Test for reducing sugar

All monosaccharides and *some disaccharides (eg maltose)* are **reducing sugars**. A reducing sugar is a sugar that is able to **donate electrons** to another chemical, in this case Benedict's reagent.

Benedict's reagent is an *alkaline solution* of *copper (II) sulfate*. When a reducing sugar is heated with Benedict's reagent, it forms an *insoluble red precipitate* of *copper (I) oxide.*

The test is carried out as follows:

- Add $2cm^3$ of the food sample to be tested to a test tube. Sample must be in *liquid form*.
- Add an *equal volume of Benedict's reagent*.
- *Heat* the mixture in a *hot water bath* for *five* minutes.
- *Blue → Brick red*

Benedict's test has a **semi-quantitative nature**. When it gives you a *colour,* the test is said to be semi quantitative.

Different concentrations of reducing sugars give *different colours.*

Blue= none, Green, Yellow, Orange, Red= High

Test for non-reducing sugar

Other disaccharides, such as sucrose, are **non-reducing sugars** because they **do not change the colour of Benedict's reagent** when *heated with it*. In order to detect a non-reducing sugar it must first be *broken down* into its monosaccharide components by *hydrolysis*.

Process:

1)

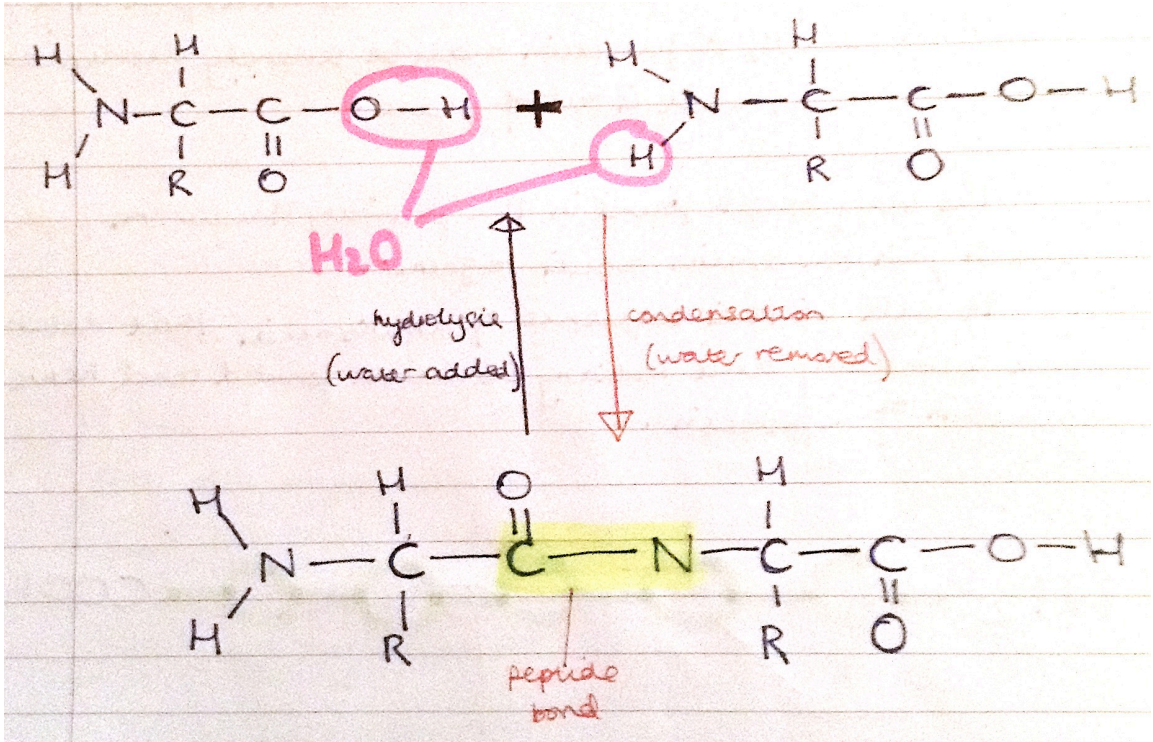
- If the sample is not already in *liquid form*, it must first be ground up in water.
- Add $2cm^3$ of the food sample being tested to $2cm^3$ of Benedict's reagent, and *heat in a boiling water bath* for 5 minutes. If the Benedict's reagent *doesn't change colour*, a reducing sugar *is not present.*

2)

