

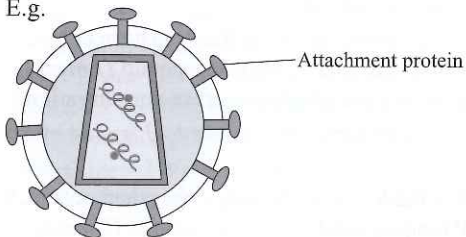
Pages 31-34: Cells and the Immune System — 1

- 1.1 A protein that binds to a specific antigen [1 mark].
- 1.2 B-cells/B-lymphocytes / plasma cells [1 mark]
- 1.3 It has four variable regions, which form the antigen-binding sites [1 mark]. The tertiary structure of the variable regions varies between antibodies [1 mark], giving the binding sites of each antibody a specific shape that is complementary to a specific antigen [1 mark].
- 1.4 E.g. it allows the antibody to bind to more antigens at once [1 mark], so there is a greater chance of agglutination occurring / more pathogens can be phagocytosed at once [1 mark].
- 1.5 $2000 \times 60 \times 60 = 7200000$ [1 mark]
 $= 7.2 \times 10^6$ [1 mark]

There are 60 seconds in a minute and 60 minutes in an hour. So, to work out how many molecules could be produced in an hour, multiply the number that can be produced in one second by 60, and then by 60 again.

- 2.1 Any two from: e.g. their age. / Their ethnicity. / Their sex. / If they are generally healthy/have a disease. / If they are currently taking any medication. / If they have previously been infected with the virus. [2 marks]
- 2.2 percentage change = $\frac{\text{final value} - \text{original value}}{\text{original value}} \times 100$
 $((90 - 10) \div 10) \times 100 = 800\%$ [1 mark]
- 2.3 It means that there is a greater than 5% probability that the results are due to chance [1 mark], so there is no significant difference between the means [1 mark].
- 2.4 The children who aren't vaccinated can be protected through herd immunity [1 mark]. If enough people are immune to a pathogen, it won't be able to spread easily through a population (even if not everyone is immune/has been vaccinated) [1 mark].
- 2.5 Antigen variability means that a pathogen's antigens can change [1 mark]. If antigens change, memory cells produced as a result of a vaccine won't recognise them [1 mark]. Therefore, there won't be a fast secondary response to the pathogen / the person won't be immune to the pathogen [1 mark].

3.1 E.g.



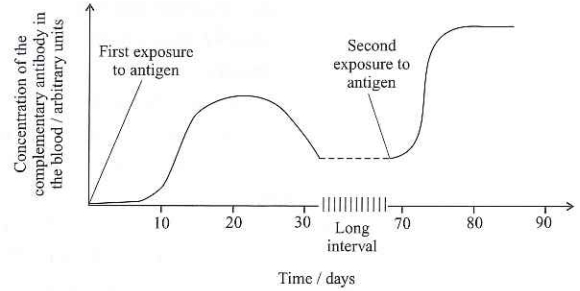
[1 mark]

- 3.2 The attachment proteins on the virus have a specific tertiary structure [1 mark] which allows them to bind to the complementary CD4 cell-surface receptor [1 mark], but not any other receptors/membrane proteins [1 mark].
- 3.3 The HTLV-I genetic material is RNA [1 mark]. Once inside the cell, reverse transcriptase is used to make a complementary DNA copy of the viral RNA [1 mark]. From this, double-stranded DNA is made and inserted into the T-cell DNA [1 mark]. The T-cell enzymes are then used to make HTLV-I proteins from the viral DNA, including Tax [1 mark].
- 4.1 ELISA/enzyme-linked immunosorbent assay [1 mark]
- 4.2 Only antibodies that are complementary to the *Leishmania* antigen can bind to it [1 mark].

