Higher Human Biology

Unit 4 Pupil Notes

Chapter 21

Body defends itself against pathogens, toxins and cancer cells by means of its Immune System

Immunity – ability to resist infection by a pathogen or to destroy it if it invades

Body has 3 lines of defence

First two are non-specific, work against any type of disease causing agent

First line of defence, mechanism employed

1. Skin (physical and chemical defence)

2. Acid secretions by stomach

3. Mucus secretion, epithelial lining of trachea

First line of defence

Skin surface composed of epithelial cells

This provides physical protection against bacteria and viruses

Mucous membranes (digestive and respiratory tracts) composed of epithelial cells forming a physical barrier

Skin and mucous membranes also produce chemical defences

Secretions from sweat and sebaceous glands keep skin at pH too low for microbes to thrive

Secretions from tears and saliva contain the enzyme lysozyme, digests bacterial walls

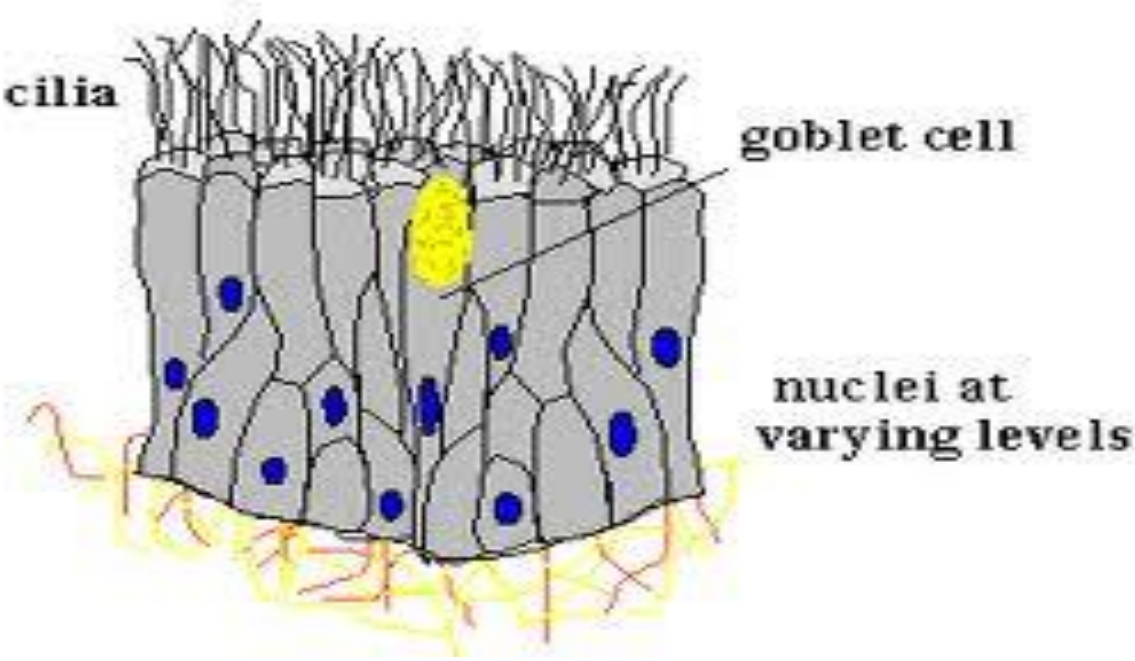
Acid

Acid is secreted by the epithelial lining of the stomach and destroys many microbes

Tracheal lining

Cells in mucous membranes secrete sticky mucus to trap microbes

Ciliated epithelial cells in trachea sweep the mucus and trapped microbes away from lungs



Second line of defence, mechanism employed

1. Inflammatory response

2. Phagocytosis

3. Natural killer cells

Inflammatory response

This is a localised defence mechanism at affected site

Mast cells, specialised immune cells, are present in connective tissue

Mast cells contain histamine which causes vasodilation and capillaries to become more permeable

Following an injury

Mast cells release histamines

Blood vessels undergo vasodilation

Capillaries become swollen with blood

Additional blood supply makes area red and inflamed

Injured area swells up because capillary walls are more permeable and leak fluid into tissues

Cytokines

'Cell signalling' proteins, stimulate movement of cells towards trauma site

Cytokines are secreted by many cells including w.b.c.

