

BMSH4302

Introduction to Chronic Disease

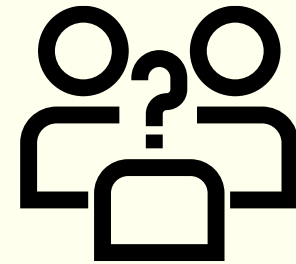
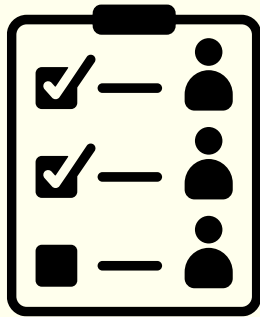
Level 4



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BMSH4302 Introduction to Chronic Disease

Housekeeping:



Cardiovascular Disease Recap

Objectives for Today:

- Understand the **Prevalence** of key pulmonary diseases in the UK and Wales specifically
- Explain the **aetiology** and basic **pathology** of **COPD** (emphysema and chronic bronchitis) **asthma, bronchiectasis, occupational lung diseases, pulmonary hypertension**
- Identify the key signs, symptoms, and comorbidities of COPD and asthma
- Outline the aetiology, pathology, risk factors and QoL impact of bronchiectasis, occupational lung diseases, and pulmonary hypertension
- Identify shared and distinct risk factors across pulmonary conditions
- Discuss the impact of pulmonary disease on quality of life across physical, psychological and social domains
- Recognise the significance of industrial heritage on respiratory health in Wales

Pulmonary Disease

Pulmonary Disease

PULMONARY = relating to the lungs (from Latin 'pulmo' = lung)

Also called the **RESPIRATORY system** (from Latin 'respirare' = to breathe again)

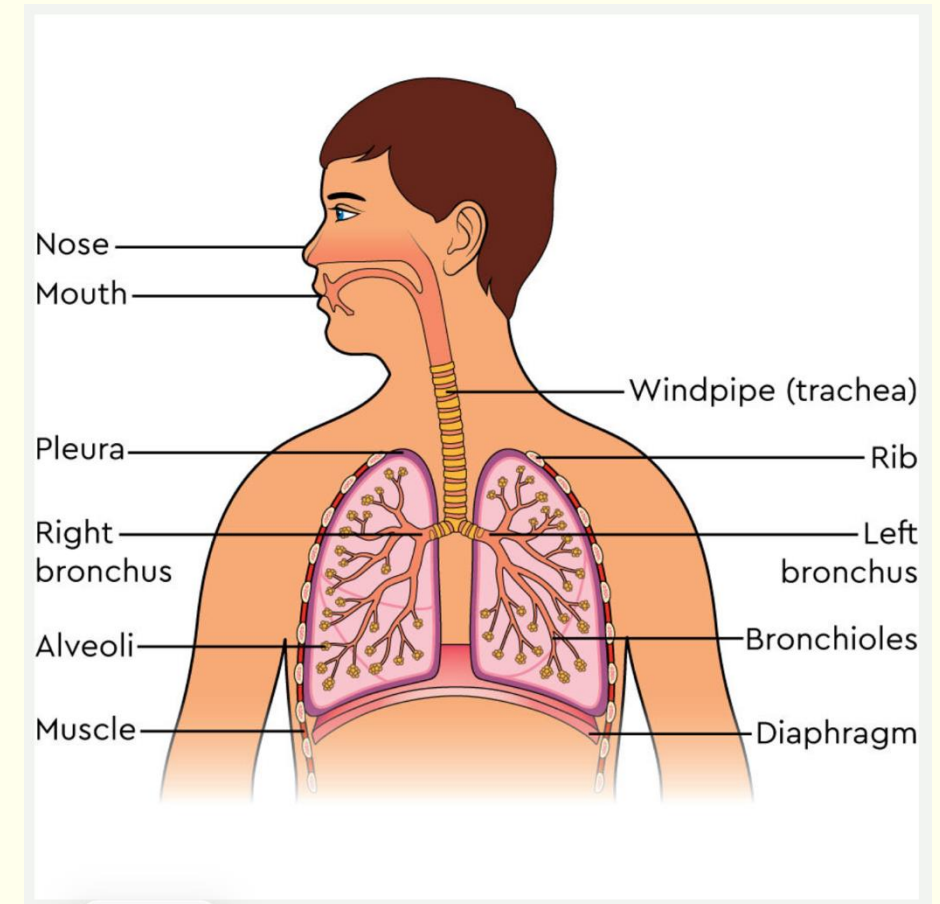
Pulmonary disease = diseases affecting the lungs and airways

The Respiratory System

What does it Do?

