Pathogens
- BACTERIA
- FUNGI
- PROTOZOA
- VIRUS
- PRION
↳ misfolded protein

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VCE Biology ¾
The Innate Immune System [3.2]
Workbook

Outline:

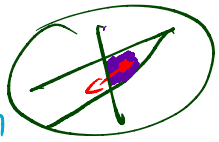
Study Design Key Knowledge:

Study Design: Responding to Antigens

The innate immune response including the steps in an inflammatory response and the characteristics and roles of macrophages, neutrophils, dendritic cells, eosinophils, natural killer cells, mast cells, complement proteins, and interferons.

The initiation of an immune response, including antigen presentation.

good



PLANTS

PHYSICAL

- Bark → prevents pathogen entry
- Waxy cuticle → seals off the leaf
- Galls → killing healthy cells around infection to prevent
- Sunken stomata

CHEMICAL

↳ Plant produces chemicals (toxins) or enzymes to kill pathogens

MICROBIOLOGICAL

↳ normal flora "good bacteria"

↳ harm pathogens

↳ outcompetes pathogenic bacteria

↳ secretes toxins to kill other bacteria

Learning Objectives:

- ☐ **BI34 [3.2.1] -** Explain how the Innate Response is Triggered in Response to Infection & its Defining Characteristics
- ☐ **BI34 [3.2.2] -** Identify & Describe the Function of the Cellular Components of the Innate Immune Response - Macrophages, Neutrophils, Dendritic Cells, Mast Cells, Eosinophils
- ☐ **BI34 [3.2.3] -** Identify & Describe the Role of Non-Cellular Components of the Innate Immune Response - Complement Proteins & Interferons
- ☐ **BI34 [3.2.4] -** Describe Key Innate Immune Processes - Phagocytosis, Inflammatory Response
- ☐ **BI34 [3.2.5] -** Apply Knowledge of Innate Immune Response to Unfamiliar Contexts such as Pathogen Infection
- ☐ **BI34 [3.2.6] -** Explain the Symptoms Associated with the Inflammatory Response

ANIMALS → mucociliary clearance → elevator

PHYSICAL

- Intact skin
- mucous membranes
  - ↳ traps pathogens
- cilia
  - ↳ hairlike projection on the ends of cells that beat rhythmically to expel mucus

CHEMICAL → saliva, sweat, tears

- Lysozymes - antibacterial enzymes
- Stomach Acid
- Antimicrobial Peptides

MICROBIOLOGICAL

GOOD BACTERIA → NORMAL FLORA

preventing colonisation

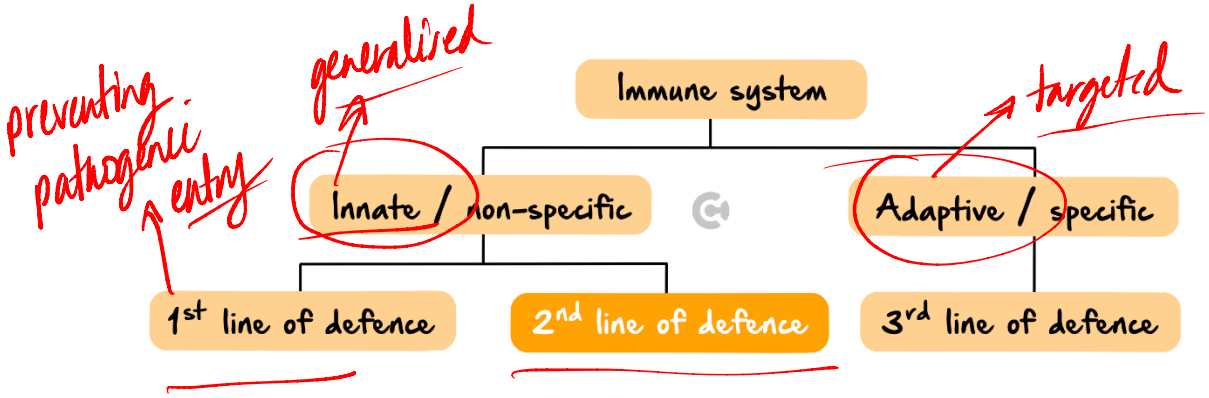
↳ competition

↳ secreting chemicals

Section A: Introduction to the Innate Immune System

Overview

- The innate immune system is the first layer of defence against pathogens.
  - Barriers prevent the entry of the pathogen, this lesson will focus on what occurs next.
- The innate immune system is non-specific and delivers a fast response to any pathogens that make it through to the intracellular space.



