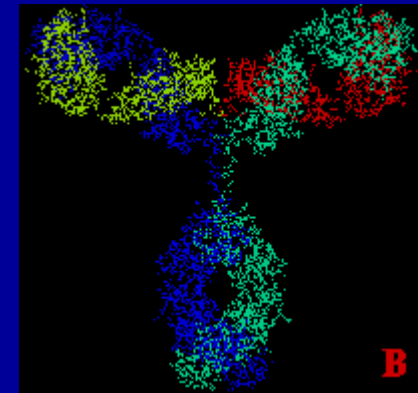
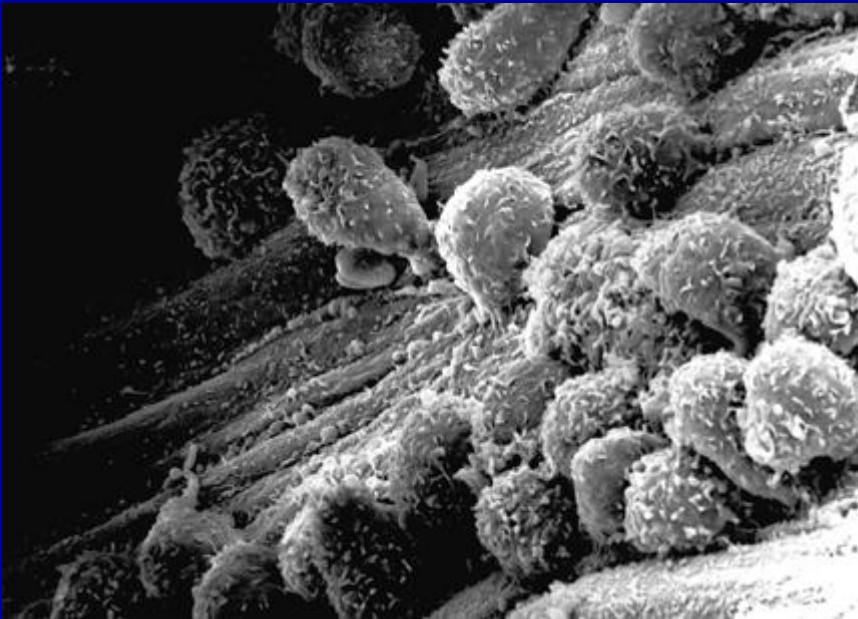
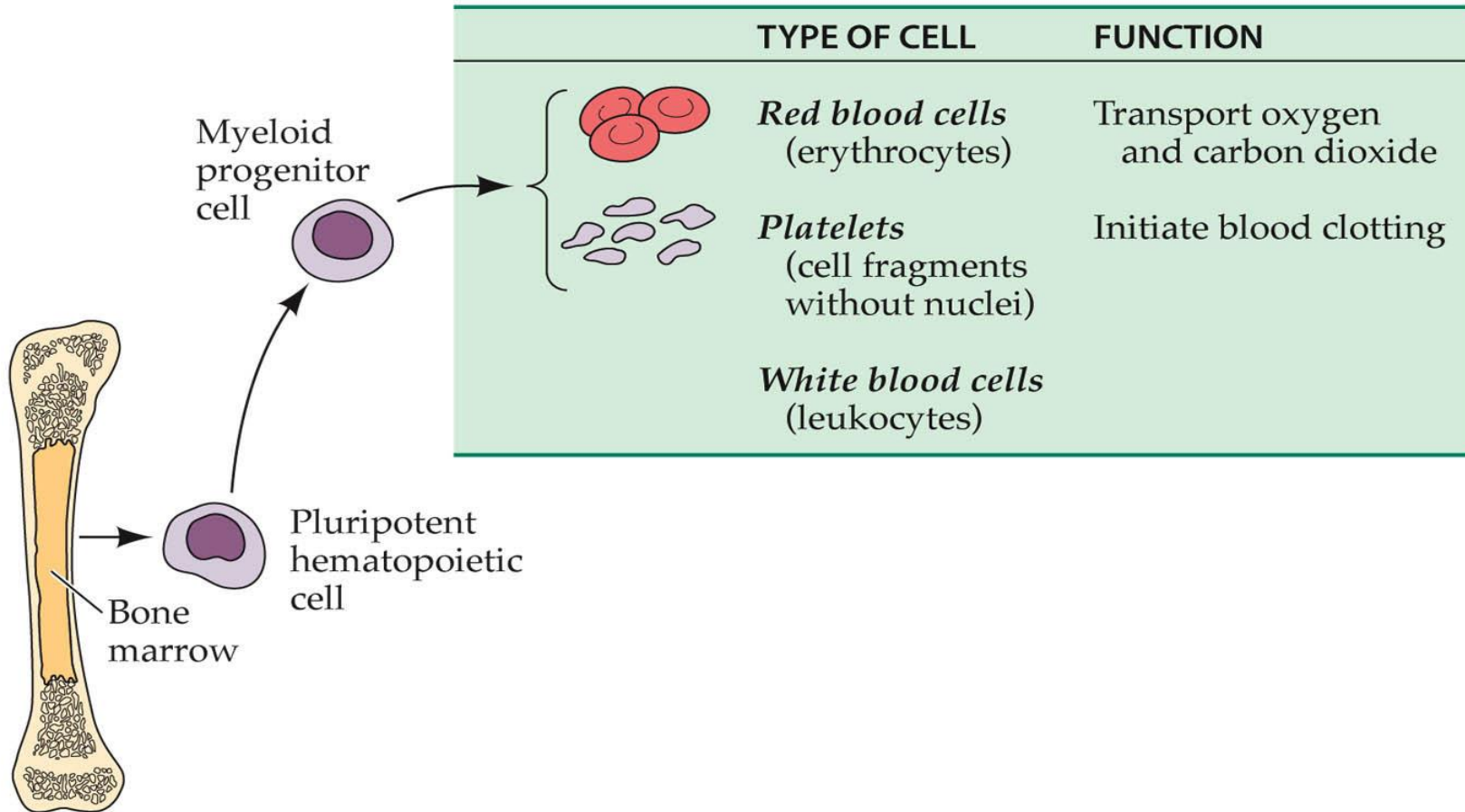


THE IMMUNE SYSTEM



Blood Cells



Cells of the Immune System

White Blood Cells

- Phagocytes - Neutrophils
 - Macrophages
- Lymphocytes

Phagocytes

- Produced throughout life by the bone marrow.
- Scavengers – remove dead cells and microorganisms.

