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Introduction to Psychology (BMHP5023)

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1. Introduction

Asthma is a chronic, reversible obstructive airway disease, characterised by inflammation, bronchoconstriction and hyperresponsiveness of the bronchi and bronchioles, it causes episodic wheeze, breathlessness, chest tightness, and cough (Kumar, Abbas and Aster, 2021). It also counts as one of the more common long-term conditions that are managed in UK primary care, and it brings with it a noticeable public health burden, in many places preventable too, sort of there in the background most of the time.

Asthma + Lung UK (2025) estimates that about 7.2 million people in the UK are currently living with the condition, which is roughly the same as around 8 in every 100 people, but prevalence shifts quite a lot across the four nations. For example, it ranges from about 7.6% in Northern Ireland to 16.4% in Wales (Whittaker et al., cited in Usher Institute, 2025). Asthma also represents an estimated 2–3% of primary care consultations, around 60,000 hospital admissions and 200,000 bed-days every year, and it links with an average of four deaths per day in the UK, with two-thirds of those thought to be preventable (Asthma + Lung UK, 2025).

2. Physiology of the Respiratory System

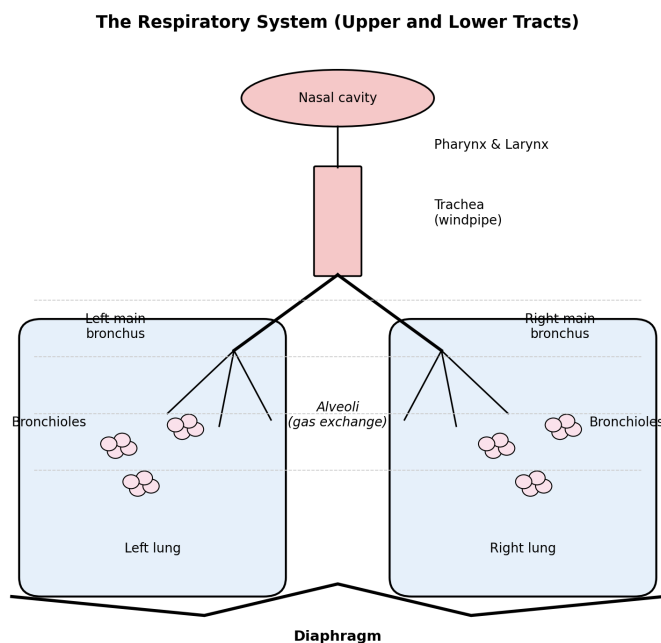


Figure 1: The upper and lower respiratory tract (author's own diagram, adapted from Tortora and Derrickson, 2021).

The upper respiratory tract kind of includes the nasal cavity, pharynx and larynx, kind of. So air that enters the nasal cavity gets warmed, made more humid, and filtered by a ciliated, mucus-secreting epithelium before it goes on to travel through the pharynx, and then the larynx, and after that into the trachea (Marieb and Hoehn, 2022). The lower respiratory tract

really starts at the trachea, which is a flexible tube supported by C shaped hyaline cartilage rings that help keep it from collapsing during pressure shifts. Then the trachea split into the left and right main bronchi, then the branches go on to become smaller bronchi, and later into bronchioles.

One key anatomical detail for asthma is that even though the larger bronchi have cartilage support, the bronchioles do not. Their walls are mainly made up of smooth muscle, and that smooth muscle is under autonomic nervous control (Tortora and Derrickson, 2021). Parasympathetic, (vagal) stimulation acting on M3 muscarinic receptors results in bronchoconstriction. Meanwhile sympathetic stimulation at β_2 -adrenergic receptors causes bronchodilation. Since bronchiolar calibre depends mostly on smooth muscle tone rather than on firm cartilaginous scaffolding, these air passages end up being much more prone to narrowing whenever the smooth muscle contracts too much, which is basically what happens in asthma.

The bronchioles end up in alveolar ducts and little alveoli, those tiny thin-walled sacks that are basically where gas exchange happens. The alveolar walls are made mostly of flat type I pneumocytes, which allow quick diffusion, and type II pneumocytes, which release surfactants to cut down surface tension, and so the alveoli don't collapse (Guyton and Hall, 2020).

