

BMSH4302

Introduction to Chronic Disease

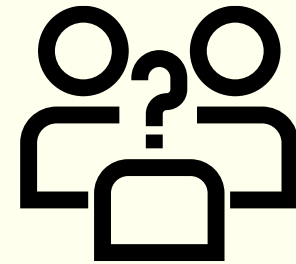
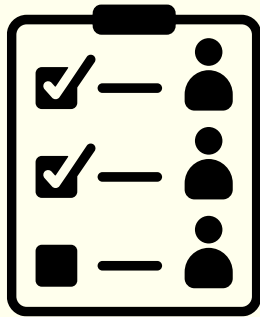
Level 4



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BMSH4302 Introduction to Chronic Disease

Housekeeping:





Metabolic Diseases Recap

Musculoskeletal (MSK) Diseases

What does Musculoskeletal mean?

MUSCULUS = Latin 'muscle' plus

SKELETAL = 'of the skeleton'

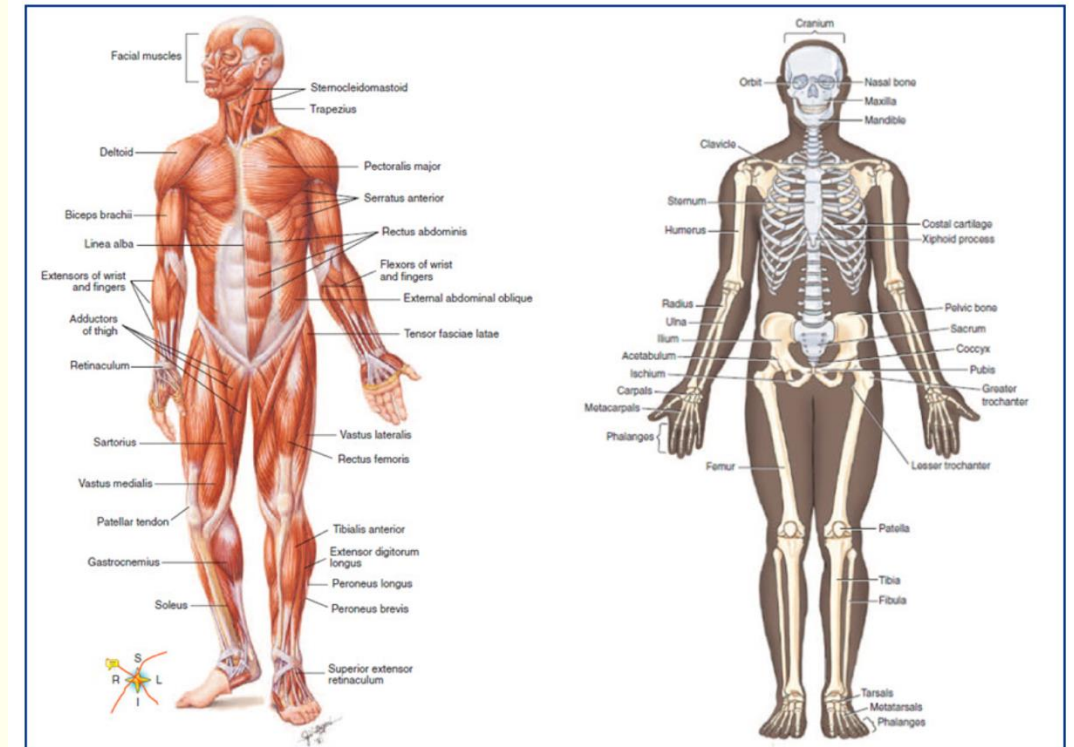
Musculoskeletal health refers to the performance of the locomotor system, comprising intact muscles, bones, joints and adjacent connective tissues (WHO)

Musculoskeletal System

The musculoskeletal system consists principally of:

- Bone
- Muscle
- Tendon
- Ligament
- Articular cartilage tissue
- Joints

These are arranged throughout the body to provide internal support and allow motion to occur



Musculoskeletal System

Bones: provide the rigid framework, protect organs, and store minerals

Muscles: fibrous tissues that contract to produce movement (skeletal, cardiac, and smooth types)

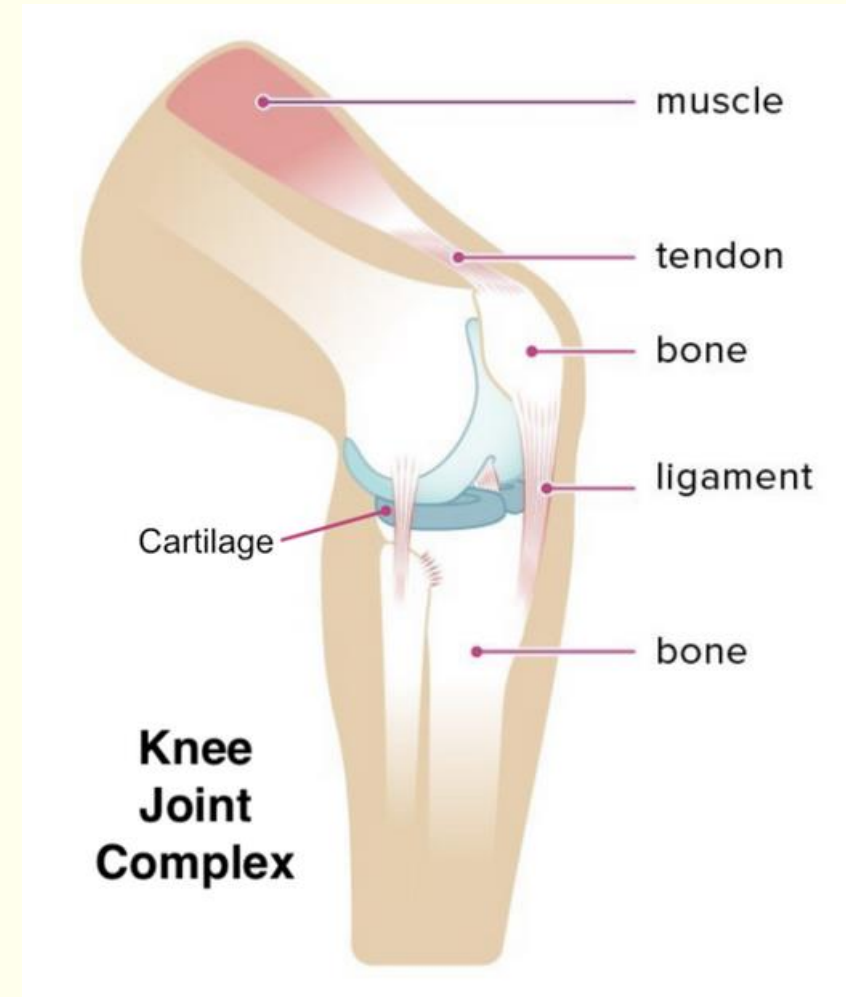
Ligaments: strong bands of fibrous connective tissue that connect bones to other bones, stabilizing joints

Tendons: tough, flexible bands of fibrous tissue that attach muscles to bones, transmitting force for movement

Cartilage: firm, flexible connective tissue that cushions joints, reduces friction, and supports structures like the nose and ribs

Joints: Points where bones meet, classified by mobility (e.g., hinge, ball-and-socket)

Musculoskeletal System: The Anatomy of the Knee



Musculoskeletal Diseases

Musculoskeletal (MSK) conditions affect the bones, joints, muscles, connective tissues and spine

Global: 1.71 billion people have MSK conditions worldwide (WHO, 2022)

