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# VCE Biology ¾
## The 1st & 2nd Line of Defense [0.12]
## Workshop Solutions

**Error Logbook:**

| New Ideas/Concepts | Didn't Read Question |
|---|---|
| Pg / Q #: _____<br>Notes: | Pg / Q #: _____<br>Notes: |
| **Algebraic/Arithmetic/ Calculator Input Mistake** | **Working Out Not Detailed Enough** |
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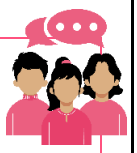
## Section A: Revision and Recall



**Discussion:** Let's create a mind map about the immune system that we have learned so far!

> Head Tutor's Comment: "please ensure to cover all CELLULAR/NON CELLULAR/PROCESS BASED COMPONENTS."

## Section B: Starter Questions (21 Marks)

INSTRUCTION: 21 Marks. 15 Minutes Writing.

**Question 1** (2 marks)

Identify and describe two physical barriers preventing infection in plants.

Refer to the workbook, this is just direct recall from that list. With questions such as these, no need to overcomplicate things – the easy marks in biology are like these which are determined directly off memory.

**Question 2** (2 marks)

Identify and describe physical barriers preventing infection in animals.

INTACT, KERATINISED skin – use this one no matter what!!!

Mucous secretions physically trap pathogens before they are expelled using the beating action of cilia on epithelial cells.

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**Question 3** (2 marks)

Define chemical barriers preventing infection in plants, giving an example of chemical defence.

- Barriers that involve the production of chemicals which are harmful to the pathogen, designed to prevent its proper functioning or development.
  - Chitinases – enzymes that break down fungal cell walls.
  - Phenols – secreted by injured plants, kill microorganisms.
  - Defensins – peptides that are toxic to microbes and fungi.
  - Saponins – disrupt cell membranes of fungi.
  - Oxalic Acid – a substance that can be toxic if it is ingested.
  - Glucanases – defend plants against fungi.

**Question 4** (2 marks)

Identify and describe chemical barriers preventing infection in animals.

- Producing chemical substances that either make an environment unsuitable for a pathogen or kill it.
  - Acidic and basic nature of many places in the body – including sweat and other secretions.
  - Antimicrobial peptides.
  - Lysozyme enzymes.
  - Stomach acid.

**Question 5** (2 marks)

Explain how mucous secretions provide a defence against pathogenic infection in animals.

Mucous secretions trap pathogens and immobilise them – mostly and these trapped pathogens are expelled using the cilia. In the respiratory tract, this occurs via coughing up mucous or swallowing it to destroy pathogens in stomach acid.

**Question 6** (2 marks)

*Akkermansia muciniphilia* secretes molecules that degrade mucin, the functional protein component of mucous. Explain how this may be beneficial to its ability to infect a host.

Degrading mucin will result in damage to the structural integrity of the mucous, which may prevent its function of trapping and immobilising pathogens, or may impact its ability to be removed from the tissue. Hence, there is a decrease in mucous effectiveness, which makes it easier for the bacteria to overcome this barrier, and increase its ability to infect a host.

**Question 7** (1 mark)

Explain what a prion is and why it is non-cellular.

A misfolded protein, that induces a misfold in proteins it comes into contact with. It is non-cellular as it is a protein.

**Question 8** (3 marks)

What is meant by self and non-self? Explain how cells in the immune system differentiate between self and non-self.

Self-cells are cells that are a part of the organism itself, whereas non-self is anything foreign.

Major Histocompatibility Complex. All nucleated cells have MHC (1 mark), therefore, all cells without that marker could be considered as foreign and non-self.

**Question 9** (2 marks)

With reference to immune function, explain why having a completely sterile body would be detrimental for an individual.

A completely sterile body would mean completely lacking any bacteria or other microorganisms. Although this may seem like removal of all pathogen and it will be positive, in reality, it deprives the body of its critical microbiological barrier against pathogens, which prevents infection via competition and secretions.

**Question 10** (1 mark)

Explain the difference between a cellular and a non-cellular pathogen, giving an example of each.

A cellular pathogen is a disease-causing agent that is a cell or a multicellular organism itself, whereas a non-cellular pathogen is not. A non-cellular pathogen includes a virus, whereas a cellular pathogen could be a bacteria.

**Question 11** (2 marks)

Compare the similarities and differences of barriers to infection in plants and animals.

- Both are non-specific responses that are designed to prevent consolidation of a pathogen in the internal environment.
- Involve secretions of chemicals, microbiota and physical prevention mechanisms.
- Differences include the specific ways they go about it – i.e., lack of mucous secretions.

