

BMSH4302 Introduction to Chronic Disease

Examination Revision Guide (Semester 2 2025/2026)

About This Guide

This guide will help you prepare for the BMSH4302 written examination. It sets out what the exam looks like, what you are expected to know, and how to approach each type of question. Read it alongside your lecture notes and any reading you have done across the module.

The exam covers all conditions and concepts introduced in lectures from Week 1 through to Week 10.

Exam Structure

The exam lasts 60 minutes and is worth 50 marks in total. Your final percentage is calculated by doubling your mark.

Section A: Multiple Choice (20 marks)

- 20 questions, each worth 1 mark.
- Answer ALL questions.
- Choose the single best answer for each question.
- Questions test your understanding of definitions, terminology, and key facts from across the module.
- There is no penalty for a wrong answer, so always give an answer.

Section B: Short Answer (30 marks)

- 5 questions. Answer TWO only.
- Each question is worth 15 marks, split into parts (a) and (b).
- Part (a) is worth 8 marks. Part (b) is worth 7 marks.
- Write your answers in the answer booklet provided.
- Questions ask you to apply your understanding, not just recall facts. Use your own words.
- Read all five questions before choosing which two to answer.

Key Terminology: Week 1 Foundations

These concepts underpin the whole module and may be tested directly in Section A.

Chronic disease

- A condition that is long-lasting (generally more than three months) and usually cannot be cured.
- Distinct from acute conditions, which develop suddenly and resolve within days or weeks.

Prevalence and incidence

- Prevalence: the total number of people living with a condition at a given point in time.
- Incidence: the number of new cases arising within a defined time period.
- Prevalence is always higher than incidence for conditions people live with for a long time.

Aetiology

- The study of the causes or origins of a disease.
- From Greek: aition (cause) + logos (study).

Risk factors: modifiable and non-modifiable

- Modifiable risk factors can be changed: for example, smoking, physical inactivity, diet, alcohol use.
- Non-modifiable risk factors cannot be changed: for example, age, sex, family history, ethnicity.

Comorbidity

- When a person lives with two or more conditions at the same time.
- Conditions often interact: having one condition increases the risk of developing others.

Autoimmune disease

- A condition where the immune system attacks the body's own healthy tissue.
- The fundamental mechanism is the same across all autoimmune conditions: what differs is the target tissue.

The immune spectrum (Week 4 framework)

- Three patterns of immune dysfunction relevant to this module.
- Autoimmune: the immune system attacks the body's own healthy tissue (Type 1 diabetes, Hashimoto's, Graves', RA, MS).
- Immune hypersensitivity: the immune system overreacts to a harmless trigger (asthma).
- Chronic inflammation: persistent low-grade inflammatory response to ongoing exposure or damage (COPD, occupational lung disease).

Understanding which pattern applies to a given condition helps explain why the treatment approach differs. Removing the trigger is central to managing chronic inflammation; dampening the immune response is central to managing autoimmune conditions and hypersensitivity.

Conditions by Week

Week 2: Cardiovascular Disease

Coronary Heart Disease (CHD)

- The coronary arteries become narrowed or blocked, reducing blood supply to the heart muscle.
- Caused by a build-up of fatty deposits (atherosclerotic plaques) in the arterial walls.
- Modifiable risk factors: smoking, physical inactivity, poor diet, hypertension, high cholesterol, obesity, diabetes.
- Non-modifiable risk factors: age, sex, family history.
- Note on gender: women's heart disease risk is often underrecognised. Symptoms in women can differ from the classic male presentation and are more likely to be attributed to other causes.

Heart Failure

- The heart muscle is damaged or weakened, reducing its ability to pump blood around the body.
- Not the same as cardiac arrest (the heart stopping completely) or a blocked coronary artery (CHD).
- Often develops as a consequence of CHD, hypertension, or other cardiac conditions.
- Symptoms include breathlessness, fatigue, and fluid retention (oedema).

Hypertension

- Persistently elevated blood pressure in the arteries.
- A major risk factor for CHD, stroke, and heart failure.
- Often has no symptoms until complications develop.

Stroke

- Interruption to the blood supply to part of the brain.
- Ischaemic stroke: caused by a blockage. Haemorrhagic stroke: caused by a bleed.
- Shared risk factors with CHD: hypertension, atrial fibrillation, diabetes, smoking.

Week 3: Pulmonary Disease

Pulmonary conditions are classified by the mechanism of damage. Three types are relevant to this module: obstructive (airways narrowed or blocked), restrictive (lung tissue scarred and stiff), and vascular (blood vessels affected).

COPD (Chronic Obstructive Pulmonary Disease)

- An obstructive lung disease in which airflow is permanently and progressively limited.
- Most commonly caused by long-term smoking. Also associated with occupational dust and fume exposure.
- Airway narrowing in COPD is largely irreversible: this distinguishes it from asthma.
- Symptoms: breathlessness, chronic cough, increased mucus production.
- Wales has one of the highest COPD prevalence rates in the UK, partly reflecting the coal mining industrial heritage.

Asthma

- A condition characterised by airway inflammation and episodes of airway narrowing.
- Key distinction from COPD: airway narrowing in asthma can be reversed, for example with a bronchodilator inhaler.
- Classified as immune hypersensitivity rather than a degenerative condition.
- Wales has the highest asthma prevalence of any UK nation at approximately 16.4% (nearly 1 in 6).

Bronchiectasis

- A chronic lung condition in which the airways become permanently widened, scarred, and thickened.
- From Greek: bronkhos (windpipe) + ektasis (widening or stretching).
- Damaged airways cannot clear mucus effectively. Mucus pools, creating a breeding ground for bacteria, leading to recurrent infection and further damage: a vicious cycle.
- Causes include severe childhood respiratory infections (pneumonia, whooping cough, TB), immune deficiency, cystic fibrosis, and autoimmune conditions including RA.
- Symptoms: persistent productive cough, recurrent chest infections, breathlessness, haemoptysis (coughing up blood).
- More common in women than men; 60% of diagnoses are in people over 70.

Occupational Lung Diseases

- Chronic respiratory conditions caused by inhaling dusts, chemicals, or fumes in the workplace. Entirely preventable but usually irreversible once established.
- Types: pneumoconiosis (dusty lung) from mineral dust; coal workers' pneumoconiosis (black lung); silicosis (silica dust); asbestosis (asbestos fibres); occupational asthma; hypersensitivity pneumonitis.
- Common mechanism: inhaled particles trigger chronic inflammation and fibrosis (scarring) that progressively destroys lung tissue.
- Wales carries a disproportionate historical burden of pneumoconiosis from the coal mining industry.
- Occupational exposure accounts for approximately 15% of COPD cases and 9 to 15% of asthma cases in working-age adults.

