

Immunity

A-Level Biology 9700 · Topic 11 revision notes

Everything from the 4 lessons of this topic on a few pages: the key idea of each lesson, the diagram to remember, the points to learn and the traps examiners set. Read it, cover it, test yourself.

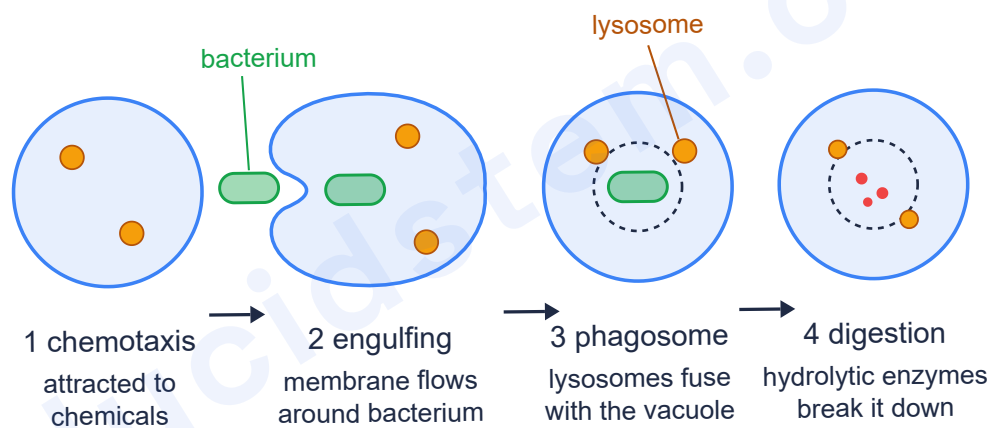
1. Phagocytes and antigens
2. Lymphocytes and the immune response
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1 Phagocytes and antigens

Syllabus 11.1

Phagocytes are white blood cells made in the bone marrow that engulf and digest pathogens. **Neutrophils** are short-lived, very numerous and the first to arrive at an infection; **macrophages** are larger, longer-lived cells that develop from monocytes in the tissues and, after digesting a pathogen, **present** its antigens to lymphocytes. Phagocytosis is **non-specific**: it is attracted by chemicals (chemotaxis), the pathogen is engulfed into a phagosome, lysosomes fuse with it and hydrolytic enzymes digest it. An **antigen** is a molecule, usually a protein or glycoprotein on a cell surface, that stimulates an immune response. **Self antigens** are the body's own and are tolerated; **non-self antigens** are foreign and trigger a response.



phagocytosis by a neutrophil or macrophage: non-specific

Fig. 1: the four stages of phagocytosis

Non-specific first, specific later.

- ✓ **Chemotaxis**, attach, engulf into a **phagosome**, lysosomes fuse, hydrolyse.
- ✓ **Neutrophils**: many, short-lived, first to arrive.
- ✓ **Macrophages**: long-lived, **present antigens** to lymphocytes.
- ✓ **Antigen** = molecule that triggers a response; **self** tolerated, **non-self** attacked.

Watch out!

Phagocytosis is **non-specific**: the same neutrophil will engulf any pathogen it recognises as non-self. Do not say a phagocyte has receptors for one specific antigen; that is true of lymphocytes, not phagocytes.

An antigen is not a pathogen. A pathogen carries many different antigens, and a single antigen is the molecule that a lymphocyte receptor or an antibody binds to.

2 Lymphocytes and the immune response

Syllabus 11.1

In a **primary immune response** a **macrophage** engulfs a pathogen and presents its antigens. The one **T-helper cell** with complementary receptors binds (**clonal selection**) and divides by mitosis (**clonal expansion**); its clones release **cytokines**. The **B-lymphocyte** with complementary receptors, stimulated by cytokines, divides into a clone that differentiates into **plasma cells**, which secrete specific antibodies, and **memory cells**. Cytokines also activate **T-killer cells**, which destroy infected body cells displaying the antigen. Because selection and cloning take days, the primary response is slow. **Memory cells** stay in the blood for years, so a second exposure to the same antigen triggers a **secondary response** that is faster and much larger: this is long-term immunity.