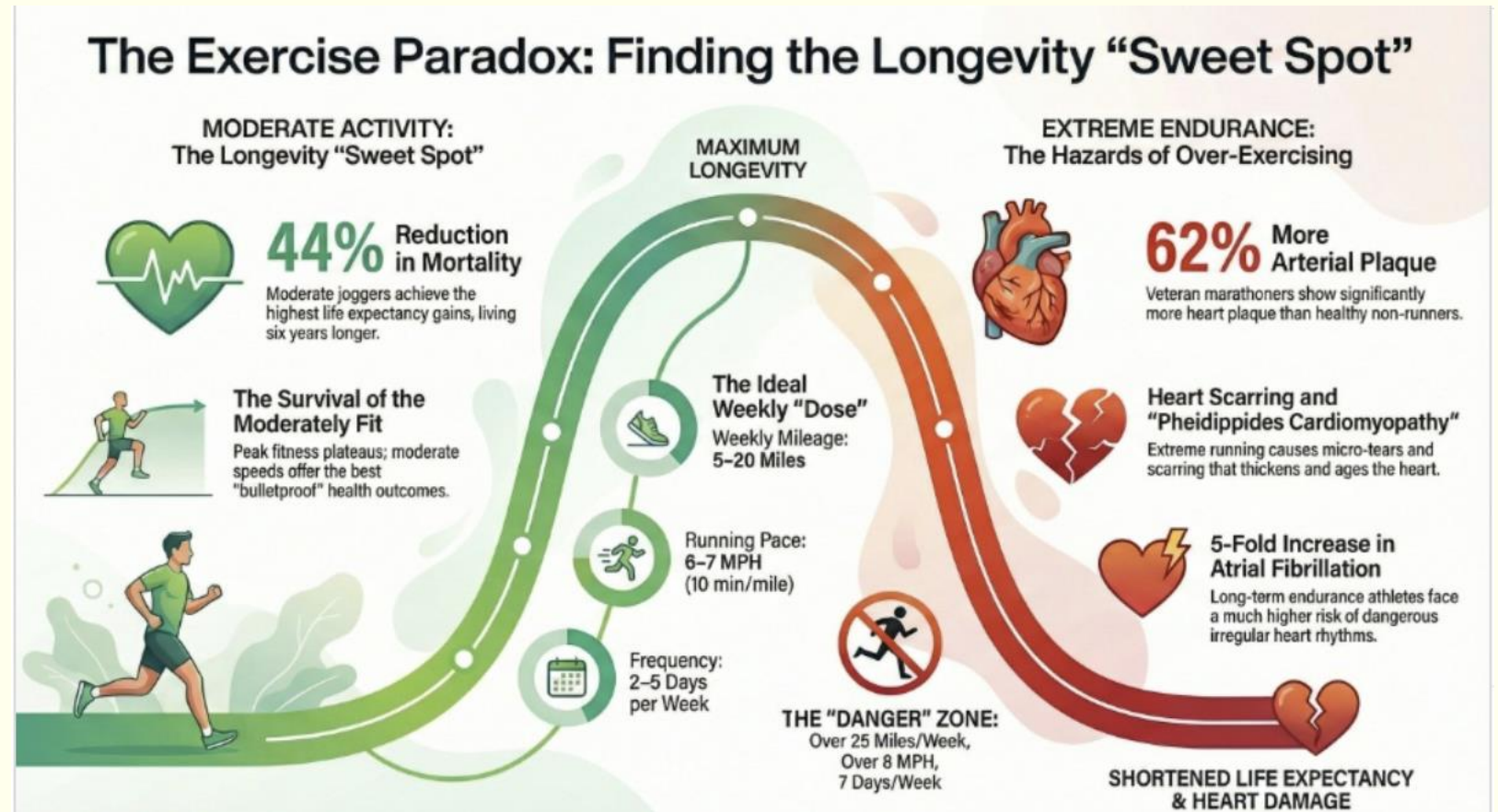
- MSK is the leading contributor to disability worldwide, accounting for 17% of all years lived with disability (YLDs)
- Low back pain alone affects 570 million people and is the single leading cause of disability in 160 countries

Musculoskeletal (MSK) Diseases

UK: 20.8 million people have MSK conditions (1 in 3 adults over 20)

- MSK accounts for 21% of all years lived with disabilities (YLDs) in the UK and 23.4 million working days lost in 2022
- Prevalence increases with age: 5% (under 20) to 63% (85+).
- Women more affected: 36% vs 28% men
- The most deprived 20% of the population have significantly higher MSK prevalence

The Exercise Paradox



The Physical Activity Paradox

Physical activity is the single most effective protector against MSK decline across the lifespan

- It builds bone density, maintains muscle mass, protects cartilage, supports joint stability, and reduces falls risk

At the same time, physical activity is a source of MSK injury and degeneration: repetitive loading, impact injuries, occupational wear

- Elite and recreational athletes experience higher rates of Osteoarthritis in loaded joints (knees, hips)

The Physical Activity Paradox

Occupational physical demands (manual labour, construction, mining) increase OA and chronic back pain risk

- **The dose matters:** moderate, varied activity is protective. Extreme, repetitive, or poorly managed loading can be destructive
- For your professional practice: prescribing physical activity means understanding both its protective and its damaging potential

Today's Session

Today we will be covering six MSK conditions:

- **Rheumatoid Arthritis** (autoimmune attack on the joints)
- **Osteoarthritis** (degenerative joint disease)
- **Osteoporosis** (metabolic bone disease: loss of bone density)
- **Sarcopenia** (age-related loss of muscle mass and function)
- **Chronic Low Back Pain** (multifactorial symptom complex)
- **Sarcopenic Obesity** (muscle loss and excess adiposity)

MSK Classification Framework: Mechanism of Damage

Category	Mechanism	Key Condition	Target
Autoimmune	Immune system attacks the body's own joint tissue	Rheumatoid Arthritis	Synovial membrane of joints
Degenerative / Mechanical	Wear, loading, and failed repair of joint structures over time	Osteoarthritis	Articular cartilage and subchondral bone
Metabolic Bone Disease	Imbalance in bone remodelling: resorption exceeds formation	Osteoporosis	Bone density and microarchitecture
Age-related Loss	Progressive loss of skeletal muscle mass, strength and function	Sarcopenia	Skeletal muscle
Combined Syndrome	Co-existence of low muscle mass/function and excess adiposity, driven by shared mechanisms (insulin resistance, chronic inflammation, inactivity)	Sarcopenic Obesity	Skeletal muscle and adipose tissue
Multifactorial / Biopsychosocial	Structural, psychological, and social factors interact to produce persistent pain	Chronic Low Back Pain	Lumbar spine and surrounding structures

The Broader MSK Landscape

Today's six conditions are the focus, but the MSK landscape is broader. Conditions to be aware of:

- **Fibromyalgia:** widespread chronic pain, fatigue, and cognitive disturbance. Affects approximately 2 to 5% of the population. Predominantly diagnosed in women. Frequently dismissed as psychological, particularly in women, connecting to the gender bias theme from Weeks 2 and 4.
- **Psoriatic arthritis:** inflammatory arthritis associated with psoriasis (autoimmune)

The Broader MSK Landscape

- **Ankylosing spondylitis:** inflammatory arthritis primarily affecting the spine (autoimmune)
- **Gout** is a crystal arthropathy caused by the deposition of uric acid crystals in joints (most commonly the first metatarsophalangeal joint). Strongly linked to diet, alcohol intake, obesity, and kidney function
- **Lupus (SLE):** systemic autoimmune condition that can affect joints, skin, kidneys, brain, and other organs

Any of these may appear in clinical contexts you encounter in practice

The Autoimmune Thread Continues

The autoimmune pattern from Week 4 now extends into MSK:

- Week 4: **Type 1 diabetes** (beta cells of the pancreas), **Hashimoto's thyroiditis** (thyroid gland), **Graves' disease** (thyroid gland)
- Week 5: **Rheumatoid Arthritis** (synovial membrane of joints)
- Week 6: **Multiple Sclerosis** (myelin sheath of nerve fibres)
- Same fundamental mechanism every time: the immune system mistakes healthy tissue for an enemy and attacks it
- The target tissue determines the disease. The mechanism is the same.

Autoimmune conditions cluster: people with one autoimmune disease are at increased risk of developing others (Conrad et al., 2023, The Lancet)

Rheumatoid Arthritis (RA)

What does Rheumatoid Arthritis mean?

RHEUMA = Greek “flow”/”flux” + “oid” (adj)

Refers to the ancient belief that disease was caused by the flowing of harmful humours through the body

ARTHRON = Greek “joint” + suffix “itis” (inflammation)

Literally: 'resembling the flow of disease in the joints'

What is Rheumatoid Arthritis?

RA is a chronic, systemic autoimmune disease in which the immune system attacks the synovial membrane (the lining of the joints), causing inflammation, pain, swelling, and progressive joint destruction

- It is systemic: it can also affect the lungs, heart, blood vessels, eyes, and skin
- It is symmetrical: it typically affects the same joints on both sides of the body
- It is distinct from osteoarthritis, which is degenerative, not autoimmune

Rheumatoid Arthritis

Prevalence

- **Global:** 17.6 million people worldwide (GBD 2021). Prevalence increased 14.1% since 1990. Projected 31.7 million by 2050 (80.2% increase)
- **UK:** Approximately 450,000 adults with recorded diagnosis (Versus Arthritis, 2025). Prevalence: 1.16% women, 0.44% men. 60,632 YLDs in the UK
- **Sex difference:** Female to male ratio 2.45:1 globally. RA is 2 to 3 times more common in women
- **Age:** Peak diagnosis age 45 to 70 years but can occur at any age including childhood (juvenile idiopathic arthritis)

Rheumatoid Arthritis

Incidence

Global: the age-standardised incidence rate is approximately 14.9 per 100,000 (GBD 2021)

UK: 3.2 to 3.9 new cases per 10,000 adults per year (OpenSAFELY 2019 to 2022)

- Incidence in women has been declining in recent decades, but overall but overall prevalence continues to rise because people are living longer with RA
- Incidence peaks between ages 45 and 70, with female to male ratio approximately 3:1

