Edexcel (A) Biology A-level

6.5 to 6.9 + 6.11 to 6.15 - Infection and the Immune System

Flashcards

Describe the structure of a bacterium.

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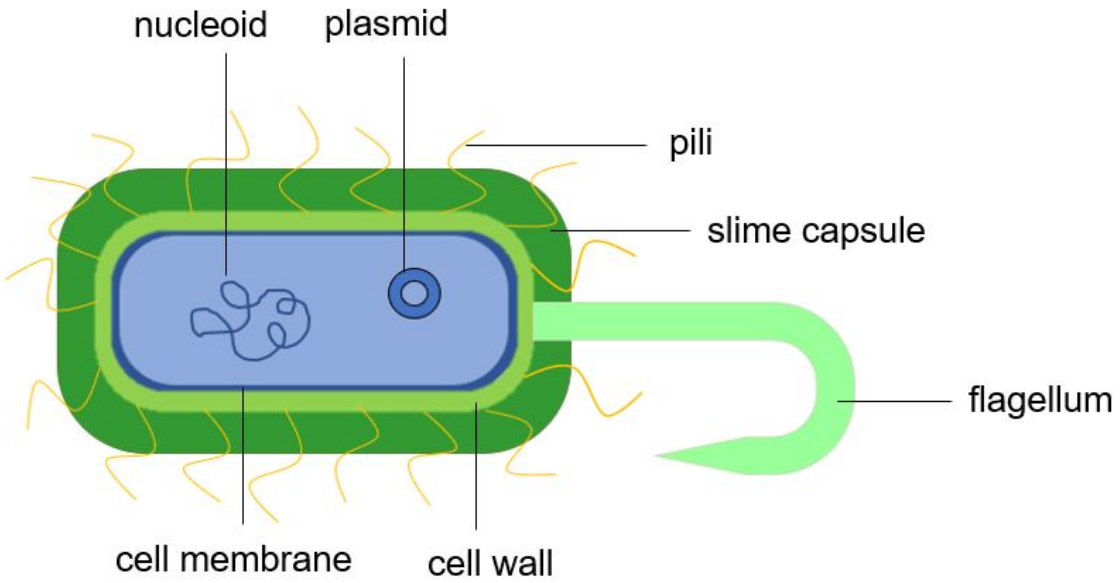

Describe the structure of a virus.

Describe the structure of a virus.

Linear genetic material (DNA or RNA) & viral enzymes e.g. reverse transcriptase.

Surrounded by **capsid** (protein coat made of capsomeres).

No cytoplasm.

Describe the structure of an enveloped virus.

Describe the structure of an enveloped virus.

Simple virus surrounded by **matrix protein.**

Matrix protein surrounded by **envelope** derived from cell membrane of host cell.

Attachment proteins on surface.

Why are viruses referred to as ‘particles’ instead of cells?

Why are viruses referred to as ‘particles’ instead of cells?

Acellular & non-living: no cytoplasm,

cannot self-reproduce, no metabolism.

How does *Mycobacterium tuberculosis* cause disease?

How does *Mycobacterium tuberculosis* cause disease?

1. Triggers inflammatory response by infecting phagocytes in lungs.
2. Infected phagocytes are sealed in waxy-coated tubercles so bacteria remain dormant. First infection has no symptoms.
3. If another factor weakens immune system, bacteria become active & destroy lung tissue. May cause death.

How does HIV result in the symptoms of AIDS?

How does HIV result in the symptoms of AIDS?

1. Attachment proteins bind to complementary CD4 receptor on T_H cells.
2. HIV particles replicate inside T_H cells, killing or damaging them.
3. AIDS develops when there are too few T_H cells for the immune system to function.
4. Individuals cannot destroy other pathogens & suffer from secondary diseases/infections. May cause death.

What is an antigen?

What is an antigen?

Cell-surface molecule can stimulate immune response.

Usually (glyco)protein, sometimes (glyco)lipid or polysaccharide.

Immune system recognises as “self” or “non-self” = enables identification of cells from other organisms of same species, pathogens, toxins & abnormal body cells.

Name 4 ways the nonspecific immune system responds to infection.

Name 4 ways the nonspecific immune system responds to infection.

- inflammation
- phagocytosis
- digestive action of lysozymes
- production of interferon (antiviral agent)

Outline the process of inflammation.

Outline the process of inflammation.

1. Damaged vessels release histamines, causing vasodilation.
2. Blood flow & permeability of blood vessels increase.
3. White blood cells & plasma flow into the infected tissue.

How does phagocytosis destroy pathogens?

How does phagocytosis destroy pathogens?

1. Phagocyte moves towards pathogen via **chemotaxis**.
2. Phagocyte engulfs pathogen via endocytosis to form a **phagosome**.
3. Phagosome fuses with lysosome (**phagolysosome**).
4. Lysozymes **digest pathogen**.
5. Phagocyte absorbs the products from pathogen hydrolysis.

What are lysozymes?

What are lysozymes?

Digestive enzymes. Often found in secretions e.g. tears & mucus. Damage bacterial cell walls, causing osmotic lysis.

Explain the role of antigen-presenting cells (APCs).

Explain the role of antigen-presenting cells (APCs).

Macrophage displays antigen from pathogen on its surface (after hydrolysis in phagocytosis).

Enhances recognition by T_H cells, which cannot directly interface with pathogens/antigens in body fluid.

Name the 2 types of specific immune response.

Name the 2 types of specific immune response.

- cell-mediated
- humoral

Outline the process of the cell-mediated response.