Ventilation, meanwhile, is more mechanical than it sounds, it is powered by the diaphragm and the intercostal muscles. During inspiration the diaphragm contracts and kind of flattens, and the external intercostals lift the ribcage, which increases thoracic volume. With Boyle's Law in mind, that expansion drops intrapulmonary pressure below atmospheric levels, so air is pulled into the lungs. Expiration when you are at rest is mostly passive, the diaphragm just relaxes, and the elastic recoil of lung tissue pulls thoracic volume down, which then increases intrapulmonary pressure and pushes air out (Guyton and Hall, 2020). But in an asthma flare-up, expiration tends to become active and drawn out, because accessory muscles get recruited to beat the higher airway resistance that comes from bronchoconstriction.

3. Physiological Process: Gas Exchange

Gas Exchange at the Alveolar-Capillary Membrane

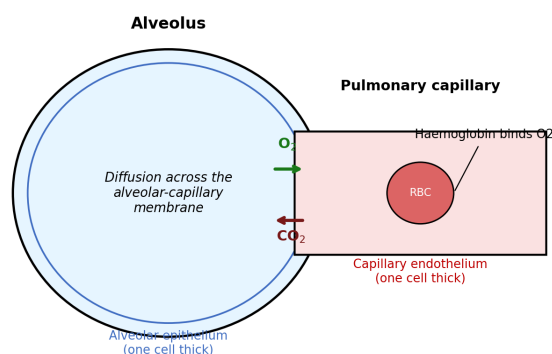


Figure 2: Diffusion of oxygen and carbon dioxide across the alveolar-capillary membrane (author's own diagram).

The alveolar capillary membrane is kind of perfectly made for diffusion really, it's less than one micrometre in thickness, and it gives a large, combined surface area, about 50–70 m² when you add both lungs together (West and Luks, 2020). Oxygen moves by diffusion from alveolar air, where its partial pressure sits high, into pulmonary capillary blood, where partial pressure is lower in comparison. After it enters the blood, most oxygen reversibly attaches to haemoglobin inside red blood cells, creating oxyhaemoglobin, and then this is ferried out to peripheral tissues so it can help with aerobic metabolism. Carbon dioxide as a sort of metabolic by-product diffuses along its own concentration gradient from the blood toward the alveoli and then during expiration it is taken away, directly (Guyton and Hall, 2020).

What also matters a lot for efficient gas exchange is how well ventilation lines up with perfusion. Ventilation means airflow reaching the alveoli, perfusion means blood moving through pulmonary capillaries, and together this is often called the ventilation perfusion or V/Q ratio. In asthma you can get widespread but uneven bronchoconstriction, so airflow drops in some alveolar units, while blood flow keeps going for the most part. That imbalance causes V/Q mismatch. When alveoli are poorly ventilated, they end up receiving blood that doesn't get properly oxygenated, and during more severe or longer attacks this may swing into hypoxaemia. So, there is this clear cause-and-effect relationship between mechanical airway narrowing, and impaired gas exchange all the way down at the cellular level (West and Luks, 2020).

4. Disease Mechanism: Asthma Pathophysiology

Asthma pathophysiology, kind of centres on chronic airway inflammation and yes, this is now recognised in the 2024 BTS/NICE/SIGN guideline as the main diagnostic and therapeutic target. Objective markers, like blood eosinophil count and fractional exhaled nitric oxide (FeNO), are used to back up eosinophilic airway inflammation (British Thoracic Society, NICE and Scottish Intercollegiate Guidelines Network [BTS/NICE/SIGN], 2024).

In allergic, or atopic, asthma exposure to an allergen basically starts off a T-helper type 2 (Th2) driven immune response and then cytokines are released including interleukin (IL) 4, IL 5 and IL 13. These cytokines steer immunoglobulin E (IgE) production forward, they also support mast cell and eosinophil recruitment. That recruitment kind of keeps the whole inflammatory cycle going, so the airway inflammation doesn't really turn itself off (Kumar, Abbas and Aster, 2021). When mast cells degranulate, histamine, plus leukotrienes are released and those mediators act on the small airways, directly influencing bronchiolar smooth muscle, which drives bronchoconstriction. On top of that parasympathetic activity at M3 receptors is increased, mainly via acetylcholine, and that adds yet another push toward airway narrowing.

