Biology A level - Communicable Diseases Cheat Sheet

by Anais (Anais_Pe) via cheatography.com/151793/cs/43598/

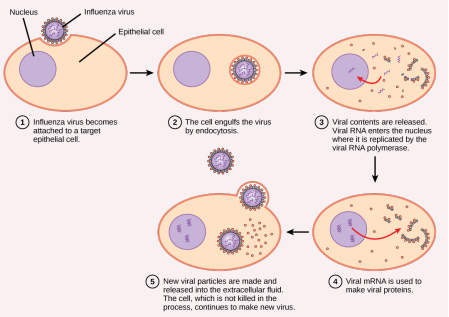
Types of pathogens

Bacteria	Prokaryotes. Classified by shape and cell walls (gram positive / negative bacteria). Gram positive bacteria (e.g. MRSA) look purple after staining, gram negative (e.g. E.coli) looks red.
Viruses	Non-living infectious agents. Rapidly reproduce. Pathogenic, attack other organisms (bacteriophages attack bacteria).
Protoctista (protista)	Eukaryotic organism
Fungi	

Viral infection

1. Attachment of virus.
2. Virus implants DNA (viral nucleic acid) in host cell.
3. Cell replicates viral nucleic acid.
4. Synthesis of viral proteins.
5. Assembly of virus particles.
6. Virus leaves cell. Lysis of host cell.

Viral infection diagram



Transmission of animal pathogens

Physical contact e.g. HIV, bacterial meningitis	Direct contact, increased risk if overcrowding, poor hygiene...
Droplet infection e.g. influenza, TB	Indirect contact, increased risk if overcrowding, poor ventilation...
Vectors e.g. malaria, bubonic plague, rabies	Indirect contact, increased risk if poor waste disposal (breeding ground for vectors), climate change...
Fomites e.g. athlete's foot, gas gangrene	Infected surfaces, indirect contact, increased risk if living in unclean conditions...
Digestion e.g. cholera, food poisoning	DIirect contact, increased risk if sewage water, poor diet / nutrition...
Innoculation e.g. malaria, rabies	Direct contact, increased risk if sharing needles...

Animal diseases

Disease	Type of pathogen	Symptoms
Bacterial meningitis	Gram neg. **bacterium**	Fever, drowsiness, light sensitivity, vomiting...

Animal diseases (cont)

Tuberculosis (TB)	Gram pos. **bacterium**	Weight loss, fever, chest pain, coughing...
HIV / AIDS	**Virus**	Fever, sore throat, rash, muscle pain...
Influenza	**Virus**	Fever, headache, dry cough, cold symptoms...
Athlete's foot	**Fungus**	Itchy red rash between toes, flaky dry skin.
Ringworm	**Fungus**	Silvery ring-like rash.
Malaria	**Protist**	Fever, sweats, vomiting, diarrhoea...

Plant diseases

Disease	Type of pathogen	Symptoms
Black sigatoka **(bananas)**	**Fungus**	Premature ripening, brown + shriveled leaves.
Tobacco Mosaic Virus (TMV) **(tomatoes, cucumbers...)**	**Virus**	Stunting, leaf curling, yellowing plant...
Ring rot **(potatoes, tomatoes...)**	Gram pos. **bacterium**	Vascular wilt, discolouration and loss of texture...

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Plant diseases (cont)

Potato / tomato blight	**Protist**	Shrivel + turn brown, fine white fungal growth around lesions...

Recognising attack on plants

Molecules from pathogen / products from pathogenic enzymes recognised by cell by receptors in membrane.

Signalling molecules alert nucleus to attack.

Defensive molecules directly attack

AND / OR defensive chemicals alert other cells through plasmodesmata

AND / OR strengthen cell walls with callose and lignin

Callose - polysaccharide (beta 1,3 and beta 1,6 glycosidic bonds).
- can block plasmodesmata
- can thicken infected cell walls
- seals sieve plates in phloem to isolate cell

Chemical defenses in plants

Insect repellent	e.g. pine resin, citronella
Antibacterial compound	e.g. phenols (antiseptics), antibacterial gossypol (in cotton), defensins (plant protein which disrupts bacterial / fungal mambranes)
Antifungal compounds	e.g. chitinase, saponins, antifungal gossypol, phenols

Chemical defenses in plants (cont)

Anti-oomycetes	e.g. glucanases (enzyme which breaks down glucans)
General toxins	e.g. chemicals broken down into cyanide
Insecticides	e.g. caffeine, pyrethrins (in chrysanthemums, act as insect neurotoxins)

Physical defenses in plants

Physical defenses	Waxy cuticle, cell wall...
Callose	Block sieve plates, thicken walls...

Non-specific animal defences

Keeping the pathogens out

Skin	Physical barrier, healthy microorganisms to outcompete pathogens, sebum secretion.
Mucous membranes	Contains phagocytes.
Tears / urine / acid in stomach	Contain lysozymes.
Coughing / sneezing	Ejects pathogen-containing mucus.
Vomiting / diarrhoea	Expel pathogen-containing gut contents.
Inflammatory response	Localised response to pathogen characterised by pain / heat / redness / swelling (separate block).