- Add another $2cm^3$ of the food sample to $2cm^3$ of **dilute hydrochloric acid** in a test tube and place the test tube in a *boiling water bath for 5 minutes*. The dilute hydrochloric acid will **hydrolyse** any disaccharide present into its *constituent monosaccharides.*
- Slowly add some **sodium hydrogencarbonate** to *neutralise the hydrochloric acid*. Test with *pH paper* to ensure solution is now *alkaline.*
- *Re-test* the resulting solution by *heating it with $2cm^3$ of Benedict's reagent* in a boiling water bath for 5 minutes.
- If a non-reducing sugar was previously present, Benedict's reagent will now turn an *orange-brown colour* due to the *reducing sugars* produced as a result of the hydrolysis.

Peptide bonds *join amino acids together*. Two amino acids combine to form a *dipeptide* by a condensation reaction between the amino group of one and the carboxyl group of the other. Further amino acids join together to form a *polypeptide chain*. Proteins consist of one or more polypeptide chains.

The *specific sequence of amino acids*, controlled by DNA code, will determine *which protein* will be formed.



Digestion

In humans, digestion takes place in two stages; physical and chemical digestion.

Physical digestion

If the food is *large*, it is broken down into *smaller pieces* by means of structures such as *teeth*. This also provides a *large surface area for chemical digestion*. Food is also churned up by muscle in the stomach wall.

Chemical digestion

Chemical digestion breaks down *large, insoluble molecules* into *smaller, soluble ones*. It is carried out by enzymes called **hydrolases**- enzymes that *split molecules up by hydrolysis*.

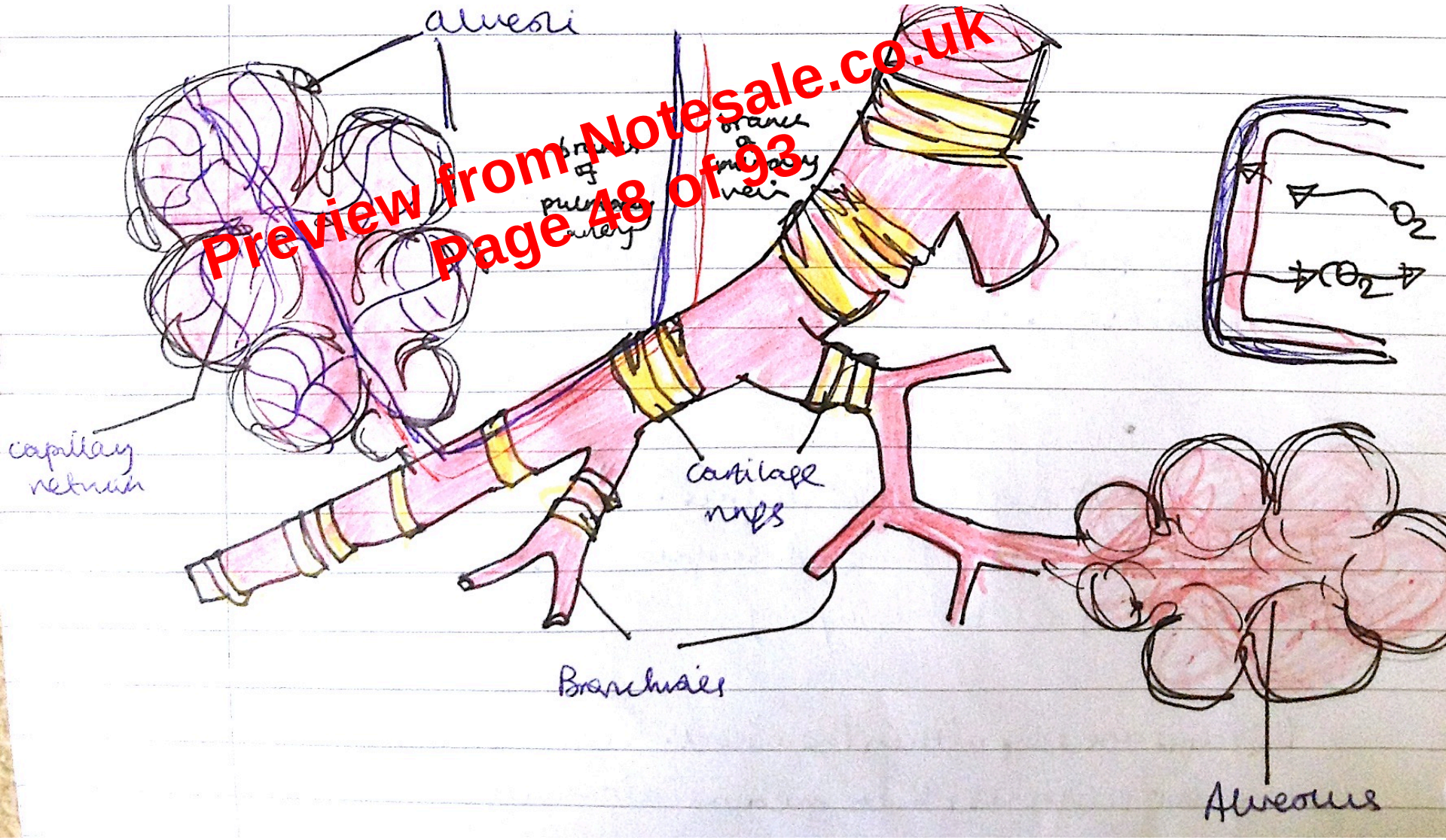
Carbohydrases break down *carbohydrates*.

