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VCE Biology ¾

Lymphatics & Adaptive Immunity (Humoral) [3.3]

Test Solutions

44 Marks. 1 Minute Reading. 35 Minutes Writing

Results:

Test Questions	_____ / 40
Extension Questions	_____ / 4

Section A: Test Questions (40 Marks)

Question 1 (5 marks)

Tick whether the following statements are **true** or **false**.

Statement	True	False
a. The lymphatic system relies on [illegible] through vessels. It relies on *skeletal muscle* contractions and one-way valves.		✓
b. Lymph nodes are considered secondary lymphoid tissues and act as sites for antigen recognition.	✓	
c. The adaptive immune response [illegible] each time. It is *specific* to antigens and shows *immunological memory*, responding more effectively upon re-exposure.		✓
d. Antigen-presenting cells display antigens using [illegible] cells. APCs use MHC-II to activate *helper T cells*.		✓
e. B cells can [illegible] clonal expa Both clonal selection and T cell help are necessary to activate B cells fully.		
f. [illegible] It targets *extracellular* pathogens. *Cell-mediated immunity* targets intracellular threats.		✓
g. M[illegible] pa They are formed during the *first exposure* but become active during *secondary responses*.		✓
h. Antibodies assist in neutralisation, agglutination, and complement activation to combat pathogens. These are key mechanisms of antibody action.	✓	
i. IgA antibodies are found predominantly in bodily secretions and protect mucosal surfaces.	✓	
j. The lymphati[illegible] bloodstream. Lymph nodes *filter* lymph and *trap pathogens*, preventing systemic spread.		✓

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Question 2 (1 mark)

Sarah gets a tetanus vaccine after stepping on a rust antibodies against the bacterial toxin.

Antigen recognition by a B cell and cytokine signals from a helper T cell trigger clonal expansion and antibody production.

What best explains how her immune system was able to produce these antibodies?

A. Her B cells were triggered by physical injury.

B. Antigen-presenting cells activated cytotoxic T cells to produce antibodies.

C. A B cell bound the tetanus antigen and received helper T cell signals.

D. The vaccine directly inserted antibodies into her bloodstream.

Question 3 (1 mark)

During an influenza infection, Maya's body produces antibodies that prevent the virus from entering her respiratory cells.

Which antibody mechanism is responsible for this defence?

A. Agglutination

B. Neutralisation

C. Complement activation

D. Opsonisation

Neutralisation involves antibodies coating the virus's binding sites, preventing cell entry.

Question 4 (1 mark)

A patient is recovering from a Strep throat infection. Their plasma cells are actively releasing antibodies into the bloodstream.

What is the function of these antibodies at this stage?

A. Engulf the bacteria directly.

B. Trigger histamine release from mast cells.

C. Bind to bacterial antigens and tag them for removal.

D. Convert antigens into harmless molecules.

Antibodies do not engulf but instead bind to pathogens and mark them for destruction (e.g., via phagocytosis).

Question 5 (1 mark)

After exposure to whooping cough, Daniel's B cells undergo rapid division to produce many identical cells.

What is this process known as?

A. Clonal selection.

B. Antigen presentation.

C. Memory formation.

D. Clonal expansion.

Clonal expansion is the rapid division of an activated B cell after antigen recognition and T cell help.

Question 6 (1 mark)

Alex, a child who recently recovered from chickenpox, is exposed to the virus again months later but does not get sick.

What explains his protection during this second exposure?

A. Natural killer cells remained active in his blood.

B. The virus was cleared by mucus secretions.

C. Memory B cells responded rapidly to the familiar antigen.

D. Plasma cells from the first infection are still secreting antibodies.

Memory B cells enable a faster and more efficient antibody response during secondary exposure.

Question 7 (1 mark)

A virus in the bloodstream is coated in antibodies, which causes it to clump together with other viral particles.

What is this process called, and why is it useful?

A. Neutralisation – prevents viral replication.

B. Complement activation – directly lyses the virus.

C. Agglutination – makes viral particles easier for phagocytes to engulf.

D. Opsonisation – causes inflammation at the site of infection.

Agglutination involves clustering pathogens together, aiding their clearance by immune cells.