Phagocytes and Their Relatives



Monocyte



Eosinophil



Mast cell



Macrophage



Dendritic cell



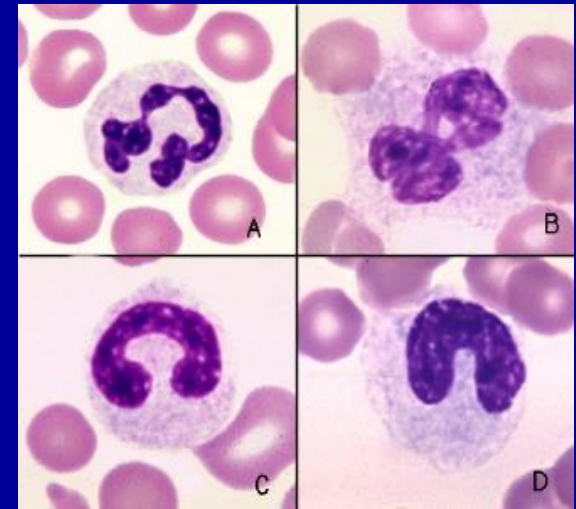
Neutrophil



Basophil

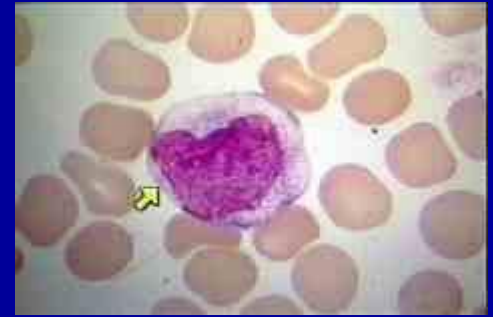
Illustration by Jennifer Kelly, ©2004

Neutrophils



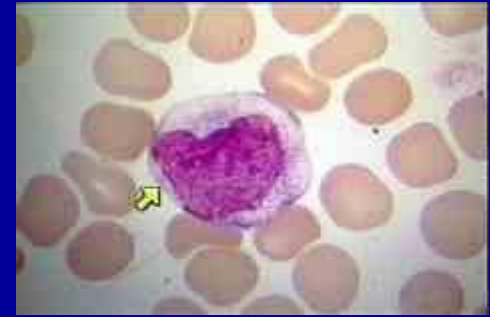
- 60% of WBCs
- ‘Patrol tissues’ as they squeeze out of the capillaries.
- Large numbers are released during infections
- Short lived – die after digesting bacteria
- Dead neutrophils make up a large proportion of puss.

Macrophages

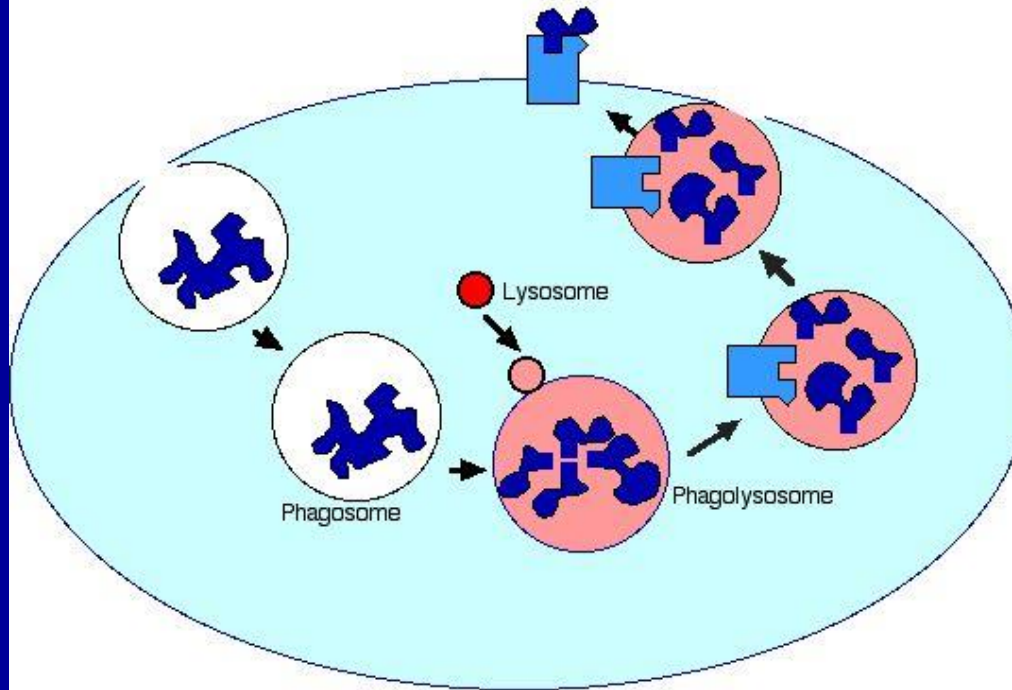


- Larger than neutrophils.
- Found in the organs, not the blood.
- Made in bone marrow as **monocytes**, called macrophages once they reach organs.
- Long lived
- **Initiate** immune responses as they display antigens from the pathogens to the lymphocytes.

Macrophages

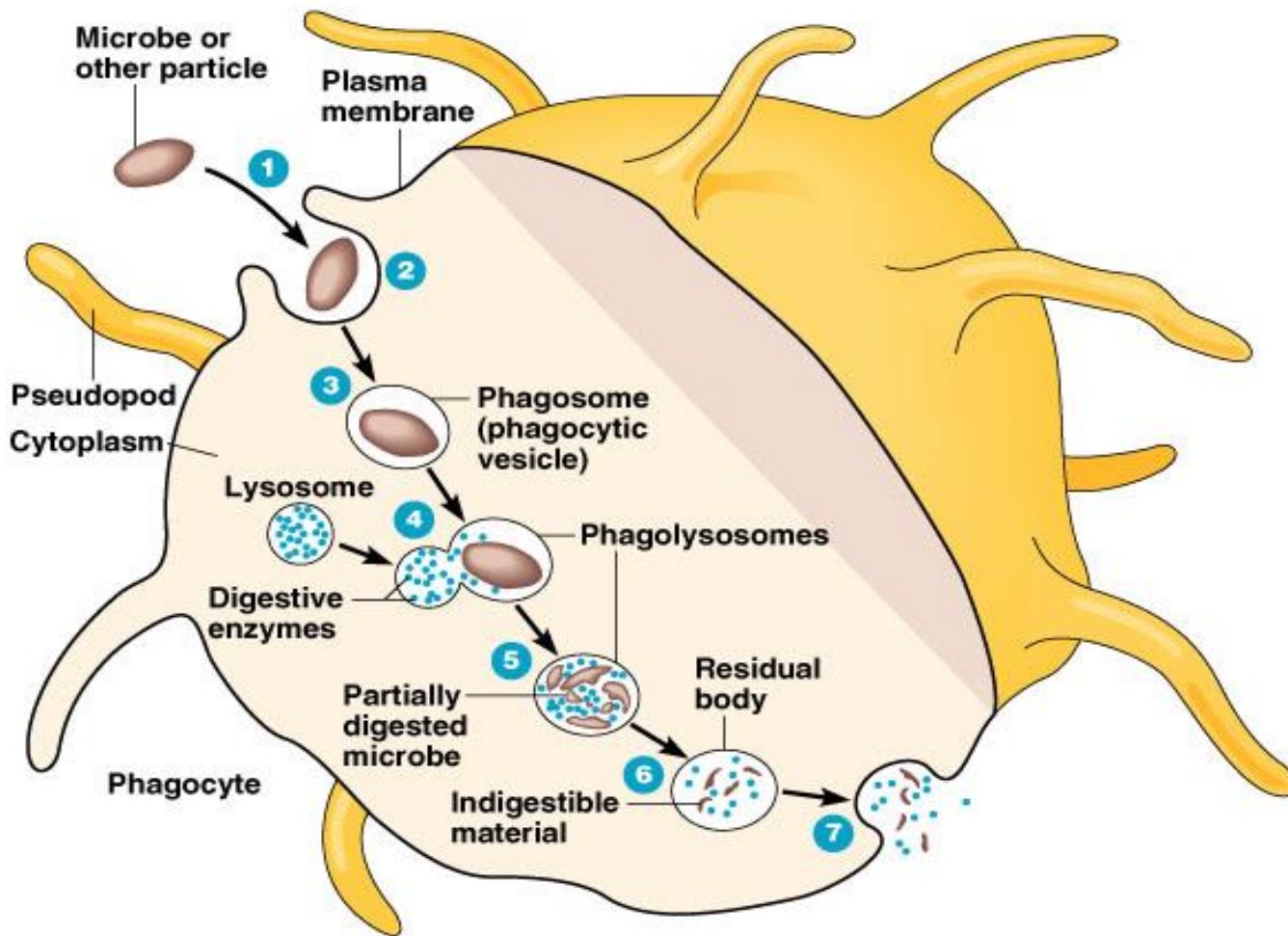


Antigen Presentation



Macrophage ("Big Eater" = big phagocyte)