antibody concentration in the blood

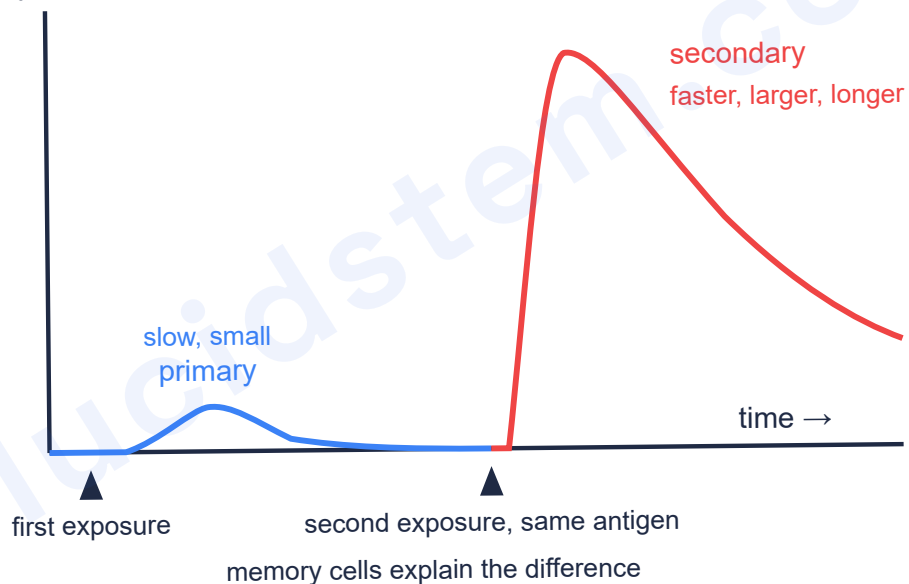


Fig. 1: antibody concentration in the primary and secondary immune responses

Every step has a named cell.

- ✓ **Macrophage** presents; **T-helper** selected, cloned, releases cytokines.
- ✓ **B cell** selected and cloned; becomes **plasma cells** + **memory cells**.
- ✓ **T-killer** cells destroy infected body cells.
- ✓ **Secondary** response: sooner, larger, longer, thanks to memory cells.

Watch out!

Clonal selection is not the lymphocyte "making" a receptor to fit the antigen. The lymphocyte with a complementary receptor already exists before the infection; the antigen only selects it.

Long-term immunity is due to **memory cells**, not to antibodies staying in the blood. Antibodies are broken down within weeks; memory cells remain.

3 Antibodies and monoclonal antibodies

Syllabus 11.2

An **antibody** is a globular glycoprotein made by plasma cells. It has **four polypeptide chains**, two long **heavy chains** and two short **light chains**, held together by **disulfide bonds**. The tips form two **variable regions** whose **antigen-binding sites** are complementary to one specific antigen; the **constant region** is the same for a class of antibodies and binds to receptors on phagocytes; a **hinge region** gives flexibility. Antibodies cause **agglutination**, **neutralise** toxins and viruses and mark pathogens for phagocytosis (**opsonisation**). **Monoclonal antibodies**, identical antibodies from one clone, are made by the **hybridoma method**: B cells from an immunised mouse are fused with myeloma cells, and the hybridoma making the wanted antibody is selected and cloned. Because they bind one antigen, they are used to **diagnose** disease (pregnancy tests, locating cancers) and to **treat** it (targeting cancer cells).

antigen-binding site
(variable region)

antigen-binding
site

light chain

heavy chain

disulfide bonds

hinge region

constant region
(binds phagocyte receptors)

four polypeptide chains: quaternary structure

Fig. 1: the structure of an antibody molecule

Link every part to a job.

- ✓ **Variable region**: antigen-binding site, complementary, two per antibody.
- ✓ **Constant region**: binds phagocytes; **hinge**: flexibility; **disulfide bonds**: hold chains.
- ✓ **Hybridoma** = B cell + myeloma: immunise, fuse, screen, clone.
- ✓ **Diagnosis**: detect one antigen; **treatment**: target one cell type.

Watch out!

The antigen-binding site is in the **variable region**, made by both a heavy and a light chain. Do not put the binding site on the constant region, and do not call the variable region the "active site": that term is for enzymes.

A hybridoma is a fusion of a **B-lymphocyte** (or plasma cell) with a **myeloma** cell. Answers that fuse "a B cell with a T cell" or "two plasma cells" lose the mark.

4 Vaccination and types of immunity

Syllabus 11.2

Active immunity develops when your own lymphocytes respond to an antigen, making plasma cells, antibodies and **memory cells**: it takes time but is long-lasting. **Passive immunity** is gained when antibodies made by another organism are received: it is immediate but short-lived, because the antibodies are broken down and no memory cells form. Immunity is **natural** when it comes from normal life (an infection, antibodies across the placenta or in breast milk) and **artificial** when it comes from medical intervention (a vaccine, an injection of antibodies). A **vaccine** contains antigens, as dead or weakened pathogens, toxoids, purified proteins or mRNA, that stimulate a primary response and memory cells without causing disease, giving **long-term immunity**. **Vaccination programmes** control the spread of disease because when a high proportion of people are immune, the pathogen rarely reaches a susceptible person: **herd immunity** protects even those who cannot be vaccinated.

	natural	artificial
active own plasma + memory cells	catching the disease	vaccination (antigens injected or swallowed)
passive antibodies given, no memory cells	antibodies across the placenta or in breast milk	injection of antibodies (for example antitoxin)

active: long-term, slow to start; passive: immediate but short-lived

Fig. 1: the four types of immunity

Who made the antibodies?

- ✓ **Active**: you made them, memory cells, long-term.
- ✓ **Passive**: given to you, no memory cells, short-term.
- ✓ **Natural** = everyday life; **artificial** = medical intervention.
- ✓ **Vaccine** = antigens; **herd immunity** = enough people immune to stop spread.

Watch out!

Passive immunity is short-lived **because no memory cells are made**, not because the antibodies are "weaker". Always mention memory cells when you compare active and passive immunity.

Herd immunity does not mean every person is immune. It means enough people are immune that the pathogen cannot keep spreading.