THREE STRUCTURES TO REMEMBER:

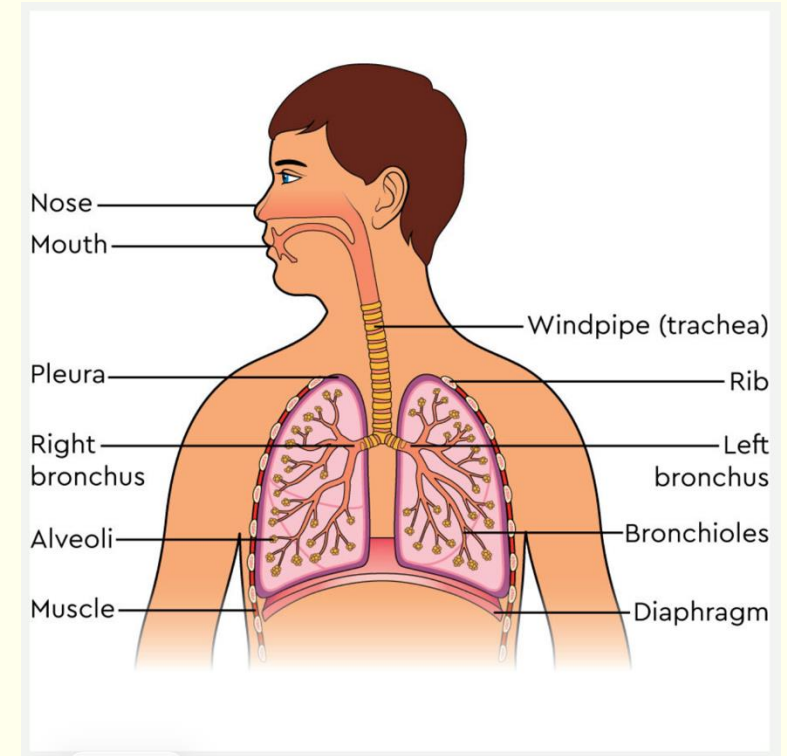
1. The AIRWAYS (bronchi and bronchioles)
2. The ALVEOLI (air sacs)
3. The BLOOD VESSELS around alveoli



The Respiratory System

What does it Do?

In pulmonary disease, ONE OR MORE of these structures is damaged



- Links to CVD: blood vessels here connect directly to the heart
- This is why CVD and pulmonary disease are often linked

What is Pulmonary Disease?

Any disorder or condition that affects:

- The lungs
- The respiratory system
- Or both

obstructing airflow and impairing gas exchange

Today's **FIVE** conditions:

1. Chronic Obstructive Pulmonary Disease (COPD)
2. Asthma
3. Bronchiectasis
4. Occupational lung diseases
5. Pulmonary hypertension

Pulmonary Disease: Prevalence

Globally: COPD is the 3rd leading cause of death (WHO)

UK: Asthma, COPD, and interstitial lung disease affect over 10 MILLION people

(Whittaker et al., 2025 – Thorax)

Bronchiectasis: ~212,000 people in the UK (British Lung Foundation, 2019)

Pulmonary hypertension: ~8,000 new cases per year in UK

WALES specifically:

Asthma prevalence: ~16.4% (nearly 1 in 6) – HIGHEST of any UK nation

COPD prevalence: ~5.1% (~185,000 people) – higher than UK average

→ Industrial heritage: coal mining legacy continues to shape respiratory health

These are NOT rare conditions – they are among the biggest health challenges we face

Chronic Obstructive Pulmonary Disease (COPD)

What is COPD?