Exploration: What do we mean by non-specific?

↳ Delivering the same response regardless of the specific pathogen

- Why might this be detrimental to the long-term prevention of pathogenic infection?

↳ might be able to adapt to and overcome those generalised responses

Exploration: Why is it necessary and important to have a fast response to pathogenic invasion?

1. To get rid of the pathogen as quick as possible
2. To prevent extensive damage / infection before eliminating pathogen

Exploration: Summarise the function of the innate immune system!

1) QUICKLY FIGHT + PREVENT INFECTION

2) ACTIVATE 3RD LINE OF DEFENSE

Analogy: The Castle

- Let's say you are a medieval lord and you've found yourself in a castle.

- To protect against your enemies, what kind of defences would you have? Would these defences change depending on the enemy? What would happen if you just had these primary defences and nothing else?

Introduction to the Innate Immune System

- ☑ Innate immunity acts immediately after pathogen entry.
- ☑ The response is non-specific and does not adapt based on pathogen type.
- ☑ Innate immunity lacks the long-term memory of pathogens.
- ☑ The main functions include preventing pathogen entry, eliminating invaders, activating adaptive responses, and recruiting immune cells.
- ☑ Non-specificity may lead to future infections if pathogens adapt.
- ☑ Fast response prevents pathogens from establishing infection and multiplying.

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Section B: Recognition of Pathogens

PAMPs and DAMPs

- The innate immune response has to still be able to tell the difference between foreign and non-foreign materials.
- What would happen if the innate immune response couldn't do this?

AUTOIMMUNE → self is attacked mistakenly

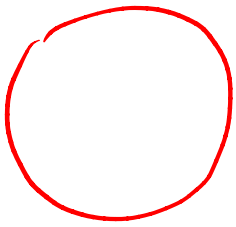
- This recognition doesn't use the MHC markers. Why?

MHC I → MHC II

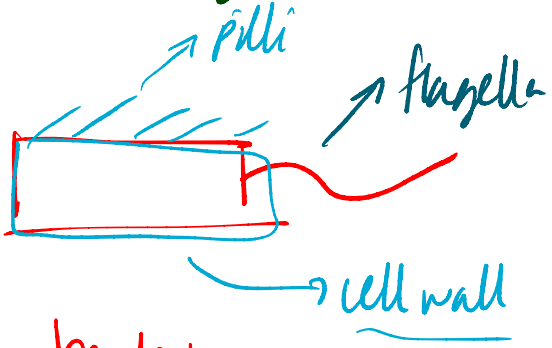
↳ only found on nucleated self cells → foreign pathogens will not have MHC I

- Instead, there are some general common molecules that are associated with pathogens and damage.
  - PAMPs – Pathogen Associated Molecular Patterns. Can you think of any examples?

↳ molecules that are commonly associated with pathogens



self cell



bacteria

  - DAMPs – Damage Associated Molecular Patterns.