Rheumatoid Arthritis

Aetiology

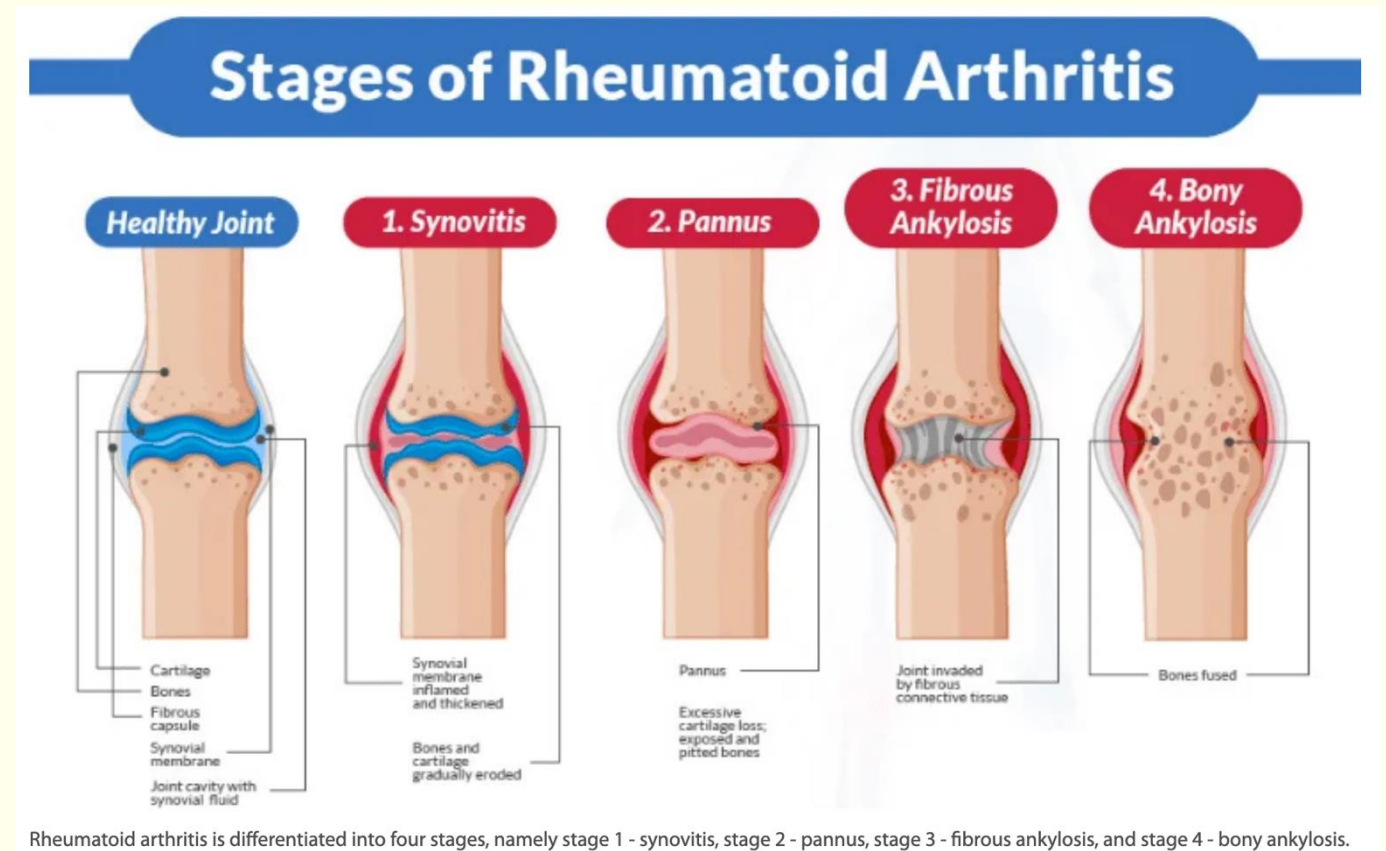
RA is an autoimmune condition. The immune system attacks the synovial membrane, the tissue lining the inside of joints.

- This is the same autoimmune pattern as Type 1 diabetes, Hashimoto's and Graves'. Different target tissue, same mechanism.
- Genetic susceptibility: Family history increases risk.
- Environmental triggers: smoking is the strongest modifiable risk factor. Infection, hormonal factors, and periodontal disease also implicated.
- Gene-environment interaction: genetic susceptibility is necessary but not sufficient. Environmental triggers initiate the autoimmune process in susceptible individuals.

Rheumatoid Arthritis

Pathology

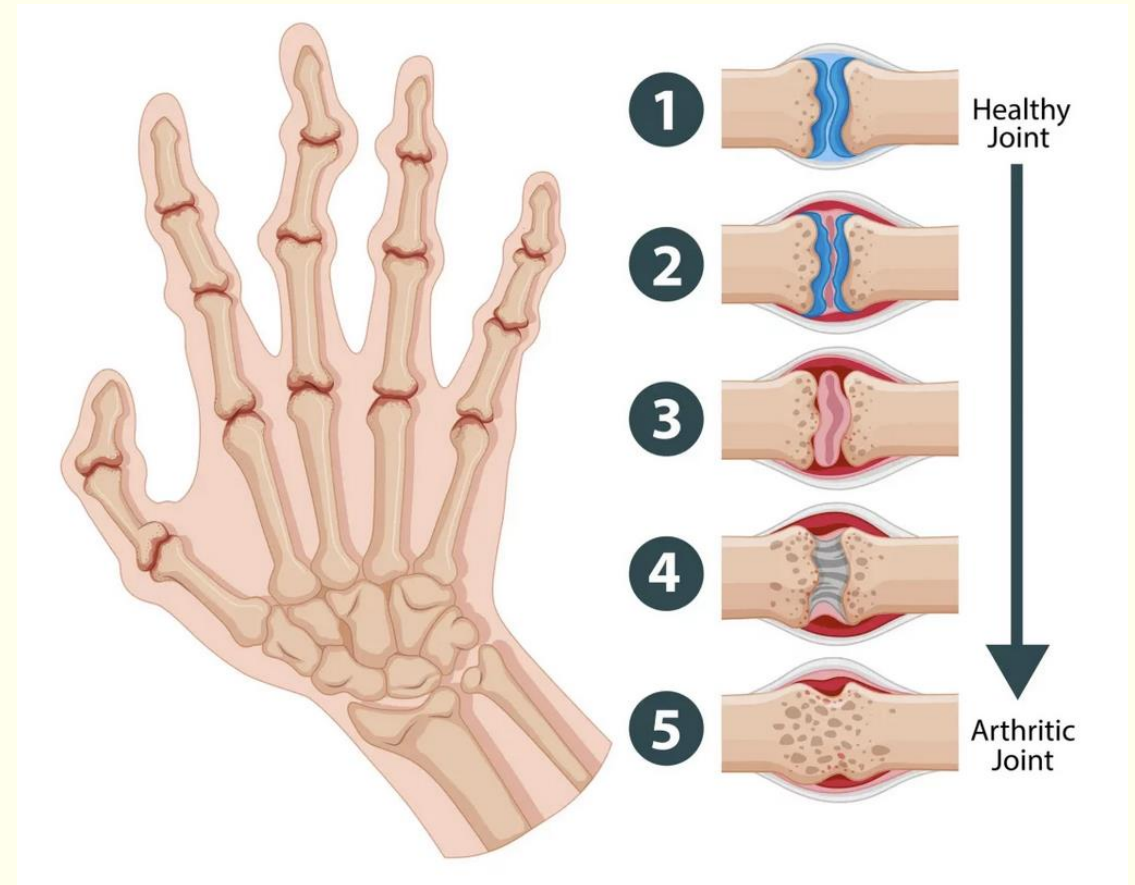
- Chronic inflammation leads to joint destruction, deformity, and loss of function
- Inflammatory cells infiltrate the synovial membrane, causing it to thicken and form a destructive tissue called pannus
- Pannus invades and erodes articular cartilage and underlying bone



Rheumatoid Arthritis

Pathology

- RA is systemic: inflammation is not limited to joints. It can affect the lungs, heart, blood vessels, eyes, and skin
- Progression: untreated RA causes irreversible joint damage, typically within the first two years. Early treatment is critical.



Rheumatoid Arthritis

Signs and Symptoms

- Symmetrical joint involvement: typically affects the same joints on both sides of the body (both wrists, both knees)
- Small joints of the hands and feet commonly affected first (MCP and PIP joints, MTPs)
- Morning stiffness lasting more than 30 minutes (often over an hour), improving with movement
- Swelling, warmth, and tenderness in affected joints

Rheumatoid Arthritis

Signs and Symptoms

- Fatigue, general malaise, and low-grade fever (systemic features of inflammation)
- Extra-articular features: rheumatoid nodules (firm lumps under the skin), dry eyes and mouth (secondary Sjogren's), interstitial lung disease, increased cardiovascular risk
- Distinguishing feature from OA: RA involves active inflammation and systemic symptoms. The joints are warm, swollen, and the stiffness is worst after rest.

Rheumatoid Arthritis

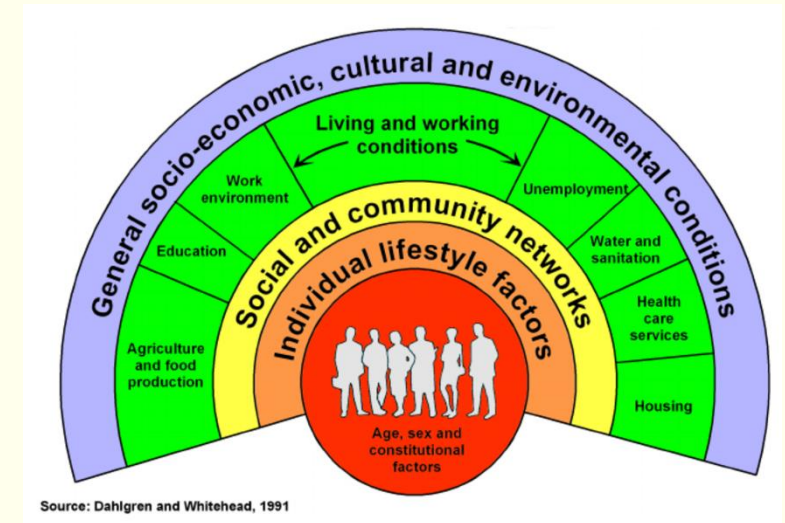
Risk Factors

Non-modifiable Risk Factors

Genetics: (HLA-DRB1)

Sex: female sex

Family history: of autoimmune disease, age (peak onset 40 to 60)