Chronic = long term, persistent

Obstructive = long term, persistent

Pulmonary = relating to the lungs

Disease = a disorder of structure or function

→ COPD is NOT reversible, damage is permanent

What is COPD?

An umbrella term for two main conditions:

Emphysema: the destruction of alveoli

Chronic Bronchitis: inflammation of the airways

→ Most people with COPD have features of both

COPD: Prevalence

Globally:

~480 million people living with COPD; 3rd leading cause of death worldwide (WHO, 2024)

UK:

~2.5 million in England;

~185,000 in Wales diagnosed

Wales:

COPD prevalence 5.1–5.9% (above UK average)

COPD: Incidence

New diagnoses falling (34% drop in Wales 2005–2019) due to reduced smoking rates

But: Prevalence still rising because people are living longer with COPD

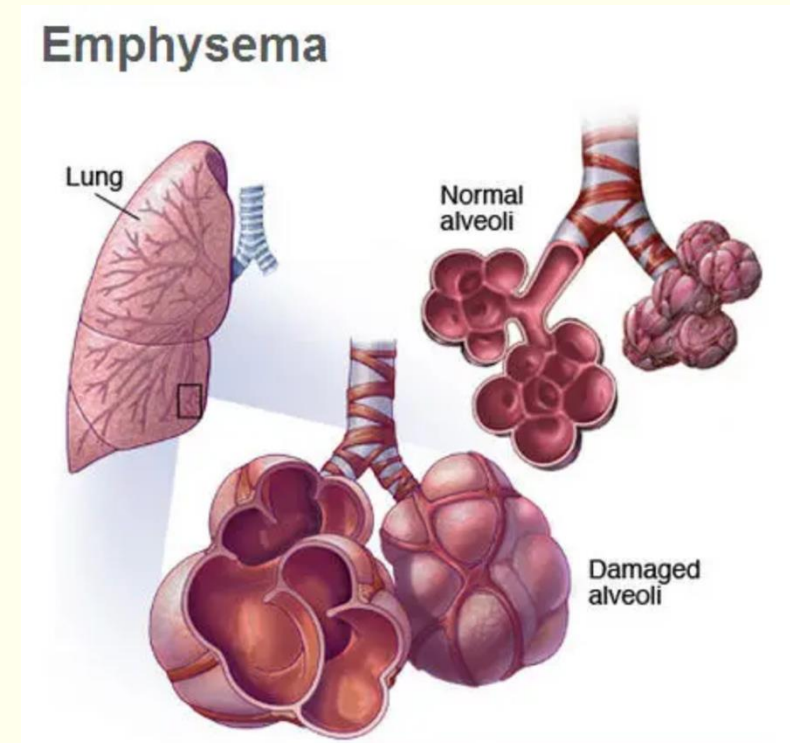
Hidden burden: Estimated 2 million undiagnosed cases across England and Wales

COVID impact: During the pandemic, new COPD diagnoses fell to just 1/3 of expected levels

COPD: Emphysema Aetiology

Chronic inhalation of irritants
(primarily cigarette smoke)
triggers persistent inflammation

