

## AQA AS Level Biology Unit 1

**Why do we calculate ratios or percentages with data?** for easier comparison, because different groups have different starting numbers/masses

**Why do we take a large sample size?** more representative, findings not due to chance

**Why do we take random samples?** avoid bias

**Why do we take repeats?** identify anomalous results and calculate a reliable mean

**Why do we have controls?** to see that what we are testing (e.g. drug) is causing the effect

**How do we treat control groups?** treat exactly the same but do not give the drug, give a placebo

**Evaluate the conclusion?**

- compare the conclusion wording to the data wording
- correlation does not mean causation, other factors may be involved
- sample size unknown
- no repeats
- no controls
- length of study unknown
- then answers specific to the data (use common sense)

**What are biological molecules?** molecules made and used by living organisms e.g. Carbohydrates, Proteins, Lipids

**What are the functions of carbohydrates?**

- energy source (glucose in respiration)
- energy store (starch in plants, glycogen in animals)
- structure (cellulose in cell wall of plants)

**What are the building blocks for carbohydrates called?** monosaccharides

**Example of monosaccharides?** glucose (alpha and beta), galactose, fructose

**Formula for monosaccharides?**  $C_6H_{12}O_6$  (isomers = same formula but different arrangement)

**Difference between alpha and beta glucose?** on Carbon 1, alpha glucose has a OH group on the bottom and beta glucose has a OH group on the top

**How are monosaccharides joined together?** condensation reaction (removing water) – between 2 OH groups

**Bond in carbohydrate?** glycosidic bond

**Example of disaccharides?** glucose + glucose = maltose, glucose + galactose = lactose, glucose + fructose = sucrose

**Formula for disaccharides?**  $C_{12}H_{22}O_{11}$

**How are polymers separated?** hydrolysis (add water)

**What is a polysaccharide?** many monosacharrides joined by condensation reaction/glycosidic bonds

**Example of polysaccharides?**

- Amylose (long chain of alpha glucose) which makes starch/glycogen
- Cellulose (long chain of beta glucose) which makes cell wall in plants

**Test for starch?** add iodine, turns blue/black

**Test for reducing sugar?** heat with benedicts, turns brick red

**Test for non-reducing sugar?**

- heat with benedicts – no change
- therefore, add dilute hydrochloric acid (hydrolyses glycosidic bond)
- then add sodium hydrogencarbonate (neutralises solution)
- heat with benedict - turns brick red

**How is starch digested?**

- Salivary Amylase in the mouth and Pancreatic Amylase in the SI breaks the starch into maltose
- Maltase on the lining of the SI breaksdown the maltose into glucose

**How is sucrose digested?** sucrase on the lining of the SI breaks it down into glucose and fructose

**How is lactose digested?** lactase on lining of the SI breaks it down into glucose and galactose

**What is lactose intolerance?** person does not have the lactase enzyme

**Symptoms of lactose intolerance?**

- Diarrhoea and Flatulence
- diarrhoea – undigested lactose lowers water potential of the lumen of the SI, so water enters the lumen by osmosis, = watery faeces
- flatulence – undigested lactose enters the LI, broken down by micro-organisms, releasing gas

**What are 2 types of proteins?** Globular and Fibrous

**What are globular proteins?** soluble proteins with a specific 3D shape e.g. enzymes, hormones, antibodies, haemoglobin

**What are fibrous proteins?** strong/insoluble/inflexible material e.g. collagen and keratin

**What are the building blocks for proteins?** amino acids

**Structure of amino acid?** central carbon, carboxyl group to the right (COOH), amine group to the left (NH<sub>2</sub>), hydrogen above and R group below

**How do amino acids differ?** have different R groups e.g. glycine has a hydrogen in its R group – simplest amino acid

**How are amino acids joined together?** by condensation reaction between the carboxyl group of one and amine group of another, leaves a bond between carbon & nitrogen (called a peptide bond) forming a dipeptide

**Define primary, secondary, tertiary, quaternary structure?**

- Primary = sequence of AA, polypeptide chain (held by peptide bonds)
- Secondary = the primary structure (polypeptide chain) coils to form a helix, held by hydrogen bonds
- Tertiary = secondary structure folds again to form final 3d shape, held together by hydrogen/ionic/disulfide bonds
- Quaternary = made of more than one polypeptide chain