Rheumatoid Arthritis

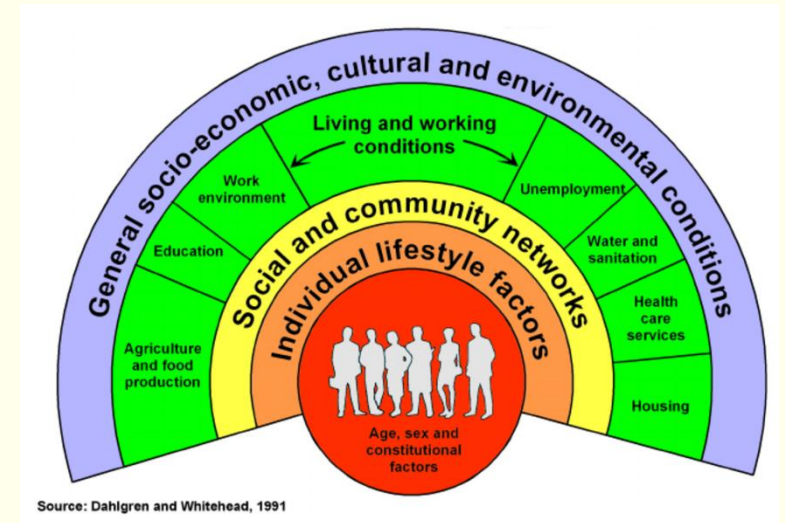
Risk Factors

Modifiable Risk Factors

Smoking: strongest environmental risk factor, doubles the risk

Obesity

Periodontal disease



Rheumatoid Arthritis

Quality of Life

- Chronic pain, fatigue, and progressive disability affecting daily tasks, work, and independence
- Treatment involves disease-modifying anti-rheumatic drugs (DMARDs) started early to prevent joint destruction
- Lifelong medication with monitoring for side effects (immunosuppression increases infection risk)

Rheumatoid Arthritis

Quality of Life

- **Significant psychological impact:** depression and anxiety are common. The unpredictability of flares adds to emotional burden
- **Employment impact:** many people with RA have to reduce hours, change roles, or stop working. 40% experience work disability within 10 years of diagnosis
- **Cardiovascular risk:** RA doubles CVD risk due to chronic systemic inflammation

Osteoarthritis (OA)

What does Osteoarthritis mean?

OSTEON = Greek “bone” plus

ARTHRON = Greek “joint” + suffix “itis” (inflammation)

Literally: 'inflammation of the bone-joint'

The name is slightly misleading: OA involves degeneration more than classical inflammation, though low-grade inflammation does play a role in disease progression

What is Osteoarthritis?

- OA is a chronic degenerative joint disease characterised by the progressive breakdown of articular cartilage, the smooth tissue that covers the ends of bones where they meet at a joint
- As cartilage wears away, bones begin to rub together, causing pain, stiffness, swelling, and reduced range of motion
- It also involves changes to the underlying bone (subchondral sclerosis, osteophyte formation), the synovial membrane, and surrounding soft tissues
- Most commonly affects the knees, hips, hands, and spine
- Often described as 'wear and tear' but this is an oversimplification: OA involves active biological processes, not simply mechanical wearing down

Osteoarthritis

Prevalence

- **Global:** 595 million people (2020), 7.6% of the global population. Projected 1 billion by 2050. The most common form of arthritis worldwide.
- **UK:** 10 million adults with probable diagnosis (Versus Arthritis, 2025). Hip and knee OA: 9 million total. 322,643 YLDs in the UK.
- **Wales:** 274,317 people with hip/knee OA. 11% of over-45s have hip OA (2019), the highest rate in the UK.
- **Age pattern:** Prevalence increases sharply with age: less than 1% under 45, rising to 42% by age 90+.
- **Joints:** Knee most common (75% increase projected by 2050), then hand (50% increase), then hip (79% increase).

Osteoarthritis

Incidence

Global: age-standardised incidence rate of 535 per 100,000 in 2021, increased from 490 per 100,000 in 1990 (GBD 2021)

- Knee OA is the most common site globally and is projected to increase by 74.9% by 2050
- High BMI contributes to 20.4% of the global OA burden

UK: incidence has been increasing significantly between 2000 and 2018:

- Hand OA incidence rising 10.9% per year
- Hip OA 3.8% per year
- Knee OA 2.9% per year

Osteoarthritis

Aetiology

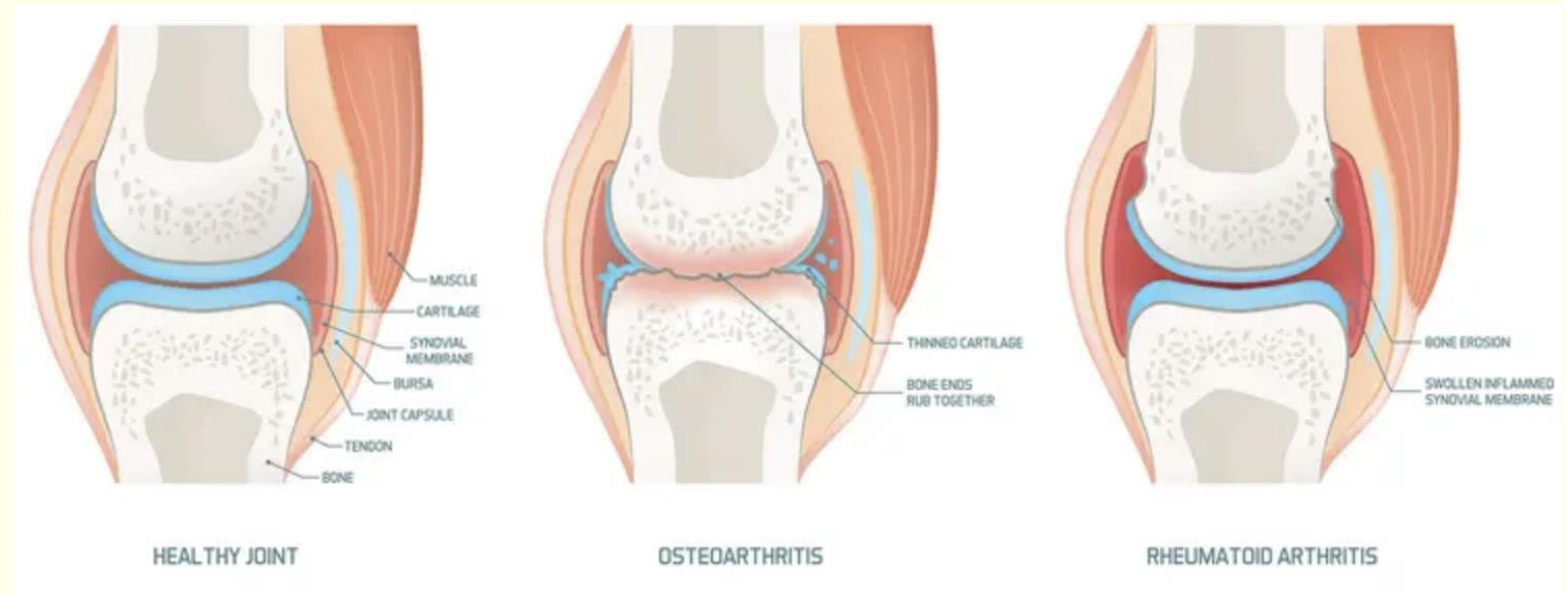
OA is a degenerative condition of the whole joint, not simply 'wear and tear'

- It involves failed repair of joint damage caused by mechanical stress, loading, and biological factors
- Risk increases with age, obesity (especially for knee OA), joint injury, occupational loading, genetics, and female sex
- OA is NOT autoimmune. There is no systemic immune attack. The damage is local and mechanical.
- Low-grade inflammation is present but is secondary to the structural damage, not the primary driver (unlike RA)

Osteoarthritis

Pathology

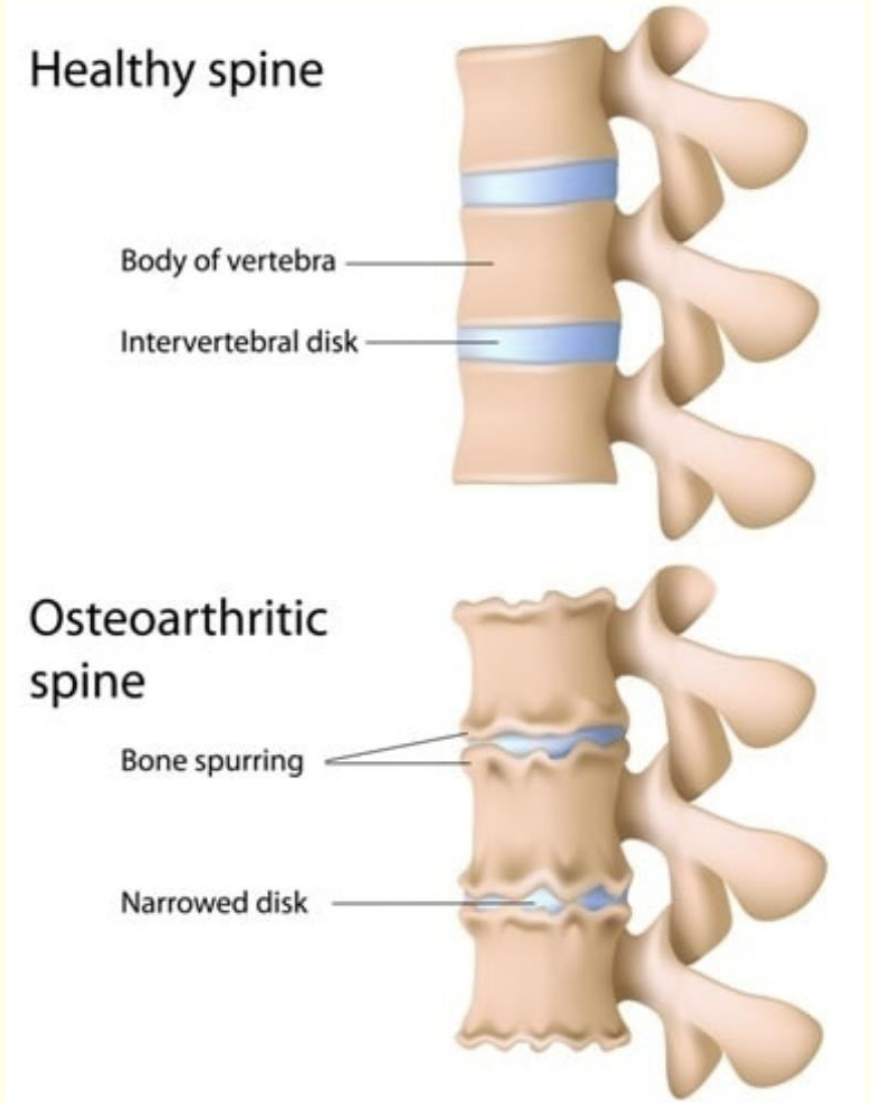
- Articular cartilage progressively breaks down and thins, losing its smooth, shock-absorbing surface
- Subchondral bone thickens and remodels. Osteophytes (bone spurs) form at joint margins.