Key concept: The problem in
emphysema is breathing OUT, not
breathing in



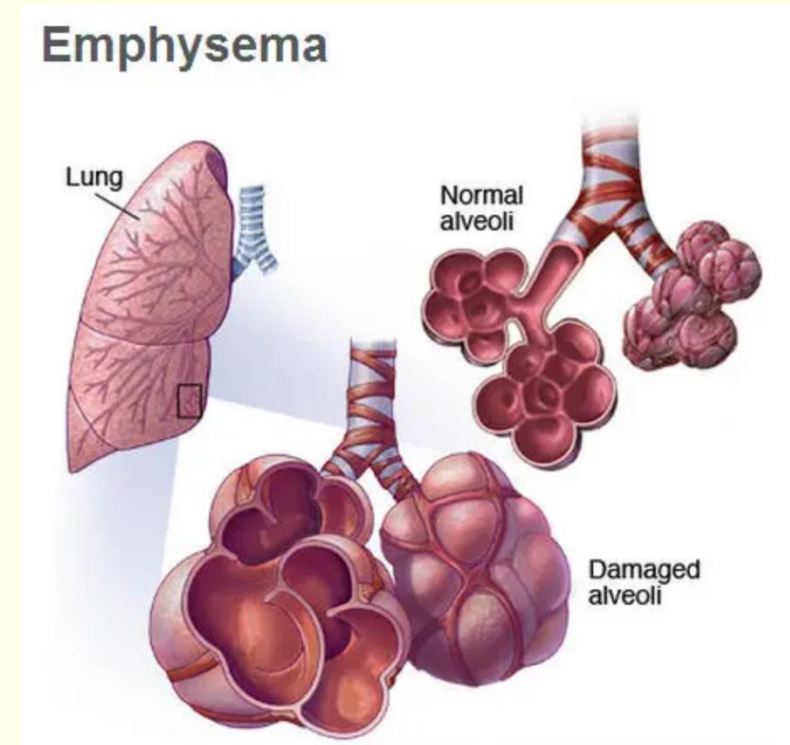
COPD: Emphysema Pathology

1. Irritants activate macrophages and neutrophils in the lungs
2. These immune cells release proteases (enzymes that break down protein)
3. Proteases destroy elastin, the stretchy protein that gives alveoli their shape
4. Alveolar walls break down, small air sacs merge into larger, less efficient spaces
5. The lung loses its elastic recoil, like a stretched-out rubber band that won't snap back
6. Air becomes trapped in the lungs (hyperinflation) you can't fully breathe OUT

COPD: Chronic Bronchitis Aetiology

Same irritants as emphysema (primarily smoking), but the damage targets the airways rather than the alveoli

Definition: A productive cough on most days for at least 3 months in 2 consecutive years



COPD: Chronic Bronchitis Pathology

1. Chronic irritation inflames the bronchial lining
2. Goblet cells (mucus-producing cells) multiply and enlarge (goblet cell hyperplasia)
3. Mucous glands in the airway walls also enlarge (submucosal gland hypertrophy)
4. This results in a massive overproduction of thick, sticky mucus

COPD: Chronic Bronchitis Pathology

5. Cilia (tiny hair-like structures that sweep mucus upward) become damaged and paralysed

6. Mucus accumulates, narrowing the airways and creating a breeding ground for infection

7. The airway walls thicken and scar (fibrosis), further narrowing the airways trapped in the lungs (hyperinflation) you can't fully breathe OUT

Key concept: Too much mucus + damaged clearance = blocked, infected airways

COPD

Signs and Symptoms

Core symptoms:

- Progressive breathlessness (dyspnoea). Initially on exertion, eventually at rest
- Chronic cough – often worse in the morning
- Sputum production – mucus, often white/grey, green/yellow during infections
- Wheeze – high-pitched whistling sound on breathing out

COPD

Signs and Symptoms

As the disease progresses:

- **Barrel chest** from chronic hyperinflation (emphysema)
- **Use of accessory muscles.** Neck and shoulder muscles recruited to help breathe
- **Tripod position** - leaning forward with hands on knees to open the chest
- **Pursed-lip breathing.** Patients instinctively do this to create back-pressure and keep airways open
- **Cyanosis** - blue tinge to lips and fingertips (from low oxygen levels)
- **Peripheral oedema** - swollen ankles (from right-sided heart strain)
- **Weight loss and muscle wasting** - the effort of breathing burns enormous energy