Phagocytosis



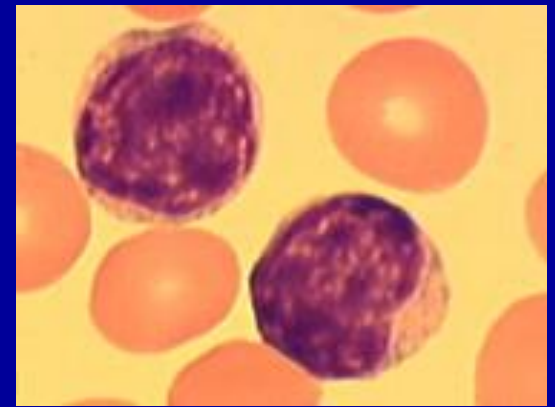
- 1 Chemotaxis and adherence of microbe to phagocyte.
- 2 Ingestion of microbe by phagocyte.
- 3 Formation of a phagosome.
- 4 Fusion of the phagosome with a lysosome to form a phagolysosome.
- 5 Digestion of ingested microbe by enzymes.
- 6 Formation of residual body containing indigestible material.
- 7 Discharge of waste materials.

(a) Phases of phagocytosis

Phagocytosis

- If cells are under attack they release **histamine**.
- Histamine plus chemicals from pathogens mean **neutrophils** are attracted to the site of attack.
- Pathogens are attached to antibodies and neutrophils have **antibody receptors**.
- Endocytosis of neutrophil membrane → phagocytic vacuole.
- Lysosomes attach to phagocytic vacuole → pathogen digested by proteases

Lymphocytes



- Produce antibodies
- **B-cells** mature in **bone marrow** then concentrate in lymph nodes and spleen
- **T-cells** mature in **thymus**
- B and T cells mature then circulate in the blood and lymph
- Circulation ensures they come into contact with pathogens and each other

B -Lymphocytes

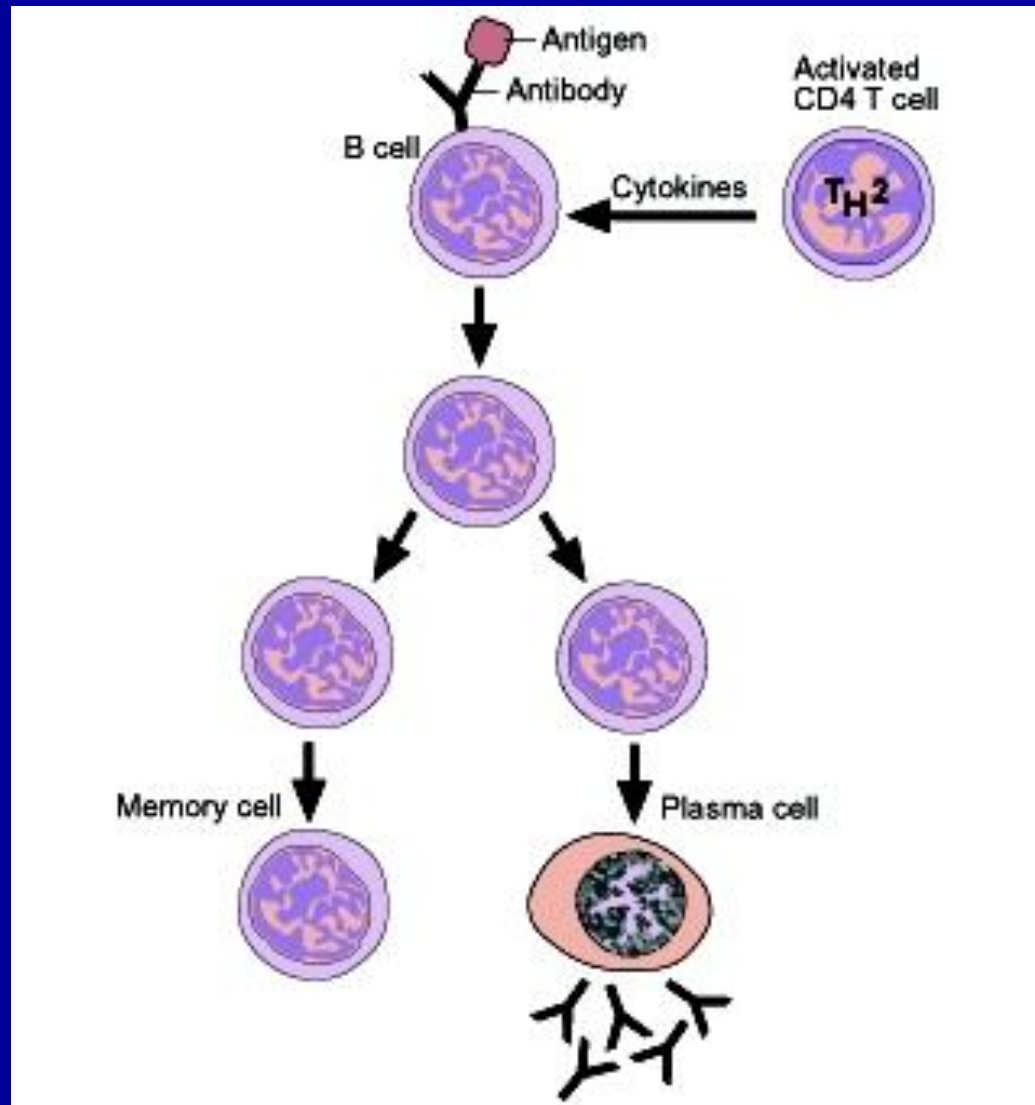
- There are c.10 million different B-lymphocytes, each of which make a different antibody.
- The huge variety is caused by genes coding for abs changing slightly during development.
- There are a small group of clones of each type of B-lymphocyte

B -Lymphocytes

- At the clone stage antibodies do not leave the B-cells.
- The abs are embedded in the plasma membrane of the cell and are called antibody receptors.
- When the receptors in the membrane recognise and antigen on the surface of the pathogen the B-cell divides rapidly.
- The antigens are presented to the B-cells by **macrophages**