Pulmonary Hypertension

- Abnormally high blood pressure in the arteries supplying the lungs. Not the same as systemic hypertension (general high blood pressure measured in the arm).
- The right ventricle must pump harder to push blood through narrowed pulmonary arteries. Over time it thickens, then weakens and fails. Right ventricular failure is the primary cause of death in pulmonary hypertension.
- Develops in 38 to 50% of people with severe COPD, making it a direct complication of a very common condition.
- Symptoms: progressive breathlessness on exertion, fatigue, dizziness, peripheral oedema. Often misdiagnosed as asthma or anxiety; average diagnostic delay is 2 to 3 years.
- Illustrates the heart-lung connection: diseased lungs increase vascular resistance, which forces the heart to work harder.

Week 4: Metabolic and Endocrine Disease

Week 4 is organised around three metabolic organs: the liver, the pancreas, and the thyroid gland. Metabolic disease occurs when one of these organs malfunctions. Because they are interconnected, dysfunction in one often affects the others.

The Constrained Energy Model (Pontzer)

- The traditional 'additive' model assumes more exercise simply burns more total calories.
- Research on Hadza hunter-gatherers in Tanzania found their total daily energy expenditure was similar to sedentary Western populations despite very high activity levels.
- Pontzer's constrained energy model proposes the body has a finite energy budget and reallocates between systems rather than simply adding more.
- Relevance: challenges the simple 'calories in, calories out' framing of obesity and metabolic disease.

Type 1 Diabetes

- An autoimmune condition in which the immune system destroys the insulin-producing beta cells in the pancreas.
- The body cannot produce insulin, so blood glucose cannot be regulated.
- Not caused by lifestyle factors. Onset is typically in childhood or young adulthood.

Type 2 Diabetes

- The body's cells become less responsive to insulin. This process is called insulin resistance.
- The pancreas initially compensates by producing more insulin, but capacity reduces over time.
- Strongly associated with modifiable risk factors: obesity, physical inactivity, poor diet.
- Non-modifiable risk factors: age, ethnicity, family history.
- Wales has the highest diabetes prevalence in the UK at approximately 8% of adults.

MASLD (Metabolic Dysfunction-Associated Steatotic Liver Disease)

- Formerly called non-alcoholic fatty liver disease (NAFLD).
- Fat accumulates in the liver in the absence of significant alcohol use, driven by metabolic dysfunction.
- Strongly associated with insulin resistance, obesity, and Type 2 diabetes: it is the hepatic manifestation of metabolic syndrome.
- Can progress from simple steatosis (fat accumulation) to steatohepatitis (inflammation), fibrosis, cirrhosis, and liver failure.
- Wales: MASLD incidence increased tenfold in 20 years, strongly tied to socioeconomic deprivation.

Hashimoto's Thyroiditis (Hypothyroidism)

- An autoimmune condition in which the immune system attacks the thyroid gland, progressively destroying it.
- Results in underactivity of the thyroid (hypothyroidism): the thyroid does not produce enough hormone.
- Because thyroid hormones control metabolic rate, everything slows down when levels are insufficient.
- Symptoms: fatigue, weight gain, cold intolerance, depression, brain fog, dry skin, bradycardia.
- Women are 5 to 10 times more likely to be affected than men. Symptoms frequently dismissed as stress, menopause, or normal ageing before thyroid function is tested.
- Diagnosed by elevated TSH with low T4. Treated with daily levothyroxine for life.

Graves' Disease (Hyperthyroidism)

- An autoimmune condition in which the immune system produces antibodies that continuously stimulate the thyroid gland to overproduce hormone.
- Results in overactivity of the thyroid (hyperthyroidism): everything speeds up.
- Contrast with Hashimoto's: both are autoimmune, both target the thyroid, but the outcomes are opposite. Hashimoto's destroys; Graves' overstimulates.
- Symptoms: weight loss despite increased appetite, heat intolerance, tachycardia (fast heart rate), palpitations, tremor, anxiety, insomnia, diarrhoea.
- Graves' ophthalmopathy (bulging eyes) is unique to Graves' disease, not seen in other causes of hyperthyroidism.

- Symptoms closely mimic anxiety disorders; women are frequently misdiagnosed with anxiety before thyroid function is tested.
- Women are 5 to 10 times more likely to be affected than men.

Week 5: Musculoskeletal Disease

The Physical Activity Paradox

- Physical activity is the single most effective protector against musculoskeletal decline across the lifespan: it builds bone density, maintains muscle mass, protects cartilage, and supports joint stability.
- At the same time, physical activity is a source of MSK injury and degeneration: repetitive loading, impact, and occupational wear all increase OA and back pain risk.
- The dose matters: moderate, varied activity is protective. Extreme or repetitive loading can be damaging.
- For professional practice: prescribing physical activity means understanding both its protective and its potentially damaging potential.

Rheumatoid Arthritis (RA)

- An autoimmune condition in which the immune system attacks the synovial membrane lining the joints.
- Causes pain, swelling, and progressive joint damage.
- A systemic condition: also affects other organs, including the cardiovascular system, through chronic systemic inflammation.
- Modifiable risk factors: smoking is the strongest environmental trigger.
- Non-modifiable: women are 2 to 3 times more likely to be affected; family history.

Osteoarthritis (OA)

- A degenerative joint condition caused by the progressive breakdown of cartilage.
- Not autoimmune. Caused by wear and tear, often worsened by obesity and previous injury.
- As cartilage is lost, bones begin to rub together, causing pain and stiffness.
- Most common in weight-bearing joints: hips, knees.

Osteoporosis

- Bones lose density and become fragile, increasing the risk of fractures from minor falls or everyday activity.
- Described as a 'silent disease' because there are typically no symptoms until a fracture occurs.

- Risk factors: age, female sex, low oestrogen post-menopause, low calcium and vitamin D, physical inactivity, smoking.
- Affects both men and women, though disproportionately affects post-menopausal women. Risk in men is sometimes overlooked.

Sarcopenia

- Progressive, age-related loss of skeletal muscle mass, strength, and function.
- Not the same as osteoporosis (bone loss) or osteoarthritis (joint degeneration).
- Increases risk of falls, frailty, and loss of independence.
- Managed through resistance exercise and adequate protein intake (1.0 to 1.2 g/kg body weight per day).