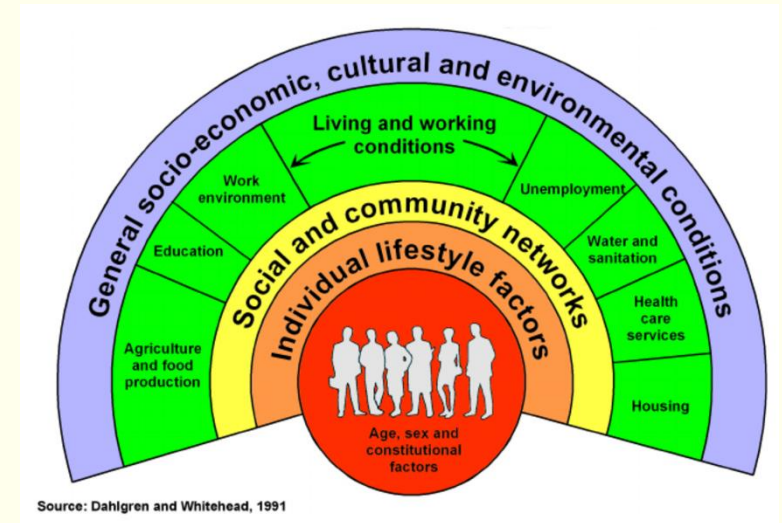
COPD: Risk Factors

Non-modifiable Risk Factors

Genetic: Alpha-1 antitrypsin deficiency: a genetic condition (~1–2% of COPD cases)

Age: symptoms typically appear after age 40+

History of childhood respiratory illness (e.g. severe pneumonia, TB)



COPD: Risk Factors

Modifiable Risk Factors

Cigarette smoking

THE dominant risk factor (~85–90% of cases)

Occupational dust/chemical exposure

accounts for ~20% of COPD cases (30% in non-smokers)

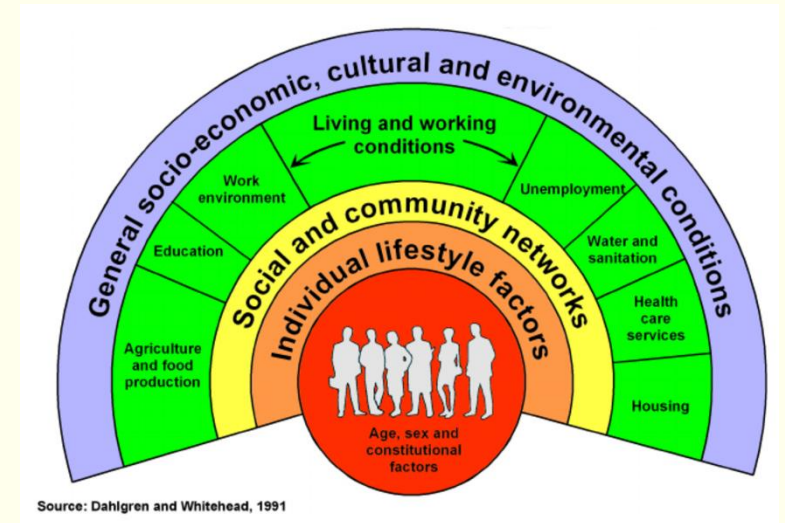
Indoor air pollution

cooking/heating with biomass fuels (globally significant)

Outdoor air pollution

particulate matter, traffic emissions

Recurrent respiratory infections in childhood



Key stat: Smoking prevalence in Wales has halved from 26% (2003) to 13% (2023)

COPD: Comorbidities

Cardiovascular:

- CHD
- Heart failure
- Stroke
- Peripheral vascular disease – shared risk factors + systemic inflammation

COPD: Comorbidities

Metabolic:

- Type 2 diabetes
- Osteoporosis (steroid use + inactivity + chronic inflammation)

Musculoskeletal:

- Muscle wasting and weakness (particularly quadriceps)

Mental health:

- Anxiety (especially panic around breathlessness) and depression (~40% of COPD patients)

COPD: Comorbidities

Respiratory:

- Lung cancer (shared smoking risk factor)
- Bronchiectasis
- Pulmonary hypertension

Other: GORD (gastro-oesophageal reflux), anaemia, cognitive impairment

NACAP Wales audit: average COPD patient has 4+ comorbidities

COPD: Quality of Life

Quality of Life Impact

- **Physical:** Progressive loss of mobility; simple daily tasks become exhausting; barrel chest, muscle wasting
- **Social:** Withdrawal from activities; can't keep up with friends/family; reluctance to go out due to breathlessness
- **Psychological:** Fear of breathlessness, panic attacks, depression, loss of identity

COPD: Quality of Life

Quality of Life Impact

- **Financial:** Often forces early retirement; costs of medications and equipment
- **Relationships:** Role changes within families; burden on carers; intimacy affected by breathlessness

"It's not the dying that's hard, it's the living"

Breathlessness Activity

What Does Breathlessness Feel Like?

