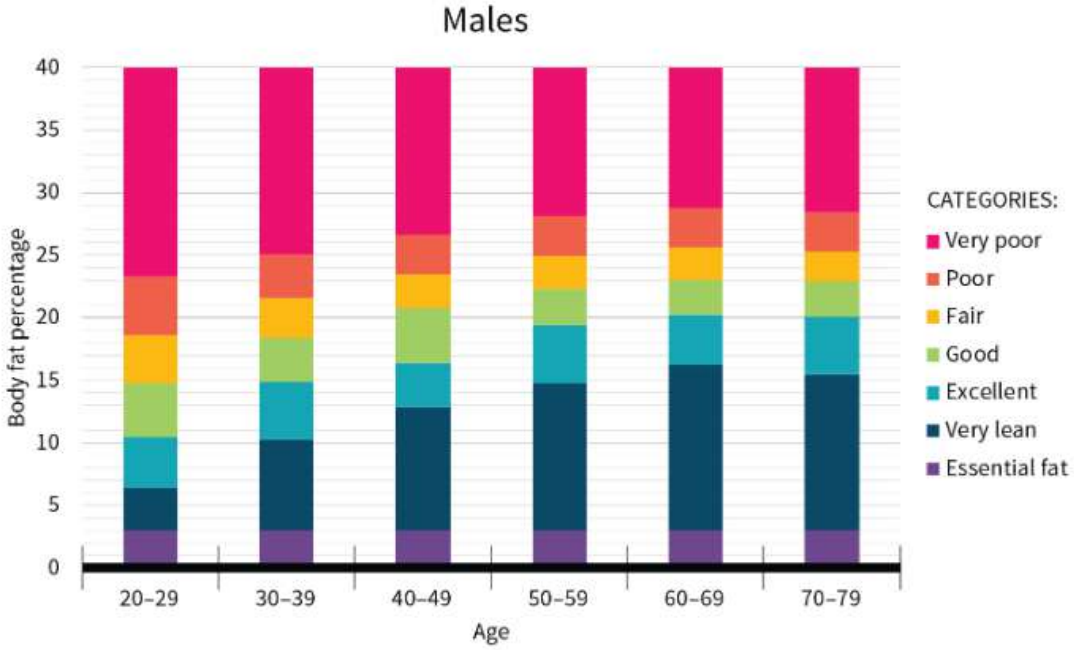


Adapted from American College of Sports Medicine. Body Composition (chapter 5 in *ACSM's Health-Related Physical Fitness Assessment Manual, 4th ed.* Lippincott Williams & Wilkins. 2013. ISBN:978-1451115680)

Why patients plateau: The core drivers

Weight loss resistance is rarely a single-factor problem. In most complex cases, multiple overlapping physiological mechanisms conspire to defend adipose tissue and suppress energy expenditure. Identifying which drivers are dominant in each patient is the first step toward a targeted protocol.

Insulin Resistance

Elevated insulin locks fat in adipocytes and impairs metabolic flexibility

Chronic Inflammation

Pro-inflammatory cytokines disrupt leptin signalling and promote fat storage

Cortisol Dysregulation

HPA axis activation drives visceral adiposity and increases appetite

Gut Dysfunction

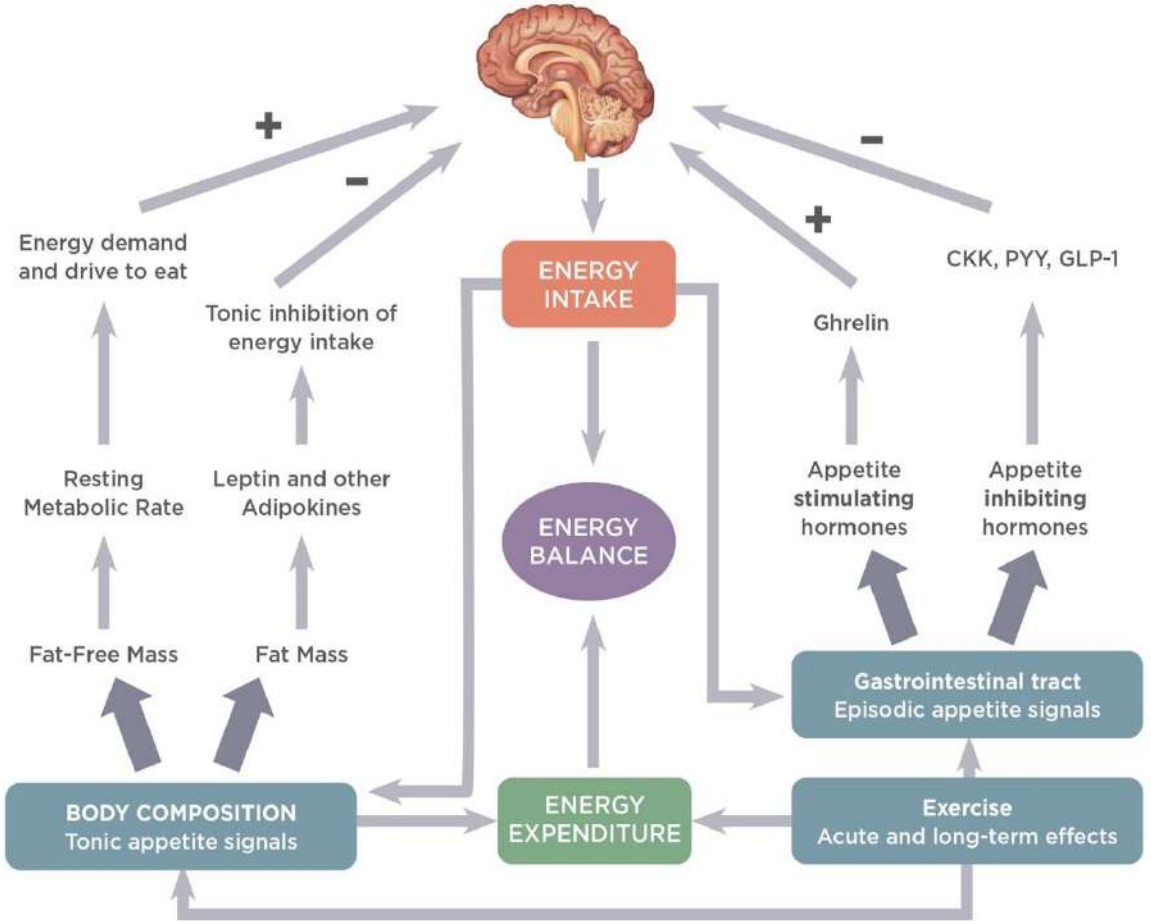
Dysbiosis and intestinal permeability impair GLP-1 secretion and energy harvest

Disrupted Appetite Signalling

Leptin resistance and ghrelin dysregulation override caloric deficit attempts

How does insulin resistance cause higher hunger?

- Shifted fuel partitioning
- Disrupted satiety signalling via the hypothalamus
- Leptin resistance
- Reduced GLP-1 signalling
- Tendency to reactive hypoglycaemia
- Altered dopamine signalling, especially with ADHD



As a major influence on RMR, FFM reflects the body's energy requirements and is a driver of eating. FM, reflecting the store of energy, can modulate this drive, but the effect is weakened as FM increases.

Episodic influences on food intake arise, in part, from the action of food in the stomach and the periodic release of peptides from the gut after eating.