Chronic Low Back Pain (CLBP)

- Persistent or recurrent pain in the lumbar region lasting 12 weeks or more.
- Importantly, CLBP is a symptom complex, not a structural diagnosis. In 85 to 90% of cases, no specific anatomical cause can be identified ('non-specific low back pain').
- Single leading cause of disability worldwide, affecting 570 million people globally (WHO).
- Chronicity is driven by psychosocial factors ('yellow flags') as much as by structural ones: fear-avoidance beliefs, catastrophising, low mood, work dissatisfaction, and social isolation.
- The biopsychosocial model is the appropriate framework: biological, psychological, and social factors all interact.
- The fear-avoidance cycle: pain leads to catastrophic thinking, which leads to avoiding movement, which leads to deconditioning, which increases pain and disability.
- Key treatment principle: graded exposure to movement, not rest. Exercise is the single most effective intervention. NICE NG59 does not recommend imaging for non-specific CLBP.
- Red flags requiring urgent referral: cauda equina syndrome (loss of bladder/bowel control, saddle anaesthesia), unexplained weight loss or fever, progressive neurological deficit.

Sarcopenic Obesity

- The co-existence of excess adiposity (high fat mass) AND low skeletal muscle mass and function.

- Defined as a distinct clinical condition by the 2022 ESPEN/EASO Consensus: not simply obesity plus sarcopenia happening to occur together.
- BMI alone cannot identify sarcopenic obesity. A person can be overweight by BMI while having dangerously low muscle mass hidden beneath excess fat.
- Shared mechanisms drive both muscle loss and fat gain: insulin resistance, chronic low-grade inflammation from excess adipose tissue, hormonal decline, and physical inactivity.
- The vicious cycle: muscle loss reduces mobility, reduced mobility promotes fat gain, increased fat increases inflammation, increased inflammation accelerates muscle loss.
- Outcomes are synergistically worse than either condition alone: nearly 3 times the risk of disability compared to people with neither condition.
- Treatment requires resistance training alongside adequate protein intake and moderate caloric restriction. Weight loss diets without resistance training cause muscle loss alongside fat loss and can worsen body composition.

Week 6: Neurological Disease

Multiple Sclerosis (MS)

- An autoimmune condition in which the immune system attacks the myelin sheath surrounding nerve fibres in the central nervous system.
- Myelin acts as insulation around nerve fibres, enabling signals to travel quickly and accurately. When damaged, signals slow down, become distorted, or fail entirely.
- Symptoms vary according to which nerves are affected: fatigue, weakness, vision problems, coordination difficulties.
- Relapsing-remitting MS is the most common form. Women are 2.5 times more likely to be diagnosed than men.

Dementia

- Not a single disease but a syndrome: a cluster of symptoms involving gradual decline in memory, thinking, and the ability to carry out daily activities.
- Can be caused by several different diseases: Alzheimer's disease (most common), vascular dementia, Lewy body dementia.
- Not a normal part of ageing, although age is the largest risk factor.
- Vascular dementia shares risk factors with cardiovascular disease: hypertension, diabetes, smoking. Stroke causes 15 to 20% of all dementia cases.

Parkinson's Disease

- A progressive neurological condition caused by loss of dopamine-producing neurons in the brain.
- Primary motor symptoms: tremor, rigidity, and slowness of movement (bradykinesia).
- Non-motor symptoms are also present: depression, sleep disturbance, cognitive changes.

Migraine

- A primary headache disorder characterised by recurrent attacks of moderate to severe headache, often with associated neurological features.
- Primary means the headache is the disease itself, not a symptom of an underlying condition.

- Affects approximately 10 million people in the UK. The second most disabling neurological condition worldwide and the leading cause of disability in people aged 15 to 49.
- Women are 3 times more likely to have migraine than men after puberty; hormonal fluctuations across the menstrual cycle are the most common trigger in women.
- Four phases: prodrome (mood and energy changes hours before), aura (reversible neurological symptoms in approximately 25 to 30% of attacks), headache (4 to 72 hours, typically one-sided, throbbing, with nausea and light/sound sensitivity), and postdrome (fatigue and cognitive slowing after the headache resolves).
- Important clinical distinction: aura symptoms spread gradually over minutes. In stroke or TIA, neurological symptoms appear suddenly and all at once. This matters for safe clinical assessment.
- Modifiable factors: regular moderate aerobic exercise is a first-line preventive recommendation. Analgesic overuse (more than 10 to 15 days per month) can transform episodic migraine into chronic daily headache.

Other neurological conditions covered briefly

- Motor Neurone Disease (MND): progressive degeneration of the motor neurons that control voluntary movement.
- Fibromyalgia: widespread musculoskeletal pain accompanied by fatigue, sleep disturbance, and cognitive difficulties. Central sensitisation is a key mechanism.
- Epilepsy: a neurological condition characterised by recurrent seizures caused by abnormal electrical activity in the brain.
- Peripheral neuropathy: damage to the peripheral nerves, often as a complication of diabetes or other systemic conditions.

Week 7: Cancer

What is cancer?

- Cancer develops when cells accumulate damage to their DNA and begin to grow and divide uncontrollably.
- Mutations can be caused by lifestyle factors, environmental exposures, inherited genetic changes, or random copying errors during cell division.
- Not the same as autoimmune disease (the immune system attacking healthy cells).
- Cancer is classified as a chronic condition: survivorship is a long-term condition requiring ongoing management. Approximately 3 million people in the UK live with or beyond a cancer diagnosis.

Lung cancer

- Most strongly associated with smoking (approximately 70% of cases). Stopping before age 30 avoids more than 90% of the tobacco-attributable risk; stopping by age 40 reduces risk by around 80%.
- The leading cause of cancer death in Wales and the UK.
- Two main types: non-small cell lung cancer (approximately 85%) and small cell lung cancer.
- Often diagnosed at a late stage because early symptoms are subtle.

Breast cancer

- Most common cancer by new cases in the UK (approximately 59,500 per year).
- Risk factors: age, family history (BRCA1/BRCA2 mutations), hormonal factors, obesity, alcohol. Oestrogen stimulates breast cell division; accumulated exposure increases risk.
- Predominantly affects women, but also occurs in men.
- Breast Test Wales provides screening from age 50. Ten-year survival in the UK exceeds 75%.

Colorectal cancer

- Cancer of the colon or rectum. Third most common cancer in the UK.
- Risk factors: age, diet high in processed meat and low in fibre, obesity, physical inactivity, family history, and inflammatory bowel disease (IBD).
- Bowel Screening Wales offers screening to individuals aged 50 to 74.