Instructions:

1. Pinch your nose closed
2. Breath ONLY through the straw
3. Try to maintain a conversation with your neighbour for 60 seconds whilst doing it

This simulates moderate COPD. Some patients live with worse than this every day.

Break

Asthma

What is Asthma?

A chronic inflammatory condition of the airways characterised by:

Variable airflow obstruction (it comes and goes)

Airway hyperresponsiveness (airways overreact to triggers)

Chronic inflammation (even when symptom-free, inflammation is present)

What is Asthma?

KEY DISTINCTION FROM COPD:

Asthma is REVERSIBLE (obstruction comes and goes)

COPD is IRREVERSIBLE (obstruction is permanent and progressive)

Asthma Prevalence

Asthma: The Scale

Globally: ~334 million people; one of the most common chronic diseases worldwide

UK: ~5.5 million people in England receiving treatment

Wales: ~590,000 people (16.4%)

→ **Wales has the highest of any UK nation (England 9.6%, Scotland 13.2%, NI 7.6%)**

Asthma Incidence

Falling ↓ 34% drop in Wales between 2005 and 2019

Demographics: More common in boys during childhood; after puberty, more common in women

COVID impact: New asthma diagnoses dropped to 1/3 of expected during the pandemic

Deaths: ~1,400 people die from asthma in the UK each year – many of these are preventable

Asthma Aetiology

Asthma is a complex condition with multiple interacting causes

Genetic

Strong hereditary component – runs in families, linked to atopy (genetic tendency to develop allergic conditions)

Environmental

The Atopic Triad: Asthma + Eczema + Hay fever. These three conditions often cluster together

Asthma Aetiology

Environmental Triggers:

Allergens: house dust mites, pollen, pet fur, mould

Irritants: cigarette smoke, air pollution, cold air, strong chemicals

Exercise (exercise-induced bronchoconstriction)

Respiratory infections (especially viral, e.g. rhinovirus)

Occupational exposures: eg bakers (flour dust); hairdressers (chemicals); painters (isocyanates)

Emotional stress (doesn't cause asthma but can trigger attacks)

Asthma Pathology: The Two-Phase Response

Early phase (minutes):

1. Trigger activates mast cells in the airway lining
2. Mast cells degranulate → release histamine, leukotrienes, prostaglandins
3. These cause: bronchoconstriction (smooth muscle tightens), mucosal oedema (swelling), increased mucus production

This is the acute attack → sudden wheeze, tightness, breathlessness

Asthma Pathology: The Two-Phase Response

Late phase (4–8 hours later):

4. Eosinophils and other immune cells are recruited to the airways
5. These release inflammatory mediators causing further swelling and damage

This phase is why symptoms can return or worsen hours after the initial trigger has gone

Asthma Pathology: The Two-Phase Response

Airway remodelling (over years):

- Repeated cycles of inflammation and repair cause permanent structural changes
- Smooth muscle thickens, basement membrane thickens, more goblet cells develop
- Airways become fixed and less responsive to treatment

Asthma: Signs and Symptoms

Classic symptoms (episodic and variable):

Wheeze: musical, high-pitched sound, typically worse on breathing OUT

Breathlessness: during attacks, not usually present between attacks (unlike COPD)

Chest tightness: described as a band around the chest

Cough: often dry, worse at night and early morning

Asthma: Signs and Symptoms

Pattern clues that suggest asthma over COPD:

- Symptoms vary over time and in intensity
- Worse at night/early morning
- Triggered by specific exposures (allergens, exercise, cold air)
- Responds to bronchodilators (reliever inhalers)
- Personal or family history of atopy

Severe/life-threatening attack signs: inability to complete sentences, silent chest (no wheeze, more dangerous), confusion, exhaustion, cyanosis

Asthma: Risk Factors

Non-modifiable Risk Factors:

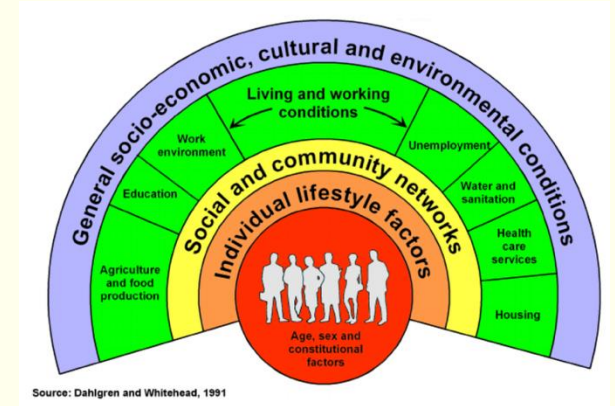
Genetic/family history of atopy (strongest predictor)

Sex:

- boys more affected in childhood;
- women more affected in adulthood

Ethnicity: some ethnic groups show higher prevalence

Low birth weight/premature birth



Asthma: Risk Factors

Modifiable Risk Factors:

- Tobacco smoke exposure (including passive smoking in childhood)
- Allergen exposure: dust mites, pets, mould
- Air pollution: particulate matter, nitrogen dioxide, ozone
- Obesity: increases risk and severity of asthma
- Occupational exposures
- Damp housing/mould → particularly relevant here in Wales

Asthma: Common Comorbidities

The Atopic Triad:

- **Eczema (atopic dermatitis)**

~70–80% of children with asthma also have eczema

- **Allergic rhinitis (hay fever)**

~80% of asthmatics also have this

These share the same underlying atopic immune mechanism

Asthma: Common Comorbidities

Other common comorbidities:

- **Gastro-oesophageal reflux (GORD)** acid reflux can trigger bronchoconstriction
- **Obesity** is both a risk factor and a comorbidity
- **Anxiety and depression** particularly in poorly controlled asthma
- Obstructive sleep apnoea
- Nasal polyps / chronic sinusitis

Asthma-COPD overlap: Some patients show features of both; increasingly recognised as a distinct phenotype

Asthma: Quality of Life

- **Physical:** Exercise limitation, night-time symptoms disrupting sleep, fatigue from broken sleep
- **Social:** Avoiding environments with triggers (pets, smoky places, cold weather); missing school/work during attacks
- **Psychological:** Anxiety about attacks, particularly in unpredictable asthma; fear of dying during severe attacks

Asthma: Quality of Life

- **Practical:** Always needing to carry inhaler; medication adherence burden; frequent GP/hospital visits
- **In children:** Missed school days, restricted play, feeling 'different' from peers, parental anxiety

Key difference from COPD: Between attacks, QoL can be near-normal. But the unpredictability itself is a stressor

Break



Prifysgol Cymru
Y Drindod Dewi Sant
University of Wales
Trinity Saint David

Trawsnewid Addysg, Trawsnewid Bywydau
Transforming Education, Transforming Lives

Final Takeaway: Wales Spotlight

- 1936–1942: Medical Research Council study in South Wales docks proved coal dust (not just silica) caused lung disease. Changed compensation law worldwide.
- 1943: Dr Archie Cochrane's Rhondda Valley research on miners' lung disease led to the founding principles of evidence-based medicine. The Cochrane Collaboration is named after him.
- The Pneumoconiosis Field Research in UK collieries became the basis for international dust exposure limits.
- Despite mine closures, former mining communities still bear the health consequences. Post-industrial deprivation (poor housing, unemployment, higher smoking rates) compounds the occupational legacy.



Next Week: Diabetes and Metabolic Disease

Questions?