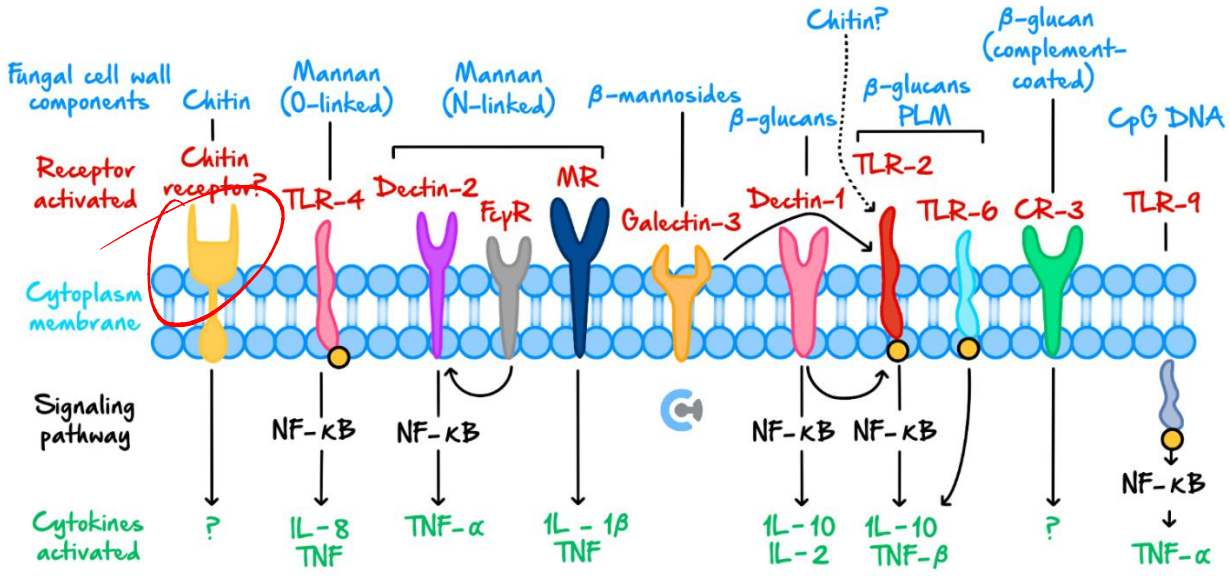
↳ molecules indicating damage

↓

DNA

Exploration: By what principle do you think that PAMPs and DAMPs are recognised?

CELL SIGNALLING



- Don't be intimidated - this isn't what is required knowledge.

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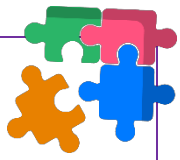
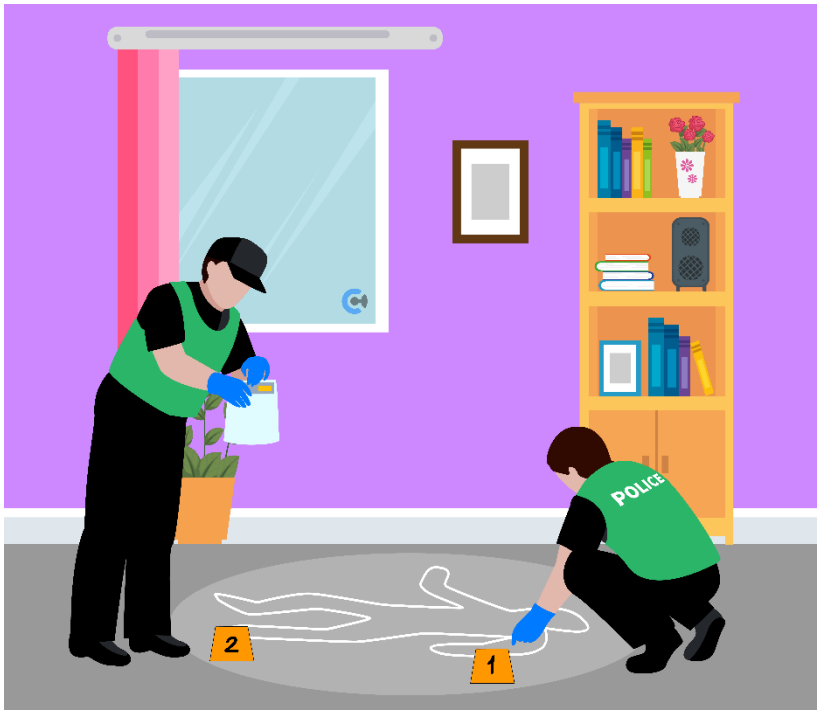


Analogy: Police Profiling

- **Innate immune cells (Police officers):**
  Patrol your body continuously, looking for threats or signs of trouble. To do this efficiently, they need rapid methods to recognise potential problems.