Prostate cancer

- Most common cancer in men in the UK, with over 62,000 new cases per year.
- Non-modifiable risk factors: age (risk doubles every 10 years after 50), ethnicity (Black men have twice the risk), family history.
- Testosterone drives continuous prostate cell division; decades of stimulation mean accumulated mutation risk.
- Often has no symptoms in early stages. Many cases detected incidentally via PSA testing.
- Ten-year survival exceeds 80%.

Week 8: Mental Health Conditions

Week 8 uses the biopsychosocial model as its organising framework: biological factors (genetics, brain chemistry, physical health), psychological factors (thoughts, emotions, past experiences), and social factors (relationships, poverty, culture, environment) all interact. No single factor explains any mental disorder.

Depression

- A mood disorder characterised by persistent low mood, loss of interest or pleasure in activities (anhedonia), fatigue, and changes in sleep and appetite.
- Diagnosed when five or more symptoms are present for at least two weeks, including low mood or anhedonia (DSM-5-TR).
- When a person with a chronic physical condition also has depression, it makes it harder to follow treatment plans and leads to worse health outcomes overall.
- The relationship is bidirectional: depression worsens physical health outcomes, and poor physical health increases depression risk.
- Group exercise is a NICE first-line treatment for less severe depression. Both aerobic exercise and resistance training have an evidence base.
- Men are half as likely as women to receive treatment for depression but die by suicide at three times the rate. This gap reflects underdiagnosis and undertreatment, not genuine lower prevalence.

Anxiety disorders

- Characterised by persistent, excessive worry and fear that interferes with daily life.
- ICD-11: excessive fear and anxiety with related behavioural disturbances.
- Subtypes include generalised anxiety disorder (GAD), panic disorder, social anxiety, and specific phobias.
- Key distinction: anxiety as a normal human experience versus anxiety disorder, which is excessive, persistent, difficult to control, and causes significant functional impairment.
- Strongly linked with chronic physical conditions: having a long-term condition significantly increases anxiety risk.

Eating disorders

- Serious mental health conditions affecting a person's relationship with food, eating, and body image. Have the highest mortality rate of any psychiatric disorder.

- Not simply about food: complex conditions with biological, psychological, and social roots.
- Relevant to all three programmes: exercise science, sports therapy, and nutrition students will encounter eating disorders in client populations.

The four subtypes covered in this module:

- **Anorexia nervosa (AN)**: restriction of energy intake, intense fear of weight gain, distorted body image. Associated with compulsive exercise and severe medical complications including bone loss and cardiac risk.
- **Bulimia nervosa (BN)**: recurrent binge eating followed by compensatory behaviour (purging), at least weekly for three months. Body weight is typically within normal range, which is why it is often invisible.
- **Binge eating disorder (BED)**: recurrent episodes of eating a large amount of food in a short time with a sense of loss of control, without compensatory behaviour. Closest to gender parity of all eating disorders (approximately 40% of sufferers are male).
- **ARFID (Avoidant/Restrictive Food Intake Disorder)**: limited food intake based on sensory characteristics, fear of aversive consequences, or low interest in food, without body image disturbance. Not driven by a desire to change body weight or shape.
- Around 25% of people with eating disorders in the UK are male. Male eating disorders are significantly underdiagnosed.
- Compulsive exercise is a clinical feature of AN and BN, not simply a risk factor. Exercise prescription in active eating disorders requires specialist clinical oversight.

Week 9: Obesity and Multi-morbidity

Week 9 covers obesity as a chronic, relapsing disease with identifiable pathophysiology and systemic consequences across multiple body systems. It also addresses multi-morbidity, clinical assessment, management pathways, and the psychosocial burden of weight stigma.

Defining the Disease: Adiposity-Based Chronic Disease

- Obesity is a chronic, complex, and relapsing condition involving dysfunctional adipose tissue. It is not a lifestyle choice or a moral failing. It is a disease with identifiable pathophysiology, systemic consequences, and evidence-based treatment pathways.
- The ABCD framework (Adiposity-Based Chronic Disease) distinguishes between fat mass disease (mechanical consequences: joint loading, respiratory compression, reduced mobility) and sick fat disease (metabolic consequences: chronic low-grade inflammation, insulin resistance, endocrine dysfunction).
- A patient can have predominantly mechanical complications, predominantly metabolic complications, or both. Understanding which pattern is present shapes the intervention approach.
- The WHO formally reclassified obesity as a chronic disease in 2025, recognising that it requires ongoing management in the same way as other chronic conditions covered in this module.

Adipose Tissue as an Active Endocrine Organ

- Adipose tissue is not passive storage. It is an active endocrine organ that, when dysfunctional, secretes inflammatory cytokines that circulate systemically and directly cause insulin resistance.
- The expandability hypothesis: individuals have a genetically determined limit to which their subcutaneous adipose tissue can safely expand. Within that limit, fat storage is metabolically benign. Beyond it, adipocytes become hypertrophic, secrete inflammatory adipokines, and excess energy spills over into organs not designed for fat storage (liver, skeletal muscle, viscera). This is called ectopic fat deposition.
- This explains why some people develop metabolic complications at relatively modest adiposity while others remain metabolically healthy at higher BMIs: the threshold differs between individuals.

Appetite Regulation and Leptin Resistance

- The hypothalamus acts as the central processing hub for energy balance. It receives hunger signals from ghrelin (from the gut) and satiety signals from leptin (from adipose tissue).
- In obesity, chronic excess leptin from expanded adipose tissue overloads the hypothalamic receptors. The brain develops cellular resistance and fails to receive the satiety signal. Despite abundant energy stores, the brain perceives starvation.
- This is why willpower-based approaches to weight loss are rarely sustainable. The person's hypothalamus is signalling starvation, driving relentless hunger and energy conservation. The biological drive to eat is not a failure of self-control.

Genetic Architecture

- Monogenic obesity is rare (approximately 5% of severe early-onset cases), involving a single mutation in genes such as LEPR or MC4R. High penetrance: the mutation almost certainly produces severe obesity regardless of environment.
- Polygenic obesity accounts for the vast majority of cases. Hundreds of genetic variants (e.g. FTO gene) each have a small individual effect but collectively create a genetic risk score. Low penetrance individually: environment is the key trigger.
- Epigenetics bridges the gap. Changes in genetic expression acquired through environmental interaction modify how polygenic variants are expressed. A high genetic risk score in a non-obesogenic environment may never manifest as obesity.