Lipases break down *lipids* into glycerol and fatty acids

Proteases break down *proteins* to amino acids

The human respiratory system

1. Air enters the airway through *either the nose or mouth*. Air entering via the nose cavity is *filtered by hairs in the nasal passages, warmed* and *moistened* by cells in the *mucous membrane* of the nasal cavity. Mucus membranes line much of the airway. These membranes contain **goblet cells** which secrete **mucus**, a slimy material rich in ***glycoproteins.***
   Air entered the buccal cavity is not warmed or moistened as much, and is *not filtered at all.*
2. The nasal and buccal cavities lead into the pharynx, a tube that conducts both food and air. When *food* is swallowed, a flap of tissue called **the epiglottis** *closes over the glottis*. This is a reflex action which *prevents food from going down the trachea.*
3. Air passes through the larynx into the trachea. This single tube forms a major airway. The trachea is *held open* by **rings of cartilage**. Without them, the trachea would *collapse during breathing out*, when the *external atmospheric pressure* is *higher than the pressure inside the trachea.*
   The **gaps** in the cartilage rings allow the trachea to be *flexible* so that *food can easily pass down the oesophagus* which runs *behind* the trachea.
4. The trachea is lined *with mucus membrane* containing *ciliated epithelium cells*, which have microscopic hair like extensions called **cilia.** These beat in a wavelike manner, moving mucus, dust and microorganisms upwards and out of the lungs.
5. The trachea *subdivides* into two main branches; the right and left bronchus. The larger bronchioles are lined by *complete rings of cartilage* but *not the very small ones,* which collapse quite easily
   Trachea, bronchi and bronchioles contain ***smooth muscle*** which enables them to *constrict.* Small bronchioles can *constrict completely* because they lack cartilage.



THE HEART

The cardiac cycle

Relaxation of the heart- diastole

- Blood *returns to the atria* of the heart from the pulmonary vein and the vena cava. As the *atria fill*, the *pressure within them rises*, ***pushing open the atrioventricular valves*** and allowing the *blood to pass* ***into the ventricles***.
- The muscular walls of both the atria and the ventricles are **relaxed** at this point. The relaxation of the ventricle wall *reduces the pressure within the ventricle*, causing the *pressure of the ventricles* to be *lower* than the *pressure of the aorta and pulmonary arteries*. This causes the ***semi-lunar valves to close*.**