**Examples of quaternary structure proteins?** collagen (3 chains), antibodies (3 chains), haemoglobin (4 chains)

**Structure of collagen?**

- strong material, used to build tendons/ligaments/connective tissues
- primary structure mainly made up of glycine (simplest amino acid)
- secondary structure forms a tight coil (not much branching due to glycine)
- tertiary structure coils again
- quaternary structure made from 3 tertiary structures wrapped around each other like rope
- = a collagen molecule
- many of these collagen molecules make the tendons/ligaments/connective tissues

**Test for protein?** add biuret, turns purple

**What is an enzyme?** a biological catalyst (substance that speeds up the rate of reaction without being used up – lowers activation energy)

**What makes an enzyme specific?** has a specific active site shape, only complementary substrates can bind to the active site to form enzyme-substrate complexes

**Lock and Key Model vs Induced Fit Model?**

- LK = active site shape is rigid, only exactly complementary substrates can bind to form ES complexes
- IF = active site changes shape, the substrate binds to the active site – the active site changes shape so the substrate fits exactly forming an ES complex

### **Affect of substrate concentration on enzyme activity?**

- increase substrate concentration, increases chance of successful collisions, increase chance of forming an ES complex, increase rate of reaction
- this continues until all the enzyme's active sites are full/saturated = maximum rate of reaction

### **Affect of temperature on enzyme activity?**

- as temperature increases
- the kinetic energy increases
- the molecules move faster
- increase chance of successful collisions
- increase chance of forming ES complex
- increase rate of reaction
- carries on till optimum
- after optimum
- bonds in tertiary structure break
- lose active site shape
- substrate no longer complementary
- cant form ES complexes
- enzyme denatured

**Affect of pH on enzyme activity?** if change pH away from optimum, bonds in tertiary structure break, lose active site shape, no longer form ES complex, enzyme denatured

### **Competitive vs Non-Competitive Inhibitors?**

- Competitive = a substance with a similar shape to the substrate and a complementary shape to the enzyme's active site, binds to the active site, blocking it, preventing ES complexes from forming
- Non-Competitive = a substance that binds to another site on the enzyme other than the active site, causes the active site to change shape, so less ES complexes can form

**Competitive vs Non-Competitive Inhibitors?** increase substrate concentration to excess (very high concentration) – with competitive inhibitor maximum rate of reaction will be reached, with non-competitive inhibitor maximum rate of reaction cannot be reached

**2 types of microscopes?** Light and Electron (transmission and scanning)

**How to judge a microscope?** by Magnification and Resolution

**Magnification?** how much larger the image size is compared to the actual size

**Which has higher magnification?** TEM > SEM > LM

**Formula for magnification?** magnification = image size/actual size

**Conversion?** 1 mm = 1000 micrometre. 1 mm = 1,000,000 nanometre

**Why can organelles appear different in images?** viewed from different angles and at different levels/depth

**Resolution?** minimum distance at which 2 very close objects can be distinguished

**Which has higher resolution?** TEM > SEM > LM

**Why does electron microscopes have a higher resolution?** Electron microscope uses electrons which have a shorter wavelength (light microscope uses light which has a large wavelength)

**Difference between TEM and SEM?** in Transmission the electrons pass through the specimen, in Scanning the electrons bounce off the specimen's surface

**Advantage and Disadvantage of TEM?**

- Advantage = highest magnification and highest resolution
- Disadvantage = works in a vacuum so can only observe dead specimens, specimen needs to be thin, black and white image, 2D image, artefacts

**Advantage and Disadvantage of SEM?**

- Advantage = produces 3D image
- Disadvantage = works in a vacuum so can only observe dead specimens, black and white image, artefacts

**Cell Fractionation?**

- Breakdown tissue into cells (cut, pestle & mortar)
- add cold/isotonic/buffer solution (*cold = reduce enzyme activity, isotonic = same water potential so organelle does not shrink or burst, buffer = maintains constant pH*)
- homogenate – breaks open cells releasing organelles
- filter = removes large debris and intact cells
- centrifuge – spin at low speed, largest organelle builds at bottom (nucleus), leaves supernatant, spin at higher speed, next heaviest organelle forms at bottom (chloroplast or mitochondria)
- (*organelle by size: nucleus, chloroplast, mitochondria, endoplasmic reticulum/golgi body/lysosomes, ribosomes*)