The Obesogenic Environment

- The obesity epidemic results from the collision between ancient genetic predispositions to conserve energy (adaptive in environments of food scarcity) and a modern environment that promotes passive over-consumption.
- Food swamps: areas with an overabundance of low-cost, ultra-processed, high fat, salt, and sugar foods. Food deserts: areas with inadequate access to nutrient-dense options at affordable prices.
- The built environment compounds this: car-reliant infrastructure, lack of safe green spaces, and urban design that reduces opportunities for physical activity.
- This is the Dahlgren and Whitehead model in action. Individual biology is shaped by social and environmental determinants that the individual cannot control. Over 60% of Welsh adults experience overweight or obesity. In the

most deprived areas of Wales, childhood obesity is 28.5% compared to 22.2% in the least deprived areas.

BMI: Utility and Limitations

- BMI categories: underweight (below 18.5), healthy weight (18.5 to 24.9), overweight (25 to 29.9), Obesity Class I/II (30 to 39.9), Obesity Class III (40 and above).
- BMI is useful as a population screening tool but is an indirect estimate. It does not discriminate between fat mass and fat-free mass, and it cannot detect ectopic fat (fat deposited in organs not designed for fat storage).
- The All-Wales Weight Management Pathway states that BMI is never the sole indicator for intervention. Clinical assessment must consider fat distribution, metabolic markers, and functional status alongside BMI.

The Non-Linear Trajectory of Risk

- Risk does not increase linearly with BMI. It accelerates. The jump from healthy weight to overweight is modest. The jump from Obesity Class II to Class III is steep and exponential (the J-curve).
- This has direct implications for intervention timing. Preventing someone from moving from BMI 32 to 40 may be more impactful than many other clinical interventions because of that exponential acceleration.

Obesity as a Cross-Cutting, Multi-morbidity Risk Factor

- Obesity elevates risk across virtually every organ system simultaneously. It is best understood as a multi-morbidity hub, not a single-system condition.
- **Metabolic:** Type 2 diabetes risk is over fourteen times higher in Obesity Class III compared to healthy weight. This is the steepest hazard ratio of any comorbidity and is driven by dysfunctional adipose tissue causing insulin resistance.
- **Cardiovascular:** hypertension, heart failure (driven by mechanical volume overload as the heart works harder to perfuse a larger body mass), and coronary artery disease (driven by metabolic atherosclerosis). Different mechanisms produce different risk gradients.
- **Musculoskeletal:** osteoarthritis risk increases through pure mechanical loading on weight-bearing joints. Sleep apnoea risk is over twenty times higher at Class III obesity; it disrupts hormonal balance and drives further weight gain, creating a vicious cycle.
- **Oncological (Cancer):** moderate but significant risk increases for endometrial cancer (driven by oestrogen excess from adipose tissue

aromatisation), kidney cancer, bowel cancer, and post-menopausal breast cancer.

- **Psychological:** individuals living with obesity have a 55% increased odds of developing clinical depression. This is driven by both endocrine mediators (hypercortisolaemia) and psychological distress from weight stigma.
- Critically, these risks compound. Someone with Class III obesity is not at higher risk for just one condition. They are at elevated risk for all of these simultaneously, resulting in severe multi-morbidity.

Psychosocial Burden and Weight Stigma

- 40.8% of the public still falsely attribute obesity to a lack of self-control. This stigma perpetuates discrimination, healthcare avoidance, and worsened outcomes.
- The biological stress loop: weight stigma causes psychological distress, which elevates cortisol, which promotes visceral fat deposition and emotional eating, which increases weight, which intensifies stigma. The loop perpetuates itself.
- Healthcare avoidance compounds the problem. Patients who have experienced bias from healthcare professionals delay seeking medical advice, allowing conditions that could have been caught early to progress.
- Stigma is gendered: women living with obesity experience significantly greater societal prejudice and reduced employment and earnings compared to men living with obesity.

Management: The All-Wales Weight Management Pathway

- The All-Wales Weight Management Pathway has four levels. Level 1: community-based prevention and early intervention. Level 2: community and primary care weight management (e.g. Foodwise for Life, the National Exercise Referral Scheme). Level 3: specialist multi-disciplinary team services with biopsychosocial assessment and trauma-informed care. Level 4: specialist medical and surgical services (bariatric surgery).
- The 5As clinical communication framework: Ask (seek permission to discuss weight), Assess (biopsychosocial status, not just weight), Advise (health risks and treatment options), Agree (collaborate on patient-led goals), Assist (connect with resources and referral pathways).
- Pharmacotherapy has evolved from orlistat (lipase inhibitor, modest efficacy) to GLP-1 receptor agonists: liraglutide (Saxenda, daily), semaglutide (Wegovy, weekly), and tirzepatide (Mounjaro, dual GIP/GLP-1 agonist). These agents target appetite regulation directly and demonstrate significant weight reduction and cardiovascular benefits.

- Bariatric surgery (Level 4) is not first-line. Eligibility: BMI 40 or above, or 35 or above with severe comorbidity. Requires mandatory progression through Levels 1 to 3 and psychological fitness assessment. Surgery is part of lifelong management, not an endpoint: post-surgical patients need monitoring for nutritional deficiencies and ongoing support.
- Adverse Childhood Experiences (ACEs) biologically and psychologically drive severe adult obesity. Trauma-informed care, not simple dietary instruction, is required for patients with trauma histories.

Professional Relevance

- For nutrition students: understanding why exercise alone rarely produces sustained weight loss (the constrained energy model from Week 4), the role of dietary change within structured pathways, and the importance of non-judgemental practice.
- For sports therapy students: mechanical loading and joint health are directly affected by adiposity. Sleep apnoea and pain can prevent patients from exercising, creating a compounding spiral that must be addressed before physical intervention can succeed.
- For exercise science students: the National Exercise Referral Scheme (NERS) is a direct professional context. Understanding the biological and psychosocial barriers to physical activity in patients living with obesity is fundamental to ethical, evidence-based practice.

Week 10: Treatment and Lifestyle Management

Week 10 brings together the conditions covered earlier in the module and addresses how they are managed in practice. Management of chronic disease has two complementary arms: pharmacology (medications and clinical interventions) and lifestyle (physical activity, nutrition, sleep, behaviour change). The two work together. Lifestyle interventions are not a soft alternative to medication; they are evidence-based treatments in their own right, and in many conditions they are first-line.

The Two-Arm Framework

Pharmacology and clinical principles: medication regimens and adherence; cardiovascular risk reduction; respiratory symptom management; metabolic control and glucose regulation. Caution: drug interactions and side effects must be monitored.