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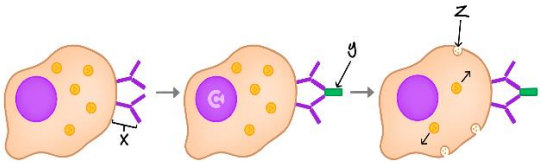
## Section C: Multiple Choice Questions (17 Marks)

**Question 12** (1 mark)

The diagram below shows an immune cell responding to a substance. This process occurs during certain types of allergic reactions.




Which type of immune cell is featured in the diagram above?

**A.** Mast cell

**B.** Neutrophil

**C.** Macrophage

**D.** Dendritic cell

**Question 13** (1 mark)

The complement system is a group of 30 serum proteins that interact as part of the immune system. The complement system:

**A.** Enhances phagocytosis in the second line of defence.

**B.** Is activated by antibodies bound to the pathogen in the first line of defense.

**C.** Results in the lysis of a bacterial pathogen in the first line of defence.

**D.** Destroys cells that are occupied by viruses in the second line of defence.

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**Question 14** (1 mark)

When specialised white blood cells called macrophages ingest and destroy bacteria, which of the following is an important step in the process?

**A.** Ingesting of the bacteria into vacuoles by endocytosis.

**B.** Fusion of the vacuoles containing the ingested bacteria with mitochondria.

**C.** Release of enzymes by lysosomes into the cell cytosol to digest the bacteria.

**D.** Expulsion of unwanted remains of digested bacteria by exocytosis.

**Question 15** (1 mark)

The innate immune response of the body involves the activity of both cells and molecules.

Which of the following is involved in the second line of defence?

**A.** Antibodies produced by memory B-lymphocytes.

**B.** Sweat secreted by the skin containing lysozyme.

**C.** Natural Killer cells that destroy virus-infected cells.

**D.** Cytokine secreted by helper T-cells.

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**Question 16** (1 mark)

Sarah was gardening when she accidentally cut her finger on a thorn. Shortly thereafter, she noticed swelling and redness around the wound. This immediate reaction is crucial in preventing infection by activating certain immune responses.

What primarily happens at the site of Sarah's injury in terms of innate immune response?

**A.** Immediate recruitment of T-cells to initiate adaptive immune responses.

**B.** Rapid mobilisation of neutrophils and macrophages to phagocytise potential pathogens entering through the cut.

**C.** Swift production of specific antibodies by plasma cells.

**D.** Formation of long-term immunological memory against common garden pathogens.

**Question 17** (1 mark)

Dr. Lewis is conducting a study on how skin integrity impacts susceptibility to infections. In his observations, a participant with a minor abrasion on their arm developed a localised infection more quickly than expected, demonstrating the critical role of the skin as an innate barrier.

What is the main consequence of compromised skin integrity in Dr. Lewis's study?

**A.** A significant reduction in lymphatic drainage leading to diminished immune response.

**B.** Increased susceptibility to infections due to a breach in the physical barrier provided by the skin, facilitating easier pathogen entry.

**C.** Immediate enhancement of adaptive immune responses due to increased antigen exposure.

**D.** Uncontrolled cytokine storm leading to systemic inflammatory responses.

Space for Personal Notes

**Question 18** (1 mark)

A health newsletter recently featured an article on the benefits of fever in fighting infections. The article included a case study of a patient with a viral infection who experienced a moderate fever, which helped reduce the severity of the illness.

Why is a moderate fever beneficial in the context of this viral infection?

**A.** It decreases metabolic activity within pathogens, slowing their replication rate.

**B.** It induces an environment less favourable for the virus, as many viruses are sensitive to increased temperatures, potentially slowing their replication.

**C.** It stimulates the infected cells to produce specific antibodies against the virus.

**D.** It speeds up tissue repair and regeneration processes, quickly healing damaged tissues.

**Question 19** (1 mark)

In a recent public health lecture, the topic of discussion was how the body's vascular system responds during inflammation. The lecturer used the example of a patient with a bacterial skin infection, emphasising the role of increased blood flow and vascular permeability in mobilising immune defences.

What is the primary advantage of increased vascular permeability in the context of the patient's bacterial skin infection?

**A.** It confines pathogens to the infected area, preventing systemic spread.

**B.** It allows more immune cells like neutrophils and proteins like complement to enter the infected tissue, enhancing the local immune response to fight the infection.

**C.** It lowers overall body temperature, which inhibits bacterial growth.

**D.** It triggers the development of specific immune responses by exposing the blood to bacterial antigens.

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**Question 20** (1 mark)

A group of medical students is exploring how the body's mucosal surfaces contribute to the first line of defence against pathogens. They focus on how saliva in the oral cavity traps and neutralises bacteria, using an experiment where they measure bacterial activity before and after exposure to saliva.