- **PAMPs – Pathogen-Associated Molecular Patterns (General Suspect Profiles):**
  These are common characteristics consistently associated with pathogens, much like general suspect profiles used by police. Just as police look for suspicious behaviours, attire, or patterns frequently exhibited by criminals, immune cells detect shared features of pathogens (like bacterial cell wall components or viral RNA). They don't identify the exact pathogen immediately but quickly recognise danger based on these familiar patterns.

- **DAMPs – Damage-Associated Molecular Patterns (Signs of Disturbance):**
  Similar to how police respond urgently to signs like broken windows, activated alarms, or smoke, DAMPs indicate internal cellular damage or distress. While these signs don't specifically reveal the exact cause, they clearly signal that something harmful has happened internally, prompting immune cells to rapidly investigate and address the issue.



Exploration: What happens next?

Recognition of Pathogens

- ☑ Innate system identifies threats using general molecular patterns.
- ☑ Pathogen-associated molecular patterns (PAMPs) are found on microbial surfaces.
- ☑ Damage-associated molecular patterns (DAMPs) originate from stressed or injured host cells.
- ☑ Pattern recognition receptors (PRRs) on immune cells detect these patterns.
- ☑ Recognition relies on conserved structural features rather than specific antigens.

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IMMUNE SYSTEM

1ST – innate – prevent entry of pathogen → BARRIERS

2ND – innate – quick, non specific (stop spread quickly) → not targeting an antigen

3RD – adaptive slow, specific → look for general features – signify disease + damage

CELLS OF THE INNATE RESPONSE

PHAGOCYTES → eat pathogens (engulf + destroy) via phagocytosis

NEUTROPHILS → expendable
- performs phagocytosis 1-2 times → before they die
- abundant – 60% of WBC in blood → catching pathogen
- 1st responders → neutrophil extracellular traps

MACROPHAGES
- longer lived and larger → cleaning tissues – cleaning debris
- Broader range of function
- permanent part of some tissue
- release cytokines to further coordinate → chemical signals

DENDRITIC CELLS → Antigen Presentation → more specialised for antigen presentation

MAST CELLS → inflammation
- release histamine + other cytokines to stimulate / coordinate immune response

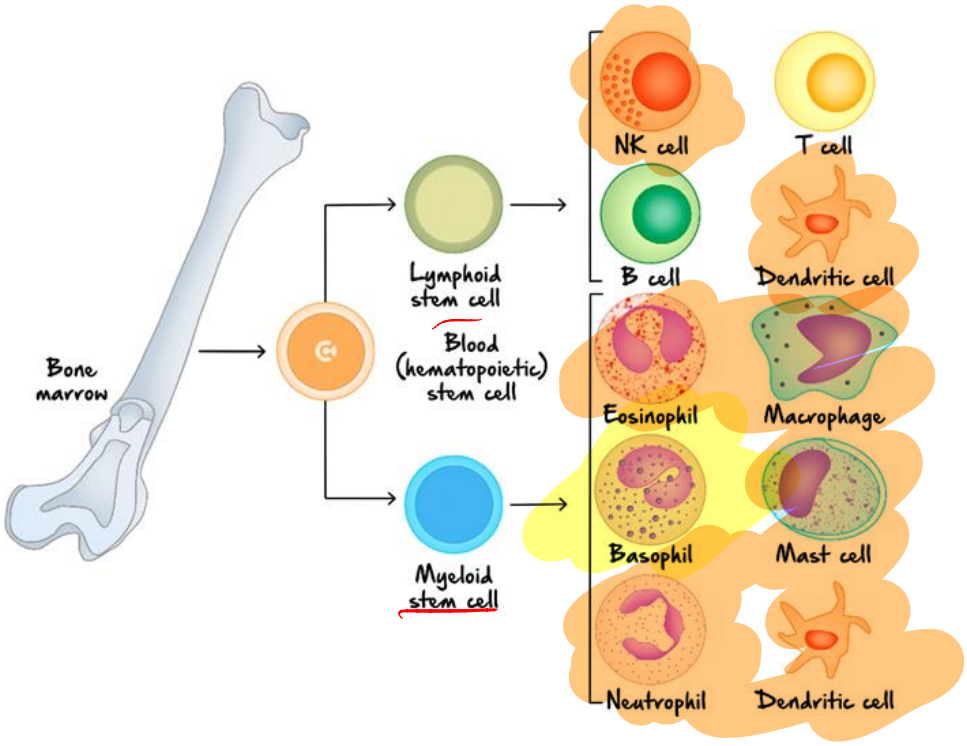
NATURAL KILLER → virally infected cancerous
↳ TARGET UNHEALTHY SELF CELLS
Recognise through abnormal or missing MHC I
Induce apoptosis through release of perforins + granzymes