B -Lymphocytes



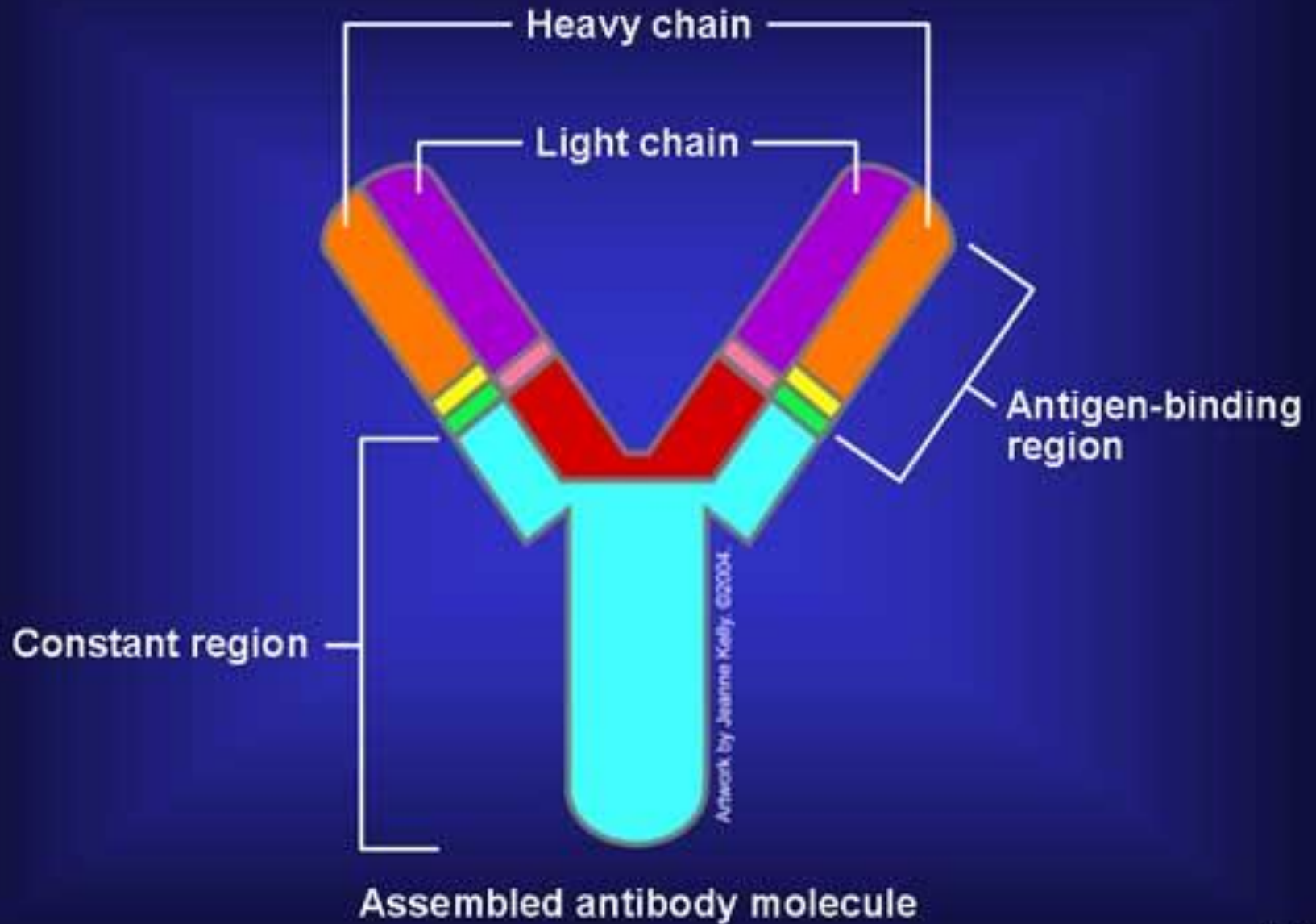
B -Lymphocytes

- Some activated B cells → **PLASMA CELLS** these produce lots of antibodies, < 1000/sec
- The antibodies travel to the blood, lymph, lining of gut and lungs.
- The number of plasma cells goes down after a few weeks
- Antibodies stay in the blood longer but eventually their numbers go down too.

B -Lymphocytes

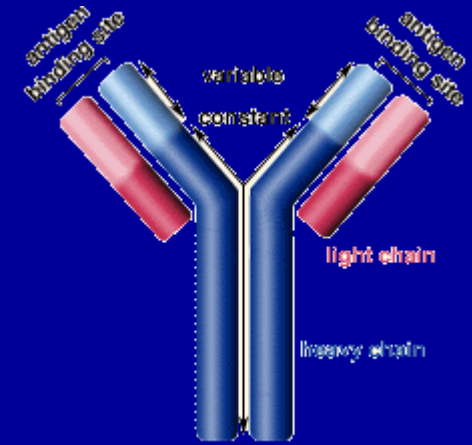
- Some activated B cells → **MEMORY CELLS.**
- Memory cells divide rapidly as soon as the antigen is reintroduced.
- There are many more memory cells than there were clone cells.
- When the pathogen/infection infects again it is destroyed before any symptoms show.

Antibody

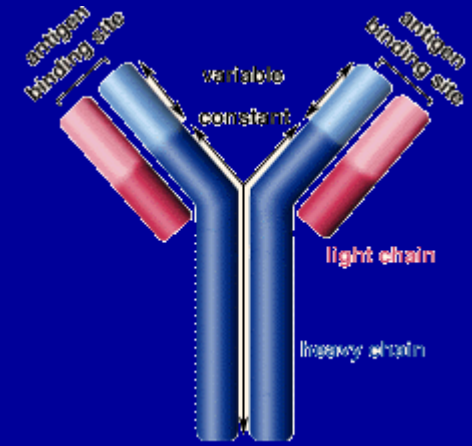


Antibodies

- Also known as **immunoglobulins**
- Globular glycoproteins
- The heavy and light chains are polypeptides
- The chains are held together by disulphide bridges
- Each ab has 2 identical ag binding sites – variable regions.
- The order of amino acids in the variable region determines the shape of the binding site

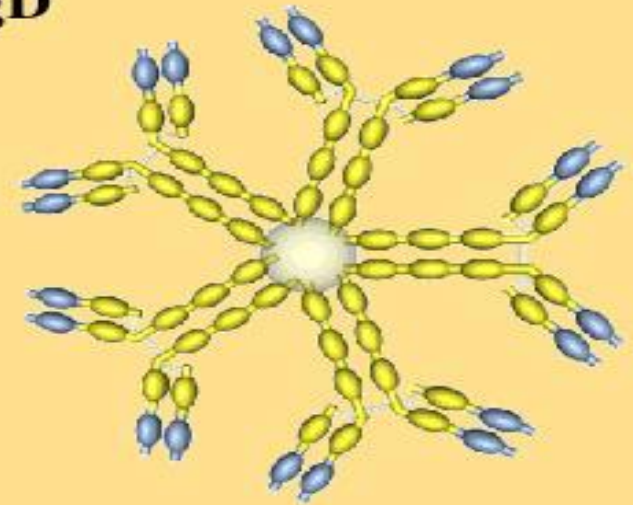
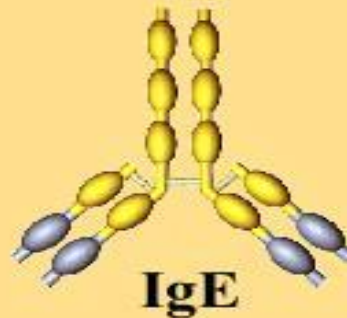
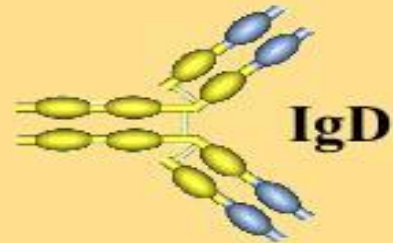
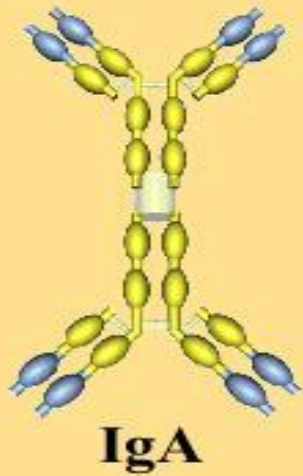