Then the chronic inflammation role doesn't really stop there, it also helps goblet cell enlargement and submucosal gland hypertrophy, and it results in excess, thick mucus that can plug up small airways which are already kind of narrowed. In the longer-term disease, especially if control is poor, structural changes inside the airway may show up, and this is often referred to as airway remodelling: there's subepithelial fibrosis, smooth muscle hypertrophy, and also angiogenesis. Over time, these kinds of changes can lead to a more fixed, less reversible decline in airflow. (Kumar, Abbas and Aster, 2021; Guyton and Hall, 2020).

5. Monitoring and Recording Data

Effective monitoring helps, in an underlying way, with diagnosis and with long-term self-management of asthma. Peak expiratory flow monitoring, or PEF monitoring, using a simple handheld peak flow meter is cheap, and it lets patients match their numbers against their own personal best. The 2024 BTS/NICE/SIGN guideline notes that PEF variability of 20% or more over a two-week stretch supports a diagnosis of asthma, particularly if spirometry is hard to access (BTS/NICE/SIGN, 2024). The main catch is that it's effort dependent, so if technique is off even a little the readings can drift, reliability then goes down, especially in children or when someone is in a bout of acute distress.

A symptom diary works differently, it helps patients and clinicians detect looping trends, possible triggers, and how frequently flare ups show up over time, which supports more tuned daily management. But it relies a lot on patient buy in and on careful self-reporting, so it can wobble if people are rushing to write entries, or if notes are forgotten now and then. The Personalised Asthma Action Plan, often shortened to PAAP, is a written and individualised roadmap that pulls together symptoms and/or PEF limits, with very explicit instructions for how to tweak medication and when to seek extra assistance. Asthma + Lung UK (2025) reports that people who keep a well organised action plan are about four times less likely to be hospitalised because of asthma, so overall this style of monitoring plan is among the most evidence supported tools around.

The refreshed NICE guideline (NG245) kind of nudges clinicians toward Maintenance and Reliever Therapy, often tagged as MART, sometimes also called SMART, where one inhaler that sort of brings together an inhaled corticosteroid with a fast-acting formoterol is used for both day-to-day maintenance and when symptoms pop up. This is intended for people 12 years old and over; it makes the routine easier and it has been shown to lower how often exacerbations happen compared with older fixed dose setups (BTS/NICE/SIGN, 2024).

But importantly, while these approaches are evidence-based and they support patient empowerment, plus earlier recognition of worsening, in real life the benefit is held back by adherence issues, problems with inhaler technique, and uneven access to those scheduled review check-ups. Asthma + Lung UK's (2025) national survey reported that almost three quarters of people hit some kind of barrier to diagnosis, and that only around one third got the recommended basic standard of care. That basically highlights a stubborn gap between what the guideline says and what plays out in day-to-day services.

6. Evidence-Based Lifestyle Recommendations

Current NICE guidance NG245, plus the supporting evidence sorts out a bunch of modifiable lifestyle factors that can improve asthma control, and lower the risk of exacerbation, in a direct way. Smoking cessation is strongly recommended; smoking tends to make symptom control worse, it also speeds up the decline in lung function and seems to reduce responsiveness to inhaled corticosteroids, which means clinicians should keep asking about smoking and vaping, and then signpost to NHS stop smoking services (BTS/NICE/SIGN, 2024). Weight management is just as key, because obesity is independently tied to worse asthma control, through mechanical restriction of lung expansion, as well as pro-inflammatory adipokine activity; and even small weight loss has been shown to ease symptoms and cut down the amount of medication people need. Regular physical activity should be encouraged too, mostly for the broader cardiorespiratory benefits, and if exercise induced bronchoconstriction is known to be a trigger, then a short acting reliever should be used before exercise, rather than waiting until symptoms start.

With diet, the proof isn't quite as rock-solid as it is for some other risk factors, but it does sort of point to the idea that eating patterns rich in fruit, vegetables and omega 3 fatty acids might nudge modest anti-inflammatory effects and also could help people keep a healthy weight. NICE 2024 (though).

On top of that, cutting back on exposure to environmental triggers is part of the advice too, including common allergens, damp mould, and indoor air pollutants, such as wood burning stoves. Asthma + Lung UK 2025 said that more than three quarters of people with a lung condition felt that air pollution touched their day-to-day life, which really backs up the notion that environmental changes matter alongside personal behaviour tweaks, not instead of them (BTS/NICE/SIGN 2024).

7. Conclusion

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