Outline the process of the cell-mediated response.

1. **Complementary** T_H lymphocytes bind to foreign antigen on APC.
2. Stimulates:

a. Clonal expansion of complementary T_H cells: become **memory cells** or trigger **humoral response.**

b. Clonal expansion of **cytotoxic T cells** (T_C): secrete enzyme **perforin** to destroy infected cells.

What is clonal expansion?

What is clonal expansion?

T/B cells that are complementary to an antigen undergo rapid mitotic division to form many cloned cells.

Outline the process of the humoral response.

Outline the process of the humoral response.

1. **Complementary** T_H lymphocytes bind to foreign antigen on antigen-presenting T cells.
2. Release cytokines that stimulate clonal expansion of **complementary B lymphocytes**.
3. B cells differentiate into **plasma cells**.
4. Plasma cells secrete **antibodies** with complementary variable region to antigen.

What is an antibody? Describe its structure.

What is an antibody? Describe its structure.

proteins secreted by plasma cells

quaternary structure: 2 '**light chains**' held by disulfide bridges to the 2 longer '**heavy chains**' which are also held together by disulfide bridges.

Binding sites on **variable region** of light chains have specific tertiary structure **complementary to an antigen**.

The rest of the molecule is known as the **constant region**.

How do antibodies lead to the destruction of a pathogen?

How do antibodies lead to the destruction of a pathogen?

- Formation of antigen-antibody complex results in agglutination.
- Activation of complement.
- Opsonisation (marks microbes for phagocytes).
- Precipitation/neutralisation (makes toxins insoluble).

What are memory cells?

What are memory cells?

Specialised T_H/B cells produced from primary immune response.

Remain in low levels in the blood.

Can divide very rapidly by mitosis if organism encounters the same pathogen again.

Contrast the primary and secondary immune response.

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secondary response (compared to primary response):

- Faster rate of antibody production.
- Shorter time lag between exposure & antibody production.
- Higher concentration of antibodies.
- Antibody level remains higher after the secondary response.
- Pathogen usually destroyed before any symptoms.

Compare passive and active immunity.

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- both involve antibodies
- can both be natural or artificial

Give examples of passive and active immunity.

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Passive natural: antibodies in breast milk/ across placenta.

Passive artificial: anti-venom, needle stick injections.

Active natural: humoral response to infection.

Active artificial: vaccination.

Contrast passive and active immunity.

Contrast passive and active immunity.

Passive	Active
no memory cells & antibodies not replaced when broken down = short-term	memory cells produced = long-term
immediate	time lag
antibodies from external source	lymphocytes produce antibodies
direct contact with antigen not necessary	direct contact with antigen necessary

How do pathogens enter the body?

How do pathogens enter the body?

- Inhalation (droplet infection).
- Skin-to-skin contact or exchange of fluids.
- Consumption of contaminated food & drink.
- Penetrate skin actively using enzymes or passively through wounds, hair follicles or sweat glands.
- Via a vector e.g. mosquitoes transmit *Plasmodium* parasite.

Name 3 barriers to infection.

Name 3 barriers to infection.

- Skin is tough keratin layer.
- Hydrochloric acid in stomach kills bacteria.
- Harmless bacteria in gut & on skin surface increase interspecific competition with pathogens.

How do bactericidal antibiotics work?

How do bactericidal antibiotics work?

Prevent formation of peptidoglycan cross-links in bacterial cell wall, causing osmotic lysis.

How do bacteriostatic antibiotics work?

How do bacteriostatic antibiotics work?

- Prevent protein synthesis by binding to small subunit of ribosome so tRNA cannot attach
- and/or inhibit nucleic acid formation.

Therefore inhibit growth & division.

Why are some bacteria resistant to treatment by antibiotics?

Why are some bacteria resistant to treatment by antibiotics?

1. Random genetic mutation, often on plasmid, confers resistance e.g. antigen shape changes.
2. These bacteria have selective advantage in the presence of antibiotics, reproduce & pass allele for resistance to offspring.
3. Directional selection results in resistant strain.

How do hospitals minimise the spread of antibiotic resistant bacteria?

How do hospitals minimise the spread of antibiotic resistant bacteria?

- Screening & quarantine of affected patients.
- Hygiene code of practice e.g. alcohol-based antibacterial gels.
- Antibiotics prescribed only when necessary & course completed to minimise selection pressure.

Outline some causes of hospital-acquired infections.

Outline some causes of hospital-acquired infection.

- Close contact with infected patients (particularly increases rate of droplet infection).
- Poor hygiene: pathogen remains on hands & equipment.

How do some pathogens evade the body's defence systems?

How do some pathogens evade the body's defence systems?

- Use mucinase to destroy mucin.
- Use proteases to destroy mucosal secretions of IgA antibody & antimicrobial peptides.
- Use phospholipase to destroy cytoskeleton to prevent fusion with lysosome.
- Use urease to produce ammonia in stomach to neutralise hydrochloric acid.
- Acid-resistant capsid.

How might bacteria evade phagocytosis?

How might bacteria evade phagocytosis?

- Antiphagocytic polysaccharide capsules mask antigens.
- Leukocidins destroy leukocytes.
- Antigen variability reduces effect of opsonisation/agglutination by antibodies.

Why is there an ‘evolutionary race’ between pathogens and their host?

Why is there an ‘evolutionary race’ between pathogens and their host?

Host defences are selection pressure for bacteria.

Random genetic mutations may enable bacteria to evade these defences.

Hosts with phylogenetic characteristics that reduce likelihood & symptoms of infection have a selective advantage.