Edexcel (A) Biology A-level

Topic 6: Immunity, Infection and Forensics

Notes

Forensics and Time of Death

Time of death of a mammal can be determined by looking at the following:

- **Extent of decomposition** – bodies usually follow the same **pattern of decay and decomposition,** and therefore a stage of decomposition can be used to determine how long a body has been dead for.
- **Stage of succession** - as the body decays, **the species colonising the body change.** Therefore, analysis of the community of species present can be used to determine time of death.
- **Forensic entomology** – each species of insects has a **specific life cycle.** Determining the age of insects present enables the time of death to be determined.
- **Body temperature** – temperature of the body begins to decrease after death as **heat-producing metabolic reactions stop.** However, temperature can only be used to determine time of death in the first 24 hours, until the body reaches the temperature of its surroundings.
- **Degree of muscle contraction** – after death muscles begin to stiffen. This is called **rigor mortis,** and the extent of rigor mortis can be used to determine time of death. However, the stiffness only lasts around 36 hours, so is only useful to an extent in determining time of death.

Microorganisms such as **bacteria and fungi** play an important role in the **decomposition of organic matter and the recycling of carbon**. Bacteria and fungi **secrete enzymes** that **decompose dead organic matter** into small molecules which they then use as **respiratory substrates** – **carbon dioxide and methane** are released in this process, thus recycling carbon.

DNA Profiling

DNA profiling is a forensic technique used for **identification and determining genetic relationships** between organisms:

1. Fragments of DNA are cut with **restriction endonuclease enzymes** (either side of satellites).
2. These fragments are separated and visualised using **gel electrophoresis** - fragments are placed in wells in agarose gels and dyed with ethidium bromide so they fluoresce under UV light. A current is then applied to the gel. DNA is negative but fragments of different sizes move differently according to mass so 'bands' appear.
3. **Southern Blot** - alkaline buffer solution added, nylon filter - dry absorbent material draws solution containing DNA fragments to the filter - fragments visible as 'blots'. Gene probes (complementary sequences labeled with genes like fluoresce or radioactivity) are added and bind with DNA (hybridisation).

4. 'Blots' compared and number of satellites visualised as a graph (repeated sequences of DNA in introns are referred to as **mini/microsatellites** depending on their size. The more closely related two people/species are, the **more similar the repeats** are).

Before the sample can be analysed via DNA profiling, the sample needs to be **amplified** using **Polymerase Chain Reaction**:

1. A reaction mixture is set up by mixing the **DNA sample, primers, free nucleotides and DNA polymerase** which is the enzyme involved in creating new DNA strands.
2. The mixture is then **heated to 95 degrees** to break the hydrogen bonds and to separate the two strands.
3. The mixture is then **cooled to a temperature between 50-65 degrees** depending on the type of primers used, so that they can bind to the strands.
4. Temperature is increased to about **70 degrees** as this is the temperature DNA polymerase works at.
5. **DNA polymerase** creates a copy of the sample by **complementary base pairing using the free nucleotides**
6. **This cycle is repeated around 30 times** and gives rise to an amount of DNA sufficient to create a DNA profile.

Bacteria and Viruses

Viruses are **non-living structures** which consist of a **nucleic acid** (either DNA or RNA) enclosed in a protective protein coat called the **capsid**, sometimes covered with a lipid layer called the **envelope**.

Bacteria and viruses are the main disease-causing pathogens in humans. Even though they both cause disease, they vary in many ways:

- Bacteria are prokaryotic cells, meaning that they **do not have a nucleus** – their genetic material is stored in the form of a circular strand of DNA. Viruses consist of just nucleic acid (DNA or RNA) enclosed in a protein coat.
- Bacteria **do not require a host** to survive, whereas viruses are entirely dependent on their hosts and cannot survive without them.
- Viruses are **significantly smaller** than bacteria.
- Bacteria have a **cell membrane, cell wall and cytoplasm**, as well as other organelles such as **ribosomes, plasmids, flagellum and pili**. Viruses possess no such structures.

An example of a bacterial disease is **tuberculosis** (TB). TB is caused by a bacteria called ***Mycobacterium tuberculosis*** which infects phagocytes in the lungs:

- First infection is symptomless. Infected phagocytes are sealed in **tubercles** as a result of an **inflammatory response** in the lungs.
- Bacteria lie **dormant** inside the tubercles. They are not destroyed by the immune system, as tubercles are covered with a thick **waxy coat**.
- When the immune system becomes weakened, the bacteria become active again, and slowly destroy the lung tissue, thus leading to breathing problems, coughing, and weight loss, as well as fever**.**
- TB can be **fatal**.

An example of a viral infection is **Human Immunodeficiency Virus** (HIV), which leads to AIDS:

- The first symptoms of HIV include fevers, tiredness and headaches.
- After several weeks **HIV antibodies** appear in blood, thus making a person HIV positive.
- After this period, the symptoms disappear until the **immune system becomes weakened** again, thus leading to AIDS.
- Symptoms of AIDS include weight loss, diarrhoea, dementia, cancers and opportunistic infections such as TB.

Response to Infection

Physical barriers to infection include:

- **Skin** is a tough physical barrier consisting of **keratin**.
- **Stomach Acid** (hydrochloric acid) which **kills bacteria.**
- **Gut and skin flora –** natural bacterial flora **competes with pathogens** for food and space.

Non-specific responses of the body to infection include:

- **Inflammation –** histamines released by damaged white vessels cause vasodilation, which increases the flow of blood to the infected area and increases permeability of blood vessels. As a result of that antibodies, white blood cells and plasma leak out into the infected tissue and destroy the pathogen.
- **Fever –** the hypothalamus sets body temperature higher. This decreases speed of pathogen reproduction and increases rate of specific immune response.
- **Lysozyme action –** lysozyme is an enzyme found in secretions such as tears and mucus which kills bacterial cells by damaging their cell wall

- **Phagocytosis** is a process in which white blood cells engulf pathogens thus destroying them by fusing a pathogen such as bacteria enclosed in a phagocytic vacuole with a lysosome.