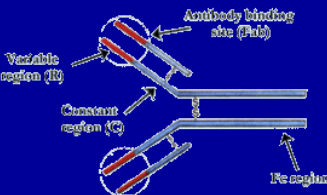
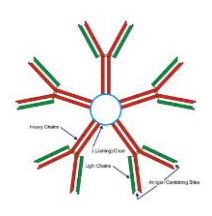
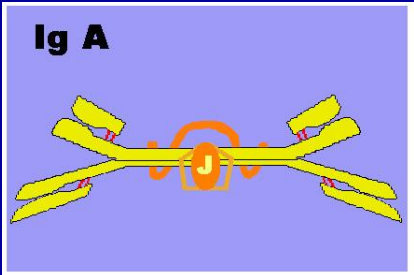
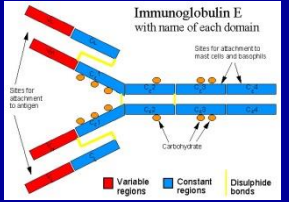
How Abs work

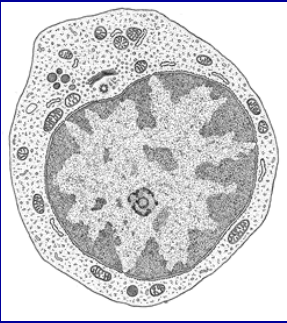


- Some act as **labels** to identify antigens for phagocytes
- Some work as **antitoxins** i.e. they block toxins for e.g. those causing diphtheria and tetanus
- Some attach to bacterial flagella making them less active and easier for phagocytes to engulf
- Some cause **agglutination (clumping together)** of bacteria making them less likely to spread

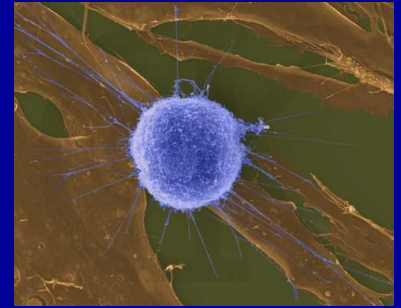
Different Immunoglobulins



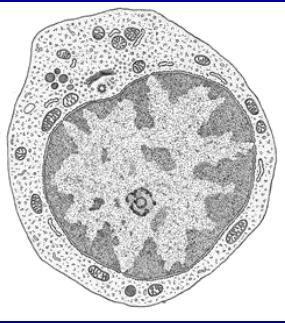
Type	Number of ag binding sites	Site of action	Functions
IgG 	2	<ul style="list-style-type: none"> •Blood •Tissue fluid •CAN CROSS PLACENTA 	<ul style="list-style-type: none"> •Increase macrophage activity •Antitoxins •Agglutination
IgM 	10	<ul style="list-style-type: none"> •Blood •Tissue fluid 	Agglutination
IgA 	2 or 4	<ul style="list-style-type: none"> •Secretions (saliva, tears, small intestine, vaginal, prostate, nasal, breast milk) 	<ul style="list-style-type: none"> •Stop bacteria adhering to host cells •Prevents bacteria forming colonies on mucous membranes
IgE 	2	Tissues	<ul style="list-style-type: none"> •Activate mast cells → HISTAMINE •Worm response



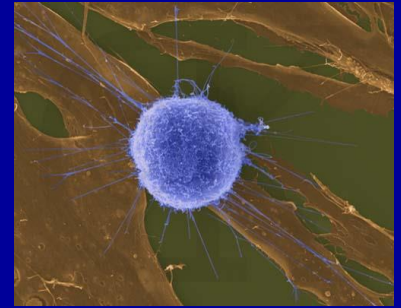
T-Lymphocytes



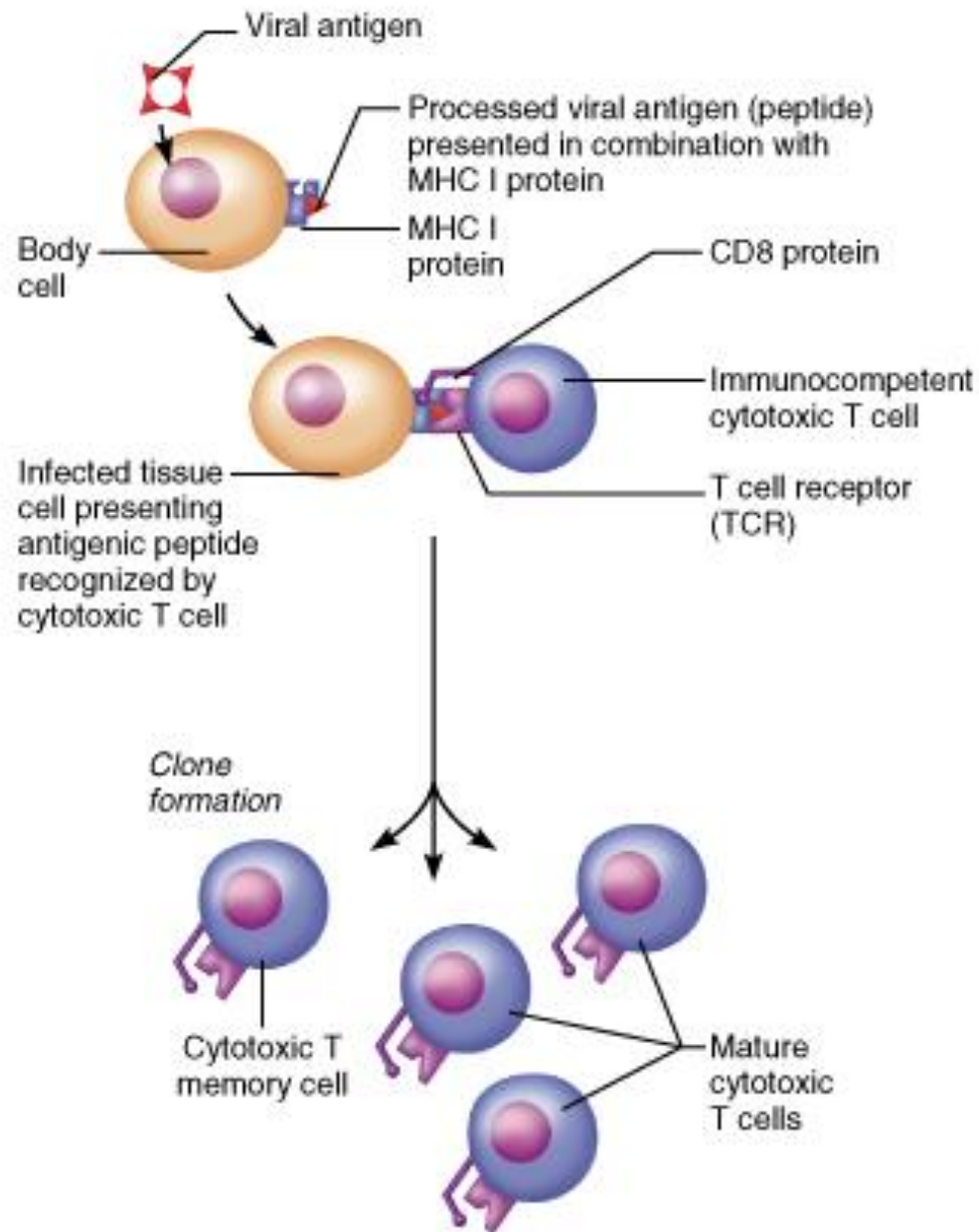
- Mature T-cells have T cell receptors which have a very similar structure to antibodies and are specific to 1 antigen.
- They are activated when the receptor comes into contact with the Ag with another host cell (e.g. on a macrophage membrane or an invaded body cell)