Metabolic flexibility: The underdiagnosed deficit

Metabolic flexibility — the capacity to switch efficiently between glucose and fatty acid oxidation — is a critical determinant of fat loss success.[3] Insulin-resistant, inflexible metabolisers are trapped in a glucose-dependent state,[1] making fat mobilisation physiologically difficult regardless of caloric intent. Mitochondrial adaptation and metabolic inflexibility in obesity further impair this switching capacity.[2]

Signs of poor metabolic flexibility

- Energy crashes between meals or on waking
- Strong carbohydrate cravings, especially in the afternoon
- Inability to fast comfortably beyond 12 hours
- Fatigue with moderate-intensity exercise

Restoring flexibility: Clinical levers

- Protein-forward, lower-glycaemic dietary architecture
- Time-restricted eating to improve mitochondrial efficiency
- Resistance training to upregulate GLUT4 translocation
- Aerobic training to enhance mitochondrial lipid metabolism
- Magnesium and B-vitamin repletion to support oxidative metabolism

References

[1] Hansen M, et al. Are Individuals With Type 2 Diabetes Metabolically Inflexible? Endocrinol Diabetes Metab. 2025;8(3):e70044.
[2] Karampela I, et al. The Role of Mitochondrial Adaptation and Metabolic Flexibility in the Pathophysiology of Obesity and Insulin Resistance. Curr Obes Rep. 2021;10(2):191–207.
[3] Farías J, et al. Association Between Adipose Tissue Characteristics and Metabolic Flexibility in Humans: A Systematic Review. Front Nutr. 2021;8:744187.

Gender-specific differences in metabolic phenotype

Female metabolic profile

- Higher proportion of subcutaneous adipose tissue; more hormonally active
- Greater sensitivity to oestrogen-driven fat redistribution, particularly peri-menopause
- Lower baseline muscle mass reduces resting metabolic rate
- Thyroid dysfunction and PCOS/PMOS are more prevalent; both impair fat mobilisation
- Increased susceptibility to cortisol-driven cravings and emotional eating patterns

Male metabolic profile

- Higher visceral adiposity burden; greater cardiometabolic risk at equivalent BMI
- Testosterone decline accelerates after 40, reducing lean mass and metabolic rate
- Greater insulin resistance associated with central adiposity patterns
- Stronger anabolic response to resistance training and protein
- Sleep apnoea is more prevalent — a major overlooked driver of cortisol and ghrelin

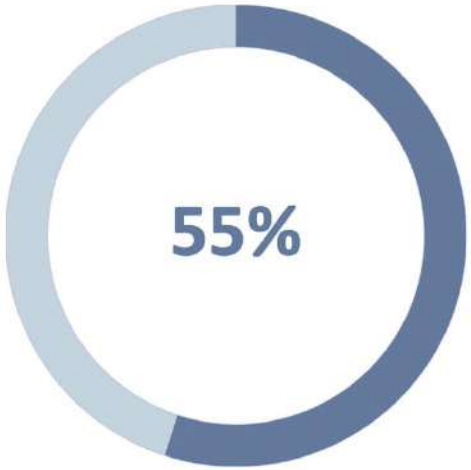
Overlooked fat loss drivers

The clinical levers most commonly missing from weight management protocols

- Sleep and circadian rhythm misalignment
- Cortisol and chrono nutrition
- Appetite hormones
- NEAT – non-exercise activity thermogenesis

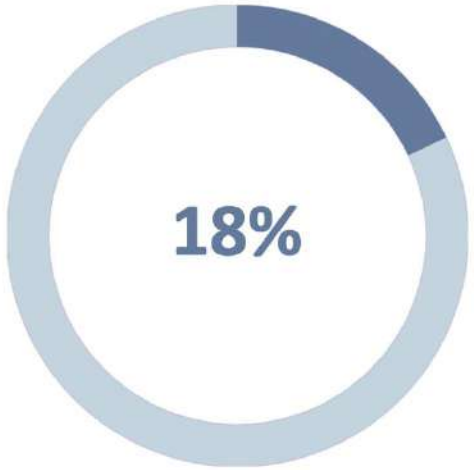
Sleep: The non-negotiable metabolic lever

Sleep duration and quality have a profound and bidirectional relationship with body composition, appetite regulation, and metabolic rate. The evidence is unequivocal: insufficient sleep is a significant independent risk factor for weight gain[1], insulin resistance, and treatment resistance.



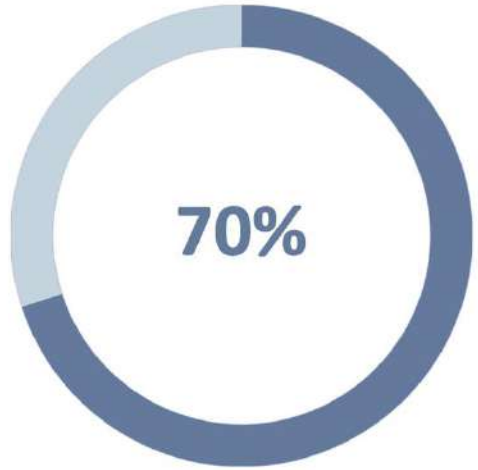
Ghrelin Increase

Rise in appetite-stimulating ghrelin after just two nights of restricted sleep (≤5 hours)[2]



Leptin Reduction

Decrease in satiety hormone leptin observed with chronic sleep restriction — creating a double appetite burden[2]



Lean Mass Loss

Proportion of weight lost as lean mass in caloric restriction combined with sleep deprivation vs. ~40% with adequate sleep[1]

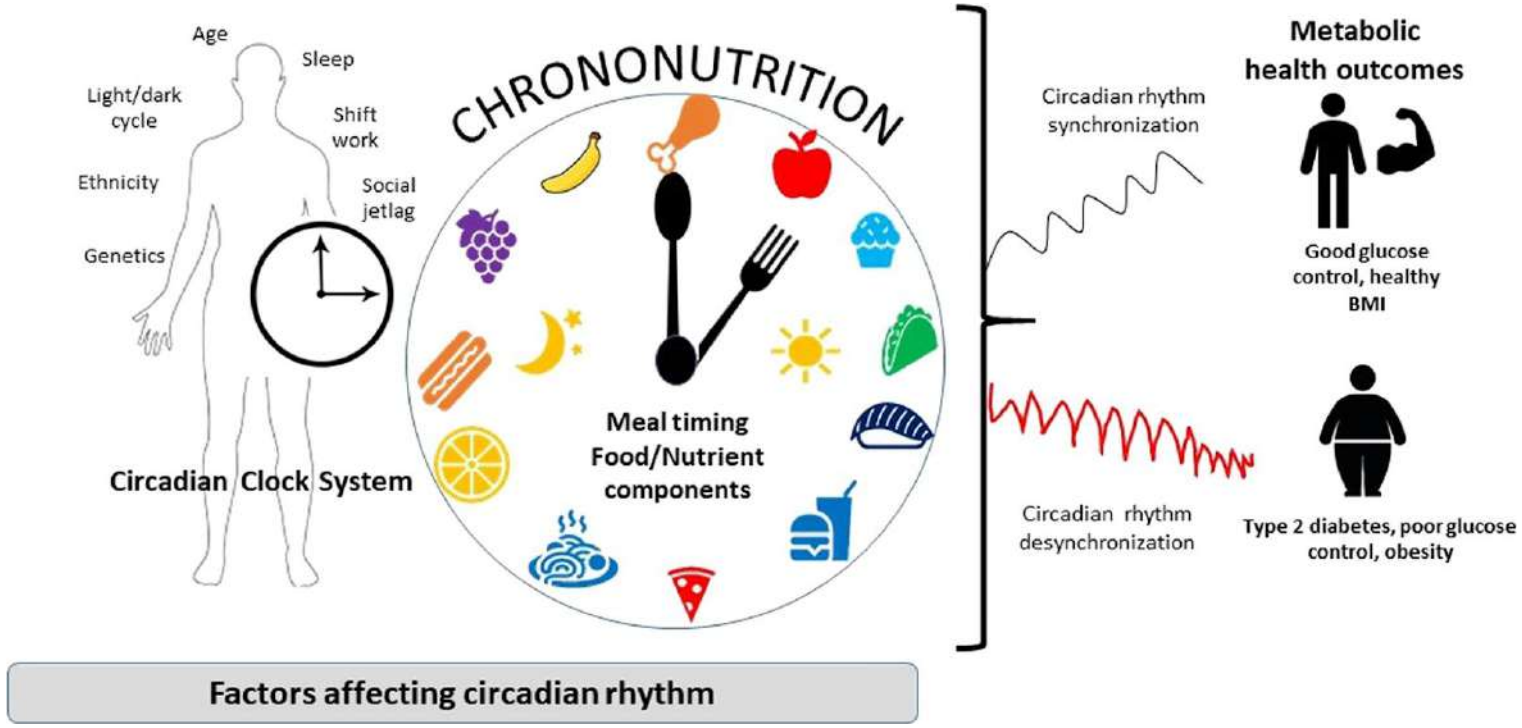
[1] Calvin AD, et al. Effects of Experimental Sleep Restriction on Caloric Intake and Activity Energy Expenditure. Chest. 2013;144(1):79–86.
[2] Taheri S, et al. Short Sleep Duration Is Associated with Reduced Leptin, Elevated Ghrelin, and Increased Body Mass Index. PLoS Med. 2004;1(3):e62.
[3] Covassin N, et al. Effects of Experimental Sleep Restriction on Energy Intake, Energy Expenditure, and Visceral Obesity. J Am Coll Cardiol. 2022;79(13):1254–1265.