**Eukaryotic vs Prokaryotic?**

- Eukaryotic = animal/plant cell, has membrane bound organelles (nucleus, endoplasmic reticulum, golgi body, lysosome, mitochondria)
- Prokaryotic = bacteria, has no membrane bound organelles

## What is an Animal Cell made of?

- Organelles (nucleus, endoplasmic reticulum, golgi body, lysosomes, mitochondria, ribosomes) – all have membrane except the ribosomes
- Cytoplasm (site of chemical reaction)
- Cell Membrane (holds cell contents together, controls what enters/leaves cell, cell signalling)

## Structure of Nucleus?

- contains DNA (made of genes, genes code for making proteins)
- DNA wrapped around histones to form Chromatin
- nucleus has a double membrane, called Nuclear Envelope, which contains pores
- at centre of nucleus is Nucleolus – produces mRNA (copy of a gene)
- rest of nucleus made of Nucleoplasm (contains the dna/chromatin)

## Endoplasmic Reticulum?

- 2 types = Rough and Smooth
- Rough Endoplasmic Reticulum has ribosomes on it, makes proteins
- Smooth Endoplasmic Reticulum has no ribosomes on it, makes lipids/carbohydrates

## Golgi body?

- modifies and packages proteins
- packages them into vesicles for transport
- digestive enzymes are placed into lysosomes (vesicles with membranes around them)

## Mitochondria?

- site of respiration, releases energy, produces ATP (energy carrier molecule)
- has a double membrane, inner membrane folded into Cristae (increases surface area for enzymes of respiration)
- middle portion called Matrix

## Ribosomes?

- attached to RER
- site of protein synthesis

## What are the 3 types of Lipids?

- Triglycerides (fat for energy store, insulation, protection of organs)
- Phospholipids (to make membranes)
- Cholesterol (for membrane stability and make hormones)

### **Structure of triglyceride?**

- made of 1 glycerol and 3 fatty acids
- joined by condensation reaction, ester bonds
- bond is COOC
- there are 2 types of triglycerides: saturated fat and unsaturated fat

### **Saturated vs Unsaturated Fat?**

- Saturated = has no carbon double bonds in the R group of the fatty acid
- Unsaturated = has carbon double bonds in the R group of the fatty acid

### **Structure of phospholipid?**

- made of 1 glycerol, 2 fatty acids and 1 phosphate
- phosphate forms a hydrophilic head, fatty acids form hydrophobic tails
- forms a phospholipid bilayer, basic structure of membranes

### **Where are Membranes found?**

- around organelles (membrane bound organelles) = to hold organelles contents together, control what enter/leaves organelles
- around cells (cell surface membranes) = to hold cell contents together, control what enters/leaves cell, cell signalling

### **Basic structure of a membrane?**

- made of a phospholipid bilayer (a double layer of phospholipids)
- hydrophilic heads face water/fluid
- hydrophobic tails face towards each other, protected from water/fluid

### **Additional structures in membranes? Proteins, Carbohydrates, Cholesterol**

#### **Role of proteins in the membrane?**

- Extrinsic and Intrinsic
- extrinsic = found in one layer
- intrinsic = found in both layers – forms transport proteins (carrier and channel)

**Role of carbohydrates in the membrane?** form Glycoprotein and Glycolipid (if carbohydrate attaches to protein or lipid) – acts as receptors to hormones in cell signalling

**Role of cholesterol in the membrane?** sits between the phospholipids in the bilayer, provides membrane stability

**Why are membranes defined as having a Fluid-Mosaic Model?** fluid = describes the phospholipids in the bilayer – flexible, mosaic = describes the appearance of the proteins

**Define Diffusion?** net movement of molecules from an area of high concentration to an area of low concentration until equilibrium is reached (down the concentration gradient)

**Simple vs Facilitated Diffusion?**

- Simple = molecules move directly through the phospholipid bilayer
- Facilitated = molecules pass through transport proteins (large use carrier, charged use channel)

**Factors that affect rate of diffusion?**

- surface area (increase = increase rate of diffusion)
- concentration gradient (increase = increase rate of diffusion)
- thickness (decrease = decrease diffusion distance = increase rate of diffusion)
- temperature (increase = increase kinetic energy = molecules move faster = increase rate of diffusion)
- size of molecules (smaller molecules = increase rate of diffusion)

**What is Ficks Law?** (Surface Area x Concentration Gradient)/Thickness

**Define Osmosis?** movement of water molecules from an area of high water potential to an area of low water potential through a partially permeable membrane