T-Lymphocytes



- After activation the cell divides to form:
- **T-helper cells** – secrete CYTOKINES
 - help B cells divide
 - stimulate macrophages
- **Cytotoxic T cells** (killer T cells)
 - Kill body cells displaying antigen
- **Memory T cells**
 - remain in body



T Cells

Resting helper T cell



Activated helper T cell

Artwork by Jeanne Kelly ©2004

Resting cytotoxic T cell



Activated killer cell

Active and Passive Immunity

Active immunity

Lymphocytes are activated by antigens on the surface of pathogens

Natural active immunity - acquired due to infection

Artificial active immunity – vaccination

Takes time for enough B and T cells to be produced to mount an effective response.

Active and Passive Immunity

Passive immunity

B and T cells are not activated and plasma cells have not produced antibodies.

The antigen doesn't have to be encountered for the body to make the antibodies.

Antibodies appear immediately in blood but protection is only temporary.

Active and Passive Immunity

Artificial passive immunity

Used when a very rapid immune response is needed e.g. after infection with tetanus.

Human antibodies are injected. In the case of tetanus these are antitoxin antibodies.

Antibodies come from blood donors who have recently had the tetanus vaccination.

Only provides short term protection as abs destroyed by phagocytes in spleen and liver.

Active and Passive Immunity

Natural passive immunity

A mother's antibodies pass across the placenta to the foetus and remain for several months.

Colostrum (the first breast milk) contains lots of IgA which remain on surface of the baby's gut wall and pass into blood

Immunity: Active and Passive

Active immunity



Naturally acquired



Artificially acquired

Passive immunity



Naturally acquired



Artificially acquired

History of Immunization

- 1st century BC in India and China
=> Variolation
 inoculation of dried exudate from
smallpox
- In 1796 (1798) Edward Jenner (British)
 Vaccination with cowpox to prevent
smallpox

- Lewis pasture
 - 1879 Importance of **reducing the ability** of an immunizing organism (accidentally discovered *pasturella multocida* vaccine)
 - 1885 the 1st successful vaccine against rabies (by **drying** spinal cord of rabied dog)

- Salmon (in US)
 - Vaccine from **dead** mic org (heat killed culture) develop against *Salmonella cholerasuis*
- Van Behring & Kitasato (in Germany)
 - Develop **filters** from *Cl. tetani* (bacteria toxin and not the bacteria) can serve as vaccine



Vaccination



A preparation containing antigenic material:

- Whole live microorganism
- Dead microorganism
- Attenuated (harmless) microorganism
- Toxoid (harmless form of toxin)
- Preparation of harmless ags



Vaccination



- Injection into vein or muscle
- Oral



Vaccination



Why aren't they always effective?

- Natural infections persist within the body for a long time so the immune system has time to develop an effective response, vaccinations from dead m-os do not do this.
- Less effective vaccines need booster injections to stimulate secondary responses



Vaccination



Why aren't they always effective?

- Some people don't respond well/at all to vaccinations
- Defective immune systems
- Malnutrition particularly protein



Vaccination



Why aren't they always effective?

- **Antigenic variation** caused by mutation
- Antigenic drift – small changes (still recognised by memory cells)
- Antigenic shift – large changes (no longer recognised)



Vaccination



Why aren't they always effective?

- No vaccines against **protoctists** (malaria and sleeping sickness)
- Many stages to *Plasmodium* life cycle with many antigens so vaccinations would have to be effective against all stages (or be effective just against infective stage but given in very small time period).



Vaccination



Why aren't they always effective?

- Sleeping sickness – *Trypanosoma* has a thousand different ags and changes them every 4-5 days

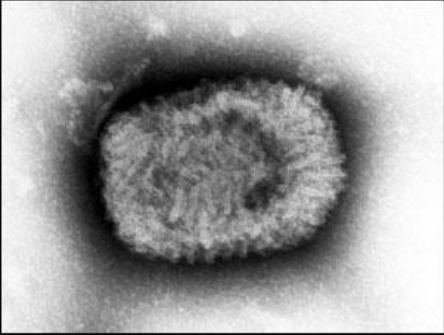


Vaccination



Why aren't they always effective?

- **Antigenic concealment** parasites live inside body cells
- *Plasmodium* – liver and blood cells
- Parasitic worms – cover themselves in host proteins
- HIV – live inside T-helper cells

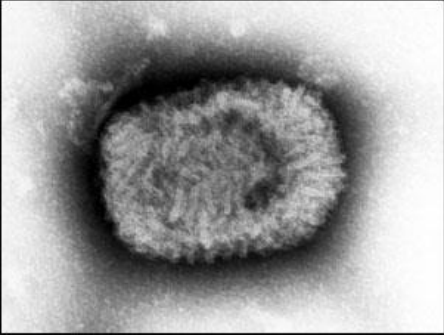


Smallpox



Symptoms

- Red spots containing transparent fluid all over body.
- Spots fill with pus
- Eyelids swell and become glued together

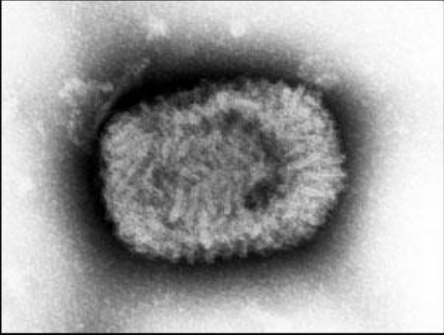


Smallpox



Mortality

- 12-30% died
- Survivors often left blind and disfigured with scabs.