Osteoarthritis

Pathology

- Synovial inflammation occurs but is secondary and milder than in RA
- Joint space narrows as cartilage is lost. The joint becomes stiff, painful, and less mobile.
- Unlike RA, OA is typically localised to weight-bearing or previously injured joints. It does not cause systemic illness.



Osteoarthritis

Signs and Symptoms

- Joint pain worsened by activity and relieved by rest (opposite pattern to RA)
- Stiffness after rest, but typically less than 30 minutes (unlike RA's prolonged morning stiffness)
- Reduced range of motion, crepitus (grinding or crackling sensation in the joint)
- Bony enlargement of joints (Heberden's nodes at DIP joints, Bouchard's nodes at PIP joints in the hands)
- No systemic symptoms: no fatigue, no fever, no malaise (distinguishing it from RA)

Osteoarthritis

Risk Factors

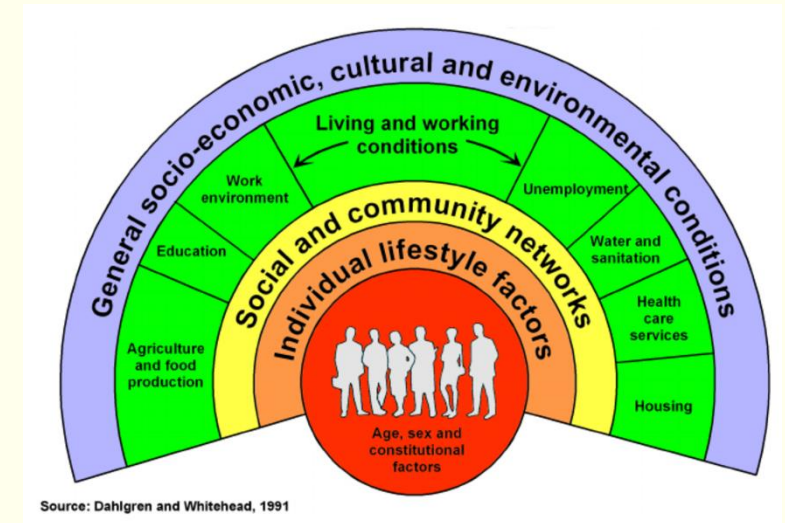
Non-modifiable Risk Factors

Genetic: (HLA-DRB1)

Age

Sex: female sex (especially post-menopause)

Previous Joint Injury



Osteoarthritis

Risk Factors

Modifiable Risk Factors

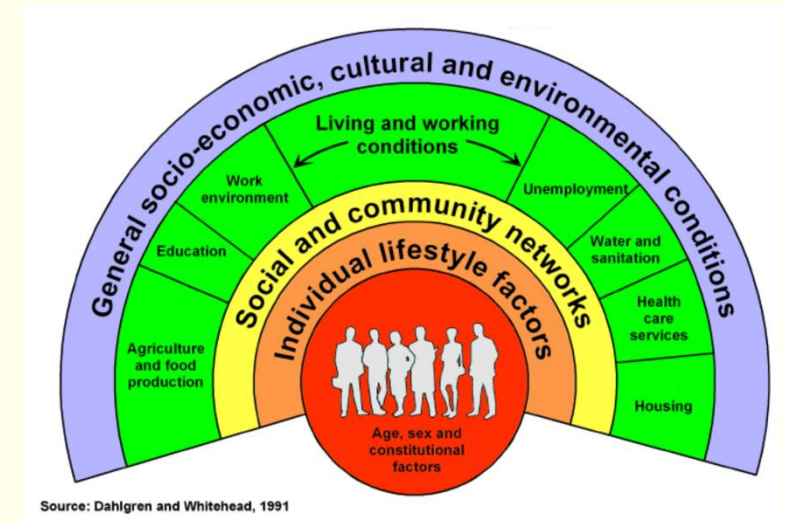
Smoking: (doubles the risk)

Obesity

Occupational Joint Loading

Physical Inactivity: deconditioning weakens joint support

Muscle Weakness



Osteoarthritis

Quality of Life

- Pain and stiffness limit mobility, work capacity, and independence, especially in advanced disease
- **No cure.** Management focuses on pain relief, exercise, weight management, and eventually joint replacement.
- Hip and knee replacements are among the most common elective surgeries in the NHS
- Physical activity (heavy loading) is both a risk factor and a treatment (strengthening, weight management) for OA. This is the physical activity paradox in action.

RA vs OA: The Key Distinctions

Feature	Rheumatoid Arthritis	Osteoarthritis
Mechanism	Autoimmune: immune system attacks synovial membrane	Degenerative: mechanical wear and failed repair
Inflammation	Primary and systemic. Drives the disease.	Secondary and local. Response to damage.
Joint pattern	Symmetrical. Small joints first (hands, feet).	Asymmetrical. Weight-bearing joints (knees, hips, spine).
Morning stiffness	Prolonged: more than 30 minutes, often over an hour	Brief: less than 30 minutes
Pain pattern	Worst after rest, improves with movement	Worst after activity, improves with rest
Systemic features	Yes: fatigue, malaise, fever, extra-articular involvement	No systemic features
Age of onset	Any age, peak 40 to 60	Increases with age, rare under 45
Treatment approach	Immunosuppression (DMARDs). Early aggressive treatment to prevent joint destruction.	Pain management, exercise, weight loss, joint replacement for advanced disease.

Osteoporosis: The Silent Bone Disease

What does Osteoporosis mean?

OSTEON = Greek “bone”

POROS = Greek “pore”/“passage” + “osis” (condition)

Literally: 'porous bone condition'

- The name perfectly describes the pathology: bones become increasingly porous and fragile as mineral density is lost

What is Osteoporosis?

- Osteoporosis is a systemic skeletal disease characterised by low bone mineral density and deterioration of bone microarchitecture, leading to increased bone fragility and susceptibility to fracture
- Diagnosed using dual-energy X-ray absorptiometry (DXA scan): a T-score of -2.5 or below at the femoral neck or lumbar spine indicates osteoporosis
- Often called 'the silent thief' because bone loss occurs without symptoms until a fracture happens
- The most common fragility fracture sites are the hip, spine (vertebral compression fractures), and wrist

Osteoporosis

Prevalence

- **Global:** 200 million people worldwide, with a global prevalence of approximately 18.3% (23.1% in women, 11.7% in men)
- **UK:** 3.5 million people affected. 500,000+ broken bones annually attributable to osteoporosis.
- **Sex difference:** 21.8% of women and 6.8% of men over 50 have osteoporosis
- **Fracture risk:** 50% of women over 50 and 20% of men over 50 will suffer an osteoporotic fracture in their remaining lifetime.

Osteoporosis

Prevalence

- **Cost:** 4.5 billion pounds annually to UK health and social care. Hip fractures alone cost 2 billion pounds and occupy 1 million acute hospital bed days per year.
- Fourth worst cause of disability and premature death (measured in DALYs) in the UK.

Called 'silent' because bone loss occurs without symptoms until a fracture happens.