Why is chronobiology important?

To understand WHEN our body should rest or be exposed to stress/exercise, etc.

It influences:

- Energy/fatigue levels
- Digestive function/symptoms
- Hormonal balance
- Stress levels
- Body composition
- Mental health & immunity
- Development of chronic disease



Factors affecting circadian rhythm

www.precisionhealthalliance.org

Circadian rhythm & metabolic health risk

- In a 6-week intervention, women restricted to an average of **5.6 hours of sleep per night** exhibited a **significant reduction in whole-body insulin sensitivity**, measured via frequently sampled intravenous glucose tolerance tests (FSIGTs).
- Sleep restriction may impair insulin signalling via increased sympathetic activity, elevated evening cortisol, systemic inflammation, and disruption of **circadian gene expression (e.g., CLOCK, BMAL1, PER, CRY)**
- The increase in insulin resistance occurred **independently of weight gain**, suggesting that sleep loss exerts **direct physiological effects** on glucose metabolism through neuroendocrine and circadian pathways.

- The decline in insulin sensitivity was **more pronounced in postmenopausal participants**, implicating estrogen deficiency and age-related metabolic shifts as potential amplifiers of sleep-related insulin dysregulation.

(Schrader etal, 2015 & Zuraikat et al, 2023)

Cortisol patterns: The visceral fat driver

Chronic cortisol elevation is one of the most overlooked drivers of visceral adiposity and weight loss resistance.

It operates across multiple pathways simultaneously, causing increased appetite, poor sleep, promoting muscle catabolism and stimulating visceral fat deposition

Clinical signs of cortisol dysregulation

- Central weight gain despite caloric deficit, Elevated fasting glucose
- Afternoon energy crashes and carbohydrate cravings at 3–4 pm
- Poor sleep onset or early waking (cortisol awakening response disruption)
- High perceived stress, anxiety, and low mood

Clinical interventions

- Circadian alignment and sleep support using magnesium, herbal medicine such as Withania, Saffron, Passionflower, Mellissa, California Poppy, etc.
- Protein focus, meal timing, phenotype support and appropriate exercise and rest/HPA axis support
- Address lifestyle stressors

Circadian rhythm and chrono nutrition

The timing of food intake relative to circadian biology has meaningful effects on insulin sensitivity, fat oxidation, and appetite regulation — independent of total caloric intake. Circadian misalignment (irregular meal timing, late-night eating, shift work) is a clinically relevant but underappreciated driver of weight loss resistance.

Front-load calories

Insulin sensitivity is highest in the morning. A larger breakfast and smaller evening meal improves glycaemic control and supports fat oxidation overnight.

Eating window

A 10–12 hour eating window aligned to daylight hours supports circadian entrainment of metabolic hormones, liver function, and gut motility.

Avoid late-night eating

Eating within 2–3 hours of sleep suppresses melatonin, disrupts growth hormone release, and shifts lipid metabolism towards storage rather than oxidation.

NEAT – Non-Exercise Activity Thermogenesis

Accounts for 15–50% of total daily energy expenditure in active individuals.

- NEAT is highly variable between individuals.
- Is profoundly suppressed by caloric restriction diets.

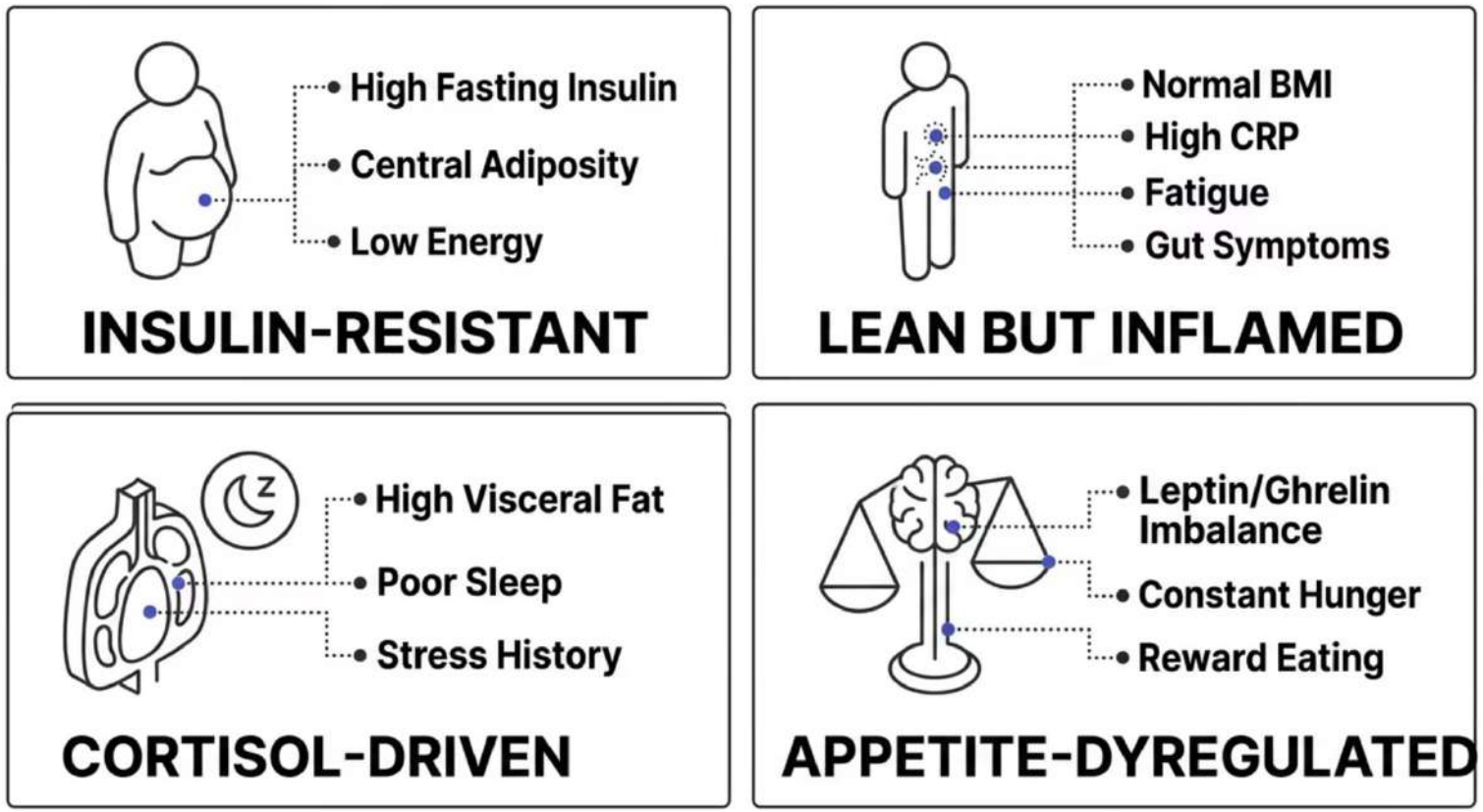
Adaptive thermogenesis reduces NEAT by up to 300–500 kcal/day during caloric restriction — often negating the caloric deficit itself.