Question 8 (1 mark)

During a blood infection, bacteria are coated with antibodies that attract phagocytes, increasing their uptake.

This process is best described as:

A. Agglutination.

B. Opsonisation.

C. Complement activation.

D. Clonal expansion.

Opsonisation enhances recognition and engulfment of pathogens by phagocytes.

Question 9 (1 mark)

A cancer patient's lymph nodes are removed during surgery. Months later, they suffer from persistent swelling in the leg.

What is the most likely reason for this swelling?

A. Plasma cells are not producing enough antibodies.

B. The lymphatic vessels cannot transport oxygen.

C. Fluid from tissues cannot drain effectively without lymph nodes.

D. Memory cells cannot be formed without lymph nodes.

Lymph nodes and vessels are essential for draining interstitial fluid; without them, swelling (oedema) occurs.

Question 10 (1 mark)

While recovering from a viral infection, Luke's lymph nodes near his throat swell.

Which of the following best explains this swelling?

A. Plasma cells burst and release antibodies.

B. Fluid accumulates due to blocked arteries.

C. Lymphocytes are being activated and proliferating.

D. Cytokines from B cells cause inflammation in arteries.

Swelling in lymph nodes during infection is due to activation and rapid multiplication of immune cells inside them.

Question 11 (1 mark)

Zara is infected with the Epstein-Barr virus, which spreads in extracellular fluid before entering host cells. Despite this, her B cells don't begin producing antibodies immediately.

What is the most likely explanation for this delay in antibody production?

A. The virus must first be neutralised by neutrophils.

B. B cells require both antigen binding and helper T cell ac

C. Plasma cells must leave the bone marrow before releasin

D. B cells must first engulf the virus to be activated.

B cells need to bind antigen (clonal selection) and receive cytokine signals from activated helper T cells to begin clonal expansion and antibody production.

Question 12 (1 mark)

After receiving a hepatitis B vaccine, James produces memory B cells but no symptoms of illness. Months later, when exposed to hepatitis B at a hospital placement, he showed no sign of infection.

Which best explains this immune protection?

A. The presence of cytotoxic T cells.

B. Ongoing inflammation from the vaccine.

C. A rapid secondary humoral response triggered by memory B cells.

D. Direct killing of virus-infected cells by antibodies.

Memory B cells allow a faster and stronger secondary response, preventing viral spread and symptoms.

Question 13 (1 mark)

Mia has a genetic disorder where her B cells can bind antigens but cannot receive cytokine signals from helper T cells. She is frequently sick with bacterial infections.

Which immune step is most likely impaired?

A. Clonal selection

B. Phagocytosis

C. Clonal expansion and plasma cell differentiation

D. MHC-II antigen presentation

Without T cell cytokines, B cells can't undergo clonal expansion or become plasma / memory cells—even if they bind antigen.

Question 14 (1 mark)

A researcher injects mice with a harmless virus and later re-injects them with the same virus. The second injection causes a much faster antibody spike than the first.

Which cell type and process is most responsible for this enhanced secondary response?

A. Naïve B cells undergoing clonal expansion.

B. Helper T cells producing more cytokines.

C. Memory B cells rapidly differentiating into plasma cells.

D. Cytotoxic T cells identifying infected host cells.

Memory B cells remain after the primary exposure and quickly activate to produce antibodies upon re-exposure.