What crucial role does saliva play in the oral cavity as part of the innate immune defence?

**A.** It contains cells that adaptively respond to microbial exposure.

**B.** It is rich in enzymes like lysozyme and antibodies such as IgA, which trap and destroy bacteria, serving as an effective chemical barrier.

**C.** It rapidly generates large quantities of specific antibodies.

**D.** It neutralises bacterial toxins directly through chemical breakdown.

**Question 21** (1 mark)

Which of the following outlines the function of dendritic cells?

**A.** Form an important role as an antigen-presenting cell.

**B.** Communicate with foreign particle's MHC.

**C.** Communicate with accessory cells.

**D.** All of the above.

Also known as accessory cells, the main function of dendritic cells is to recognise and process antigen material (MHC) and present it on the cell surface to the T cells of the immune system. They act as messengers between the innate and the adaptive immune systems.

Space for Personal Notes

**Question 22** (1 mark)

**Role of Mucus in Pathogen Defense**

**Context**: After spraining her ankle, Emily catches a cold. Increased mucus production in her respiratory tract is noted.

**Question**: How does mucus contribute to the innate immune defence against respiratory pathogens during Emily's cold?

**A.** By creating a slippery surface that prevents pathogen adherence to the respiratory lining.

**B.** By trapping pathogens and debris, which are later expelled by ciliary action.

**C.** By enhancing the activation of local immune cells that release more cytokines.

**D.** By altering the pH of the respiratory tract to inhibit pathogen growth.

**Question 23** (1 mark)

**Complement System Activation**

**Context**: Emily's immune vigilance increased after her ankle sprain, particularly for monitoring bacteria that might enter through skin abrasions.

**Question**: How does the complement system function to combat potential bacterial infection near the site of Emily's ankle sprain?

**A.** By facilitating the clearance of dead cells and debris to prevent secondary infection.

**B.** By mediating the lysis of bacteria through the formation of the membrane attack complex.

**C.** By binding to pathogens and marking them for destruction by other immune cells (opsonisation).

**D.** By blocking pathogen entry into cells through receptor inhibition.

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**Question 24** (1 mark)

**Activation of the Adaptive Immune System**

**Context**: If the innate immune response near Emily's sprained ankle is overwhelmed, the adaptive immune system may need to engage.

**Question**: Which process best describes the signalling from the innate to the adaptive immune system in response to an infection threat?

**A.** Dendritic cells capture and present antigens to T-cells in the nearest lymph node.

**B.** Neutrophils release chemicals that directly stimulate the maturation of B-cells.

**C.** Complement components activate macrophages to produce antibodies.

Solution Pending

**D.** Cytokines released by mast cells promote the differentiation of memory cells.

**Question 25** (1 mark)

The table below shows the proportion of each type of white blood cell as a percentage of the total number of white blood cells that are typically present in a healthy, adult human body.

| White blood cell | Proportion present (%) |
|---|---|
| Neutrophil | 50 |
| Lymphocyte | 30 |
| Macrophages | 4 |
| Eosinophils | 2 |

Reading from the table, the cells involved in the innate immune response are:

**A.** Neutrophils, Macrophages and Eosinophils only.

**B.** Lymphocytes and Eosinophils only.

**C.** Lymphocytes, Macrophages and Eosinophils only.

**D.** Neutrophils, Lymphocytes, Macrophages and Eosinophils.

**Question 26** (1 mark)

Characteristics of inflammation include:

**A.** Leaky capillaries.

**B.** Drop in temperature.

**C.** Vasoconstriction.

**D.** Swollen lymph nodes.

Leaky capillaries are a characteristic of inflammation. Additionally, vasodilation and an increase in temperature are often associated with inflammation. Swollen lymph nodes generally occur when the specific response has been activated.