**Which liquid has the highest water potential?**

- distilled/pure water
- has a value of 0kPa
- lower water potential by adding solutes (makes water potential negative)
- water moves from less negative water potential (e.g. -35 kPa) to more negative water potential (e.g. -75 kPa)

**Surround animal cell with pure water?** swells and burst (water enters by osmosis)

**Surround plant cell with pure water?**

- swells but does not burst
- cell wall prevents it from bursting
- made of cellulose – strong material
- the cell is Turgid

**Surround animal cell with concentrated sugar/salt solution?** shrinks (water leaves by osmosis)

**Surround plant cell with concentrated sugar/salt solution?**

- water leaves by osmosis
- cell wall prevents cell from shrinking, keeps it rigid
- the protoplast (cell membrane plus contents) shrink
- the cell is Plasmolysed

**Define Active Transport?** movement of molecules from an area of low concentration to an area of high concentration using ATP and carrier proteins (against concentration gradient)

**Describe the process of active transport?**

- molecules (in area of low concentration) bind to carrier protein
- ATP breakdown to ADP, Pi and Energy
- the Pi and Energy cause the carrier protein to change shape
- carrier protein releases molecules on opposite side (in area of high concentration)
- the carrier protein releases the attached Pi to return to its original shape

**Adaptations of SI?**

- folded to form Villus (large surface area)
- cells lining SI have Microvilli (large surface area)
- wall of SI is thin (short diffusion distance)
- rich blood supply (maintains concentration gradient)
- cells lining SI have transport proteins and mitochondria

**Active Transport of Glucose in SI?**

- sodium ions are actively transported from the cells lining the SI into the blood
- lowers the sodium ion concentration in the cell
- therefore sodium ions move from the lumen of the SI into the cell
- this pulls in glucose via a cotransport protein
- therefore glucose builds up in the cell and moves into the blood by diffusion

**Structure of Bacteria?**

- No nucleus – loose DNA in the form of a single loop and plasmid
- No membrane bound organelles: smaller ribosomes, mesosomes – infolding of cell membrane for respiration
- Cytoplasm
- Cell Membrane & Cell Wall (made of peptidoglycan)
- some have a Capsule (reduce water loss, protect from phagocytosis) and Flagella (movement)

**Cause of Cholera?**

- involves a bacteria called *Vibrio Cholerae* entering the SI
- the bacteria produces toxins which cause the cells lining the SI to release Chloride ions into the lumen of the SI
- this lowers the water potential of the lumen
- so water enters the lumen by osmosis leading to diarrhoea (watery faeces)

## **Treatment for Cholera?**

- ORT (Oral Rehydration Therapy)
- contains Sodium ions – absorbed by cells lining the SI lowering the water potential of the cells so they absorb water by osmosis
- contains Glucose – provides energy (given in the form of starch, as starch is insoluble)
- contains Potassium ions – increases appetite

**Function of Lungs?** site of gas exchange (oxygen into blood – used in cells for respiration, carbon dioxide out of the blood – toxic waste product of respiration)

**What is Lungs made up of?** Trachea, Bronchi, Bronchioles, Alveoli (+ capillaries)

**Function of trachea, bronchi, bronchioles?** transport of air and filter air, (bronchioles also controls amount of air reaching alveoli)

## **Structure of trachea/bronchi?**

- wall made of c-shaped cartilage
- cartilage is strong so trachea/bronchi do not collapse
- cartilage is c-shaped to give flexibility
- lining made of goblet cells and ciliated epithelial cells
- goblet cells make mucus, which traps pathogens/particles
- ciliated epithelial cells have cilia, which pushes mucus up and out of lungs

## **Structure of bronchioles?**

- wall made of smooth muscle
- smooth muscle contracts, lumen narrows, bronchiole constricts
- (occurs when surrounded by noxious gases – reduces amount reaching alveoli)
- lining made of goblet cells and ciliated epithelial cells

## **Adaptation of alveoli?**

- millions of tiny alveoli that are folded (large surface area)
- thin wall/one cell thick/squamous epithelial cells (short diffusion distance)
- elastic tissue in wall (stretches when breathing in to increase surface area, recoils when breathing out to push the air out)
- ventilation maintains concentration gradient (high oxygen, low carbon dioxide)