Lifestyle and exercise interventions: physical activity prescription; dietary modification and nutrition; sleep hygiene and rest; stress management and behavioural change. Clinical consideration: exercise clearance must be individualised, particularly in people with multi-morbidity or on multiple medications.

Cardiovascular Disease and Heart Failure

Pharmacology

- Antihypertensives: ACE inhibitors, ARBs, beta-blockers, diuretics.
- Lipid management and clot prevention: statins, antiplatelets, anticoagulants.
- Heart failure 'Core 4': ACE inhibitor or ARB, beta-blocker, mineralocorticoid receptor antagonist, SGLT2 inhibitor.
- Angina symptom relief: GTN spray.

Lifestyle

- Cardiovascular exercise: 3 to 5 days per week, RPE 11 to 13 (light to moderate), 30 to 60 minutes per session.
- Resistance training: 2 to 3 days per week, 8 to 15 reps at 50 to 70% 1RM, avoiding the Valsalva manoeuvre.
- Diet: Mediterranean or DASH pattern, sodium under 6 g per day, fluid restriction in heart failure.

Red flags and considerations

- Beta-blockers blunt heart rate, so use Rate of Perceived Exertion (RPE) rather than HR for intensity prescription.
- Diuretics require session hydration planning.

- Statins (simvastatin, atorvastatin) interact with grapefruit and grapefruit juice.
- Warfarin requires consistent vitamin K intake; sudden changes in green vegetable consumption can destabilise anticoagulation.

The Airways: COPD and Asthma

Pharmacology

- Relievers: short-acting beta agonists (SABA) such as salbutamol.
- Maintenance: long-acting beta agonists (LABA), long-acting muscarinic antagonists (LAMA), inhaled corticosteroids (ICS).
- COPD advanced: triple therapy or oxygen for chronic hypoxaemia.
- Asthma advanced: biologics for severe eosinophilic variants.

Lifestyle

- COPD cardio: interval focus with a 3:1 rest-to-work ratio, building up to 30 minutes; target RPE 11 to 13.
- Asthma cardio: 3 to 5 days per week, RPE 11 to 15.
- Resistance training: peripheral muscle focus in COPD; endurance focus in asthma.

Red flags and considerations

- Maintain SpO₂ above 88% during COPD activity.
- Asthma preparation: SABA 2 puffs 10 to 15 minutes pre-exercise, plus a 15-minute warm-up.
- Cold or dry air can trigger bronchoconstriction. Pursed-lip breathing helps in COPD.
- Rinse the mouth after using ICS to prevent oral candidiasis.

Diabetes Mellitus: Type 1 versus Type 2

Type 1 Diabetes (autoimmune)

- Pharmacology: basal-bolus insulin or pump; continuous glucose monitoring (CGM) is now standard.
- Lifestyle: daily activity bursts adding up to around 150 minutes per week; resistance training on 2 to 3 days; carbohydrate counting matched to insulin dosing.
- Strict rules: aim for 6 to 10 mmol/L pre-exercise. Below 6, take 15 to 30 g of fast-acting carbohydrate. Blood ketones above 1.5 means defer exercise. Do not inject insulin into actively exercising muscles.

Type 2 Diabetes (metabolic)

- Pharmacology: metformin first-line; SGLT2 inhibitors and GLP-1 agonists; sulfonylureas.
- Lifestyle: resistance training is critical because muscle acts as a glucose buffer; RPE 11 to 13. Structured weight loss has been shown to produce remission (DiRECT trial). Reduce ultra-processed food intake.
- Strict rules: hypoglycaemia risk with sulfonylureas and insulin; metformin should be taken with food; check footwear regularly because peripheral neuropathy raises foot ulcer risk.

Shared red flags

- Always carry fast-acting carbohydrate.
- Heart rate is unreliable as an intensity guide if the person is also on beta-blockers (more common in T2DM).

Obesity Management

Pharmacology

- Traditional: orlistat, the NHS mainstay (lipase inhibitor).
- Modern: GLP-1 agonists (semaglutide) and dual GIP/GLP-1 agonists (tirzepatide), accessed through the specialist All-Wales Pathway.
- Surgical: bariatric surgery for severe disease.

Lifestyle

- Cardiovascular exercise: 5 to 7 days per week, RPE 11 to 13, 40 to 60 minutes; low-impact options preferred to protect joints.
- Resistance training: 2 to 3 days per week, full body.
- Diet: structured energy deficit using a Mediterranean or DASH pattern; cut ultra-processed foods.

Cautions

- The GLP-1 rule: appetite suppression risks sarcopenia. Protein intake must be protected at 1.2 to 1.6 g per kg per day to preserve muscle mass.
- Screen for sleep apnoea if BMI is above 35 with daytime sleepiness.
- Psychosocial: weight stigma is clinically significant. Adjust language and the physical environment of the consultation room.

Structural Degradation: Osteoporosis versus Sarcopenia

Osteoporosis (bone)

- Pharmacology: bisphosphonates (alendronate) first-line; calcium and vitamin D adjuncts.
- Lifestyle: weight-bearing rhythmic activity, progressive loading, graded impact.

Sarcopenia (muscle)

- Pharmacology: no licensed pharmacotherapy.
- Lifestyle: resistance training is non-negotiable on 3 days per week; balance training twice per week. Protein focus 1.2 to 1.5 g per kg per day, spread across meals (around 30 g per meal) with a leucine-rich evening meal.

Shared red flags

- Bisphosphonates require plain water, an empty stomach, and remaining upright for 30 minutes after the dose.
- Avoid spinal flexion under load in severe disease.
- In sarcopenic obesity, prioritise resistance training over caloric restriction.
- Watch for cognitive decline, which raises falls risk.

Joint Pathology: Rheumatoid versus Osteoarthritis

Rheumatoid arthritis (autoimmune, systemic)

- Pharmacology: DMARDs (methotrexate, sulfasalazine), then biologics; short-term steroids and NSAIDs for flares.
- Lifestyle: low-impact cardiovascular exercise; isometric work during joint inflammation; daily mobility.
- Cautions: immunosuppression risk; pace activity around unpredictable flares; minimise alcohol with methotrexate.

Osteoarthritis (mechanical, localised)

- Pharmacology: paracetamol; topical NSAIDs; oral NSAID short courses.
- Lifestyle: strengthen the surrounding musculature (e.g. quadriceps for knee OA); aquatic therapy.
- Cautions: avoid deeply flexed loaded knee positions in early rehabilitation; modest weight loss dramatically reduces joint load.