Osteoporosis

Incidence

Global: over 10 million hip fractures in people aged 55+ in 2019. Hip fractures projected to nearly double between 2018 and 2050

UK: approximately 549,000 new fragility fractures per year (105,000 hip fractures, 86,000 vertebral fractures, 358,000 other fragility fractures)

- 33% of all fragility fractures occur in men vs 67% in women
- Lifetime risk: 1 in 2 women and 1 in 5 men over 50 will sustain a fragility fracture
- NHS cost: 4.7 billion pounds annually (2.6 billion in direct post-fracture costs). Hip fractures alone account for 2 billion pounds and 1 million acute hospital bed days per year

Osteoporosis

Aetiology

- Bone is living tissue in constant remodelling: osteoblasts build new bone, osteoclasts break down old bone
- Osteoporosis occurs when resorption (breakdown) exceeds formation, leading to net bone loss
- Peak bone mass is reached by approximately age 30. After this, bone density gradually declines.
- In women, oestrogen withdrawal at menopause accelerates bone loss dramatically (up to 2 to 3% per year in the first 5 to 10 years post-menopause)
- Nutrition (calcium, vitamin D, protein) and weight-bearing physical activity are critical modifiable factors in both building and maintaining bone

Osteoarthritis

Pathology

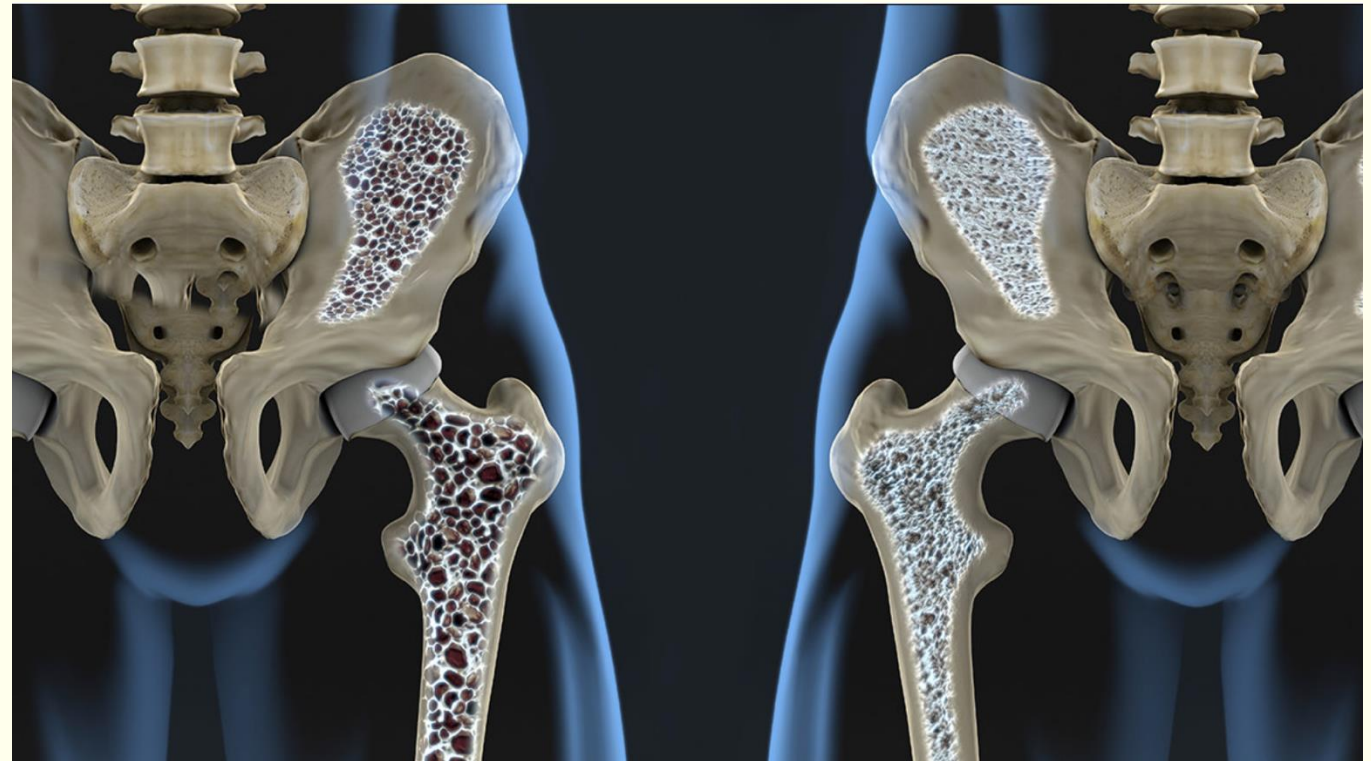
- Bone becomes porous, thin, and structurally weak. Microarchitecture deteriorates.
- Trabecular (spongy) bone is affected first, particularly in the vertebrae and wrist



Osteoarthritis

Pathology

- Bones become fragile enough to fracture from minimal trauma (fragility fractures): a fall from standing height, bending, coughing
- Common fracture sites: hip, vertebrae (compression fractures), wrist (Colles' fracture)



Osteoporosis

Signs and Symptoms

- Usually asymptomatic until fracture occurs (hence 'silent disease')
- Loss of height and kyphosis (curvature of the upper spine) from vertebral compression fractures
- Fracture from minimal trauma is often the first sign
- Chronic pain following vertebral fractures

Osteoporosis

Risk Factors

Non-modifiable Risk Factors

Age

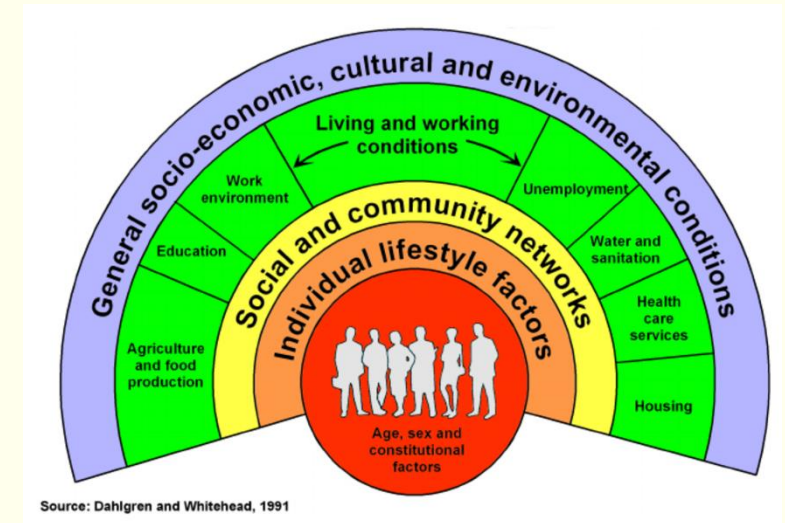
Family History

Small Body Frame

Previous Fragility Fracture

Sex: female sex (early menopause/post-menopause)

Ethnicity: higher in White and Asian populations



Osteoporosis

Risk Factors

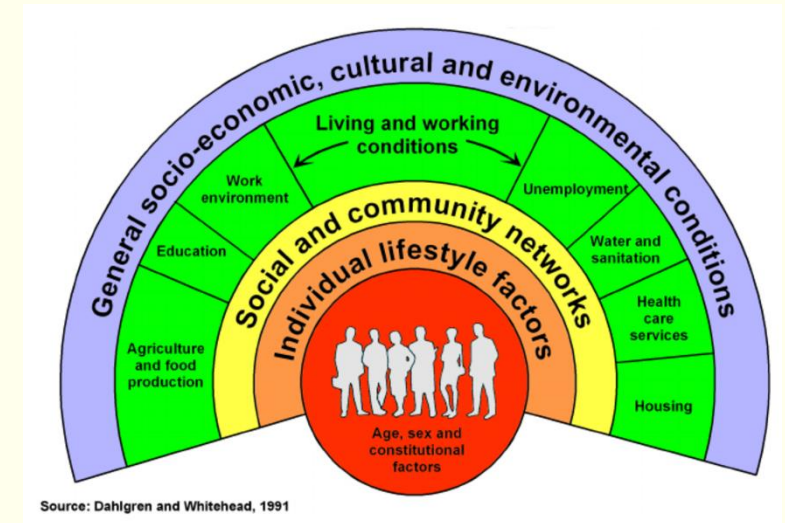
Modifiable Risk Factors

Nutrition: low calcium and vitamin D intake, excessive alcohol intake

Smoking

Low Body Weight

Physical Inactivity: especially lack of weight-bearing exercise

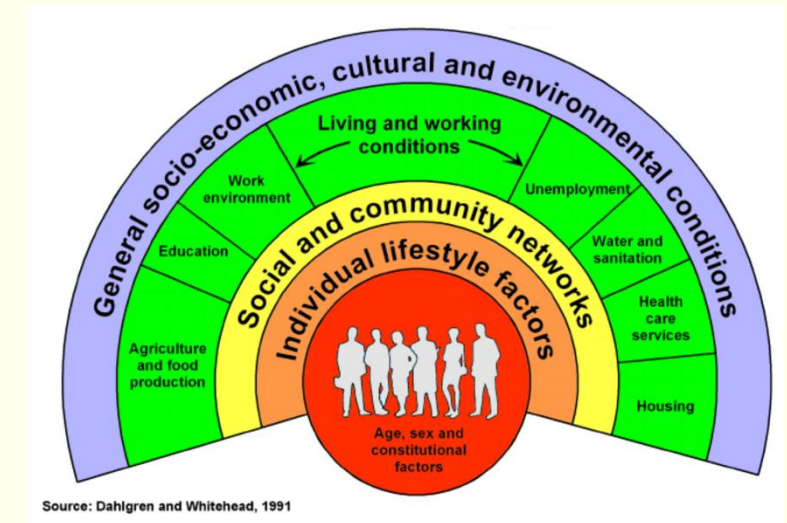


Osteoporosis

Risk Factors

Modifiable Risk Factors

Prolonged corticosteroid use



Secondary causes: conditions and medications that accelerate bone loss (thyroid disease, RA, coeliac disease, eating disorders)

Osteoporosis

Quality of Life

- Hip fractures have devastating consequences: approximately 10% die within one month, 30% within one year. 50% lose independence.
- Fear of falling restricts activity, leading to social isolation and deconditioning (a vicious cycle)
- Vertebral fractures cause chronic pain, height loss, and kyphosis affecting breathing, eating, and self-image
- Treatment includes bisphosphonates, calcium/vitamin D supplementation, and crucially, weight-bearing exercise and falls prevention
- Only 51% of NHS Trusts provide Fracture Liaison Services. 90,000 people miss treatment annually.