- Patients become less fidgety, reduce spontaneous movement.
- Daily step targets of 8,000–10,000 are strongly associated with improved body composition.

Standing desks, walking meetings, post-meal walks (10 minutes reduces postprandial glucose by up to 22%), and breaking prolonged sitting every 30 minutes.

Identifying the metabolic phenotype

Not all patients with elevated body fat present the same metabolic picture. Distinguishing phenotypes allows for precision protocol design rather than a one-size-fits-all approach. The two most clinically relevant categories are the insulin-resistant phenotype and the lean-but-inflamed phenotype — each requires a distinct therapeutic emphasis.



We have differences in our optimal stress

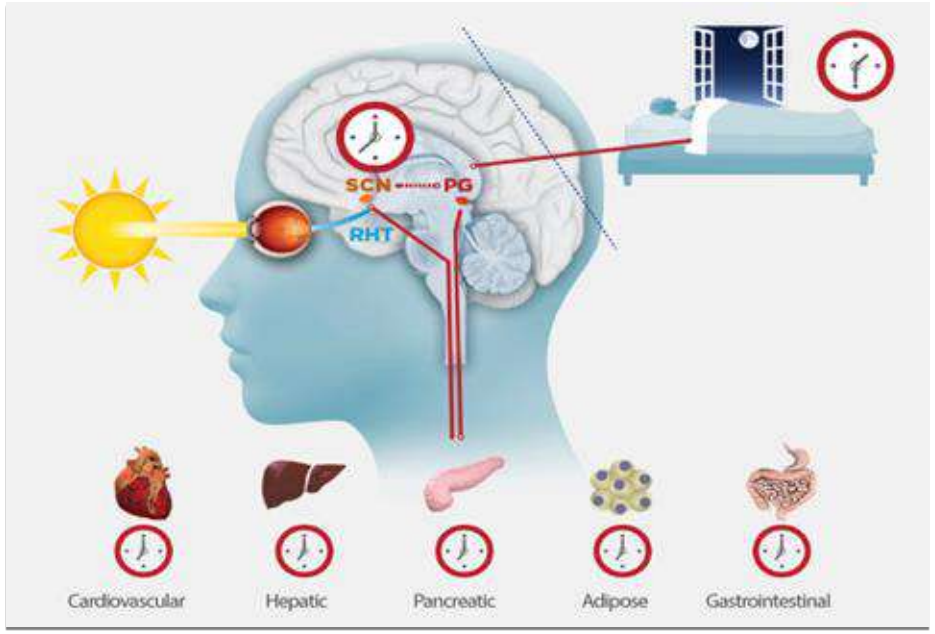
EARLY BIRD

Greater tolerance of morning stress

Body temp rises faster in the am.

Higher cortisol & lower melatonin.

CHRONOTYPES



PHA
PRECISION HEALTH ALLIANCE

NIGHT OWL

Needing calm in the morning

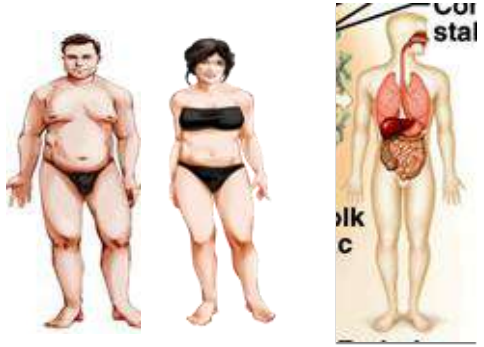
More sensitive to light.

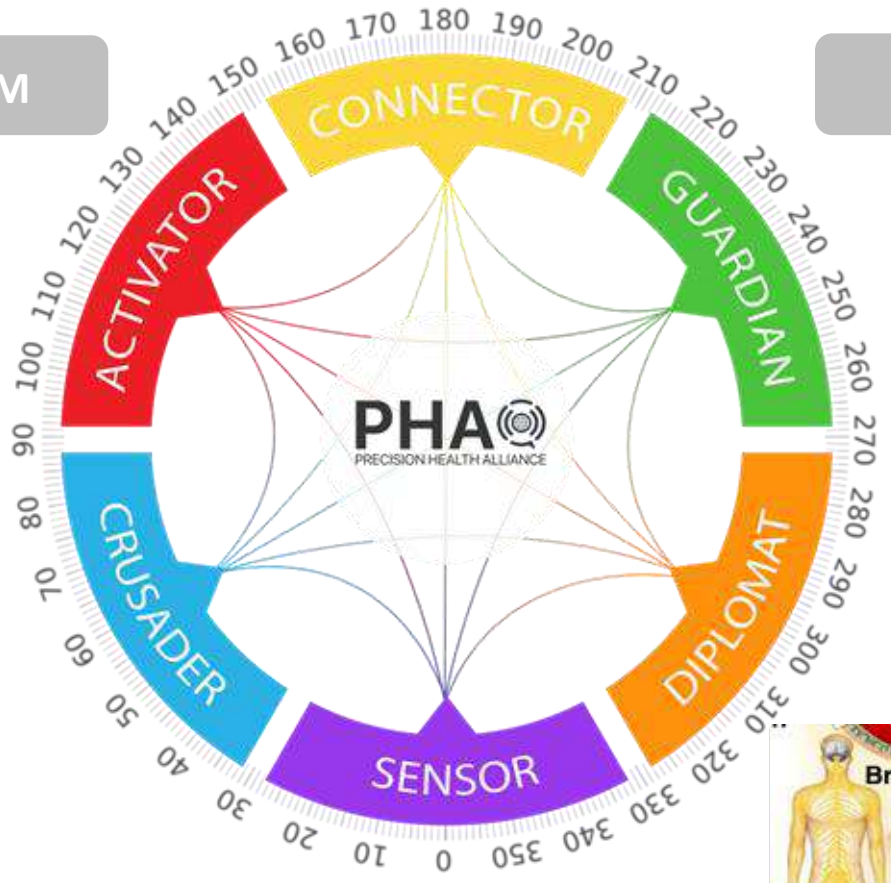
More sensitive to stress in the morning.