Shared red flags

- NSAIDs must be taken with food.
- Sudden, severe new joint symptoms warrant immediate GP referral.

Neurological: Dementia (Alzheimer's and Vascular)

- Pharmacology: cholinesterase inhibitors (donepezil). Vascular dementia requires aggressive cardiovascular risk management. Antipsychotics are avoided because of a mortality signal in this population.
- Lifestyle: group-based, meaningful activity; familiar settings; simple compound movements.
- Cautions: hydration and appetite oversight is the practitioner's responsibility because clients may forget. Use simple verbal cues. Watch for bradycardia with cholinesterase inhibitors.

Cancer

- Pharmacology: surgery, radiotherapy, and systemic therapies (cytotoxic chemotherapy, targeted therapies, endocrine treatments such as tamoxifen).
- Lifestyle: prehabilitation in the 4 to 6 weeks before treatment improves outcomes. Resistance training during chemotherapy preserves lean mass.
- Cautions: anthracycline cardiotoxicity means strictly RPE-led intensity. Platinum chemotherapy is associated with peripheral neuropathy and balance risks. Strict food hygiene during chemotherapy: no raw meat or unpasteurised dairy.

General Principles for Professional Practice

- Exercise prescription must always be individualised, particularly when multi-morbidity is present.
- Drug-exercise interactions matter. The most common example: beta-blockers blunt heart rate, so RPE rather than HR is the appropriate intensity guide.
- Protein adequacy is a thread across multiple condition areas: sarcopenia, sarcopenic obesity, cancer, and obesity pharmacotherapy with GLP-1 agonists.
- Adherence to medication is part of management. Knowing the practical rules (take with food; consistent vitamin K; rinse mouth after ICS) helps clients stick with treatment and avoid avoidable side effects.
- Recognise scope of practice. Lifestyle prescription is your domain; medication management is not. Working in collaboration with the wider healthcare team is part of safe practice.

Cross-Cutting Themes

The exam rewards students who can draw connections across the module. These themes run throughout all weeks and are directly relevant to Section B.

The autoimmune thread

The same underlying mechanism (the immune system attacking healthy tissue) produces very different diseases depending on the target tissue:

- Type 1 diabetes (Week 4): beta cells in the pancreas are destroyed. The body cannot produce insulin.
- Hashimoto's thyroiditis (Week 4): the thyroid gland is attacked. The thyroid becomes underactive.
- Graves' disease (Week 4): the thyroid is stimulated continuously by antibodies. The thyroid becomes overactive.
- Rheumatoid arthritis (Week 5): the synovial membrane of joints is attacked. Joints become painful and damaged.
- Multiple sclerosis (Week 6): the myelin sheath around nerve fibres in the CNS is attacked. Nerve signal transmission is disrupted.

For Section B Question 4, you need to identify two autoimmune conditions from different weeks and explain what each attacks and what effect this has. Higher marks are awarded for noting that the fundamental process is the same across all autoimmune conditions, but the disease differs because the target tissue differs.

Comorbidity

Conditions rarely exist in isolation. These are the key comorbidity links taught in this module:

- Rheumatoid arthritis increases cardiovascular disease risk through chronic systemic inflammation damaging blood vessel walls over time.
- Type 2 diabetes increases cardiovascular disease risk through high blood glucose damaging arterial walls and accelerating atherosclerosis.
- Obesity increases risk of Type 2 diabetes (through insulin resistance), osteoarthritis (through mechanical load on joints), and cardiovascular disease.
- Depression worsens outcomes for almost every chronic condition by reducing treatment adherence.
- COPD leads to pulmonary hypertension in 38 to 50% of severe cases.
- Stroke causes 15 to 20% of all dementia cases (vascular dementia).

Treatment principles across conditions

Week 10 brings out a set of principles that apply across many conditions:

- Pharmacology and lifestyle work together. Lifestyle interventions are first-line in some conditions (mild hypertension, Type 2 diabetes) and adjuncts in others (heart failure, RA), but they are evidence-based treatments throughout, not soft alternatives.
- Resistance training appears as a core intervention across many conditions: sarcopenia, osteoporosis (via Wolff's Law), Type 2 diabetes (muscle as glucose buffer), obesity (preserving lean mass), and cancer (preserving function during treatment).
- Drug-exercise interactions matter. Beta-blockers blunt heart rate, so RPE replaces HR as the intensity guide; diuretics affect hydration; statins interact with grapefruit; warfarin requires consistent vitamin K intake.
- Protein adequacy is a recurring concern: sarcopenia and sarcopenic obesity, GLP-1 agonist therapy in obesity (because appetite suppression risks muscle loss), and cancer treatment.
- Scope of practice is part of safe management. Lifestyle prescription sits within the domain of nutrition, sports therapy, and exercise science professionals; medication prescribing does not. Recognising when to refer is part of being a safe practitioner.

Social determinants of health

Health is shaped by the conditions in which people are born, grow, live, work, and age. The Dahlgren and Whitehead rainbow model is the framework used in this module.

- Inner layers: age, sex, genetic factors (non-modifiable).
- Middle layers: individual lifestyle behaviours.
- Outer layers: social networks, living and working conditions, socioeconomic and environmental factors.
- Wales has significant health deprivation in some communities, producing higher chronic disease prevalence, poorer outcomes, and persistent health inequalities.

Gender bias in diagnosis

Several conditions in this module are more commonly recognised in one sex, or present differently between sexes, affecting clinical recognition:

- **Cardiovascular disease:** historically framed as a male condition. Women's symptoms can differ and are more likely to be attributed to other causes.
- **Thyroid conditions (both Hashimoto's and Graves')**: far more common in women, but symptoms are frequently attributed to menopause, stress, or anxiety before thyroid function is tested.
- **Rheumatoid arthritis:** women are 2 to 3 times more likely to be affected.
- **Osteoporosis:** disproportionately affects post-menopausal women but risk in men is often overlooked.
- **Eating disorders:** approximately 25% of those affected are male, but male eating disorders are significantly underdiagnosed because presentations differ and men are less likely to seek help.
- **Depression:** men are half as likely to receive treatment but die by suicide at three times the rate.

Section B: How to Answer Each Question

You answer TWO out of five questions. Each is worth 15 marks. Read all five before choosing. Pick the two where you can write in the most depth, not the two that look shortest.