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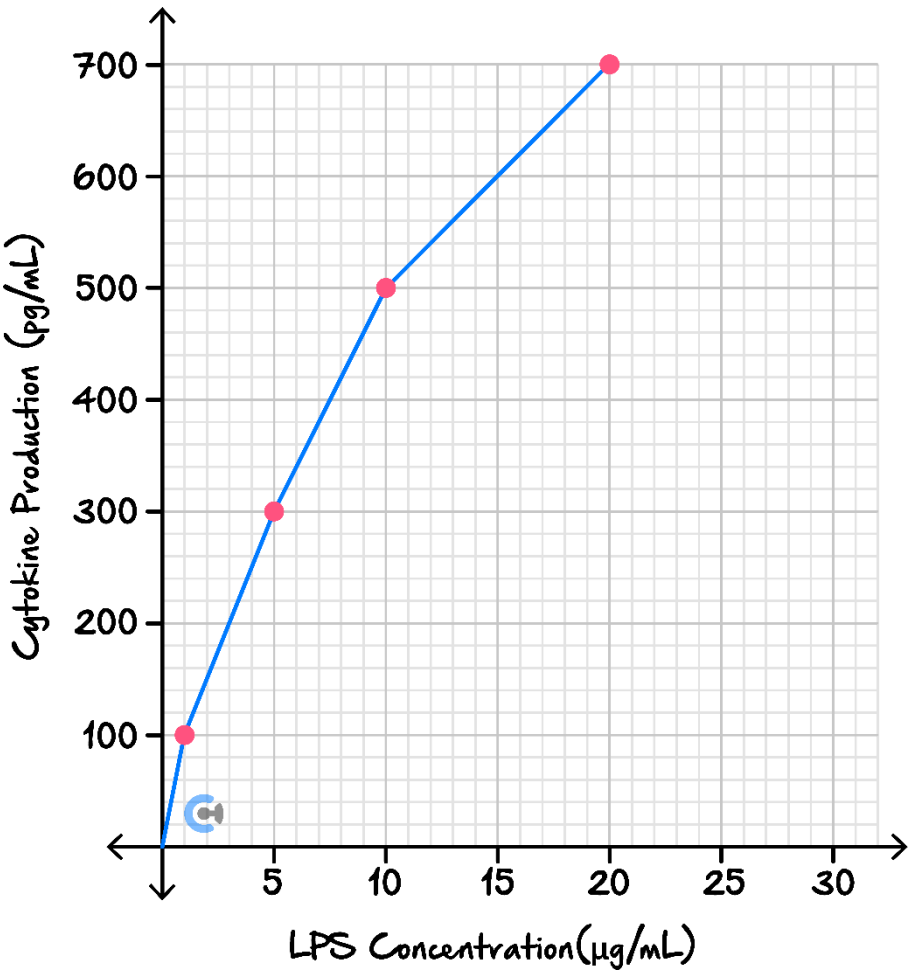
**Question 27** (1 mark)

Researchers are examining the role of macrophages in responding to bacterial infections. They conducted an experiment where macrophages were exposed to varying concentrations of bacterial Lipopolysaccharide (LPS) and measured the production of pro-inflammatory cytokines over time.




What does the increasing cytokine production in response to higher concentrations of LPS suggest about macrophage activation?

**A.** Macrophages are activated in a dose-dependent manner by LPS, leading to increased cytokine production which plays a critical role in initiating and propagating inflammatory responses.

**B.** Macrophages decrease cytokine production as a negative feedback mechanism to prevent overactivation of the immune response.

**C.** LPS concentrations are irrelevant to macrophage activation, and the observed increase in cytokines is likely due to other uncontrolled experimental variables.

**D.** Increasing cytokine production indicates a malfunction of macrophages, suggesting a pathological response rather than a protective one.

**Question 28** (1 mark)

Considering the role of cytokines in the inflammatory response, what potential effects could this macrophage activation have on the affected tissue?

**A.** Enhanced cytokine production can lead to increased blood flow, permeability, and recruitment of other immune cells, intensifying the inflammatory response to effectively contain and eliminate bacterial pathogens.

**B.** Excessive cytokine production might suppress the inflammatory response, leading to inadequate immune activation and the potential spread of infection.

**C.** Cytokine production in this scenario is likely to cause immune tolerance, reducing the effectiveness of the immune response.

**D.** The increase in cytokines would specifically activate adaptive immune components, which is contradictory to the innate nature of macrophage responses.

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## Section D: Exam Style Questions (80 Marks)

**Question 29** (8 marks)

One of the most common infection-causing bacteria is *Staphylococcus aureus*, and it is responsible for a variety of different diseases, depending on its location of infection.

It is found as one of the "normal flora" in our body, commonly found residing on the skin, and has been described as causing "opportunistic infections."

**a.** Is *S. aureus* a cellular pathogen? Explain. (1 mark)

Yes, as it is a bacterium and can replicate on its own accord outside a host cell and causes disease.

**b.** Name the main physical barrier that prevents *S. aureus* from entering the body. (1 mark)

Intact Keratinised Skin

**c.** Explain what is meant by the term 'opportunistic infection.' (1 mark)

An infection caused by bacteria or fungi that are part of the bodies' normal flora.

**d.** Describe why it is useful for the body to retain *S. aureus* on its skin, despite its ability to cause infection. (1 mark)

*S. aureus* can help provide a microbiological barrier against pathogenic bacteria, such as through increasing competition OR by providing an acidic environment through secretions.