EOSINOPHILS
- Through exocytosis, release toxic chemicals that will target pathogens too large to phagocytose!

Section C: The Cells of the Innate Immune Response

WHITE BLOOD CELLS (LEUCOCYTES)

Where do these cells arise from?



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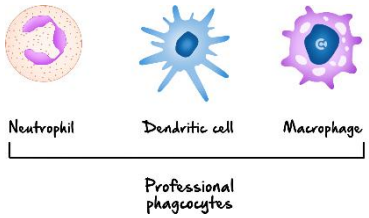
Sub-Section: Phagocytes

- These are cells involved responsible for **phagocytosis**, as the name suggests!



What are neutrophils?

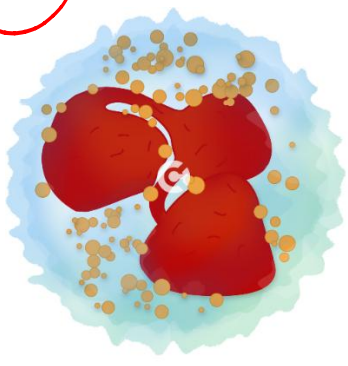
What role do neutrophils play in the innate response?

Definition

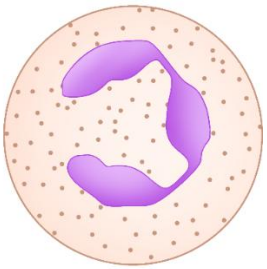
Neutrophils

- They are classed as a phagocyte - engulfing and destroying pathogens.
  - The vast majority of the immune cells in your body are neutrophils - 60%.
- First responders, but also are very expendable.
- Common component of pus.

→ only perform phagocytosis once or twice



3D rendering of a neutrophil



NETosis → neutrophil extracellular traps

How else might neutrophils eliminate pathogens?