For Questions 1, 2, 3, and 5, you may choose any condition covered in this module from Week 1 through to Week 10. This includes obesity, which can be used as a standalone condition (for example, explaining its pathophysiology, its modifiable and non-modifiable risk factors, or its management through lifestyle and clinical pathways) or as a cross-cutting risk factor linking to conditions in other body systems. You are not limited to the conditions used in the worked examples below.

Q1: Condition, cause, and what happens in the body

(a) 8 marks: Explain what the condition is and what causes it.

- Name the condition clearly and describe what part of the body is affected.
- Explain the cause in detail. A single word earns no marks. 'Autoimmune' alone is not enough. Explain what the immune system is doing and what the consequence is.

(b) 7 marks: Explain what happens in the body as a result.

- Describe the disease process and connect it to symptoms a person would experience.

Q2: Modifiable and non-modifiable risk factors

(a) 8 marks: One modifiable risk factor.

- Name the condition and the risk factor. Explain the mechanism: how does this risk factor increase the risk?

(b) 7 marks: One non-modifiable risk factor.

- Explain the mechanism in the same way. Do not just name the factor.
- Example: age increases osteoporosis risk because bone density naturally declines from the mid-30s and accelerates after menopause in women.

Q3: Comorbidity and quality of life

(a) 8 marks: How one condition increases risk of another in a different body system.

- Name both conditions. Explain the mechanism linking them. This is where most marks are earned.

(b) 7 marks: How multi-morbidity affects quality of life.

- Cover more than one dimension. Physical alone is not sufficient for full marks.
- Physical: pain, fatigue, reduced mobility, reduced independence.
- Psychological: anxiety, depression, frustration, loss of identity.
- Social: difficulty maintaining relationships or employment, isolation, financial strain.

Q4: Autoimmune diseases

(a) 8 marks: One autoimmune condition.

- Identify the condition as autoimmune, name the correct target tissue, and explain what effect attacking that tissue has on the body.

(b) 7 marks: A second autoimmune condition from a different week of the module.

- Weeks with autoimmune content: Week 4 (Type 1 diabetes, Hashimoto's, Graves'), Week 5 (RA), Week 6 (MS).
- For higher marks: note that the fundamental process is the same across all autoimmune conditions, but the disease differs because a different tissue is targeted.

Q5: Management and professional relevance

(a) 8 marks: How the condition can be managed through lifestyle, nutrition, or exercise.

- Be specific. Name the intervention and explain why it works.
- Week 10 gives you condition-specific lifestyle prescriptions to draw on. (b) 7 marks: Why this matters for your professional practice.

- Be specific to your programme and role. Vague answers such as 'it is important to understand diseases' receive zero marks for this component.
- Example: as a Sports Therapist, understanding the physical activity paradox in osteoarthritis is essential for prescribing exercise that reduces pain without accelerating joint damage.
- For Nutrition students: in the UK, Nutritionists work with healthy populations and provide general nutritional advice. Therapeutic management of clinical conditions sits with dietitians. Working within scope of practice and signposting appropriately are part of safe practice.

Revision Checklist

Use this to check your preparation before the exam.

Terminology

- I can define: chronic disease, prevalence, incidence, aetiology, comorbidity, autoimmune disease, modifiable and non-modifiable risk factors.
- I understand the three types on the immune spectrum and can give an example of each.

Conditions

- Cardiovascular (Week 2): CHD, heart failure, hypertension, stroke.
- Pulmonary (Week 3): COPD, asthma (and the reversibility distinction); bronchiectasis, occupational lung diseases, pulmonary hypertension.
- Metabolic (Week 4): T1 diabetes, T2 diabetes (insulin resistance), MASLD, Hashimoto's thyroiditis, Graves' disease.
- Musculoskeletal (Week 5): RA (autoimmune), OA (degenerative), osteoporosis (silent disease), sarcopenia, CLBP (biopsychosocial model, fear-avoidance), sarcopenic obesity.
- Neurological (Week 6): MS (myelin sheath), dementia (syndrome not single disease), Parkinson's (dopamine loss), migraine.
- Cancer (Week 7): uncontrolled cell division from DNA damage; lung, breast, colorectal, prostate. Cancer as a survivorship condition.
- Mental health (Week 8): depression (bidirectional with physical health), anxiety disorders, eating disorders (AN, BN, BED, ARFID).
- Obesity and multi-morbidity (Week 9): obesity as a chronic relapsing disease (ABCD framework); adipose tissue as an endocrine organ; leptin resistance; the obesogenic environment; BMI utility and limitations; obesity as a multi-morbidity hub (metabolic, cardiovascular, musculoskeletal, oncological, psychological); psychosocial burden and weight stigma; the All-Wales Weight Management Pathway (Levels 1 to 4); the 5As framework.
- Treatment and lifestyle management (Week 10): the two-arm framework (pharmacology + lifestyle); condition-specific management for cardiovascular disease and heart failure, COPD and asthma, Type 1 and Type 2 diabetes, obesity, osteoporosis and sarcopenia, RA and OA, dementia, and cancer.

Cross-cutting themes

- I can name all five autoimmune conditions from the module, state what each attacks, and explain the effect.
- I can give at least three comorbidity links and explain the mechanism for each.
- I can discuss quality of life impact across physical, psychological, and social dimensions.
- I understand the physical activity paradox and its implications for professional practice.
- I can explain gender bias in diagnosis for at least three conditions.
- I can explain why obesity is described as a cross-cutting risk factor and give specific examples of how it increases risk across multiple body systems through different mechanisms (insulin resistance, mechanical loading, oestrogen excess, chronic inflammation).

Treatment and lifestyle management (Week 10)

- I can describe the two arms of management: pharmacology and lifestyle, and how they work together.
- I can give the lifestyle prescription parameters for at least three conditions (e.g. cardio days per week, RPE range, resistance training frequency, dietary pattern).
- I can name the main pharmacological classes used in cardiovascular disease, COPD/asthma, T2 diabetes, and obesity.
- I can give at least three drug-exercise interactions or red flags (e.g. beta-blockers blunt HR, statins and grapefruit, ICS and oral candidiasis, NSAIDs with food).
- I can explain why protein adequacy matters across sarcopenia, sarcopenic obesity, GLP-1 therapy in obesity, and cancer treatment.
- I understand the limits of my scope of practice and when to refer.

Section B approach

- I know which two question types suit my knowledge best.
- I know that explaining the mechanism earns more marks than listing facts.
- I know how the marks are allocated in each part: 2 marks for identification and 6 marks for explanation in part (a); 1 mark for identification and 6 marks for explanation in part (b).