THE IMMUNE SYSTEM
Blood Cells

<table>
<thead>
<tr>
<th>TYPE OF CELL</th>
<th>FUNCTION</th>
</tr>
</thead>
<tbody>
<tr>
<td>Red blood cells (erythrocytes)</td>
<td>Transport oxygen and carbon dioxide</td>
</tr>
<tr>
<td>Platelets (cell fragments without nuclei)</td>
<td>Initiate blood clotting</td>
</tr>
<tr>
<td>White blood cells (leukocytes)</td>
<td></td>
</tr>
</tbody>
</table>

Myeloid progenitor cell

Pluripotent hematopoietic cell

Bone marrow

*LIFE: THE SCIENCE OF BIOLOGY, Seventh Edition, Figure 18.2 Blood Cells (Part 1)*

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
- Macrophage
- Dendritic cell
- Neutrophil
- Eosinophil
- Mast cell
- Basophil
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 pus.
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

(a) Phases of 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.

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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 $\rightarrow$ phagocytic vacuole.
- Lysosomes attach to phagocytic vacuole $\rightarrow$ 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 $\rightarrow$ 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.
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

IgA

IgG

IgE

IgM
<table>
<thead>
<tr>
<th>Type</th>
<th>Number of ag binding sites</th>
<th>Site of action</th>
<th>Functions</th>
</tr>
</thead>
<tbody>
<tr>
<td>IgG</td>
<td>2</td>
<td>Blood, Tissue fluid, CAN CROSS PLACENTA</td>
<td>Increase macrophage activity, Antitoxins, Agglutination</td>
</tr>
<tr>
<td>IgM</td>
<td>10</td>
<td>Blood, Tissue fluid</td>
<td>Agglutination</td>
</tr>
<tr>
<td>IgA</td>
<td>2 or 4</td>
<td>Secretions (saliva, tears, small intestine, vaginal, prostate, nasal, breast milk)</td>
<td>Stop bacteria adhering to host cells, Prevents bacteria forming colonies on mucous membranes</td>
</tr>
<tr>
<td>IgE</td>
<td>2</td>
<td>Tissues</td>
<td>Activate mast cells, HISTAMINE, Worm response</td>
</tr>
</tbody>
</table>
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

Resting cytotoxic T cell

Activated killer cell
## Active and Passive Immunity

<table>
<thead>
<tr>
<th>Active immunity</th>
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<tbody>
<tr>
<td>Lymphocytes are activated by antigens on the surface of pathogens</td>
</tr>
<tr>
<td><strong>Natural active immunity</strong> - acquired due to infection</td>
</tr>
<tr>
<td><strong>Artificial active immunity</strong> – vaccination</td>
</tr>
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</table>

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.
### 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

Mumps 12/9/79
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 *Plamodium* 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

- 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
First exposure

[Diagram showing the process of allergen interaction with immune cells]

Second or later exposure

- Sensitization of mast cells and basophils
  - IgE binding
  - Granules

(d)

- Degranulation
- Histamines, leukotrienes, and other mediators

(e)

Capillary dilation, increased capillary permeability, airway constriction, mucus secretion, pain, and itching

Responses such as hay fever and anaphylaxis
**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.
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
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
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

- Interleukins
- Allergen
- IgE
- Mast cell
- Mediators
- Symptoms

- B cell
- Plasma cell
- Mature helper T cell

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