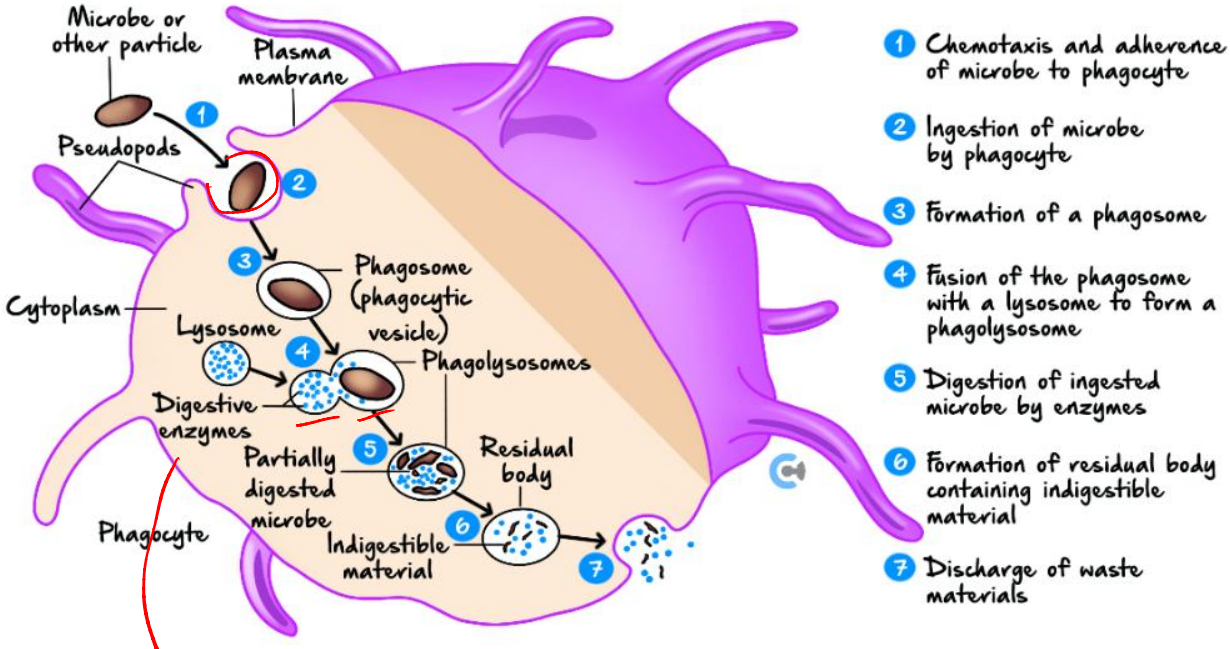
Analogy: The Expendable Foot Soldier

- Medieval soldiers, like Ali pictured below, were often mere pawns in the larger scheme of war and conquest, sacrificed for mere metres of land.
- You could say that neutrophils are like Ali, the common soldier, sacrificed for the greater good!

- The body can generate neutrophils every 2-3 days, so it can just sacrifice them on the field of battle!

Any resemblance to real persons is purely coincidental 😉!

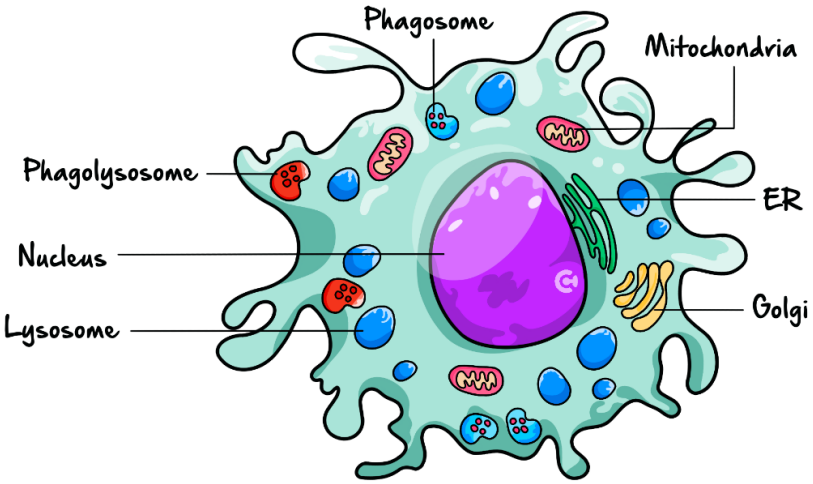
Exploration: Phagocytosis



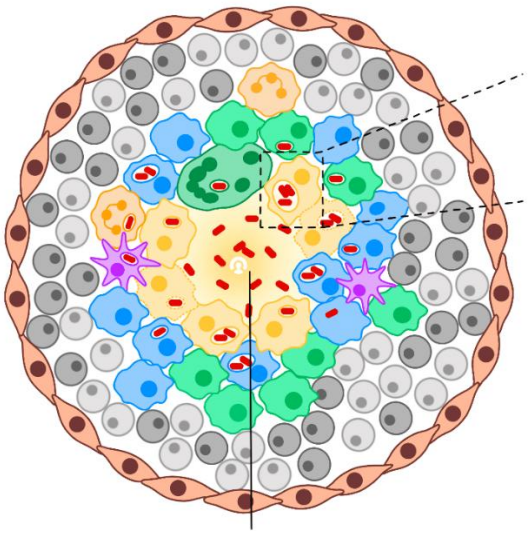
enzymes that break down pathogen

Macrophages

- These are larger immune cells - also perform phagocytosis.
  - Broader range of functions and longer lived than compared to neutrophils.
  - Can be found as a permanent part of some tissues. → clean up cellular debris
  - Can also release cytokines to induce other responses of the immune system.
- Involved with antigen presentation. → chemical signals (messenger)