**e.** Briefly explain how the immune system would recognise *S. aureus*, in order to engage a ***non-specific*** response. (2 marks)

*S. aureus* is a bacterium, so then it would have certain characteristics common to bacteria, such as pentaglycine and a cell wall. This could be recognised as Pathogen Associated Molecular Patterns (PAMPs) through pathogen recognition receptors on innate immune cells.

OR could also talk about lack of MHC recognition.

**f.** Would interferons be useful for the innate immune response to fight *S. aureus* infection? Explain. (2 marks)

No, they would not be useful. *S. aureus*, as mentioned, would cause a bacterial infection, whereas interferons are more useful in cases of viral infection as they alert surrounding cells to increase resistance mechanisms to viral invasion.

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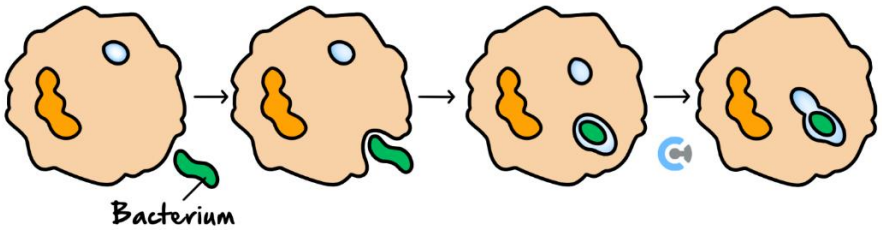
**Question 30** (3 marks)




**a.** Use one word to describe the process shown in the diagram above. (1 mark)

Phagocytosis

**b.** Is this response specific or non-specific? Explain your selection. (2 marks)

This is a non-specific response as the cell is a phagocyte which is not initiated into action through the specific recognition of an antigen; they are generalised leucocytes that will attach to any foreign substance.

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**Question 31** (11 marks)

As the weather gets colder in winter, many people find themselves sick with a 'cold', and although the name is misleading, what people typically associate a 'cold' with is an upper respiratory tract viral infection, caused commonly by rhinovirus. This generally impacts epithelial cells in the nose and throat – those lining the external surfaces.

**a.** Identify and describe a barrier to viral infection of those epithelial cells. (2 marks)

Mucous production and ciliary clearance. Mucous is produced which lines and covers the epithelial cells, trapping pathogens before they can infect them. Next, the cilia on these epithelial cells beat the mucous into the throat where it can be coughed out or swallowed with the stomach acid destroying pathogens.

**b.** Compare and contrast the roles of macrophages and natural killer cells in the response against viral infection. (4 marks)

Natural Killer cells will recognise virally infected cells, and trigger their destruction via the release of perforins and granzymes, triggering apoptosis. Macrophages can work together with these natural killer cells, as they will engulf and destroy the apoptotic bodies that remain after natural killer action. Macrophages may also be able to recognise and destroy viral particles themselves, compared to natural killer cells which are limited to a host cell function.

NEED TO HAVE SOME COMPARISON
"DIFFERENT ROLES AND FUNCTIONS BUT WORK TOGETHER."

Some individuals often mistake these symptoms for hay fever and take antihistamines to avoid the symptoms.

**c.** Naming the cell that releases histamine, describe the process of the inflammatory response and its role in preventing and limiting infection. (4 marks)

Mast Cells

Initiation – mast cells detect cytokines and injury markers, or infection markers from damaged cells at the site of infection, and they are stimulated to release histamine.
Histamine will bind to receptors on the blood vessel wall and trigger vasodilation, widening the blood vessels, which increases the amount of fluid that is being received at the site of infection, carrying immune cells particularly phagocytes.
This will also increase the permeability of the blood vessel which allows more fluid to leak into the surrounding tissue where there has been an injury and/or infection.

There is ultimately an increase of fluid and immune cells being delivered to the site of infection, which results in an increased innate immune response at the local site of injury and or infection.

Some people may also get a fever with an infection.

**d.** Explain why this may be beneficial to the immune response. (1 mark)

This increases the temperature of the body which may improve the activity and function of immune cells whilst also disrupting the pathogen from its optimal temperature.

**Space for Personal Notes**

**Question 32** (10 marks)

Sepsis, or infection of the blood, is an extremely urgent and life-threatening medical situation, which is why it is extremely important for the innate immune system to quickly eliminate any pathogens in the blood.

The complement system is an example of one of these innate immune systems which can act quickly to eliminate pathogens.

**a.** Describe how the complement system functions, with reference to each of the three possible outcomes of complement activation. (4 marks)

The complement system functions as a complement cascade – after the initial protein is activated, it activates the next one and so on to result in 3 main outcomes.