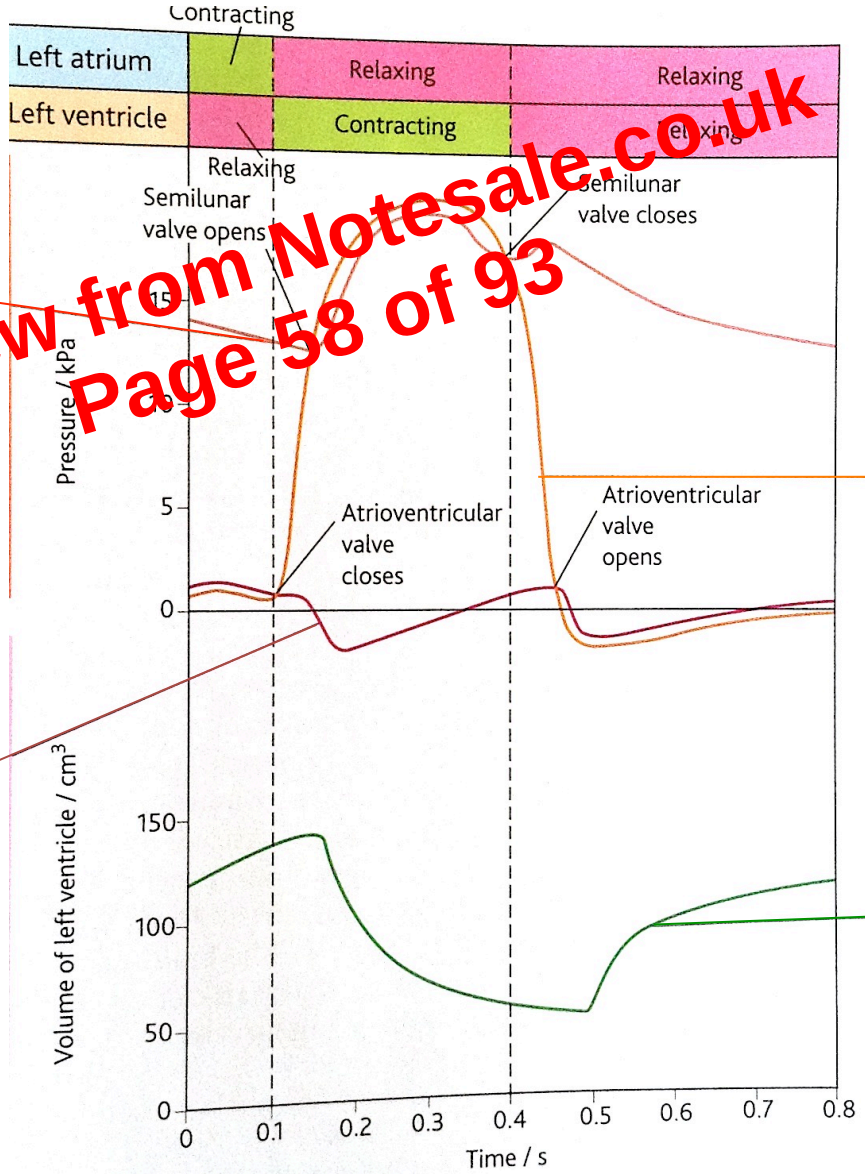
Contraction of the atria- atrial systole

- The *muscle of the atria walls* **contract**, *forcing the remaining blood* that they contain *into the ventricles*.
- The blood has only to be pushed a *very short distance* and therefore the muscular walls of the atria are *very thin*.
- Muscle of the *ventricle wall* remains **relaxed**

Contraction of the ventricles- ventricular systole

- After a short delay to *allow the ventricles to fill with blood*, their walls **contract simultaneously**. This *increases the blood pressure* within them, *forcing* ***closed the atrioventricular valves*** and *preventing backflow* of blood into the atria.
- With the atrioventricular valves closed, the *pressure rises further*, ***forcing open the semi-lunar valves*** and *pushing blood into the pulmonary artery and aorta*.
- The walls of the ventricles are *much thicker* than those of the atria, as they have to *pump blood much further*. The wall of the *left ventricle* has to pump blood to the *extremities* of the body, and so is *much thicker* than that of the right ventricle.

Aortic pressure *rises when ventricles contract*, as blood is *forced into the aorta*. It then *gradually falls*, but never below 12kPa because of the *elasticity of its wall*, which creates **a recoil action**. The recoil produces a *temporary rise in* pressure at the *start of the relaxation phase*



Ventricular pressure is low at first but *gradually increases* as the *ventricles fill with blood* as the *atria contract.* The left atrioventricular valves close and *pressure rises dramatically* as the ***thick muscular walls of the ventricle contract.*** As pressure rises ***above that of the aorta***, *blood is forced into the aorta* past the semilunar valves. Pressure *falls* as the ventricles *empty and the walls relax*

Atrial pressure is *always relatively low* because the ***thin walls*** *of the atrium cannot create much force.* It is *highest when they are contracting*, but drops when the left atrioventricular valve closes and its walls relax.

Ventricular volume *rises* as the atria contract and the ventricles *fill with blood*, and then *drops suddenly as blood is forced out into the aorta* when *the semilunar valve opens*. Volume increases again as ventricles fill with blood.

