

Station B: Sarcopenic Obesity

What is Sarcopenic Obesity?

- Sarcopenic obesity (SO) is the co-existence of excess adiposity (high fat mass) AND low skeletal muscle mass and function
- Defined by the 2022 ESPEN/EASO Consensus as a distinct clinical condition, not simply obesity plus sarcopenia happening to occur in the same person
- Someone with SO may have a normal or even elevated body weight but simultaneously have reduced muscle mass, impaired strength, and excess body fat
- Outcomes are synergistically worse than either condition alone: SO has nearly 3 times the risk of disability and 3 times the risk of cognitive impairment compared to people with neither condition
- BMI alone cannot identify sarcopenic obesity. A person classified as 'overweight' by BMI could have dangerously low muscle mass hidden beneath excess fat.

Prevalence

- Global pooled estimate: approximately 11% of older adults (pre-2022 criteria; ranges widely from 0.1 to 48% depending on the definition used)
- Using the 2022 ESPEN/EASO standardised criteria: 4.5% in a German population study (KORA-Age), rising to 15% in some cohorts
- Prevalence increases substantially after age 70
- Higher in hospitalised populations and people with severe obesity
- Formal incidence data is limited because standardised diagnostic criteria were only published in 2022

Aetiology and Pathology

Shared mechanisms drive both muscle loss and fat gain simultaneously. This is not a coincidence; these are connected pathways:

- **Insulin resistance:** impairs muscle protein synthesis (insulin is one of the signals that tells muscle to build protein) while simultaneously promoting fat storage. The same metabolic dysfunction that drives Type 2 diabetes also drives the muscle-fat imbalance of SO. (Connection to Week 4 metabolic disease.)

- **Chronic low-grade inflammation:** adipose tissue, particularly visceral fat, actively secretes inflammatory cytokines (IL-6, TNF-alpha) that circulate through the body and promote muscle protein breakdown. The excess fat is not inert; it is actively destroying muscle.
- **Hormonal dysregulation:** declining anabolic hormones (testosterone, growth hormone, IGF-1) reduce muscle building capacity while favouring fat deposition.
- **Physical inactivity:** a sedentary lifestyle accelerates both muscle loss and fat gain simultaneously.

The Vicious Cycle

Muscle loss reduces mobility. Reduced mobility reduces physical activity. Reduced activity promotes fat gain. Increased fat produces more inflammation. Increased inflammation accelerates muscle loss. This is a self-reinforcing spiral that is very difficult to break without targeted intervention.

Signs and Symptoms

- Often clinically 'invisible': the person may appear overweight or obese on standard assessment, masking the underlying muscle loss
- Reduced functional capacity disproportionate to body weight: difficulty rising from a chair, climbing stairs, carrying objects
- Low grip strength (measurable with a dynamometer)
- Slow gait speed
- Poor balance and increased falls risk
- Fatigue and reduced exercise tolerance
- May have metabolic complications: insulin resistance, dyslipidaemia, hypertension (metabolic syndrome features)
- Weight may appear 'stable' or even elevated while functional decline progresses

Diagnosis (ESPEN/EASO 2022 Algorithm)

- **Step 1 (Screening):** identify at-risk individuals (older adults, those with chronic disease, people with significant weight changes)
- **Step 2 (Diagnosis):** requires ALL THREE of:
 - Reduced muscle function (handgrip strength or sit-to-stand test)
 - Reduced skeletal muscle mass relative to body weight (not height squared, to account for excess weight)

- Elevated fat mass
- **Step 3 (Staging):** assess for clinical complications (metabolic syndrome, CVD, respiratory disease)

Key difference from isolated sarcopenia diagnosis: muscle mass is adjusted by body weight rather than height squared. If someone weighs 120 kg, they need more muscle to support and move that body mass than someone who weighs 60 kg. Adjusting by height alone would miss sarcopenia in obese individuals.

Risk Factors

Non-modifiable

- Age (risk increases substantially after 70)
- Female sex (hormonal decline at menopause accelerates both muscle loss and fat redistribution)
- Chronic disease burden (diabetes, COPD, heart failure, cancer all contribute)

Modifiable

- Physical inactivity (the strongest modifiable factor: drives both muscle loss and fat gain)
- Inadequate protein intake (older adults need 1.0 to 1.2 g/kg/day; those with sarcopenia need up to 1.5 g/kg/day)
- Caloric restriction without exercise (weight loss diets that do not include resistance training cause muscle loss alongside fat loss, potentially worsening body composition)
- Vitamin D deficiency
- Sedentary behaviour independent of exercise (prolonged sitting even in people who exercise)

Quality of Life

- Disability: nearly 3 times the risk compared to people with neither condition
- Multimorbidity: odds ratio 2.59
- Cognitive impairment: odds ratio 3.03
- Arthritis: odds ratio 2.66
- Also associated with increased cardiovascular risk, frailty, loss of independence, and mortality

- Treatment requires an integrated approach addressing both muscle loss AND excess fat simultaneously:
 - Resistance training is essential (not just aerobic exercise)
 - Adequate protein intake with leucine-rich sources distributed across the day
 - Caloric restriction must be moderate and combined with resistance training to protect muscle
- Body weight alone tells you almost nothing about muscle and bone health. Grip strength, sit-to-stand tests, and balance assessments tell you far more than a bathroom scale.

Connections to Other Weeks

- Week 4 (Metabolic Disease): insulin resistance, chronic inflammation, and the metabolic syndrome are shared pathways. SO is where metabolic disease meets MSK disease.
- Week 5 (Sarcopenia): SO builds directly on the sarcopenia content. Students should apply what they have just learned about muscle loss mechanisms.
- Week 5 (Osteosarcopenia): a person can simultaneously be losing bone (osteoporosis), losing muscle (sarcopenia), and gaining fat (obesity). Three processes, all driven by shared mechanisms, all making each other worse.
- The Dahlgren and Whitehead model: diet quality, physical activity access, food environment, socioeconomic status all influence SO risk.

Sources

- ESPEN/EASO Consensus Statement: Definition and Diagnostic Criteria for Sarcopenic Obesity. Clinical Nutrition (2022); Obesity Facts (2022)
- KORA-Age Study, Germany: prevalence and clinical associations using 2022 criteria
- Luo Y et al. (2024) Prevalence of sarcopenic obesity in the older non-hospitalised population: a systematic review and meta-analysis. BMC Geriatrics
- Barazzoni R et al. (2018) Sarcopenic obesity: Time to meet the challenge. Clinical Nutrition, 37(6): 1787 to 1793