Lysis – forms a membrane attack complex which punches a hole in the membrane, causing lysis of the pathogen.

Opsonisation – complement proteins coat the pathogen's membrane, allowing phagocytes to recognise and destroy them.

Chemotaxis – complement proteins further along the cascade serve to act as signals to immune cells elsewhere in the bloodstream to migrate to the site of infection.

**b.** Identify two cells which use different mechanisms to kill pathogens, and compare and contrast them. (4 marks)

Any phagocyte – uses phagocytosis to ingest and engulf the pathogen, destroying it using enzymes.

Eosinophil – releases toxic chemical mediators which kill larger pathogens that cannot be engulfed.

Natural Killer – releases chemicals which trigger apoptosis.

Compare the target, the methods (inside or outside cell), and the result of any two. More depth than what is given here CHECK WITH AALIYAN.

**c.** Pus can form in some cases when the infection spreads elsewhere. Explain what pus is and with reference to its component explain why infections can result in pus being generated. (2 marks)

Pus is made of dead immune cells that have been involved in fighting infections. Infections can cause an increase in immune cells to a specific location, and pathogens can result in their death, so the accumulation of these large numbers of dead pathogens can cause the formation of pus.

Space for Personal Notes

**Question 33** (6 marks)

With many infections, our body can handle them efficiently and we do not even notice that we have been infected. However, in some infections, particularly bacterial ones, we often require antibiotics to ensure that we don't die.

**a.** How might excessive antibiotic use be detrimental to immune responses against pathogens? Explain. (2 marks)

Talk about antibiotic resistance = 2 marks!

Main answer could be referring to the fact that this will get rid of the microbiological barrier to pathogenic infection.

**b.** Does the innate immune response have the same response for all pathogens? Describe how pathogen recognition occurs with the innate immune response, including natural killer cell recognition. (4 marks)

The innate immune response has the same broad response for all pathogens, however certain types of classes/pathogens have different specialised responses.

For example, natural killer cells fight viral infection by targeting infected host cells and killing them.

Recognition occurs via excitatory and inhibitory receptors, as well as MHC I receptors.

By contrast, phagocytes engulf and destroy pathogens, recognising them using a broad class of common pathogenic molecules known as PAMPs (Pathogen Associated Molecular Patterns), which serve as the trigger for the innate destruction of them via phagocytosis.

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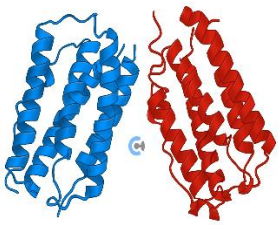
**Question 34** (6 marks)

Interferons (IFNs) are proteins released by host cells in response to the presence of pathogens. The image shows a representation of interferon beta 1.



**a.** What type and level of protein structure is most evident in the image of interferon beta 1 ? (1 mark)

α-helices of the secondary structure

**b.**

**i.** What is a pathogen? (1 mark)

A non-cellular or cellular agent of disease

**ii.** Identify a pathogen that is likely to trigger a response by interferon. (1 mark)

Pathogens likely to trigger a response by interferon are viruses or bacteria.

**c.** Describe how protein signalling molecules, such as interferon, can trigger the protective defences of the immune system in response to infection by a pathogen. (2 marks)

Interferons are proteins (lipid insoluble) and bind specifically to external receptors on the cell membrane. Binding of the interferon activates second messengers in the cytosol which amplifies the initial signal leading to a response from effector proteins which are already present in the cytosol.

**d.** Suggest why some pathogens may show resistance to interferon. How might this occur? (1 mark)

There may be a mutation that confers resistance **OR** they can block signalling events that occur after the cytokine binds to its receptor.

**Question 35** (4 marks)

Tumour Necrosis Factor (TNF) is a naturally occurring protein secreted by cells of the immune system, especially macrophages. This molecule regulates the production of several pro-inflammatory molecules.

**a.** Name the group of molecules that TNF belongs to. (1 mark)

*Cytokines.*

**b.** What is the role of the group of molecules named in **part a.** in protecting the individual against pathogens? (1 mark)

*Cytokines are chemical messengers that can stimulate cell movement to sites of inflammation and infection.*

**c.** What part of the immune system would TNF belong to? (1 mark)

*The second line of defence.*

TNF can bind to two different cell surface receptors TNF1 and TNF2. The result is a different outcome depending on which receptor is activated.