During inflammatory response

Enhanced migration of phagocytes to damaged area

Speedy delivery of antimicrobial proteins to affected site

Rapid delivery of blood clotting chemicals to injury site

Phagocytosis – phagocyte is motile

Bacterium releases chemicals

Phagocyte moves to it

Engulfs the invader in a vacuole

Lysosomes fuse with vacuole, release digestive enzymes into it

Following digestion of microbe, phagocyte releases cytokines (signalling proteins) which attract more phagocytes

Natural killer cells

NK cells are not phagocytes

They attack virus affected cells and cancer cells

NK cell releases a protein that creates pores in target cell's membrane

'Signal molecules' from the NK cell enter target cell

This causes target cell to produce self destructive enzymes (degradation enzymes)

Results in cell's DNA being broken down to useless fragments

And vital proteins being broken down into useless fragments

Cell shrinks and dies

Process of programmed cell death = Apoptosis

NK cells and phagocytes release cytokines into the blood stream to activate lymphocytes

Higher Human Biology

Unit 4 Pupil Notes

Chapter 22

Third line of Defence

3rd line of defence is the specific immune response

Brought about by lymphocytes

Lymphocytes are derived from stem cells in bone marrow

Some lymphocytes pass to thymus and develop into T lymphocytes (T cells)

Others remain in bone marrow, B lymphocytes (B cells)

Immune surveillance

A range of white blood cells move around the body in the circulatory system

If tissue is damaged or invaded some types of w.b.c. release cytokines, cell signalling protein molecules, into bloodstream

Results in large numbers of phagocytes and T cells accumulating at site

Antigen

Is a foreign molecule recognised by a lymphocyte

Antigens include –

- Viruses
- Bacteria
- Cancer cells
- Molecules on surface of transplant cells

Body has enormous number of lymphocytes

Each lymphocyte has antigen receptors on its surface

Each lymphocyte is able to become attached to one type of antigen – the lymphocyte is said to have been 'selected' by the antigen

Lymphocyte responds to selection by dividing repeatedly to form a clonal population – this is called 'clonal selection'

Antigen signature

Body cells have cell surface proteins unique to that person, their 'antigen signature'

Critical that a person's lymphocytes do not regard these cell surface proteins as antigens

T cells are able to distinguish between the body's own cells and cells with 'foreign' molecules on their surface

Autoimmunity

Body no longer tolerates the antigens that make up 'self' message on cell surfaces and T lymphocytes attack body's own cells

Causes Autoimmune Diseases

M.S. rheumatoid arthritis and type1 diabetes

Allergy

This happens when the immune system over-reacts, with B lymphocytes responding to harmless substances

Such substances include pollen, dust, feathers, Penicillin

Allergic reaction

Certain B cells are stimulated

Antibodies are produced

Antibodies become attached to 'mast' cells

Mast cells secrete histamine

Runny nose, constriction of bronchioles etc.

Two types of T lymphocytes (T cells)

Helper T cells

Helper T cells secrete cytokines

This activates phagocytes, cytotoxic T cells and B cells

Cytotoxic T cells

These destroy infected cells by inducing apoptosis

How do helper T cells activate cytotoxic T cells and B cells?