- 4.3 To remove any unbound antibodies [1 mark] so that they don't affect the result / cause a false positive result [1 mark].
- 4.4 The enzyme catalyses the reaction of solution X/its substrate, causing a colour change that indicates a positive result [1 mark].
- 4.5 E.g. individuals may see colour change differently. / Colour change may be hard to detect by eye. [1 mark]
- 4.6 E.g. colorimetry [1 mark]

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1.1



[1 mark]

A second exposure to the same antigen will always produce a quicker response, with a greater concentration of complementary antibodies.

- 1.2 E.g. the antibody concentration remains low for several days after exposure to the antigen because there aren't many B-cells that can produce the complementary antibody [1 mark]. Antibody concentration increases as activated B-cells divide to produce plasma cells, which start to rapidly produce lots of antibodies [1 mark]. Antibody concentration reaches a gradual peak and then falls as plasma cells die off [1 mark]. It doesn't fall back to pre-exposure levels because memory cells remain in the body [1 mark].
- 2.1 E.g. so that the woman produces antibodies against the bacteria, which would be transferred to her baby (via the placenta) before it is born [1 mark].
- 2.2 Any five from: e.g. the vaccine contains GBS antigens [1 mark]. Antigen-presenting cells present these antigens to helper T-cells [1 mark]. When the antigens bind to receptors on helper T-cell membranes, the helper T-cells are activated [1 mark]. The helper T-cells then activate B-cells [1 mark], which divide to produce plasma cells that secrete antibodies against GBS [1 mark]. The activated T-cells and B-cells both produce memory cells [1 mark]. Memory cells remain in the body for a long time and result in a faster response to GBS antigens if they appear in the body again [1 mark].
 [Maximum of 5 marks available]
- 2.3 Any two from: e.g. the immunity the baby receives from its mother is immediate, whereas immunity from a vaccine takes time to develop. / A vaccine involves exposing the baby to an antigen, whereas breastfeeding provides immunity without needing to expose the baby to an antigen. / The baby does not produce memory cells as a result of breastfeeding, but memory cells are produced as a result of a vaccine. / The immunity the baby gets from breastfeeding only lasts for a short time, whereas the immunity from a vaccine lasts much longer. [2 marks]
- 2.4 Viruses do not have cell walls [1 mark].

E.g. the monoclonal antibodies bind to the (beta-amyloid) proteins in the plaque and form antigen-antibody complexes [1 mark]. This labels the proteins for destruction by phagocytosis, which breaks down the plaque [1 mark].

The vehicle acted as a control [1 mark] so that the scientists could ensure that the drug caused the observed effect on plaque number and not the vehicle itself [1 mark].

Any one from: e.g. gantenerumab cleared plaques that were less than $300 \mu\text{m}^2$ in size. / Compared to the vehicle/control, gantenerumab reduced the number of plaques formed that were less than $600 \mu\text{m}^2$ in size. / Gantenerumab had no effect on plaques that were greater than $600 \mu\text{m}^2$ in size. [1 mark]

The number of plaques in treated mice was less than the baseline, so you can conclude that the drug prevented plaque formation and removed some plaques. If it was less than the control but not the baseline, then the drug reduced the number of new plaques formed but didn't remove any.

Any four from: e.g. because the study was carried out in mice, not humans, so you don't know how effective the drug would be in humans [1 mark]. The data shows how the drug affected the number of beta-amyloid plaques, not how it affected the symptoms of Alzheimer's [1 mark]. Scientists don't know for certain that amyloid plaques cause Alzheimer's [1 mark]. The gantenerumab did not remove all the plaques in the mice's brains [1 mark]. The data does not record any side effects experienced by the mice, which might make the drug a less effective medical treatment [1 mark]. [Maximum of 4 marks available]

Different blood types have red blood cells with different antigens [1 mark]. The immune system of someone receiving the wrong blood type would not recognise the antigens on the donated red blood cells / would view the antigens on the donated red blood cells as foreign [1 mark]. This would stimulate an immune response, destroying the blood cells [1 mark].