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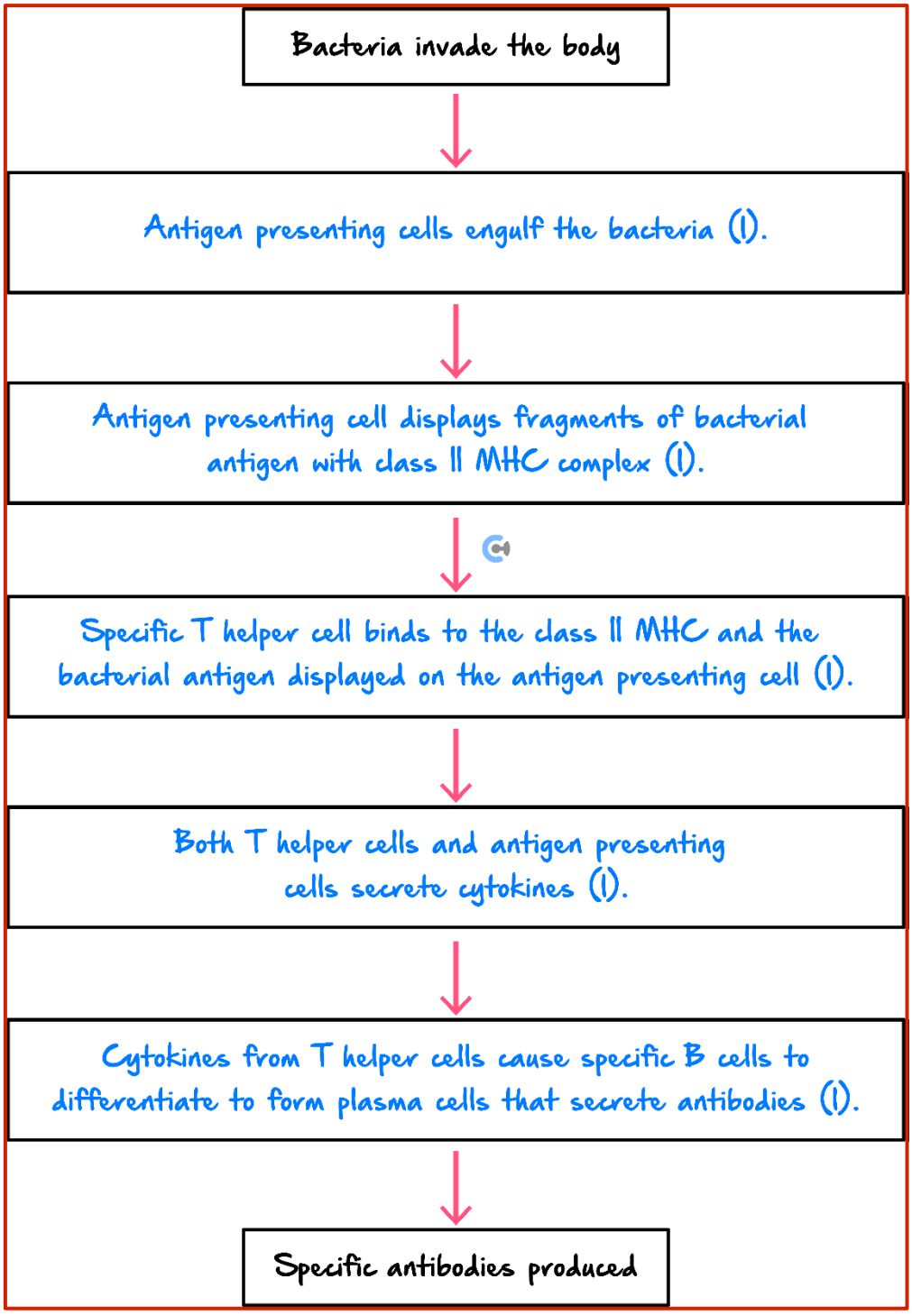
Question 15 (5 marks)

When an individual becomes infected by a pathogenic bacterium it results in a humeral response with the production of the appropriate antibodies. In the spaces below, complete the flow chart to clearly show the steps involved leading to the production of the appropriate antibodies to fight the invading bacteria. The following words **must** be included in your flow chart.

Antigen Cytokines Plasma cell B cells T helper cell Antigen presenting cell Class II MHC



Question 16 (9 marks)

A 45-year-old woman, **Sarah**, has a history of recurrent urinary tract infections (UTIs) caused by Escherichia coli. Her doctor has been concerned about the frequency of her UTIs, which have occurred six times in the past year. Sarah's blood test shows low levels of antibodies specific to *E. coli*, suggesting her immune system is not producing enough antibodies to fight the recurrent infections effectively. Her doctor suggests an immunological evaluation and recommends a personalised treatment plan to address her immune deficiencies.

a. Describe how B cells are activated to produce antibodies in response to an *E. coli* infection. (4 marks)