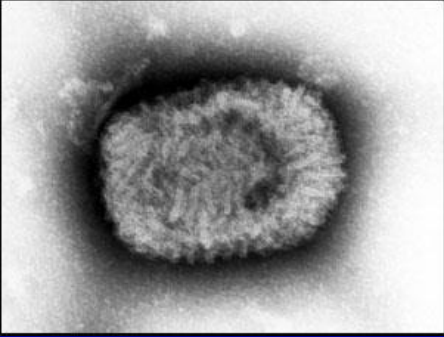


Smallpox



Eradication programme

- Started by WHO in 1956
- Aimed to rid world of smallpox by 1977
- Involved vaccination and surveillance
- Over 80% of populations at risk of the disease were vaccinated
- After any reported case everyone in the household and 30 surrounding households vaccinated – **RING VACCINATION**

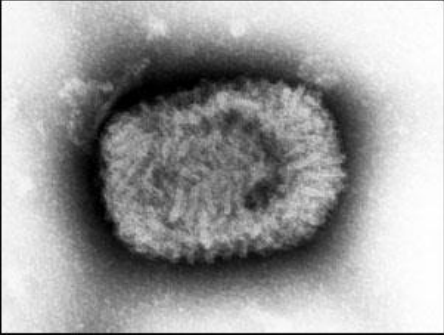


Smallpox



Eradication programme

- Last case of smallpox reported in Somalia in 1977
- World declared free of smallpox in 1980

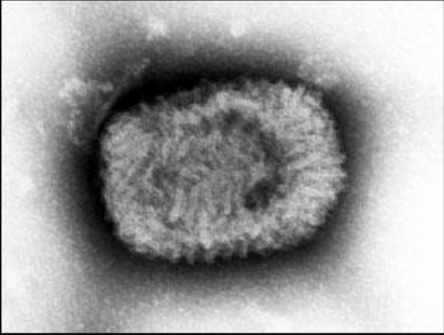


Smallpox



Eradication programme – why was it successful?

- Variola virus stable -> cheap as everyone used same vaccine
- Vaccine made from harmless strain of similar virus (vaccinia)
- Vaccine could be used at high temperatures
- Easy to identify infected people
- Smallpox doesn't lie dormant in body



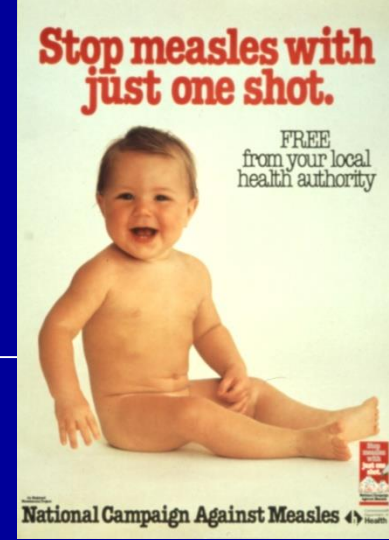
Smallpox



Eradication programme – why don't all work?

- Political instability
- Poor infrastructure
- Unstable m-os

Measles



- Caused by an airborne virus
- 9th leading cause of death worldwide
- Causes rash and fever
- Can have fatal complications
- Passive immunity from mothers in infants under 8 months
- Now quite a rare disease in developed countries due to vaccination

Measles

Stop measles with
just one shot.

FREE
from your local
health authority

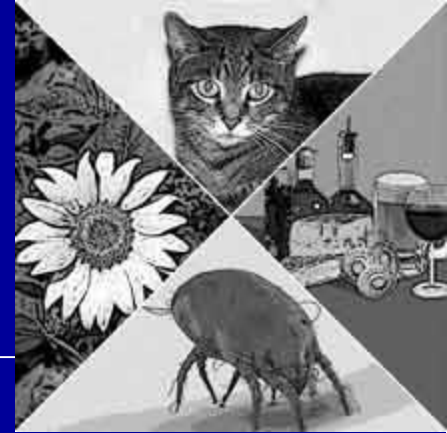


National Campaign Against Measles

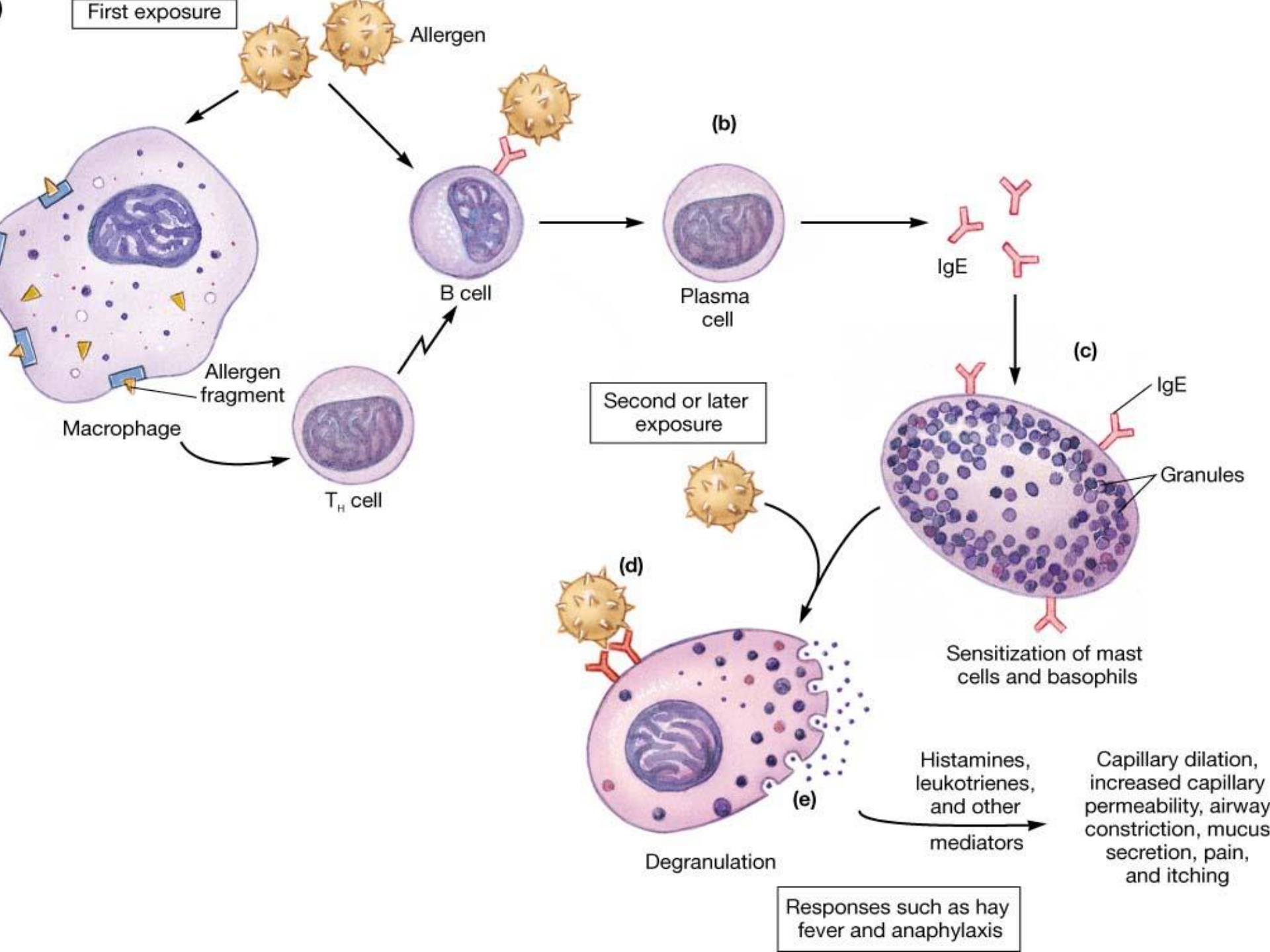
- Transmitted easily in overcrowded, insanitary conditions
- Mainly affects malnourished infants with vitamin A deficiencies
- Responsible for many cases of childhood blindness and can cause severe brain damage
- Herd immunity of 93-95% needed to prevent transmission within a population.