## **Adaptation of capillaries?**

- millions of tiny capillaries (large surface area)
- thin wall/one cell thick/squamous epithelial cells (short diffusion distance)
- narrow lumen (increases diffusion time, decreases diffusion distance)
- circulation maintains concentration gradient (low oxygen, high carbon dioxide)

**How O<sub>2</sub> moves from the alveoli to the capillaries?** by simple diffusion passing thru the alveolar epithelium and capillary epithelium

**How CO<sub>2</sub> moves from capillaries to the alveoli?** by simple diffusion passing thru the capillary epithelium and alveoli epithelium

**Describe the process of Breathing/Ventilation?**

- Breathing In/Inhalation = external intercostal muscles contract (rib cage moves up and out) & diaphragm contracts (flattens), therefore increase in volume in chest and decrease in pressure, so air moves in
- Breathing Out/Exhalation = external intercostal muscle relax (rib cage moves down and in) & diaphragm relaxes (back to dome shape), therefore decrease in volume in chest and increase in pressure, so air pushed out (aided by elastic recoil in the alveoli)

**Formula for Pulmonary Ventilation?**

- $PV = \text{tidal volume} \times \text{ventilation rate}$
- tidal volume = volume of air breathed in/out in one breath
- ventilation rate = number of breaths per minute
- Pulmonary Ventilation = volume of air breathed in/out per minute

**Pulmonary Tuberculosis?**

- infectious disease
- caused by a bacterial pathogen called Mycobacteria Tuberculosis
- spread in air droplets
- infected person coughs/sneezes, releases air droplets containing pathogen
- requires prolonged exposure
- pathogen enters lungs
- pathogen becomes trapped by white blood cells (primary infection)
- if person's immune system weakens (old age, hiv, medical conditions, malnourished, immunosuppressant drugs) the pathogen may escape causing disease (post primary infection)
- causes damage to lung tissue
- symptoms = fever, cough up blood, night sweats, severe weight loss
- treatment = long course of antibiotics (6-9 months)

**Pulmonary Fibrosis?**

- formation of scar tissue (in alveoli)
- increases diffusion distance (so less gas exchange)
- reduces elasticity (so less air pushed out of lungs)
- reduces surface area (so less gas exchange)
- symptoms:
- shortness of breath (rapid breathing but shallow breaths)
- chronic dry cough (due to presence of scar tissue)
- discomfort in chest

- fatigue/tiredness

### **Emphysema?**

- caused by smoking
- alveoli becomes permanently stretched (shortness of breath)
- alveoli walls breakdown (reduced surface area)
- alveoli burst (reduced surface area)
- symptoms = shortness of breath, chronic cough (more mucus), bluish skin colouration

### **Asthma?**

- localised (lungs) allergic reaction
- main allergens = pollen, animal fur, dust
- other factors = air pollution, stress, cold air, exercise
- allergens/factors cause white blood cells to release histamine, histamine causes inflammation, so bronchioles constrict & goblet cells release more mucus (airways narrow)
- symptoms = difficulty breathing, wheezing (whistling sound), tight feeling in chest, cough

### **Heart?**

- job is to pump blood around the body (delivers nutrients to cells and remove waste)
- made of 4 muscular chambers (2 atria, 2 ventricles)
- atria pumps blood to ventricles, ventricles pump blood out of heart (R to lungs, L to body)
- ventricles thicker than atria (has to pump blood further)
- left ventricle has a thicker muscular wall than right ventricle, therefore has stronger contractions, so can generate higher pressure and pump the blood further around the body

### **Blood vessels of the heart?**

- artery takes blood away from the heart, vein returns blood to the heart
- Vena Cava supplies R atrium (with deoxygenated blood from body)
- Pulmonary Vein supplies L atrium (with oxygenated blood from lungs)
- R ventricle supplies Pulmonary Artery (deoxygenated blood to lungs)
- L ventricle supplies Aorta (oxygenated blood to body)

### **Job of valves in heart?**

- Ensure one way flow of blood, no backflow
- (blood flows from atria to ventricles to arteries)
- 2 sets of valves: Atrio-ventricular Valve & Semi-lunar Valve
- AV valve = between atria and ventricles
- SL valve = between ventricles and arteries

**When are AV valves open or closed?** Open = pressure in atria greater than pressure in ventricles,  
Closed = pressure in ventricles greater than pressure in atria

**When are SL valves open or closed?** Open = pressure in ventricles greater than pressure in arteries,  
Closed = pressure in arteries greater than pressure in ventricles