Non-specific animal defences (cont)

Blood clotting	Cascade reaction (seaprate block).

Destroying pathogens

Fevers	Cytokines stimulate hypothalamus to increase temperature.
Cytokines	Cell-signalling molecules.
Opsonins e.g. immunoglobin G and M (IgG / IgM)	Bind to + tag path. Phagocytes = opsonin-binding receptors.
Phagocytosis	Phagocytes = neutrophils and macrophages (separate block).

Inflammatory response and blood clotting

Inflammatory response	Mast cells released, release histamines and cytokines. **Histamines** --> make blood vessels dilate (heat and redness) + blood vessels walls become more leaky --> blood plasma is forced out and becomes tissue fluid (swelling and pain). **Cytokines** --> attract phagocytes.

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Inflammatory response and blood clotting (cont)

Blood clotting	Damaged tissue --> acivates platelets --> **thromboplastin** released. Thromboplastin + Ca^{2+} = **prothrombin** --> **thrombin** --> catalyses **fibrinogen** --> **fibrin** --> blood clot

Phagocytosis

Phagocytes attracted by chemicals produced by path.

Phagocyte recognises path. as non-self.

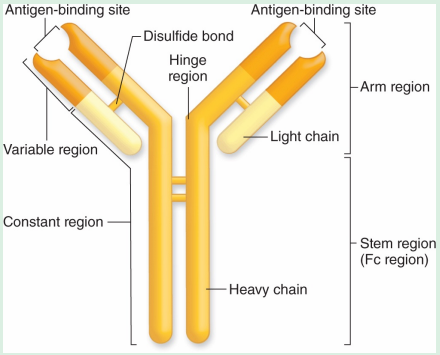
Phagocyte engulfs path. --> phagosome.

Lysozymes combines with phagosome --> phagolysosome.

Digested path. absorbed, antigen combines with MHC to form MHC complex.

Phagocyte + MHC complex = antigen-presenting cell (APC).

Antibody structure



Variable region - Changes in every antibody and is complementary to antigen.
Agglutination - Antigens bind to multiple pathogens to clump them together.
Neutralisation - Stop pathogens entering host cell OR act as antitoxins.

Cell mediated immunity - specific response

T lymphocytes respond to changed cells.

An APC is formed (phagocytosis by macrophages). T helpers bind to presented antigens.

T helper carrying the correct antibody is activated and divides by mitosis (**clonal expansion**).

T cells can then take one of four paths:

- Develop into **T memory cells**

- Produce **interleukins** to trigger phagocytosis

- Produce **interleukins** to trigger **B cell division**

- Develop into **T killer cells**

T helper cell - Produce interleukins, stimulate B cells and antibody production and attract other T cells and antibodies.
T killer cell - Kill pathogen - produce perforin which make holes in pathogen membrane.
T memory cell - Immunological memory, remain in blood for a long time. Second infection = rapid division of T killer cells.
T regulator cells - Prevent autoimmune response, repress immune system after pathogen has been destroyed.

Humoral immunity - specific response

Activated **T helper cells** bind to **B lymphocytes** presenting the correct antigen (**clonal selection**) and activates it with **interleukins**.

B cells can then become one of two things:

- Become a **B plasma cell**.

- Become a **B memory cell**.

B lymphocytes / cells - APCs, respond to antigens and APCs.
PRIMARY IMMUNE RESPONSE:
B plasma cells - Produce antibodies, act as opsonins (label cells) or as agglutinins.
SECONDARY IMMUNE RESPONSE:
B memory cells - Stay in the blood. If infected again, will divide into plasma cells.

Autoimmune diseases

Diseases	Part affected	Treatment
Rheumatoid arthritis	Joints	No cure, anti-inflam. drugs and steroids...
Lupus	Skin, joints and organs	No cure, anti-inflam. drugs, steroids...
Type 1 diabetes	Insulin-secreting parts of pancreas	Insulin injections, pancreas transplant...

Natural v. artificial, active v. passive immunity

Natural active	Secondary immune response (memory T and B cells).
Natural passive	Antibodies cross from placenta / mothers' milk.
Artificial active	Body simulated to make own antibodies (e.g. injection of weakened / dead path., isolated antigens...)
Artificial passive	Antibodies made in other animal, collected and injected.

Drug design of the future

Pharmogenetics	Personalised meds, genotype and drugs interact.
Synthetic biology	Develop bacteria to produce otherwise rare drugs, GM mammals and nanotech.

Antibiotic dilemma

Antibiotics = selective toxins, harm bacteria but not human cells.
Random mutation in bacteria could lead to antibiotic resistance --> whole population / species develops with trait (bacteria reproduce rapidly therefore antibiotic-resistant generation arrives quickly).
Specific example - *MRSA*
Research - computer models, deep sea microorganisms...
Bacterial resistance building faster than antibiotics are developed.

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