**d.** Why does TNF bind to a cell surface membrane rather than entering the cell? (1 mark)

*TNF is a protein and not hydrophobic so it is not able to enter the cell and therefore its receptor will be on the surface of the cell membrane.*

**Question 36** (7 marks)

When Emily, a wildlife biologist, returned from a field study in a tropical rainforest, she began experiencing severe abdominal discomfort and unusual fatigue. Unbeknownst to her, she had contracted a parasitic infection from contaminated water. Parasites, such as worms, can invade various body systems, leading to a range of health issues, depending on their type and the site of infection. In Emily's case, the parasites had begun to affect her digestive system, causing symptoms like nausea, diarrhoea, and bloating.

**a.** Which innate cell would be most likely to form part of the response against this pathogen? Explain. (3 marks)

Eosinophils – they will degranulate to release toxic chemical mediators which act against the worm as it is too large to be phagocytosed by other cells, and is not a self-cell to be destroyed by natural killer cells.

**b.** Explain how the inflammatory response could be responsible for some of her symptoms. (2 marks)

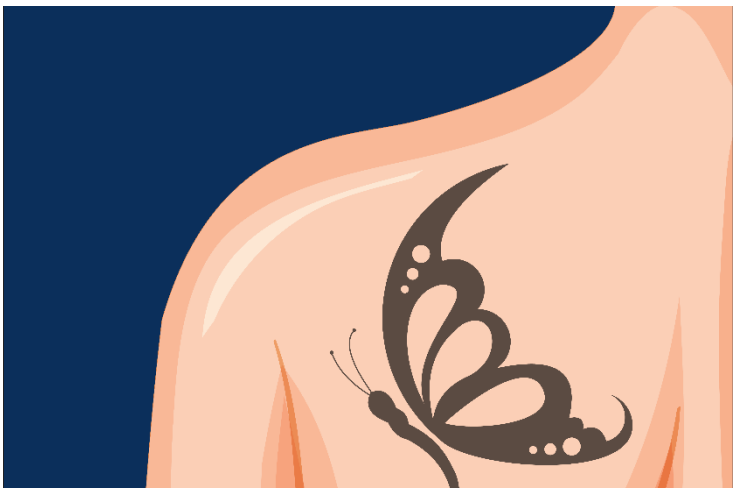
The typical signs of inflammation that we see occur at the surface at the skin – however when this takes place in specific organs, such as the GIT, this may mean different features including dysfunction (nausea, diarrhoea).

**c.** Would interferons be effective in Emily's case? Explain. (2 marks)

Solution Pending

**Question 37** (5 marks)

When a tattoo is performed, pigments that make up the ink are injected into the skin. New research has investigated the role of macrophages in tattoos. When the wound is created as a result of the skin being punctured, macrophages are present at the site of the wound. The research has found that the macrophages move to the site where they capture the pigment and remain there until they die. They become trapped, due to the size of the pigment, and the pigment is then released. This creates a cycle, that is continually repeated when another macrophage then arrives to take up the pigment and subsequently dies. Prior to this research. it was thought that dermal cells in the skin had permanently trapped the pigment.



**a.** Explain the steps of the inflammation process and its importance for tattooing. (2 marks)

*The capillaries around the wound become more leaky* (1). *This allows more phagocytes to the site to fight infection (in this case the pigment or other pathogens* (1).

**b.** By what process would the macrophage 'capture' the pigment in the ink? (1 mark)

*By phagocytosis or endocytosis.*

**c.** Explain the process for how the pigment would be identified as foreign by the macrophages. (2 marks)

*The pigment would have antigens on its cell surface* (1). *These antigens would be recognised as foreign as they are different from the self (MHC markers) antigens* (1).

**Question 38** (10 marks)

A 38-year-old woman named Hana visited her local GP after developing redness, swelling, and warmth around a cut on her lower leg. The GP suspected a bacterial skin infection and collected a sample for testing. Laboratory analysis revealed the presence of *Pseudomonas aeruginosa*, a bacterium commonly found in soil and water that can cause opportunistic infections in open wounds.

The clinic monitored Hana's condition for 10 days without antibiotics to study the natural course of her **innate immune response**. The following observations were made:

- **Day 1–2:** Local swelling, pain, and redness developed. Her temperature increased to 38.5℃.
- **Day 3–5:** Blood tests revealed increased levels of neutrophils and macrophages.
- **Day 6–8:** Complement proteins (e.g., C3b) and inflammatory cytokines peaked.
- **Day 9–10:** The redness subsided and her temperature returned to normal

**a.** Describe how Hana's innate immune system responded in the **first few days** of the infection and explain the biological function of the fever in this context. (3 marks)

- Tissue damage triggered mast cells to release **histamine**, causing vasodilation and increased permeability. *(1)*
- This allowed **phagocytes** and plasma proteins to access the infected site. *(1)*
- **Fever** elevated Hana's core temperature, which:
  - Slows bacterial replication.
  - Increases metabolic rate of immune cells *(1)*.