Break



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Trawsnewid Addysg, Trawsnewid Bywydau
Transforming Education, Transforming Lives

Sarcopenia: The Silent Muscle Disease

What does Sarcopenia mean?

- **SARX** = Greek “flesh”/”muscle”
- **PENIA** = Greek “poverty”/”deficiency”

Literally: 'poverty of flesh'

- The term was coined by Irwin Rosenberg in 1989 to describe the age-related loss of skeletal muscle mass
- Recognised as a disease with its own ICD-10 code (M62.84) since 2016

What is Sarcopenia?

- Sarcopenia is the progressive, age-related loss of skeletal muscle mass, strength, and function
- It is now classified as a disease, not just a normal part of ageing
- Diagnosis requires low muscle strength, confirmed by low muscle mass, with severity graded by low physical performance
- Muscle mass begins to decline from around age 30, at approximately 3 to 8% per decade. The rate accelerates after 60

Sarcopenia

Prevalence

Global: Prevalence ranges from 5 to 13% in adults aged 60 to 70, rising to 11 to 50% in those over 80

UK: UK Biobank data (ages 40 to 70): 5.3% overall prevalence of probable sarcopenia

- 64.8% of those with sarcopenia have multimorbidity

Under-recognised:

- Only 52% of UK healthcare organisations identify sarcopenia clinically
- Only 53% use a recognised diagnostic algorithm

Sarcopenia

Incidence

Incidence data for sarcopenia is limited because formal diagnosis is relatively new (ICD-10 code assigned 2016)

Only 52% of UK health organisations identify sarcopenia clinically, and only 47% use a recognised diagnostic algorithm

Sarcopenia

Incidence

Prevalence serves as the primary epidemiological measure: global community-based prevalence ranges from 10 to 27% of older adults depending on diagnostic criteria used (EWGSOP criteria)

Prevalence increases steeply with age:

- 2.5% at ages 40 to 49
- 4.5% at 50 to 59
- 7.6% at 60 to 70 (UK Biobank data)

Sarcopenia

Aetiology

Classifications:

1. Primary sarcopenia: caused by ageing itself, with no other identifiable cause, the default pathway if you live long enough
2. Secondary sarcopenia has three sub-types:
 - i. **Disease-related:** caused by conditions that promote inflammation or wasting (CVD, chronic kidney disease, cancer, COPD, rheumatoid arthritis)
 - ii. **Activity-related:** caused by physical inactivity, sedentary lifestyle, or prolonged immobility (bed rest, injury)
 - iii. **Nutrition-related:** caused by inadequate protein or energy intake, or malabsorption

Sarcopenia

Aetiology

Can also be classified by duration:

- acute sarcopenia (less than 6 months, usually related to illness or injury)
- chronic sarcopenia (6 months or more, associated with progressive conditions and higher mortality risk)

Sarcopenia

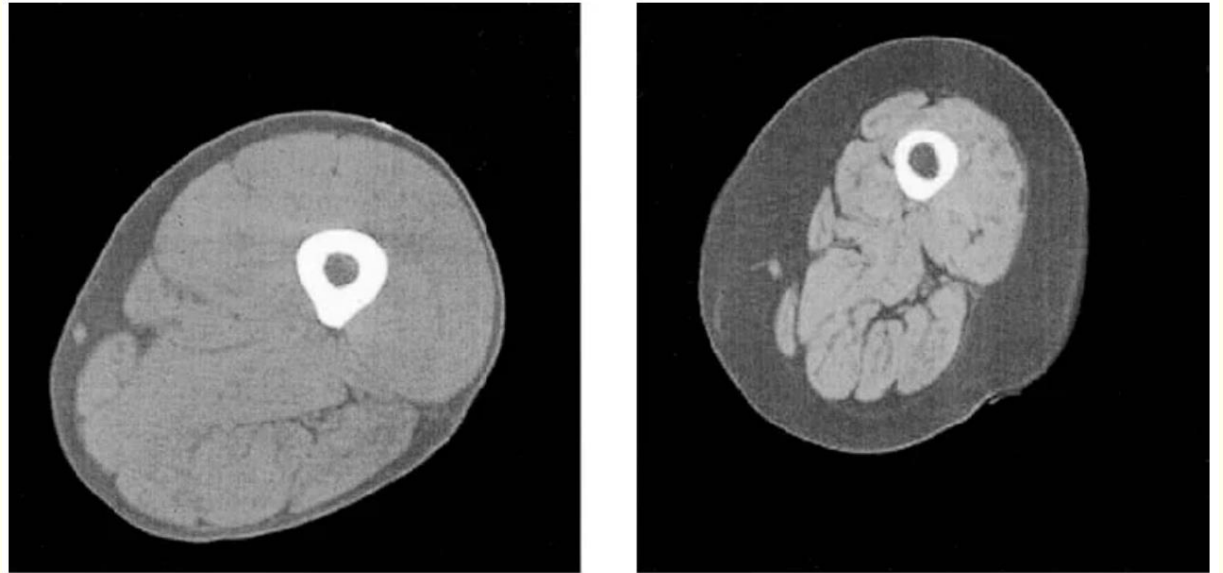
Pathology

- Multiple interacting mechanisms drive sarcopenia:
- Protein homeostasis imbalance: decreased anabolic (building) signals and increased catabolic (breakdown) signals. Muscle protein synthesis slows while degradation accelerates
- Neuromuscular degeneration: motor neurons are lost with ageing, neuromuscular junctions deteriorate, and satellite cells (muscle stem cells) lose their ability to activate for repair
- Muscle fibre type shift: preferential loss of Type II ('fast-twitch') fibres with relative preservation of Type I ('slow-twitch'). This disproportionately reduces power and strength

Sarcopenia

Pathology

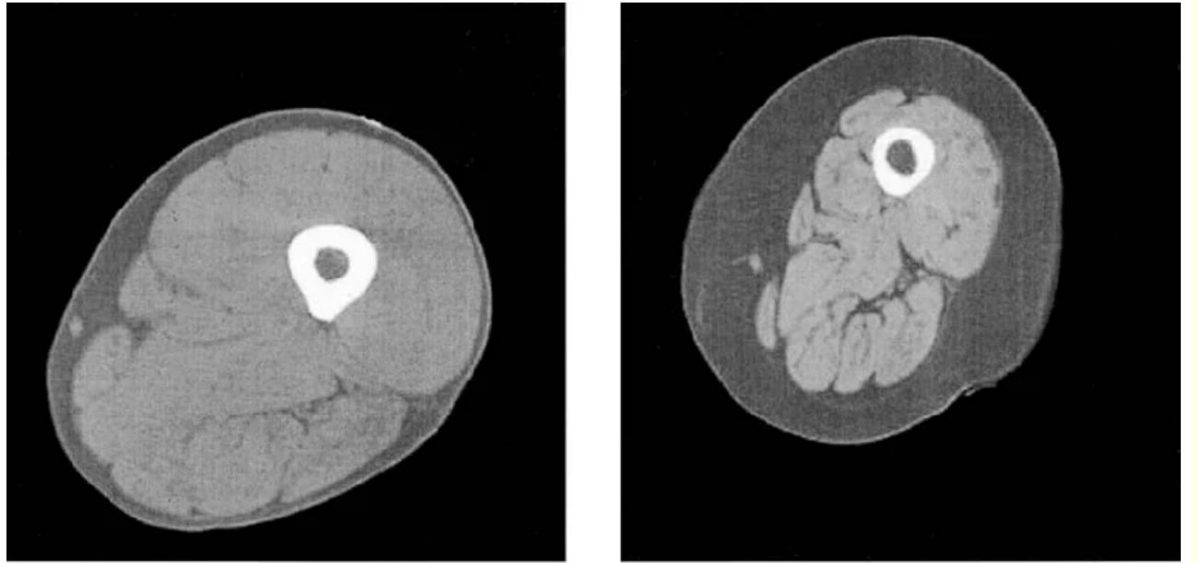
- Muscle quality changes: muscle fibres are progressively replaced by fat infiltration (myosteatorsis) and fibrous tissue (fibrosis). The muscle may not visually appear smaller but its functional capacity is reduced
- Chronic low-grade inflammation: elevated inflammatory cytokines promote muscle protein breakdown



Sarcopenia

Pathology

- Hormonal changes: declining testosterone, growth hormone, and DHEA with ageing reduce anabolic drive
- Mitochondrial dysfunction: impaired oxidative capacity and increased reactive oxygen species damage muscle cells



Sarcopenia

Pathology

Progression Rates:

After age 50, muscle mass declines at approximately 1 to 2% per year

- Muscle strength declines at 1.5 to 5% per year, which is faster than mass loss
- The relationship between muscle mass and muscle strength is non-linear: you can lose significant strength before the loss of mass becomes obvious on measurement
- This is why the diagnostic criteria prioritise function (grip strength, chair stand test) over mass alone

Sarcopenia

Signs and Symptoms

- Reduced grip strength (one of the earliest measurable signs; assessed with a hand dynamometer)
- Difficulty rising from a chair without using arms (the chair stand test is a key clinical screening tool)
- Slow gait speed (below 0.8 m/s is a diagnostic threshold in EWGSOP2)
- Reduced balance and increased falls risk (loss of Type II fast-twitch fibres impairs reactive balance)

Sarcopenia

Signs and Symptoms

- Fatigue and reduced exercise tolerance
- Difficulty with daily activities: climbing stairs, carrying shopping bags, opening jars
- Visible muscle wasting is a LATE sign. Functional impairment typically precedes visible loss of muscle bulk
- No pain, no inflammation, no swelling (distinguishing it from RA and OA)

Sarcopenia is a 'silent' progressive condition, similar to osteoporosis.