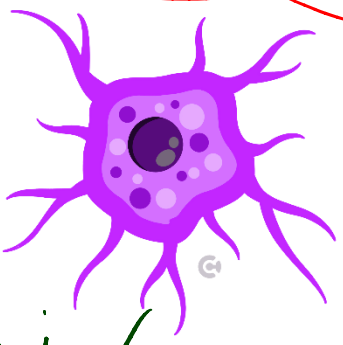
Exploration: Tuberculosis



Dendritic Cells

any molecule that activates immune response

- Another cell that engages in phagocytosis, primarily found in tissues, but can travel through the bloodstream.
  - More specialised for the process of antigen presentation. Why?

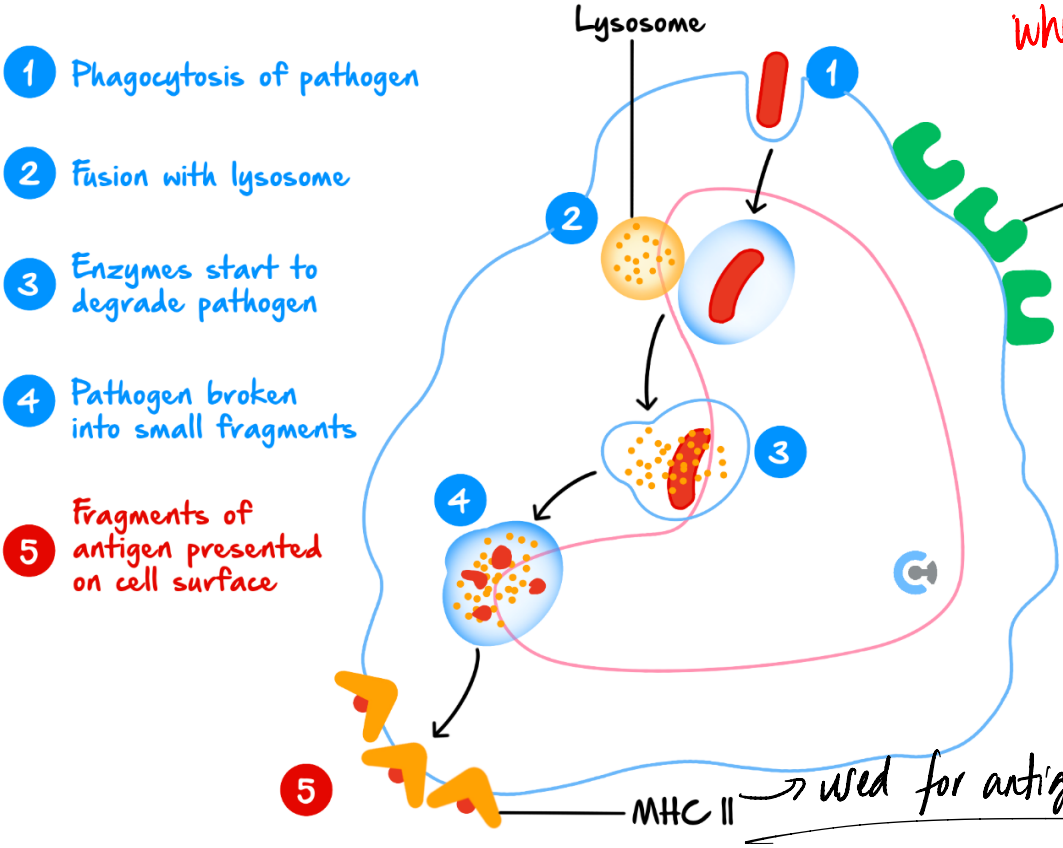


→ 3rd line adaptive cell

higher surface area to the dendrites

Exploration: Antigen Presentation

↳ showing off SPECIFIC antigens to 3rd line



whether the cell is healthy or not!