The **specific immune response** is antigen-specific and produces responses specific to one type of pathogen only. This type of immune response relies on **lymphocytes produced in the bone marrow**:

- **B cells** mature in the bone marrow and are involved in the **humoral response.**
- **T cells** move from the bone marrow to the thymus gland where they mature, they are involved in **cell-mediated response**.

Cell Mediated Response

Pathogen invades a host cell.

1. The host cell displays the antigens on its **Major Histocompatibility Complexes** and becomes an **Antigen-Presenting Cell**.
2. **T Killer cell** with complementary receptor proteins binds to the APC.
3. **Cytokines** secreted by active T Helper cell stimulates the T Killer cell to divide by mitosis.
4. T Killer cell divides to form **active T Killer cells** and T Memory cells.
5. Active T Killer cells bind to APCs and secrete chemicals which cause pores to form in the cell membrane.
6. The infected cell dies.

Humoral Response

T Helper Activation:

1. Bacterium is engulfed by a **macrophage**. Surface antigens are passed along the endoplasmic reticulum into a vesicle which are transported to the cell surface membrane.
2. Macrophage acts as an APC and presents antigens on MCPs.
3. Macrophage APC binds to T Helper cell with **complementary receptor proteins**.
4. The T Helper cell is 'activated' and divides by mitosis to form T memory cells and active T helper cells.

Effector Stage:

1. Antigens from APCs that are **complementary to the antibodies on B cells** bind and are taken in by endocytosis.
2. The B cell acts as an APC and presents antigens on MCPS.
3. An activated T helper cell (from the previous stage) with a complementary receptor protein to the antigens binds to the APC. It produces **cytokines**.
4. Cytokines stimulate the B cell to divide by mitosis and form B memory cells and **B effector cells**.
5. B effector cells differentiate into **plasma cells**.
6. Plasma cells synthesise antibodies.
7. Effects of antibodies:
    a. Agglutination (microbes clump together – makes phagocytosis easier)
    b. Lysis (bursting of bacterial cells)
    c. Opsonisation (antibodies coat microbes and mark them for phagocytes)
    d. Precipitation/Neutralisation (soluble toxins are made insoluble)
8. T Suppressor cells stop the immune response.

Immunity

Immunity can either be **active or passive**; active **immunity results from the production of antibodies by the immune system** in response to the presence of an antigen whereas passive immunity results from the **introduction of antibodies from another person or animal.**

There are also two subtypes of immunity; natural or artificial:

- **Natural active immunity** arises from being exposed to an antigen/getting the disease whereas **natural passive immunity** is the result of crossing of mother's antibodies through the placenta and their presence in breast milk.
- **Active artificial immunity** is acquired through vaccinations which stimulate the immune system and lead to production of antibodies whereas **passive artificial immunity** is where antibodies are injected into the body.

Herd Immunity = enough people have been vaccinated to make transmission of a disease very unlikely. Requires 80-90% vaccination. Immunisation is the process of protecting people from infection with passive/active artificial immunity – vaccination is the process by which this is achieved through use of attenuated antigens.

- The secondary infection has **less lag** (so less time for symptoms), is more rapid and produces more antibodies and T Killer cells than the primary response because there are Memory T and B lymphocytes in circulation from the primary infection.

RNA splicing: post-transcription modification of mRNA. Eukaryotes produce more proteins than they have genes - RNA splicing explains how, because it results in different products from a single gene.

1. Gene is transcribed which results in pre-MRNA (the transcript of the whole gene).
2. **All introns** (non-coding regions) **and some exons** (coding regions) are **removed**.
3. The remaining genes are joined back up by enzyme complexes called **spliceosomes**. The same exons can be joined in a variety of ways to produce several different versions of mature functional RNA.

Antibiotics

Antibiotics can also be used to fight infection by killing the bacteria and stopping their growth. There are two types of antibiotics:

- **Bactericidal antibiotics** kill bacteria by destroying their cell wall, thus causing them to burst.
- **Bacteriostatic antibiotics,** which inhibit the growth of bacteria by stopping protein synthesis and production of nucleic acids so the bacteria can't divide and grow.

However, some bacteria become **resistant** to antibiotics as a result of **natural selection**. The bacteria which are not killed by the antibiotic possess a **selective advantage** – resistance which enables them to survive and reproduce. Therefore the allele for **antibiotic resistance** is passed onto their offspring thus creating a **resistant strain.**

Moreover, there is an ongoing **evolutionary race** between organisms and pathogens as **pathogens evolve adaptations** which enable them to survive and reproduce. For instance, the constantly changing protein coat (antigen coat) of HIV means that the virus is not recognised and destroyed by the immune system.

Resistance to antibiotics results in **antibiotic resistant bacterial infections,** sometimes referred to as 'superbugs', **in hospitals,** such as **MRSA.** Hospitals have developed various ways of controlling the spread of antibiotic resistant infections, for example:

- New patients are screened at **arrival, isolated and treated if they are** infected to prevent the spread of bacteria between patients.
- **Antibiotics are only used when needed and their course is completed** to **ensure that all the bacteria are destroyed,** and to **minimise the selection pressure** on bacteria, to prevent resistant strains from forming.

- All staff must follow the code of practice which includes **strict hygiene regimes** such as **washing hands with alcohol based antibacterial gels** and wearing suitable clothing which **minimises the transmission of resistant bacteria.**