- **1 mark:** B cells recognise specific antigens on the surface of *E. coli* via their B cell receptor (BCR).
- **1 mark:** The antigen is internalised by B cells, processed, and presented on their surface using MHC II molecules.
- **1 mark:** Activated helper T cells (CD4 +) bind to the antigen-MHC II complex on the B cell and release cytokines to further activate B cells.
- **1 mark:** Activated B cells proliferate and differentiate into plasma cells that secrete antibodies against *E. coli*.

b. Explain how the lymphatic system aids in the detection and response to *E. coli* antigens. (3 marks)

- **1 mark:** The lymphatic system transports B cells, T cells, and antigen-presenting cells (APCs) to lymph nodes, where immune responses to *E. coli* are initiated.
- **1 mark:** Lymph nodes act as sites where antigen-presenting cells (APCs) process and display *E. coli* antigens to activate B cells.
- **1 mark:** The lymphatic system also helps distribute activated B cells and antibodies through the bloodstream to the site of infection.

c. Discuss the potential consequences of the patient's low antibody levels and how this might affect her ability to fight off future infections. (2 marks)

- **1 mark:** Low antibody levels suggest an inadequate immune response, meaning Sarah may not be able to effectively neutralise *E. coli* bacteria during her UTIs.
- **1 mark:** This may result in recurrent infections and complications such as kidney damage, as the body's ability to clear the infection is compromised.

Question 17 (8 marks)

A 50-year-old woman, **Jill**, recently recovered from a skin infection caused by *Staphylococcus aureus* after a minor wound became infected. Blood tests now show the presence of IgM and IgG antibodies specific to *S. aureus*. Jill's doctor explains that the presence of these antibodies suggests her immune system has successfully responded to the infection and is prepared to protect her in case of future exposures to *S. aureus*.

a. Explain how B cells respond during the primary immune response to *S. aureus* infection. (3 marks)

- **1 mark**: B cells recognise *S. aureus* antigens via their surface receptors (BCRs).
- **1 mark**: The antigen is internalised and processed by the B cells, then presented on the cell surface with MHC II molecules for helper T cell recognition.
- **1 mark**: Activated helper T cells release cytokines that promote B cell proliferation and differentiation into plasma cells that secrete antibodies against *S. aureus*.

b. Describe the role of antibodies in eliminating *S. aureus*. (3 marks)

- **1 mark**: Antibodies bind to specific surface antigens on *S. aureus* bacteria, neutralising their ability to adhere to host cells or tissues, which prevents further infection.
- **1 mark**: Antibodies mark *S. aureus* for destruction by immune cells such as macrophages and neutrophils through a process called **opsonisation**. This enhances phagocytosis of the bacteria.
- **1 mark**: Antibodies also activate the **complement system**, which leads to the formation of membrane attack complexes that lyse *S. aureus* bacteria, further aiding in their elimination from the body.

c. Discuss how the formation of memory B cells ensures a faster immune response if Jill is re-exposed to *S. aureus*. (2 marks)

- **1 mark**: Memory B cells are long-lived and retain the information about the *S. aureus* antigen.
- **1 mark**: If Jill is re-exposed to *S. aureus*, memory B cells quickly respond by rapidly differentiating into plasma cells that produce antibodies at a much faster rate than during the initial infection.

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Section B: Extension Questions (4 Marks)

Question 18 (4 marks)

Dendritic cells are one type of cell circulating within the lymph and lymph nodes of humans.

a. Describe how dendritic cells perform their function within the human immune system. (2 marks)

Dendritic cells:

- display antigens on their surface
- present antigens to T cells/activate T helper cells or T cytotoxic cells.

Non-Hodgkin lymphoma is a type of cancer. In most patients with non-Hodgkin lymphoma, the B lymphocytes multiply uncontrollably and are unable to differentiate. The patient can develop recurring infections from normally non-pathogenic bacteria such as *Staphylococcus epidermidis*.

b. Explain why a non-Hodgkin lymphoma patient's immune system would find it difficult to eliminate an infection by *S.epidermidis.* (2 marks)

- can't form plasma B cells
- the plasma B cells would normally produce specific antibodies, cause agglutination or target the bacteria to be destroyed.

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