Embryo to HealthType

MESODERM

ENDODERM





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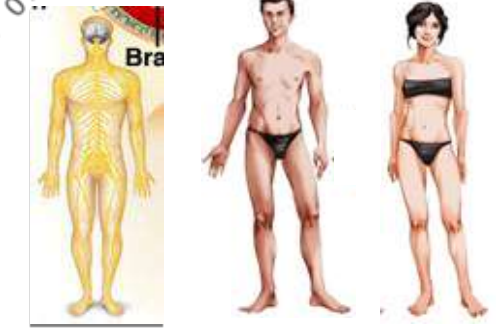

Womb environment
determines layer dominance.

PHA
PRECISION HEALTH ALLIANCE

ACTIVATOR

ACTH & Testosterone
Produce more & sensitive to it
Oxidative & Inflammatory
Early chronotype

Morphology
Shorter, Leaner, Muscular
Square face, shorter femurs
The typical gymnast

“Use then Rest Adrenals”
High intensity activity
5-6 meals/day
A midday nap
Competition, Variety, Novelty, Challenge

If they don’t get it
Irritable, agitated
Inflamed joints
Overactive immune function (Hashimoto’s)
Central weight gain

If they don’t get it
Cortisol & crash
Fluid retention, stubborn central weight, - thyroid
Degradation of collagen - injuries, premature ageing

DIPLOMAT

Serotonin
Gut-brain Serotonin signal
Low cortisol capacity
Night Owl

Morphology
Often taller (can be short)
Thicker legs, longer femurs
Often smooth, no sinewy
Longer limbs & joints more mobile

“Create Calm & Avoid Cortisol/Chaos”
Need circadian consistency
2-3 meals/day
Avoid chaos, esp in a.m.
Hard Exercise in the p.m.

CRUSADER

DOPAMINE
More sensitive & produce more
Early chronotype

Morphology
Longer, Leaner, Mod muscle
Longer face & legs
The typical triathlete

"Goal, Produce, then Rest"
High SNS output more mental activity
5 meals/day - higher carb
Evening mental rest,
Endurance & Stretching
All about progress, logic, strategy, work till u drop

If they don't get it
Disappear in endless work
Rigid neck, back, spine
Muscle loss, abdominal paunch
Burnout & depression

If they don't get it
Dorsal vagal freeze
Weight gain
Insulin resistance
Fatty liver
Non stop worry (family)

GUARDIAN

PROLACTIN & INSULIN
Gut-brain Serotonin signal
Low cortisol capacity
Night Owl

Morphology
Biggest, strongest bodies
Largest muscle, fat & bone mass
Short and tall

"Social & life stability"
Monitoring family, food & finances for stability/safety
3 regular meals (v. light dinner)
Avoid sugars, lower protein
Need high volume movement
Hardest & heaviest in p.m.

CONNECTOR

OXYTOCIN
Higher need for it

Intermediate Chronotype

Morphology
Square face, torso, more mass in chest, thicker than Activator, smaller than Guardian

"Connect at all Times"
Make it social at all times
4 meals/day - Light dinner (CHO & Salt)
Lots of movement with people
Want fun, connection, give & receive attention

If they don't get it
Anxious & high cortisol
Oestrogen disruptions
Weight gain upper body
Distracted and scattered

If they don't get it
Catabolism - sarcopenia, demineralisation
Poor absorption, lower HCl production
Anxious, overwhelm

SENSOR

VASOPRESSIN & CNS
Highly sensitive, constant alert
Intermediate Chronotype

Morphology
Most delicate body. Least bone, muscle and fat mass
Generally longer femurs, fingers (can be tall and short)

"Calm, Quiet, Warm"
5 meals, warm, well cooked
Higher complex carb
Light & mind-body exercise
External warmth & low sensory input

Evidence-based supplementation

OEA: The satiety lipid mediator

Oleoylethanolamide is an endogenous lipid mediator produced in small intestinal enterocytes in response to dietary fat ingestion. It activates PPAR-α receptors, signalling satiety to the hypothalamus via the vagus nerve and stimulating fat oxidation in peripheral tissue.[1]

Clinical mechanisms:

- Activates PPAR-α to upregulate fatty acid oxidation[1]
- Reduces meal frequency and inter-meal appetite via hypothalamic signalling[2]
- Modulates dopamine pathways involved in food reward
- Shown to reduce visceral fat in insulin-resistant phenotypes[2]

Prescribing note:

125mg 20 minutes prior to breakfast and dinner; most effective in patients with high reward-driven eating patterns.

References:
[1] Friuli M, et al. "To brain or not to brain": evaluating the possible direct effects of the satiety factor oleoylethanolamide in the central nervous system. Front Endocrinol. 2023;14:1158287.
[2] Payahoo L, et al. Oleoylethanolamide increases the expression of PPAR-α and reduces appetite and body weight in obese people: A clinical trial. Appetite. 2018;128:214–219.
[3] Tutunchi H, et al. Effects of OEA supplementation on inflammatory biomarkers and oxidative stress in obese patients with NAFLD on a calorie-restricted diet: A randomised controlled trial. Pharmacol Res. 2023;37089938

Magnesium: The metabolic master mineral

Magnesium is a cofactor in over 300 enzymatic reactions[1], including those governing insulin receptor signalling, glucose metabolism, and cortisol regulation. Deficiency — prevalent in up to 70% of the general population[2] — is strongly associated with insulin resistance[3], poor sleep quality, and impaired fat metabolism.

Insulin Sensitivity

Magnesium is essential for insulin receptor tyrosine kinase activity. Repletion improves glucose uptake and fasting insulin in deficient patients[3].

Sleep Quality

Magnesium glycinate and threonate support GABA receptor activity, deepening slow-wave sleep — critical for leptin release and cortisol resetting.

Cortisol Regulation

Magnesium attenuates HPA axis hyperreactivity. Deficiency amplifies cortisol output, promoting visceral fat deposition and appetite dysregulation.

Dose: 300-600mg/day is common

References:
[1] Chacko SA, et al. Magnesium supplementation, metabolic and inflammatory markers, and global genomic and proteomic profiling: a randomised, double-blind, controlled, crossover trial in overweight individuals. Am J Clin Nutr. 2011;93(2):463–473.
[2] Wang J, et al. Dietary Magnesium Intake Improves Insulin Resistance among Non-Diabetic Individuals with Metabolic Syndrome. Nutrients. 2013;5(10):3910–3919.
[3] Salehidoost R, et al. Effect of oral magnesium supplement on cardiometabolic markers in people with prediabetes: a double-blind randomised controlled clinical trial. Sci Rep. 2022;12:18209.

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[2] Wang J, et al. Dietary Magnesium Intake Improves Insulin Resistance among Non-Diabetic Individuals with Metabolic Syndrome. Nutrients. 2013;5(10):3910–3919.
[3] Salehidoost R, et al. Effect of oral magnesium supplement on cardiometabolic markers in people with prediabetes: a double-blind randomised controlled clinical trial. Sci Rep. 2022;12:18209.

Protein supplementation: Precision, not excess

Serves a distinct clinical role beyond what whole food alone can achieve for many patients — particularly older adults with anabolic resistance, patients in significant caloric restriction, or those with impaired appetite on GLP-1 therapy.

Whey Protein

- Highest leucine content (~11%) — optimal for MPS stimulation
- Rapidly digested; ideal post-resistance training (within 2 hours)
- Supports gut barrier function via glutathione precursors

Plant-based proteins

- Pea + rice blend provides a complete amino acid profile
- Lower leucine content; higher doses required (~35–40g/serving)
- Suitable for dairy-intolerant or vegan patients
- May offer additional prebiotic fibre benefit for gut-dysbiosis phenotypes

Collagen: Beyond joint health

Hydrolysed collagen peptides have emerged as a clinically relevant adjunct in body composition protocols — not as a primary protein source, but as a specific tool for connective tissue remodelling, satiety modulation, and joint support during resistance training. Its unique amino acid profile (glycine, proline, hydroxyproline) is distinct from complete proteins and complementary rather than competitive.

