The immune response, immunity and antibiotics

Antigens and antibodies, B and T cells, types of immunity, the evolutionary race, antibiotics and resistance, and the control of hospital-acquired infections.

The specific immune response

Antigens are molecules (usually proteins or glycoproteins) on the surface of cells that trigger an **immune response**. The body recognises its own antigens (**self**) and responds to **non-self** antigens on pathogens, transplants and cancer cells.

Antibodies are **proteins** (immunoglobulins) with four polypeptide chains and a **variable region** that is **specific** to one antigen. Each antibody binds to its complementary antigen.

Steps in the response

1. A **macrophage** engulfs a pathogen and displays its antigens on its surface, becoming an **antigen-presenting cell**.
2. A **helper T cell** with a complementary receptor binds to the antigen-presenting cell and is activated, and **divides** (clonal selection and expansion), releasing **cytokines**.
3. The helper T cells stimulate **B cells** with the matching antibody on the surface; these divide by **mitosis** into **clones**.
4. The B cells differentiate into **plasma cells** (effectors), which secrete **large numbers of antibodies** (many ribosomes and rough ER), and **memory cells**.
5. **Antibodies** bind to antigens and: **agglutinate** the pathogens (clump them together), **neutralise toxins**, **block** the entry of viruses into cells, and **act as markers** for phagocytes (opsonisation).

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B cells and T cells

Cell	Role
B effector (plasma) cells	Secrete antibodies (**humoral** immunity)
B memory cells	Long-lived. Give a **faster, stronger secondary response**
T helper cells	Release cytokines that stimulate B cells and T killer cells
T killer (cytotoxic) cells	**Destroy** body cells infected with a virus (and cancer cells) by releasing **perforin** that makes holes in the membrane (**cell-mediated** immunity)
T memory cells	Long-lived; provide memory for the cell-mediated response

The **primary response** is slow (a few days) and produces few antibodies. The **secondary response** on re-infection is **faster** and **larger**, because of the memory cells, so often no symptoms occur.

Immunity

Type	Meaning	Example
Natural active	The body makes its own antibodies after **infection**	Recovering from chickenpox
Artificial active	After **vaccination** with a dead, weakened or part of a pathogen (antigen); memory cells form	MMR vaccine
Natural passive	Antibodies **passed** from mother to baby across the placenta or in breast milk	Newborn protection
Artificial passive	Antibodies **injected**	Anti-venom, tetanus antibody injection

Passive immunity is **immediate** but **short lived** and gives no memory, because no memory cells form.

The evolutionary race

Pathogens and their hosts are in an **evolutionary race**: selection pressure on hosts favours those with better defences, and selection pressure on pathogens favours those that can **evade** them. Examples of **evasion mechanisms**:

- **HIV** mutates rapidly and **infects helper T cells**; the antigens it displays change, so antibodies are no longer effective.
- **Influenza** has **antigenic variation** (new strains need a new vaccine).
- ***Mycobacterium tuberculosis*** survives **inside macrophages**; it has a waxy coat.

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- Some bacteria have **capsules** that hide their antigens from phagocytes.

Antibiotics

Antibiotics are chemicals that **kill** or **inhibit** bacteria, with little harm to human cells (selective toxicity). They are not effective against viruses, which have no cell wall or metabolism of their own.

- **Bactericidal** antibiotics **kill** bacteria, such as **penicillin**, which inhibits formation of the cell wall, so cells **burst by osmosis** (lysis).
- **Bacteriostatic** antibiotics **stop** bacteria from multiplying (without killing them), for example by inhibiting protein synthesis. The immune system then clears the infection. An example is **tetracycline**.

Antibiotic resistance

Mutation can give a bacterium **resistance**. When antibiotics are used, **resistant bacteria survive**, reproduce, and pass on the resistance gene (including by plasmids), by natural selection. **MRSA** is an example.

Core Practical 14: effect of antibiotics

1. Spread a **bacterial lawn** on agar using aseptic technique.
2. Place **discs** containing different antibiotics (and a control disc of sterile water) on the lawn.
3. Incubate (25 °C) for 24–48 hours.
4. Measure the **diameter of the zone of inhibition**: a larger zone means the bacteria are more sensitive to that antibiotic.

Hospital-acquired infections

Contributory causes of **hospital-acquired (nosocomial) infections**: the overuse of **antibiotics** which selects for resistant strains, **poor hygiene** (hand washing), **crowding**, patients with **weakened immune systems**, invasive procedures, and the spread of bacteria on equipment and staff.

Codes of practice for **infection prevention and control**:

- **Hand washing** and alcohol gels, and clean equipment and wards,
- **Isolation** of infected patients and **screening** (e.g. for MRSA),
- **Restricted antibiotic prescribing**: use only when needed, choose a narrow-spectrum antibiotic, **complete the full course**,
- Staff and visitors wear protective clothing.

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