**Describe the processes of the cardiac cycle?**

- Filling Stage = atria relaxed, ventricles relaxed, AV valve open, SL valve closed
- Atria Contracts = the SAN located in the R atrium initiates the heart beat and sends the impulse across both atria making them contract, this pushes all the remaining blood into the ventricles so it becomes full
- Ventricles Contract = the AVN picks up the impulse, delays it (*stops the atria and ventricles contracting at the same time, so the atria empties and the ventricles fill*), sends the impulse down the septum in the Bundle of His, then at the apex the impulse goes up both walls of the ventricles in the Purkinje fibres, *so the ventricles contract from the base upwards, pushing the blood up thru the arteries*, when the ventricles start to contract the AV valve closes then the SL valve opens and blood leaves the heart
- Ventricles Relax = the SL valve closes then the AV valve opens and filling starts again

**What causes the Heart Sounds?**

- when the valves close
- 1<sup>st</sup> = AV closes
- 2<sup>nd</sup> = SL closes

**Formula for Cardiac Output?**

- $CO = \text{Stroke Volume} \times \text{Heart Rate}$
- stroke volume = volume of blood pumped out of the heart in one beat
- heart rate = number of beats per minute
- Cardiac Output = volume of blood pumped out of the heart in one minute

**Coronary Heart Disease and Myocardial Infarction?**

- high blood pressure damages lining of coronary artery
- fatty deposits/cholesterol builds up beneath the lining, in the wall = Atheroma
- the atheroma breaks thru the lining forming an Atheromatous Plaque on the lining, in the lumen
- this causes turbulent blood flow
- a blood clot (thrombus) forms
- this blocks the coronary artery
- therefore less blood flow to the heart muscle
- less glucose and oxygen delivered
- the heart muscle cannot respire
- so it dies (myocardial infarction)

## **Risk Factors of CHD?**

- Age, gender, ethnicity
- Saturated fats (increases LDL, LDL deposits cholesterol in the arteries to form atheroma)
- Salts (increases blood pressure – lowers water potential of the blood so it holds the water)
- Smoking (nicotine = increase HR and makes platelets more sticky – blood clot, carbon monoxide = permanently blocks haemoglobin)
- Obesity and Lack of Exercise

**Atheroma & Aneurysm?** atheroma weakens wall of artery, blood builds up in the wall, the wall swells then bursts = aneurysm

## **What is a pathogen?**

- a disease causing micro-organism
- e.g. bacteria, virus, fungi
- bacteria cause disease by producing toxins
- virus cause disease by dividing in cells causing them to burst

## **Body's defence against pathogens?**

- I, Barriers (prevents pathogens entering the body)
- II, Phagocytes (perform phagocytosis and stimulate specific response)
- III, Specific Response (uses lymphocytes to produce memory cells and antibodies)

## **What are the Barriers (I)?**

- Skin, an impermeable barrier made of keratin
- Cilia & Mucus in Lungs
- Stomach Acid (denatures/breaksdown pathogens)

## **Describe the process of Phagocytosis (II)?**

- pathogen releases chemicals
- this attracts the phagocyte
- the phagocyte binds to the pathogen
- the phagocyte engulfs the pathogen
- forms a phagosome around the pathogen
- lysosomes inside the phagocyte release digestive enzymes into the phagosome
- breaking down the pathogen

## **Describe the Specific Response (III)?**

- phagocytes perform phagocytosis (engulf and destroy pathogen) without destroying the antigen, they place antigens on their surface, they present antigens
- t lymphocytes (t cells) bind to the antigen and become stimulated
- they divide by mitosis to form 3 types of cells: t helper, t killer, t memory
- t helper cells stimulate b lymphocytes (b cells)

- t killer cells kill infected cells (infected by virus)
- t memory cells provide long term immunity
- b lymphocytes (b cells) engulf and present antigens on their surface, the t helper cells bind to this
- the b cells become stimulated and divide by mitosis to make 2 types of cells: Plasma Cells & B Memory Cells
- Plasma cells make antibodies
- B memory cells provide long term immunity

**What is an antigen?** a protein on the surface of a pathogen that stimulates an immune response

**How does the immune response lead to production of antibodies?** the phagocytes stimulate the t cells, the t cells form t helper cells, the t helper cells stimulate the b cells, the b cells form plasma cells, the plasma cells make antibodies