Satiety enhancement

Collagen peptides stimulate the release of GLP-1 and PYY in the gut, producing greater post-meal satiety than equivalent doses of other proteins in some trials.

Connective tissue remodelling

10–15g pre-exercise with vitamin C supports tendon and ligament synthesis — critical for patients increasing training load to preserve lean mass.

Glycine for metabolic health

Glycine supports hepatic glutathione synthesis, mitochondrial function, and insulin sensitivity — providing metabolic benefit beyond structural support.

Creatine monohydrate: An underutilised fat loss tool

- Creatine monohydrate is the most extensively researched ergogenic supplement in existence[1]
- By enabling greater training volume and intensity, it amplifies the anabolic stimulus for lean **mass preservation** during energy restriction. (2) (3)
- Role in **brain energy metabolism**; relevant for diet-related fatigue and mood disturbance that impair adherence
- Resistance training + creatine shows an additive effect on bone mineral density[3], especially relevant for peri-menopause and for GLP-1 users. Dosages can range from 3-5gm/day or higher

[1] Desai I, et al. The Effect of Creatine Supplementation on Resistance Training-Based Changes to Body Composition: A Systematic Review and Meta-analysis. J Strength Cond Res. 2024;38(10):1813–1821.
[2] Candow DG, et al. Resistance Exercise and Creatine Supplementation on Fat Mass in Adults <50 Years of Age: A Systematic Review and Meta-Analysis. Nutrients. 2023;15(21):4519
[3] Mills S, et al. Effects of Creatine Supplementation during Resistance Training Sessions in Physically Active Young Adults. Nutrients. 2020;12(6):1880.

Berberine

Improved Glycaemic Control: Berberine supplementation significantly reduced fasting blood glucose (FBG) and glycated haemoglobin (HbA1c) levels in women, particularly for women who are peri or post menopausal, with PCOS/PMOS & metabolic syndrome.

Enhanced Insulin Sensitivity: by a decrease in the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) among female participants, suggesting improved insulin sensitivity.

Lipid Profile Benefits: Reductions in total cholesterol and low-density lipoprotein (LDL) cholesterol levels, contributing to better cardiovascular health.

Ye et al, 2021, Zhao et al, 2023

Fibre

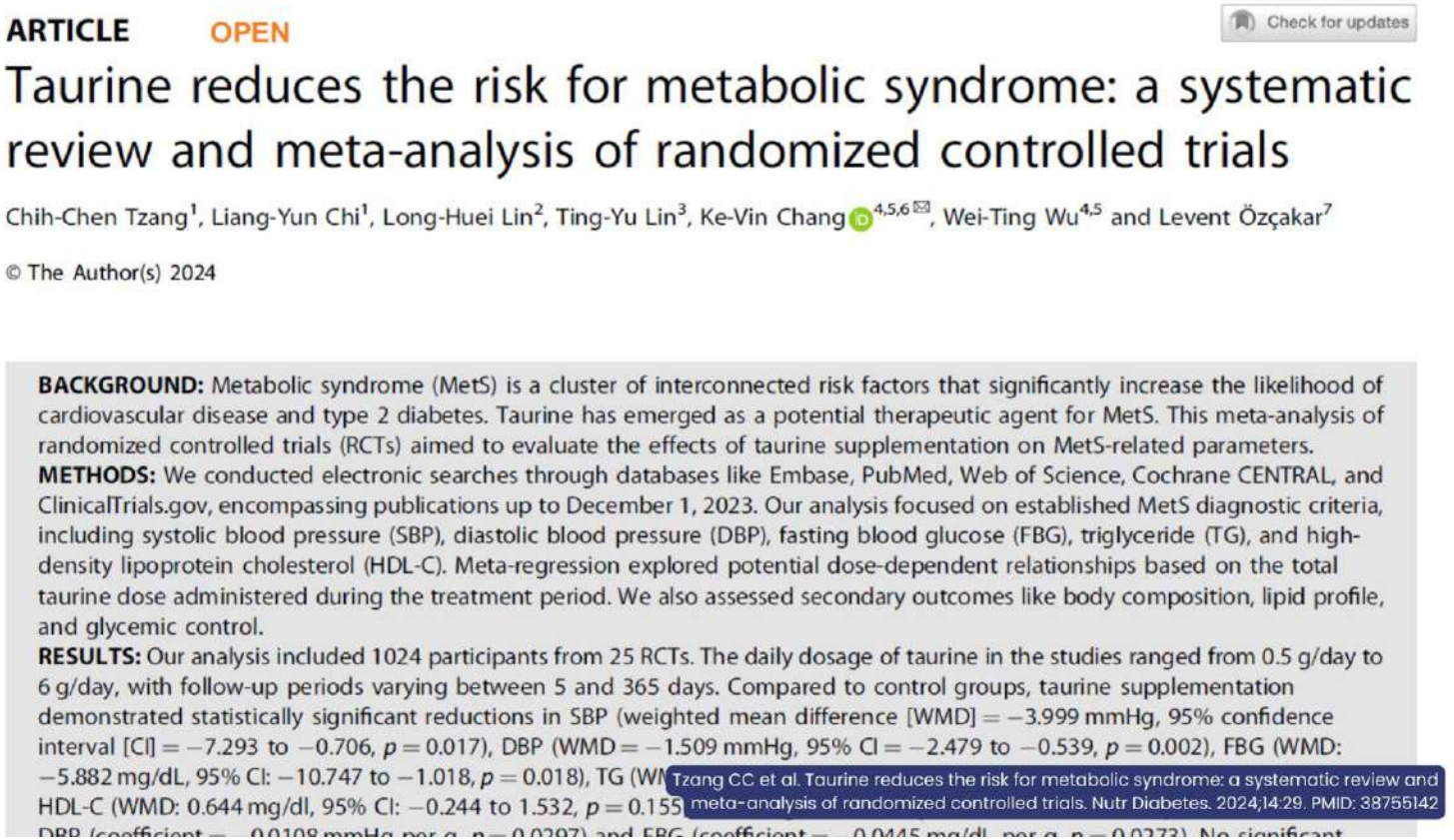
- Associated with **improved insulin sensitivity and lower insulin resistance**
- **Improves glucose metabolism**: Soluble fibre slows gastric emptying and carbohydrate absorption, leading to lower postprandial glucose spikes and improved insulin sensitivity.
- **Modulates gut microbiota**: Fermentable fibres enhance short-chain fatty acid (SCFA) production (especially butyrate), which has anti-inflammatory effects and improves insulin signalling.
- **Reduction in systemic inflammation:** Increased fibre intake is associated with lower inflammatory markers (e.g., CRP), which are known to impair insulin receptor function.
- **Improve satiety and body composition:** Fibre contributes to appetite regulation and reduced caloric intake

Fibres such as β-glucan, psyllium, and guar gum, inulin, acacia, apple pectin**,** psyllium husk, and flaxseed (which are viscous and fermentable) were shown to significantly lower fasting glucose

(Hall et al, 2024 & Xiang et al, 2025)

Other nutrients to consider

- Taurine
- Choline
- Glycine
- CoQ10/Ubiquinol
- Inositol
- Other mitochondrial supporting nutrients
- Liver and digestive herbs and nutrients



ARTICLE OPEN

Check for updates

Taurine reduces the risk for metabolic syndrome: a systematic review and meta-analysis of randomized controlled trials