When a phagocyte has done its job, it presents fragments of the pathogen on its surface

A type of T helper cell binds to the surface of this 'antigen-presenting' cell/ phagocyte

When this happens, the helper T cell becomes 'activated', it multiplies forming a clone of activated helper T cells and a clone of memory helper T cells

The clone of activated helper T cells secrete cytokines which stimulate cytotoxic T cells and B cells

A type of cytotoxic T cell has antigen receptors which bind to the surface of an antigen-presenting phagocyte

This results in the cytotoxic T cell becoming 'activated'

A clone of activated cytotoxic T cells and a clone of memory cytotoxic T cell is formed

Activated cytotoxic T cells move to the site of infection

At site of infection

Cytotoxic T cells bind to infected cells

Chemicals from the cytotoxic T cells perforate the membrane of the infected cell

Infected cell undergoes apoptosis

This is followed by phagocytosis

Cancer cells

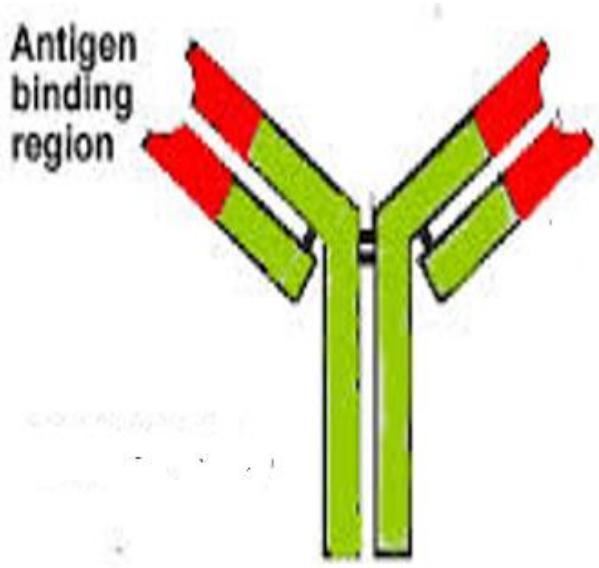
Other cytotoxic T cells recognise antigens on the surface of cancer cells and attack them

Lysis (bursting) of the cancer cells occurs

B lymphocytes (B cells)

An antibody is a Y-shaped protein molecule

Each of its 'arms' bears a receptor (binding site) specific to a particular antigen



An antibody response normally occurs with the assistance of a helper T cell, how?

The B cell displays molecules of the antigen it has taken in

These antigens are recognised by an 'activated' helper T cell

It responds by releasing cytokines

Cytokines stimulate the B cells to –

Make a clone of activated B cells

Make a clone of memory B cells

Antibodies recognise and bind with antigens

Antigen-antibody complexes formed

This complex inactivates the pathogen

Complex is engulfed and digested by phagocytosis

Immunological memory

Some T and B lymphocytes survive as memory cells

Exposure to disease causing organism brings about Primary Response

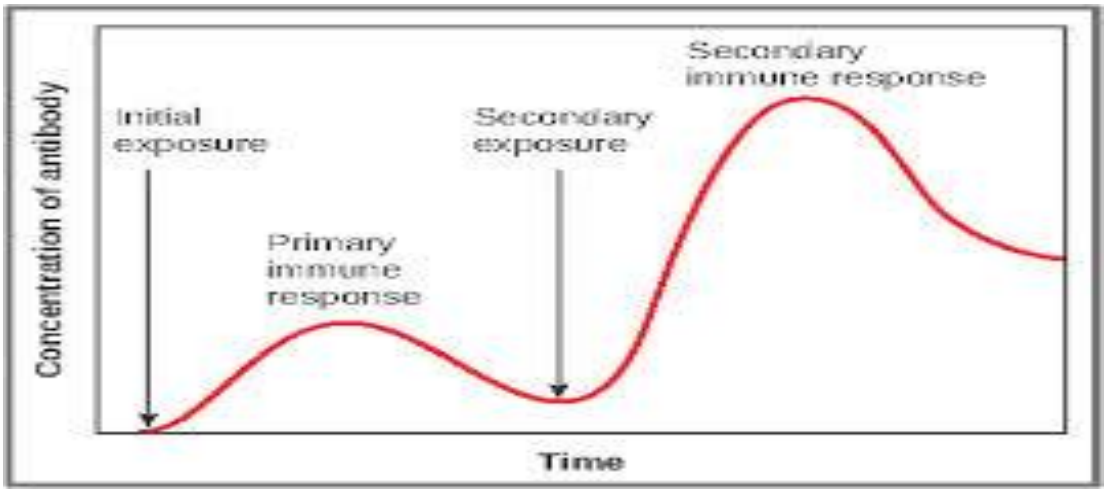
Exposure at later date brings about Secondary Response