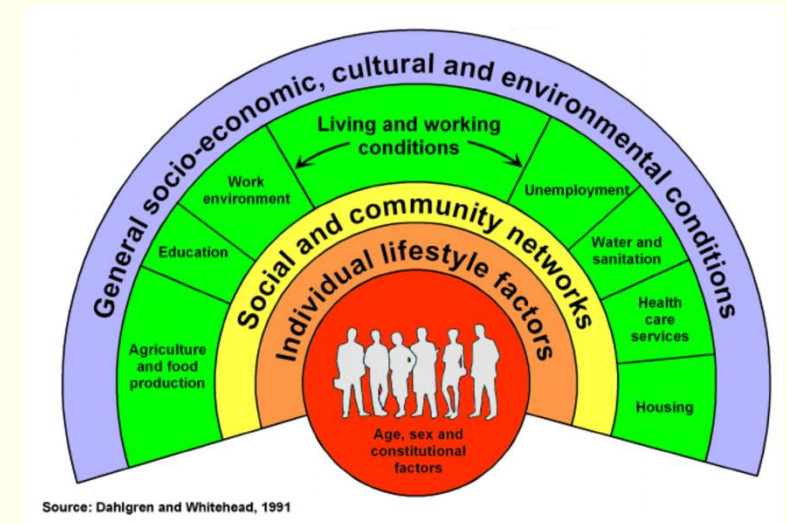
Sarcopenia

Risk Factors

Non-modifiable Risk Factors

- **Age:** muscle mass declines from around age 30
- **Sex:** women's risk increases sharply after menopause
- **Genetics:** variation in genes controlling muscle fibre composition, inflammatory response, and hormone sensitivity
- **Chronic disease burden:** diabetes, COPD, heart failure, cancer, chronic kidney disease, and liver disease all accelerate sarcopenia through inflammatory and metabolic pathways.

64.8% of people with sarcopenia have multimorbidity

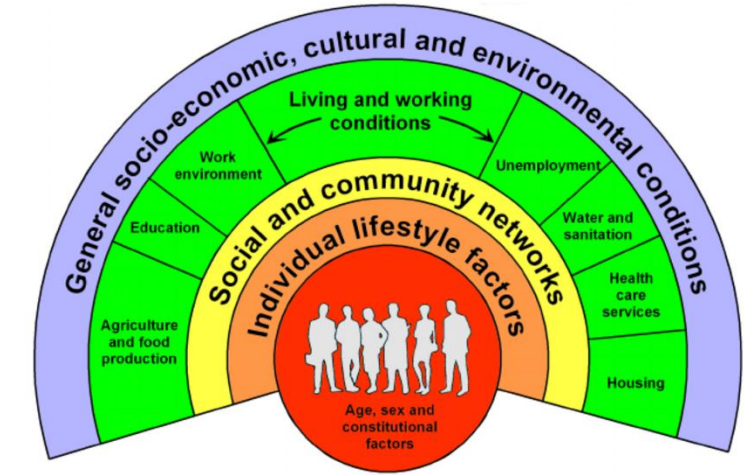


Sarcopenia

Risk Factors

Modifiable Risk Factors

- **Physical inactivity**: the strongest modifiable factor
Two weeks of bed rest can cause the same amount of muscle loss as a decade of normal ageing. Sedentary behaviour accelerates loss even in otherwise active people.
- **Inadequate protein intake**: older adults need 1.0 to 1.2 g/kg/day (healthy) or up to 1.5 g/kg/day (with sarcopenia). Many older adults do not meet even the lower recommendation.

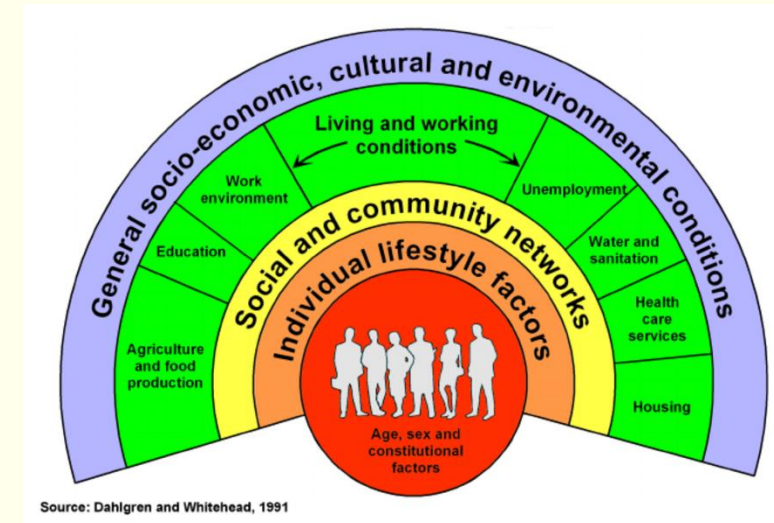


Sarcopenia

Risk Factors

Modifiable Risk Factors

- **Malnutrition and undernutrition**: overall caloric deficit accelerates muscle breakdown
- **Vitamin D deficiency**: Deficiency impairs muscle function
- **Smoking**: increases systemic inflammation, impairs blood flow to muscle tissue, and accelerates muscle protein breakdown
- **Excess alcohol intake**: directly toxic to skeletal muscle (alcoholic myopathy) and impairs protein synthesis



Sarcopenia

Quality of Life

- Increased falls risk, fracture risk, and hospitalisation
- Loss of independence in daily activities (rising from a chair, climbing stairs, carrying shopping)
- Strongly associated with frailty, disability, and mortality in older adults
- Treatment is exercise-based: resistance training is the single most effective intervention. Combined with adequate protein intake (1.0 to 1.2 g/kg/day for older adults, up to 1.5 g/kg/day for those with sarcopenia).

Directly in the professional domain of nutrition, sports therapy, and exercise science graduates

Osteosarcopenia

When Bone and Muscle Decline Together

- Osteoporosis and sarcopenia frequently co-occur in older adults, a condition increasingly termed osteosarcopenia
- Shared risk factors: ageing, physical inactivity, malnutrition, vitamin D deficiency, hormonal decline, chronic inflammation
- Bone and muscle are mechanically linked: muscle contractions stimulate bone formation (Wolff's Law). When muscle declines, bone loses its stimulus to maintain density.

Osteosarcopenia: When Bone and Muscle Decline Together

- Weak muscles increase falls risk. Weak bones mean those falls cause fractures. The combination is devastating.
- Both conditions respond to the same interventions: resistance training, adequate protein and calcium intake, vitamin D, and falls prevention strategies
- This is where your professional practice has the greatest direct impact: exercise prescription and nutritional guidance are the frontline treatments for both conditions

Group Work

Condition	Category	Target Tissue	Mechanism	Key Distinguishing Feature
RA	Autoimmune	Synovial membrane	Immune system attacks joint lining; pannus erodes cartilage and bone	Systemic, symmetrical, inflammatory. Worst after rest.
OA	Degenerative	Articular cartilage	Mechanical wear and failed repair of cartilage and joint structures	Local, asymmetrical, non-inflammatory. Worst after activity.
Osteoporosis	Metabolic bone	Bone density	Resorption exceeds formation; bone becomes porous and fragile	Silent until fracture. Fragility fractures from minimal trauma.
Sarcopenia	Age-related loss	Skeletal muscle	Progressive loss of muscle mass, strength and function with ageing	Under-recognised. Resistance training is primary treatment.

Condition	Category	Target Tissue	Mechanism	Key Distinguishing Feature
Sarcopenic Obesity	Combined syndrome	Skeletal muscle and adipose tissue	Co-existence of low muscle mass/function and excess adiposity, driven by shared pathophysiological mechanisms	Synergistically worse outcomes than either condition alone. Metabolic links to insulin resistance, CVD, disability, cognitive decline.
Chronic Lower Back Pain (CLBP)	Biopsychosocial	Lumbar spine and structures	Structural, psychological and social factors interact to produce persistent pain	Not a structural diagnosis. Yellow flags predict chronicity.

Connections to other chronic diseases

- The autoimmune thread continues: Week 6 (Multiple Sclerosis, autoimmune attack on myelin sheath of nerve fibres)
- MSK conditions connect to CVD (Week 2): RA doubles cardiovascular risk; physical inactivity is a shared risk factor
- MSK conditions connect to metabolic disease (Week 4): gout links to metabolic syndrome, diabetes and CKD. Obesity is a cross-cutting risk factor for OA, osteoporosis and chronic back pain.

Connections to other chronic diseases

- Obesity as a cross-cutting risk factor will be covered properly in Week 10 (Diagnosis, Treatment and Lifestyle Management)
- Osteoporosis and sarcopenia are directly in your professional domain. Exercise prescription and nutritional guidance are frontline interventions.
- The physical activity paradox reappears whenever you prescribe exercise: protective when well-managed, potentially harmful when excessive, repetitive, or poorly prescribed.



Next Week: Neurological Conditions

Questions?