Valves in the control of blood flow

Valves in the cardiovascular system are designed so that they *open* whenever the *difference in blood pressure either side of them favours the movement of blood in the required direction.*

When pressure differences are reversed, the valves are designed *to close.*

- **Atrioventricular valves** between the *atrium and the ventricle* prevent backflow of blood when *contraction of the ventricles* means that ***ventricular pressure exceeds atrial pressure***. Closure of these valves ensure that, when the *ventricles contract*, blood within them moves *to the aorta and pulmonary artery* rather than *back to the atria*.
- **Semi-lunar valves** in the *aorta and pulmonary artery*. These prevent ***backflow of blood into the ventricles*** when the *recoil action of elastic walls of these vessels* creates a *greater pressure in the vessels* than in the *ventricles.*
- **Pocket valves** in *veins* that occur throughout the venous system. These ensure that when the ***veins are squeezed***, blood *flows back to the heart rather than away from it.*

Koch's postulates

- The *same microorganism* must be found in *all cases* of the disease
- It must be possible to *isolate* the suspected microorganism from the diseased host and *grow it in a pure culture*
- It must be possible *to isolate* the *same microorganism* from an *experimentally infected animal*

When B Cells are activated, they *divide by mitosis*. There need to be many B Cells because pathogens reproduce quickly.

Once divided, they form:

-*More dividing cells*, which then *differentiate* into:

-**Plasma Cells**. These produce the **antibodies** with *specific antigen binding cites*. All plasma cells formed from one type of B cell secrete the *same antibody.*

-**Memory B Cells**, which will remain in the *lymphatic system* for many years.

If the same pathogen with a *specific non-self antigen* re-enters the body, due to the specific B memory cells the response would be *immediate* as the memory cells will *instantly start dividing by mitosis*, resulting in the *production of plasma cells* which in turn quickly produce *antibodies*. Therefore, you get *no symptoms* meaning you are **immune**

1) The *surface antigens* of the invading pathogen are *taken up by B Cells.*
2) The B Cells *process* the antigens and ***present them on their surface.***
3) T Helper cells *attach* to the processed antigens on the B cells, thereby **activating** them.
4) The B Cells are now *activated* to *divide by mitosis* to give a *clone* of the *plasma cells.*
5) The cloned plasma cells produce *antibodies* that *exactly fit* the antigens on the pathogen's surface.
6) The antibodies *attach to antigens* on the pathogen and *destroy them*. This is the **primary immune response.**
7) Some B Cells develop into *memory cells*. These can respond to *future infections* of the same pathogen by dividing rapidly and developing into plasma cells. This is the **secondary immune response.**

Vaccinations

Passive immunity is produced by the *introduction of antibodies* into individuals from an *outside source*.

Active immunity is produced by *stimulating the production of antibodies* by the persons *own immune system*.

Some vaccinations *do not work* because:

-Vaccinations may *fail to induce immune response* in certain individuals.

-Individuals may develop disease *immediately after vaccination*, but *before immunity levels are high enough* to prevent it.

-Pathogen may *mutate frequently*, so their *antigens change suddenly* rather than gradually. New antigens may *not be recognized* by the immune system.

-May be very *many varieties of a particular pathogen*, so almost impossible to develop a vaccine effective against them all.

Tuberculosis

Cause: Either **Mycobacterium tuberculosis** or **Mycobacterium bovis** (rarer)

Symptoms: Persistent *cough, tiredness* and *loss of appetite*. As disease progresses, *fever and coughing up of blood* may occur.

Transmission: Spread through air by *droplets*

Course of infection:

1) The bacteria grow and divide within *the upper regions of the lungs* where there is a *plentiful supply of oxygen.*
2) The body's immune system *responds* and white blood cells accumulate at the site of infection to ingest the bacteria.
3) This leads to *inflammation* and *enlargement of lymph nodes* that *drain that area of the lungs*. This is the **primary infection** and usually occurs in children.
4) In a healthy person, there are usually *few symptoms* and the infection is *controlled within a few weeks*. However, a few bacteria usually remain.
5) Many years later, these bacteria may *re-emerge* to cause a *second infection* of TB. This is called **post-primary tuberculosis** and typically occurs in adults.
6) This infection also arises in *the upper region of the lungs*, but is not so easily controlled. The bacteria *destroy the tissue of the lungs*, resulting in *cavities* and *scar tissue* where the lungs repair themselves.
7) The sufferer *coughs up* damaged lung tissue containing the bacteria, along with *blood*. Without treatment, the TB *spreads to the rest of the body* and can be fatal