Any six from: e.g. phagocytes recognise foreign antigens on type B red blood cells and engulf them [1 mark]. They present the antigens on their surface [1 mark]. Receptors on helper T-cells bind to these antigens [1 mark]. This stimulates the helper T-cells to activate more phagocytes/cytotoxic T-cells to kill the type B red blood cells [1 mark]. The helper T-cells also activate B-cells [1 mark] which divide to produce plasma cells that secrete antibodies against the type B antigens [1 mark]. The antibodies bind to the type B antigens causing the type B blood cells to clump together/agglutinate [1 mark], labelling them for destruction by phagocytosis [1 mark]. [Maximum of 6 marks available.]

Blood type O has no antigens, so no immune response will be triggered [1 mark].

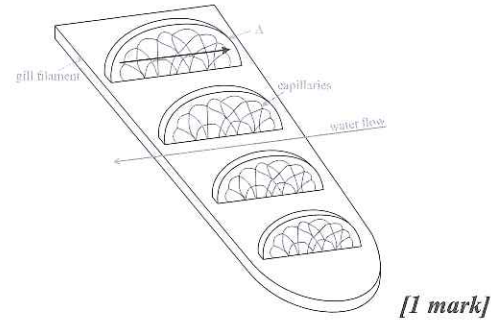
E.g. monoclonal antibodies specific to the antigen(s) of one blood type could be added to a sample of the person's blood [1 mark]. If agglutination is observed, then it can be concluded that the person has that blood type [1 mark].

Topic Three — Exchange and Transport

Pages 39-42: Exchange and Transport Systems — 1

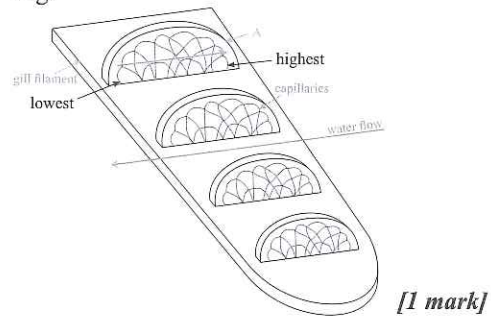
1.1 lamella [1 mark]

1.2 An arrow drawn across structure A in the opposite direction to the arrow showing water flow across the gill filament, e.g.



Fish gills have a counter-current system, meaning the blood flows in the opposite direction to the water.

1.3 E.g.



1.4 E.g. the many lamellae give the gill a large surface area [1 mark], increasing the rate of diffusion of gases [1 mark].

2.1 E.g. dissecting scissors [1 mark]

2.2 A: spiracle [1 mark]

B: tracheae [1 mark]

2.3 E.g. pipette a drop of water onto a slide [1 mark]. Use tweezers to place a section of structures B/the tracheae onto the drop of water [1 mark]. Stand a cover slip upright on the slide, next to the water drop, then carefully tilt and lower it so it covers the specimen [1 mark].

2.4 Any two from: e.g. it is able to close its spiracles when it is losing too much water [1 mark]. / It has a waterproof, waxy cuticle all over its body [1 mark]. / It has tiny hairs around the spiracles [1 mark].

3.1 Mean number of stomata per $0.025 \text{ mm}^2 = (5 + 6 + 7 + 4 + 3 + 8 + 5 + 5 + 3 + 4) \div 10 = 50 \div 10 = 5$

$150 \text{ mm}^2 \div 0.025 \text{ mm}^2 = 6000$

Number of stomata you'd expect to find in $150 \text{ mm}^2 = 5 \times 6000 = 30\,000$ stomata

[2 marks for correct answer, otherwise 1 mark for mean per $0.025 \text{ mm}^2 = 5$, or 1 mark for multiplying mean by 6000]

3.2 E.g. it is based on data from the lower epidermis only and stomata might not be evenly distributed across a leaf [1 mark]. It is based on a small sample size [1 mark].

3.3 Mesophyll [1 mark]

3.4 The stoma is sunken in a pit [1 mark], which traps moist air, reducing the concentration gradient of water between the leaf and the air [1 mark]. This reduces the diffusion and evaporation of water from the leaf [1 mark].