**b.** Explain the role of **phagocytes** during Hana's infection. Include a reference to how pathogens are identified. (2 marks)

- **Phagocytes** (e.g., neutrophils/macrophages) engulfed and digested the bacteria. *(1)*
- They recognised the bacteria using **Pattern Recognition Receptors (PRRs)** that bind to **Pathogen-Associated Molecular Patterns (PAMPs)**. *(1)*

**c.** One of the lab reports showed a significant increase in **complement protein C3b** on Day 6.

Explain one **direct effect** of complement proteins like C3b and describe how this supports the innate immune response. (2 marks)

- **C3b** acts as an **opsonin**, binding to the surface of bacteria to enhance recognition by phagocytes. *(1)*
- This leads to **faster phagocytosis** and more efficient clearance of pathogens. *(1)*

**d.** What specific innate immune system **cell type** is most responsible for initiating the release of histamine at the site of the wound? (1 mark)

**Mast cells** *(1)*

**e.** After reviewing this case, a group of researchers proposes to deliberately expose patients with minor cuts to *P. aeruginosa* under controlled conditions to study innate responses.
Suggest one bioethical principle this study might violate, and explain why. (2 marks)

- **Principle violated**: Non-maleficence *(1)*.
- Explanation: Deliberately exposing patients to *P. aeruginosa* could cause **harm or infection**, violating the principle of doing no harm. *(1)*

**Question 39** (10 marks)

Jacob, a 70-year-old man, was admitted to the hospital with chills, high fever, and confusion. His symptoms started two days after a routine bladder catheterisation. Blood cultures revealed a Gram-negative bacterium, *Escherichia coli*, had entered his bloodstream, leading to **bacterial sepsis** — a potentially life-threatening condition caused by an overwhelming immune response.

Sepsis triggers a widespread **innate immune response**, including activation of the **complement system**, **inflammatory cytokines**, and **phagocytes**. Doctors collected Jacob's immune profile over four days to monitor his response before antibiotics were administered. The table below shows a summary of the results:

| Day of Illness | C3b Levels | Neutrophil Count | Body Temperature (℃) | TNF-$\alpha$ (Cytokine) Levels |
|---|---|---|---|---|
| Day 1 | Low | Low | 37.2 | Low |
| Day 2 | High | High | 39.5 | High |
| Day 3 | High | High | 38.7 | Moderate |
| Day 4 | Moderate | Moderate | 37.8 | Low |

**a.** Explain how the **complement protein C3b** helps Jacob's immune system fight *E. coli* infection. Include both the mechanism and its impact. (3 marks)

- C3b binds to the surface of *E. coli*, acting as an **opsonin**. *(1)*
- This makes it easier for **phagocytes** (e.g., neutrophils) to identify and engulf the bacteria. *(1)*
- It enhances the speed and efficiency of **phagocytosis**, supporting bacterial clearance. *(1)*

**b.** On Day 2, Jacob's body temperature rose sharply and his TNF-$\alpha$ levels were high.

Explain the **benefits** of fever and TNF-$\alpha$ in an innate immune response. (2 marks)

- Fever increases body temperature, which **inhibits bacterial replication** and **enhances phagocyte activity**. *(1)*
- TNF-$\alpha$ is a cytokine that **promotes inflammation**, recruiting immune cells and activating the vascular response. *(1)*

**c.** Use the data in the table to describe how Jacob's **innate immune response changed** over time and what this suggests about his recovery between Day 2 and Day 4 (3 marks)

- Day 2 shows a **peak innate response**: high C3b, high neutrophils, high cytokines, and fever. *(1)*
- By Day 3, these markers begin to decline (e.g., TNF-$\alpha$ and temperature), suggesting **pathogen load is decreasing**. *(1)*
- By Day 4, levels are returning to normal, suggesting **innate defenses have started resolving the infection**. *(1)*

**d.** Neutrophils are often the first cells to respond to bacterial infections.

Explain how neutrophils recognise and destroy pathogens like *E. coli*. (2 marks)

Solution Pending

**e.** A sample from Jacob's blood was analysed using flow cytometry. The lab found that a large number of neutrophils had internalised *E. coli* cells coated in C3b.

Explain why C3b-coated bacteria were more likely to be phagocytosed than uncoated bacteria.

- C3b acts as an **opsonin**, tagging the bacteria for destruction. *(1)*
- Neutrophils **complement receptors** that recognise C3b, allowing for **faster and more efficient phagocytosis**. *(1)*

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