**What is an antibody?**

- a globular protein
- made by plasma cells
- has 3 regions: variable region, hinge region, constant region
- variable region has a different shape in each antibody, contains the antigen binding sites, these bind to complementary antigens (on a pathogen) to form an antigen-antibody complex, destroying the pathogen
- hinge region gives the antibody flexibility
- constant region the same shape in all antibodies, binds to phagocytes to help with phagocytosis

**How do Memory cells (B/T) work?**

- made during the specific immune response after a new infection by a pathogen (called a primary infection)
- B and T memory cells remain in the blood
- if person is reinfected by the same pathogen (called a secondary infection) the memory cells will recognise the pathogen and produce antibodies RAPIDLY and to a LARGE amount
- therefore the pathogen is killed before it can cause harm = immunity

**How does a vaccine produce immunity?** involves giving an injection that contains dead/weakened pathogens that carry antigens which stimulates the immune response leading to production of antibodies & memory cells

**Active vs Passive immunity?**

- Active = individual has memory cells – can make their own antibodies & provides long term immunity
- Passive = person given antibodies, these work then die, no long term immunity, no memory cells.

**How does activity immunity occur?** naturally = by primary infection, artificially = by vaccination

**How does passive immunity occur?** naturally = from mother to baby (placenta or breast milk),  
artificially = by injection

### **Successful Vaccination Programme?**

- produce suitable vaccine (effective – make memory cells, does not cause disease, no major side effects, low cost, easily produced/transported/stored/administered)
- herd immunity

**What is herd immunity?** when a large proportion of the population is vaccinated, therefore most people will be immune, only a few will not be immune, increases chance of non-immune person coming into contact with immune person, so the pathogen has no where to go, so it dies out

### **Problems with Vaccination Programmes?**

- vaccine does not work (dead form ineffective, pathogen hides from immune system)
- vaccine not safe (no weak/inactive form, causes major side effects)
- many strains of pathogen
- cannot achieve herd immunity (logistic of vaccinating large proportion)
- antigenic variability

**What is antigenic variability?** the pathogen mutates, the antigen changes shape, so the memory cells no longer complementary – do not recognise the pathogen, therefore the pathogen can re-harm

**What is a monoclonal antibody?** one type of antibody, complementary to one type of antigen, made by one type of plasma cell

**What are monoclonal antibodies used for?** identify specific antigens or antibodies in person's blood

### **How do monoclonal antibodies identify specific antigens in the blood?**

- e.g. identify PSA antigen made by prostate cancer
- place monoclonal antibodies complementary to PSA antigen on test plate
- add person's blood to test plate
- if PSA antigen is present in the blood, it will bind to the monoclonal antibodies
- then a 2<sup>nd</sup> set of monoclonal antibodies with an enzyme attached is added
- if the PSA antigen is present, this 2<sup>nd</sup> set will bind to it
- if the PSA antigen is not present, this 2<sup>nd</sup> set will not bind
- the test plate is then washed
- if PSA antigen is present, 2<sup>nd</sup> set of monoclonal antibodies will attach, this will not be washed away, so the enzyme will be present
- if PSA antigen not present, 2<sup>nd</sup> set of monoclonal antibodies will not attach, this will be washed away, so enzyme also washed away
- a colourless substrate is then added, if the enzyme is present it will breakdown the substrate causing a colour change, if the enzyme is not present there will be no colour change

- therefore: colour change occurs = enzyme present/PSA antigen is present, no colour change = no enzyme present/no PSA antigen is present

### **How do monoclonal antibodies identify specific antibodies in the blood?**

- e.g. identify TB antibodies in the blood
- place antigen complementary to TB antibodies on test plate
- add person's blood to test plate
- if TB antibodies are present in blood, they will bind to the antigen
- then a set of monoclonal antibodies (with an enzyme attached) complementary to the TB antibodies are added
- if the TB antibodies are present, the monoclonal antibodies will attach
- if the TB antibodies are not present, the monoclonal antibodies will not attach
- the test plate is then washed
- if the TB antibodies are present, the monoclonal antibodies will attach, this will not be washed away, so the enzyme will be present
- if the TB antibodies are not present, the monoclonal antibodies will not attach, this will be washed away, so the enzyme will be washed away
- a colourless substrate is then added, if the enzyme is present it will breakdown the substrate causing a colour change, if the enzyme is not present there will be no colour change
- therefore: colour change occurs = enzyme present/TB antibody is present, no colour change = no enzyme present/no TB antibody is present