This time the disease is usually prevented –

Antibody production much more rapid

Concentration of antibodies produced reaches a higher level

Concentration maintained for longer



Higher Human Biology Unit 4 Pupil Notes

Chapter 23

Infectious Disease is one that is capable of being transmitted by direct or indirect contact

Pathogens causing infectious diseases include –

Viruses

Bacteria

Fungi

Protozoa

Multicellular parasites (e.g. hookworm)

Methods include –

Direct contact- shaking hands etc.

Inhaled air, breathing in microbes released by an infected person coughing (measles virus is air-borne)

Indirect contact- sharing cups etc.

Body fluids, saliva and seminal fluid (HIV)

Vector organisms, being bitten by a mosquito infected with Malaria

Cholera is caused by a water-borne bacterium, water supply becomes polluted by sewage

Control of transmission

Quarantine – compulsory isolation of person suffering from a serious communicable disease (stops spread)

Asepsis – state of being completely free of live microorganisms

Antisepsis – inhibition or destruction of microbes that cause disease

Individual responsibility–

Good hygiene, hand washing etc.

Care in sexual health, condoms protect against STDs

Appropriate handling and storage of food

Community Responsibility

Filtration and chlorination of drinking water

Food manufacturers obliged to adopt 'Good Manufacturing Practise', including inspection and traceability of food

Appropriate waste disposal

Control of 'Vectors' of disease

Rats – the rat flea transmits bubonic plague

Malaria is caused by a protozoan carried by mosquitoes

Control of Malaria–

Draining stagnant water, removes breeding sites

Introducing sterile males

Using insecticides

Epidemiologist – studies outbreaks of infectious diseases, the epidemiology of an infectious disease includes study of

Location of initial outbreak

Pattern and speed of spread

Its geographical distribution

Spread classified as

Sporadic – occurs in scattered or isolated instances

Endemic – regular number of cases recurring in an area

Epidemic – very high number of cases in an area

Pandemic – global epidemic

Immunisation is the process by which a person develops immunity to a disease causing organism

Active Immunity – protection is gained as a result of a person producing antibodies

There are 2 types of active immunity:

Naturally Acquired Active Immunity - a person who survives an infection by pathogens has acquired active immunity

Artificially Acquired Active Immunity - achieved by vaccination

Form of antigen in vaccine	Examples of disease to which active immunity is acquired
Dead pathogens	Hepatitis A and polio
Parts of pathogens	Hepatitis B and HPV
Weakened pathogens	Rubella, mumps and measles
Inactivated bacterial toxin	Diphtheria and Tetanus

Antigen is mixed with an adjuvant – a chemical that promotes the activity of the antigen and induces the production of B and T cells (Some B and T cells persist as memory cells)

Vaccines must be subjected to clinical trials on humans to establish that they are safe and efficacious (capable of producing the intended result)

Clinical Trials

Prior to clinical trials, the treatment is tested on animals

There are three phases in clinical trials

Phase 1

Small doses of treatment are tested on small number of volunteers (25-50) to check if it is safe

Phase 2

Treatment tested on a large number (150-300) of people with the illness to find out if it is safe and effective and to find out what the optimum dose would be

Phase 3

Treatment tested on a very large number (1000-2000) of people who have the illness

If phase 3 is successful a licence is sought for the manufacture of the new vaccine

In phase 3 the patients are split into two groups, the 'test' and the 'control' group

Protocol employed at this stage is –

1. Placebo controlled
2. Double blind
3. Randomised

Placebo –takes the same form as the treatment but lacks the active ingredient being tested

The procedure is carried out to assess the 'placebo effect'

The effect from receiving the treatment that does not depend on the active ingredient

Some patients on the placebo show an improvement in their condition, why?