Allergies

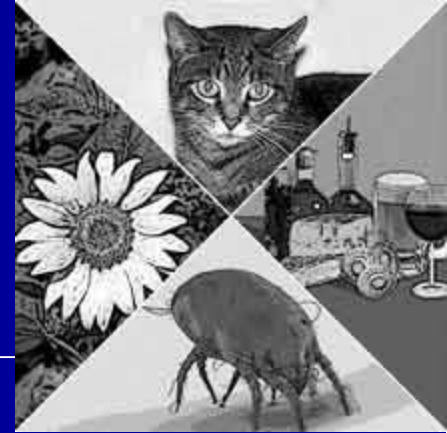


- When the immune system responds to harmless substances
- *Allergens* – antigenic substances which do no real harm
- Allergens include house dust, animal skin, pollen, house dust mite and its faeces





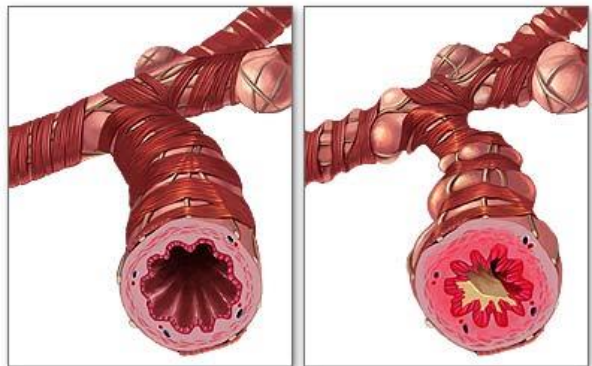
Allergies



- Histamine causes blood vessels to widen and become leaky.
- Fluid and white blood cells leave capillaries.
- The area of leakage becomes hot, red and inflamed

Normal bronchiole

Asthmatic bronchiole



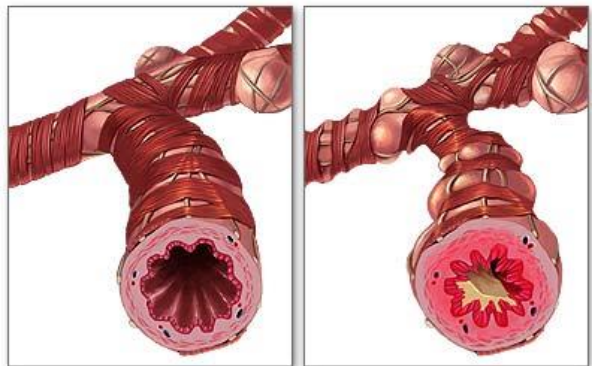
Asthma



- Attacks can occur at any time
- Genes play a role in who develops asthma
- Breathing becomes difficult, sufferers experience wheezing, coughing, a tightness about the chest and shortage of breath.
- 1/7 children in UK has asthma, number is increasing.
- >1000 people die each year from asthma every year in the UK

Normal bronchiole

Asthmatic bronchiole



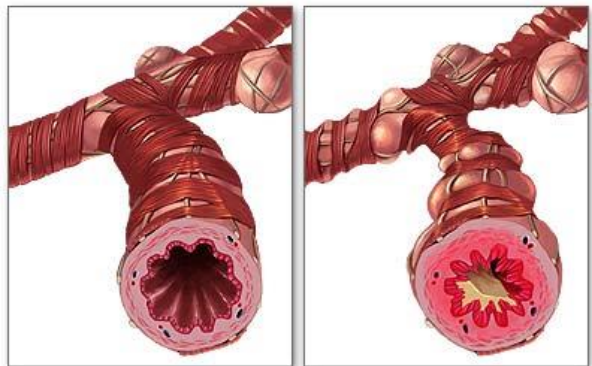
Asthma



- Airways in asthmatics are always inflamed, during an attack this worsens.
- Fluid leaks from blood into airways and goblet cells secrete lots of mucus
- Airways can become blocked
- Muscles surrounding trachea and bronchioles contract which narrows airways further

Normal bronchiole

Asthmatic bronchiole



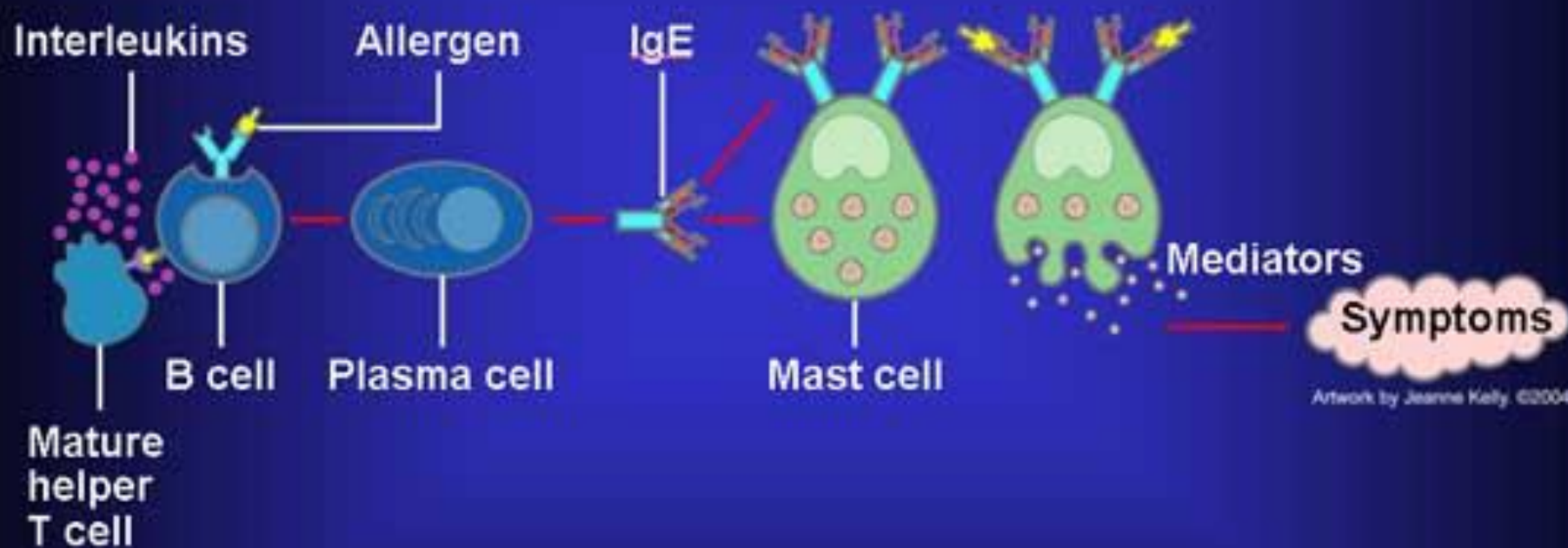
ADAM

Asthma



- Vaccines are being developed to make allergic responses less severe
- Designed to desensitise people so they do not produce antibodies to allergens
- Genetic tests may be used to screen children and then a vaccine could be given to prevent them developing asthma

Disorders of the Immune System: Allergy



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