Chih-Chen Tzang[1], Liang-Yun Chi[1], Long-Huei Lin[2], Ting-Yu Lin[3], Ke-Vin Chang[4,5,6]✉, Wei-Ting Wu[4,5] and Levent Özçakar[7]

BACKGROUND: Metabolic syndrome (MetS) is a cluster of interconnected risk factors that significantly increase the likelihood of cardiovascular disease and type 2 diabetes. Taurine has emerged as a potential therapeutic agent for MetS. This meta-analysis of randomized controlled trials (RCTs) aimed to evaluate the effects of taurine supplementation on MetS-related parameters.
METHODS: We conducted electronic searches through databases like Embase, PubMed, Web of Science, Cochrane CENTRAL, and ClinicalTrials.gov, encompassing publications up to December 1, 2023. Our analysis focused on established MetS diagnostic criteria, including systolic blood pressure (SBP), diastolic blood pressure (DBP), fasting blood glucose (FBG), triglyceride (TG), and high-density lipoprotein cholesterol (HDL-C). Meta-regression explored potential dose-dependent relationships based on the total taurine dose administered during the treatment period. We also assessed secondary outcomes like body composition, lipid profile, and glycemic control.
RESULTS: Our analysis included 1024 participants from 25 RCTs. The daily dosage of taurine in the studies ranged from 0.5 g/day to 6 g/day, with follow-up periods varying between 5 and 365 days. Compared to control groups, taurine supplementation demonstrated statistically significant reductions in SBP (weighted mean difference [WMD] = −3.999 mmHg, 95% confidence interval [CI] = −7.293 to −0.706, $p = 0.017$), DBP (WMD = −1.509 mmHg, 95% CI = −2.479 to −0.539, $p = 0.002$), FBG (WMD: −5.882 mg/dL, 95% CI: −10.747 to −1.018, $p = 0.018$), TG (WM
HDL-C (WMD: 0.644 mg/dl, 95% CI: −0.244 to 1.532, $p = 0.155$
DBP (coefficient = −0.0108 mmHg per g, $p = 0.0297$) and FBG (coefficient = −0.0445 mg/dL per g, $p = 0.0273$). No significant

Tzang CC et al. Taurine reduces the risk for metabolic syndrome: a systematic review and meta-analysis of randomized controlled trials. Nutr Diabetes. 2024;14:29. PMID: 38755142

Synergistic supplementation combinations

Creatine + Protein

The most evidence-supported combination for lean mass preservation during energy restriction. Creatine amplifies training stimulus; protein provides the substrate for muscle protein synthesis. Combine 3–5g creatine daily with 1.6–2.2g/kg protein.

OEA + Collagen (Pre-Meal)

Both independently stimulate GLP-1 and satiety peptides. Taken 30 minutes before the largest meal, this combination creates additive appetite suppression — particularly useful for patients with high reward-driven or volume-driven eating.

Magnesium Glycinate + Collagen (Evening)

Taken before bed, this combination supports deep sleep (via GABA modulation), overnight connective tissue repair, and cortisol resetting. Glycine from collagen has independent sleep-promoting effects.

GLP-1 Integration

- GLP-1 receptor agonists (semaglutide, tirzepatide, liraglutide) represent the most significant pharmacological advance in obesity management in decades.
- They are not a standalone solution.
- Without concurrent attention to lean mass, nutritional adequacy, and lifestyle architecture, patients risk losing disproportionate muscle mass and setting the stage for rebound weight gain upon cessation.

GLP-1 medications: What clinicians need to know

Mechanisms of Action

GLP-1 agonists slow gastric emptying, reduce glucagon secretion, enhance insulin release in a glucose-dependent manner, and act centrally to suppress appetite and food reward signalling in the hypothalamus and brainstem.

The Lean Mass Risk

Trials show that up to 40% of weight lost on GLP-1 monotherapy may be lean mass, particularly in patients not engaged in resistance training or consuming adequate protein.[3] This is clinically significant and often overlooked at point of prescribing.

Nutritional Adequacy on GLP-1s

Severe appetite suppression increases the risk of protein, micronutrient, and caloric inadequacy. Structured supplementation — particularly protein (whey or collagen), creatine, and magnesium — becomes even more critical in this population.

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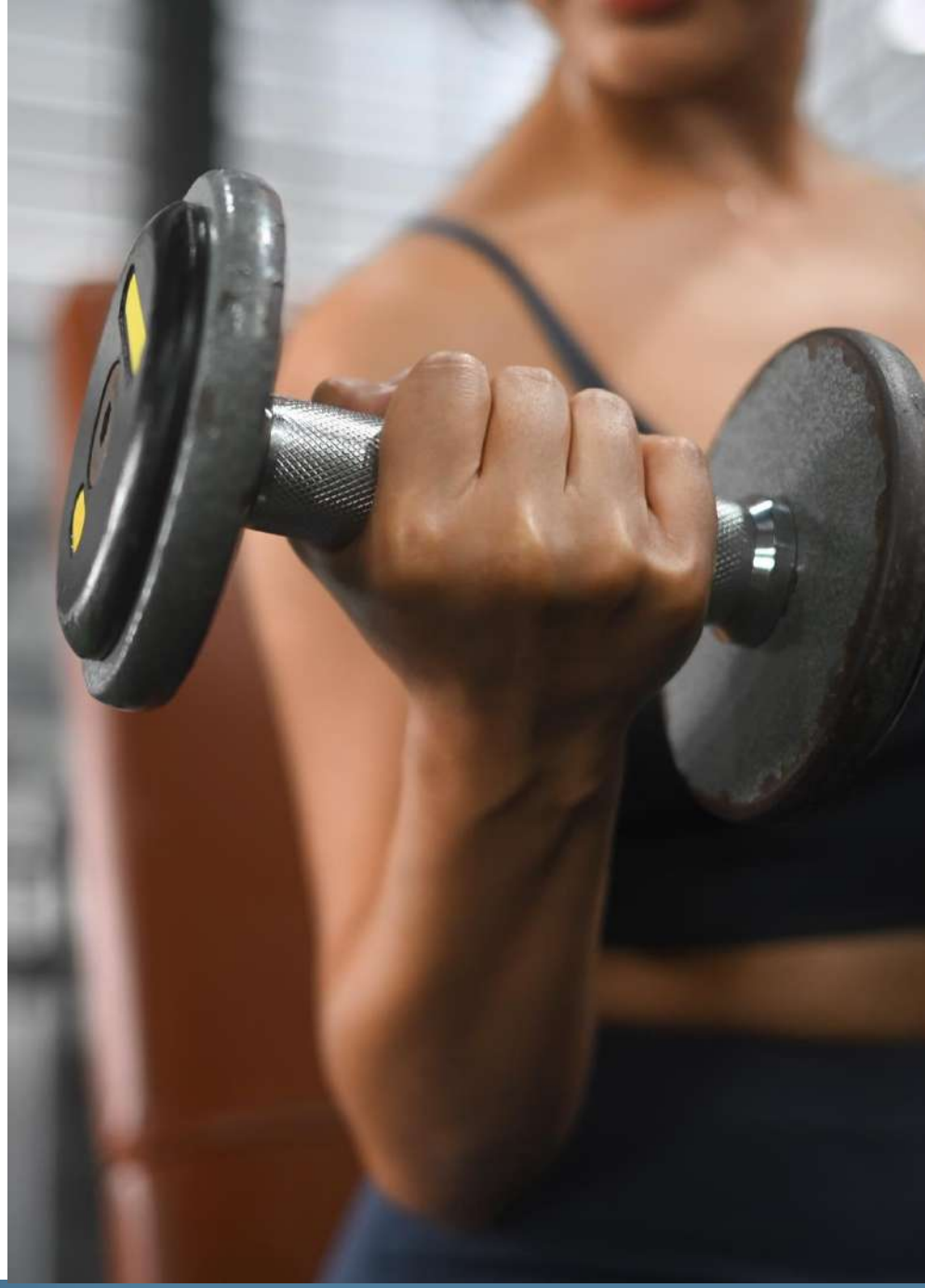
[1] Wilding JPH, et al. Once-Weekly Semaglutide in Adults with Overweight or Obesity. N Engl J Med. 2021;384(11):989–1002.
[2] Dunn JP, et al. Tirzepatide as Compared with Semaglutide for the Treatment of Obesity. N Engl J Med. 2025. doi:10.1056/NEJMoa2416394.
[3] Pujol A, et al. Impact of Semaglutide on fat mass, lean mass and muscle function in patients with obesity: The SEMALEAN study. PubMed. 2024;41068996.