This could be the result of the psychological effect of –

Thinking they were getting the 'real' treatment

Getting expert attention from health care staff

Expecting the treatment to be efficacious

Double-Blind trial

This is one in which neither the subjects nor the doctors know who is receiving what

It is used at stage 3 to eliminate bias

Randomisation

Age, gender and other details entered into a computer

This then puts each person into one or other of the two groups at random

This further eliminates bias

Higher Human Biology Unit 4 Pupil Notes

Chapter 24

Herd Immunity

Herd Immunity – this protection given indirectly to the non-immune minority

Herd immunity provides protection for 'vulnerable' groups e.g. people who must not be vaccinated because of an immune disorder

Herd immunity threshold – is the % of immune individuals in a population above which a disease no longer manages to persist

Mass vaccination programmes is the public health policy in many countries

In the UK, the vaccination schedule begins at around two months

In poor and malnourished developing countries it is not possible to introduce widespread vaccination

As a result, herd immunity cannot be established

In a developed country, herd immunity may be compromised if there is adverse publicity

Antigenic Variation

Antigenic variation – this is when new strains of a pathogen have antigens on their surface that are different from the original strain

New strain is genetically and immunologically distinct from parent strain

New antigens are not recognised by memory cells

It succeeds because it has a selective advantage

Influenza virus

Remains a public health problem

'At risk' individuals need to be vaccinated each year with a new version of the Flu vaccine

Sleeping Sickness

Trypanosoma b. is a protozoan which enters the blood and causes Trypanosomiasis, 'sleeping sickness'

The pathogen is surrounded by a glycoprotein coat, this is the antigen

Infected host makes antibodies against the antigen 'coat'

This kills 99% of pathogen

The 1% shed their coat and 'switch on' genes for a variant glycoprotein coat

Host responds by making a new set of antibodies

Again, 99% pathogens killed, 1% survive, shed coats and so on

Cycle continues until, in absence of treatment, host dies

Public Health

Malaria

Protozoan Plasmodium f. causes Malaria

The pathogen is found inside red blood cells

Antigenic variation exists in the pathogen and this enables the pathogen to evade the host's immune response

Also, the pathogen produces a protein that is transported to an infected RBC's surface

This makes the RBC stick to the lining of the blood vessels and prevents it being destroyed

T.B.

Mycobacterium tuberculosis causes T.B.

It interferes with the body's phagocytic response

The pathogen is able to survive inside phagocytes

When a macrophage engulfs the BT bacterium, the microbe prevents lysosomes fusing with the vesicle

If fusion does occur, the pathogen is not easily attacked by lysosomal enzymes because it is protected

The pathogen remains alive inside the phagocyte and avoids immune detection

AIDS (acquired immune deficiency syndrome) is a disease caused by HIV (human immunodeficiency virus)

HIV attacks helper T lymphocytes

The envelope surrounding the HIV particle fuses with the membrane of the helper T cells and the virus enters the host cell

It can remain dormant for years before directing the synthesis of new viral particles

New viral particles escape from the helper T cell by 'budding'

B cells do make antibodies but these are ineffective against viral particles 'hiding' inside helper T cells

As number of helper T cells decreases, immunological activity decreases leaving the person susceptible to infection

Remember, helper T cells are of critical importance to the immune system – they activate B cells and cytotoxic T cells

HIV is a retrovirus, it contains RNA

Along with RNA it introduces 'reverse transcriptase' into the host cell

This produces viral DNA from viral RNA

Eventually, viral DNA directs the synthesis of new viral RNA