

5.1 Defence mechanisms

<b>Pathogen</b>	a microorganism that can cause an infectious disease
<b>Immunity</b>	the ability of an organism to resist a particular infection or toxins by antibodies or white blood cells
<b>Infection</b>	process of a pathogen invading and multiplying in the body tissue of the host
<b>Antigen</b>	a foreign protein found on the cell surface membrane which triggers an immune response
<b>Phagocytosis</b>	mechanisms by which cells engulf particles to form a vesicle or a vacuole
<b>Phagocytes</b>	ingest and destroy the pathogen
<b>Lymphocytes</b>	cells involved in immune responses
<b>Antigen-presenting cell</b>	cells that display foreign antigens on the surface
<b>Monoclonal antibody</b>	a collection of identical antibodies cloned from a single antibody
<b>Vaccination</b>	introduction of a vaccine containing appropriate disease antigens injected into the body in order to induce artificial immunity
<b>Antibody</b>	a protein produced by lymphocytes (B cells) in response to the presence of a specific antigen which stimulates an immune response

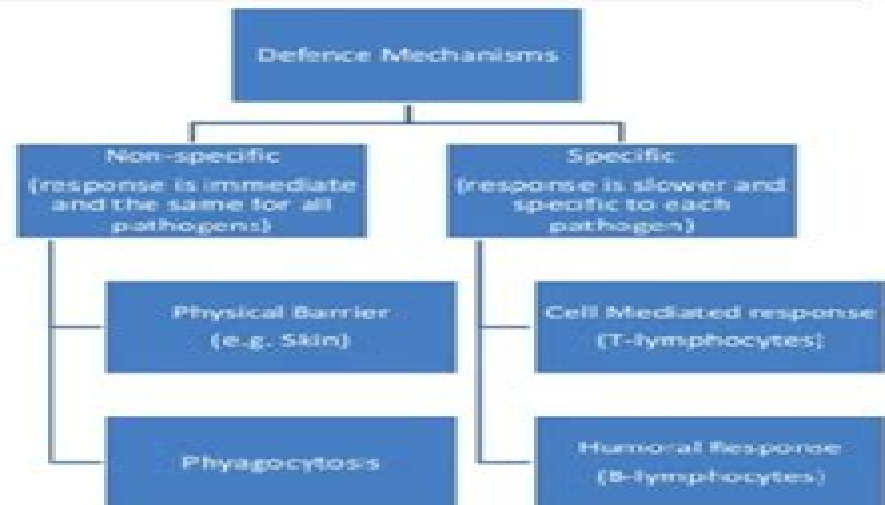
**Recognising your own cells**

- If the lymphocytes couldn't identify self from non-self-molecules then the lymphocytes would destroy the organism's own tissue.
- Each cell has specific molecules on its surface to identify it, this is why a transport donor has to be genetically similar to the recipient or the organ might be rejected and the person will die

Humoral response secretes antibodies; cell-mediated response secretes cytokines (signals cells about a potential pathogen)

**How does the body learn to identify its own molecules?**

- In the fetus lymphocytes are always colliding with other cells
- Infections are rare as the fetus is protected by a placenta
- Lymphocytes will collide mostly with own material
- Some lymphocytes will have receptors that fit exactly its own material
- These are there for suppressed or die
- In adults lymphocytes produced in the bone marrow encounter self-antigens at the start
- Any lymphocytes that show an immune response will then undergo programmed cell death (apoptosis) before they mature



5.2 Phagocytosis

**The process of phagocytosis**

1. Chemical products of pathogen act as attractants causing phagocytes to follow it
2. Phagocytes have several receptors on their cell surface membrane that recognize and attach to chemicals on the surface of the pathogen
3. The phagocyte engulfs the pathogen to form a vesicle (phagosome)
4. Lysosomes move in to fuse with the vesicle
5. Lysozymes destroy ingested bacteria by hydrolyzing the pathogen's cell walls
6. The soluble products from the breakdown of the pathogen are absorbed into the cytoplasm of the phagocyte
7. The antigen is then presented on the cell surface membrane of the phagocyte

**2 types of white blood cells:**

- Phagocytes (Ingest destroy)
- Lymphocytes (Involved in immune responses)

5.3 T lymphocytes and cell-mediated immunity

**Lymphocytes**

- produced by stem cells in the bone marrow

**The stages of T cells in response to a pathogen:**

1. The pathogens invade body cells or are taken in by phagocytosis
2. Phagocyte becomes an antigen presenting cell
3. Receptors on a specific helper T cell fit exactly onto those antigens
4. The attachment activates T cells to divide by mitosis and clone itself
5. The cloned T cells:

# A2 Level A Level Biology

**A Gutmann**



**A2 Level A Level Biology:**

## **A2 Level A Level Biology** Book Review: Unveiling the Magic of Language

In a digital era where connections and knowledge reign supreme, the enchanting power of language has become much more apparent than ever. Its capability to stir emotions, provoke thought, and instigate transformation is actually remarkable. This extraordinary book, aptly titled "**A2 Level A Level Biology**," compiled by a very acclaimed author, immerses readers in a captivating exploration of the significance of language and its profound effect on our existence. Throughout this critique, we shall delve into the book's central themes, evaluate its unique writing style, and assess its overall influence on its readership.

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