Priority interventions in patients on GLP-1 therapy

- **Protein target:** Maintain ≥1.6g/kg/day throughout treatment.
- **Resistance training:** Non-negotiable; preserves lean mass and maintains metabolic rate.
- **Creatine 3–5g daily:** Protects muscle performance and supports training quality despite reduced energy intake.
- **Magnesium:** Sleep and cortisol support; reduced dietary variety may worsen pre-existing deficiency.
- **Collagen pre-meal:** Enhances satiety at smaller meal volumes; supports connective tissue during rapid weight change.



The GLP-1 exit protocol: Preventing rebound

Patients who discontinue GLP-1 therapy without a structured exit protocol regain, on average, 2/3 of lost weight within 12 months (1)(2)

- **8-Week Taper**
- **Increase Protein**
- **Appetite Habits**
- **Resistance Training**
- **Monthly Monitoring**

[1] Quarenghi M, et al. Weight Regain After Liraglutide, Semaglutide or Tirzepatide Interruption: A Narrative Review of Randomized Studies. J Clin Med. 2025;14(11):3791.
[2] Steinberg GR, et al. Can muscle avert GLP1R weight plateau and regain? Cell Rep Med. 2025;6(9):102308.

Preventing rebound hyperphagia: The mechanism

Understanding the biological basis of post-GLP-1 rebound equips clinicians to counsel patients proactively and design preventative strategies.

Ghrelin rebound

- Medications suppress ghrelin-driven appetite. Upon cessation, ghrelin surges — often above pre-treatment baseline — driving intense hunger in the first 4–8 weeks

Reduced metabolic rate

- If lean mass has been lost during treatment, the resting metabolic rate is lower than before treatment began

Leptin dysregulation

- Rapid fat loss can paradoxically suppress leptin, signalling perceived starvation to the hypothalamus and further amplifying food-seeking behaviours post-cessation

Phenotype-matched protocol selector

Prioritise interventions for the dominant metabolic phenotype and then support the second potential driver.

Phenotype	Priority Interventions	Key Supplements	Cautions
Insulin-Resistant	Protein-forward diet, resistance training, lower-GI carbohydrate timing, 10-min post-meal walks	Magnesium, Creatine, Berberine, Protein	Avoid very low-fat diets; prioritise metabolic flexibility before caloric restriction
Cortisol-Driven	Sleep prioritisation, HPA support, stress reduction, protein adequacy	Magnesium glycinate, Withania, Collagen (evening)	Aggressive caloric restriction worsens cortisol; stabilise first
Appetite-Dysregulated	Meal structure, pre-meal satiety tools, reward pathway support	OEA, Collagen pre-meal, Protein at breakfast	Address leptin resistance (inflammation) before solely restricting calories
On GLP-1 Therapy	Protein targets, resistance training, structured exit planning from initiation	Protein, Creatine, Magnesium, Collagen	Screen for protein inadequacy at every visit; anticipate lean mass loss risk

Putting it all together: The clinical framework

Assess	Metabolic phenotype, body composition, hormones, stress, sleep, etc.
Nourish	Protein forward diet, circadian timing, monitor glycaemic load of diet, 3-500 calorie deficit or cyclical dieting
Supplement	OEA, creatine, collagen, protein, magnesium, etc.
Movement	Resistance training, zone 2 training, NEAT, 10,000 steps, etc.
Recover	Sleep 7-9 hours, HPA axis support, address hormones and cortisol

Thank you – Kira Sutherland

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https://www.kirasutherland.com.au/applied-weight-loss-nutrition

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2. Karampela I, et al. The Role of Mitochondrial Adaptation and Metabolic Flexibility in the Pathophysiology of Obesity and Insulin Resistance. Curr Obes Rep. 2021;10(2):191–207.

3. Farías J, et al. Association Between Adipose Tissue Characteristics and Metabolic Flexibility in Humans: A Systematic Review. Front Nutr. 2021;8:744187.

OEA (Oleoylethanolamide)

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Magnesium

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8. Wang J, et al. Dietary Magnesium Intake Improves Insulin Resistance among Non-Diabetic Individuals with Metabolic Syndrome. Nutrients. 2013;5(10):3910–3919.

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GLP-1 Receptor Agonists

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Trpti OEA™

The challenge with standard OEA

Oleoylethanolamide (OEA) is a naturally occurring lipid mediator synthesised in the small intestine.

However, conventional forms of OEA face significant bioavailability limitations due to poor dispersion and degradation in the gastrointestinal tract. That means delivery matters...

Table 1: Summary of Human Clinical Trials with OEA.

Population / Condition	Intervention	Dosage	Methodology	Results	Reference
Healthy overweight adults	Trpti OEA™	Single-dose 125 mg or 250 mg	Single-blind, placebo-controlled, 3-way cross-over study; 8-hour monitoring, (n=40)	250 mg significantly increased GLP-1 AUC (~3.5× vs placebo) and Cmax (1.7× vs placebo), with sustained elevation post-meals. Dose-dependent suppression of DPP-4 observed, correlating with GLP-1 preservation. No effect on glucagon or GIP. Well tolerated; only mild GI events (<5%).	Briskey & Rao, 2025. *Acute effect of Trpti (OEA) supplementation on metabolic pathways.* Publication pending.
Healthy overweight and obese adults	Trpti OEA™	2 × 125 mg (250 mg/ day)	RCT, DB, PC (n=44) 12 weeks	Change-from-baseline analyses: ↑ GLP-1 at W6 & W12 (OEA), ↑ occludin at W12 vs placebo; ↓ IL-1β at W6 vs placebo and vs baseline at W12; ↑ IL-2 at W6 vs placebo. Microbiome: ↑ beneficial taxa (*A. muciniphila*, *F. prausnitzii*) in OEA group. No serious AEs.	*Effect of OEA vs placebo on gut microbiome.* RCT. 2025 Publication pending.
Healthy adults	Trpti OEA™	2 × 125 mg (250 mg/ day)	PC, 2 arm, single-dose crossover study, (n=6)	Trend: ↑ GLP-1 (higher AUC/Cmax), ↓ GIP with OEA vs placebo. Two mild GI AEs (fasted state), resolved with food.	*Effect of OEA on GLP-1 and GIP (crossover).* 2025. Publication pending.

Meet Trpti™ OEA – a next-generation OEA with LipiSperse® delivery technology

LipiSperse® technology was developed to improve the dispersion of lipophilic ingredients, helping address the bioavailability challenges historically associated with standard OEA forms.

By enhancing dispersibility in aqueous environments, this delivery technology offers a more advanced approach to OEA formulation.