→ used for antigen presentation

→ is used to present / show antigens of things that have been phagocytosed

Sub-Section: Natural Killer Cells

What is the life cycle of a virus?

infect their genetic material → cell produces more copies

How do they kill these cells?

↳ lysis → releases viral copies

Natural Killer Cells

- These are cells that do not target foreign cells but actually focus on targeting your own unhealthy cells, or infected cells.
  - Why might cells be unhealthy?
    - Virally infected / bacteria (rare)
    - Cancer
  - How could the unhealthy cells be recognised?

non-self → viral antigens shown on MHC I

virally infected

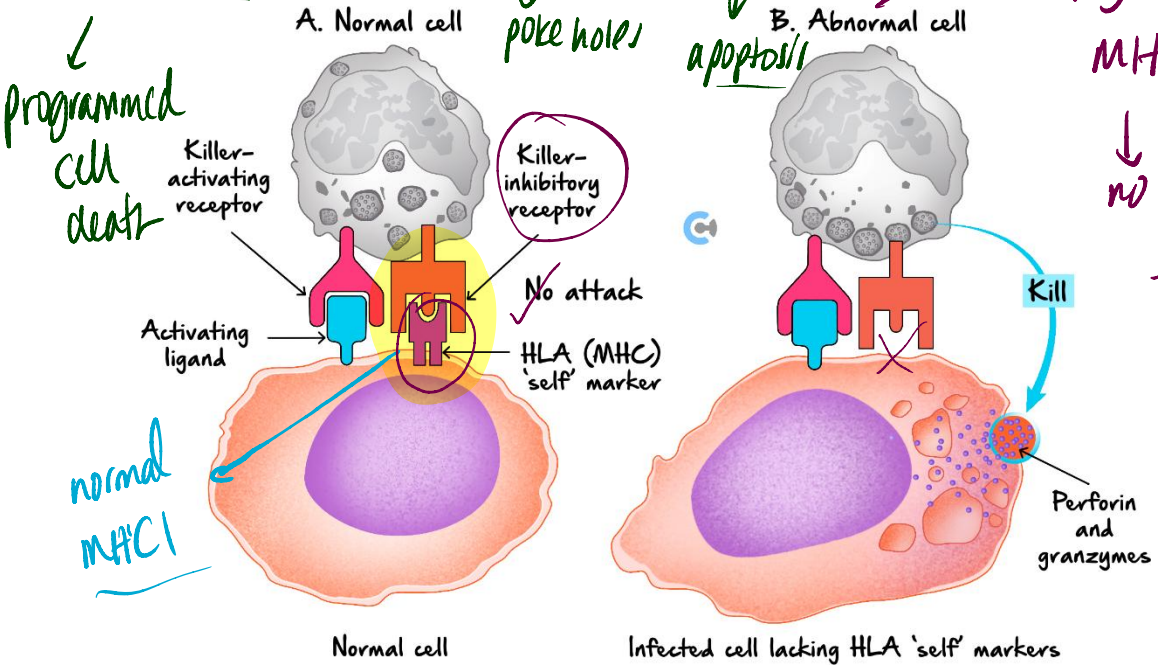
↳ MHC I markers → display antigens on what the cell makes

- This natural killer cell will then release perforins and granzymes which will trigger the cell's death by apoptosis

programmed cell death

poke holes

apoptosis



Analogy: The Secret Police

- **City** → Your body.
- **Citizens** → Your own cells.
- **Secret Police (NK cells)** → Patrol the body looking for abnormal or suspicious cells.
- **Identification Papers/Social Credit Score (MHC markers)** → Used to distinguish healthy cells from infected or cancerous ones.
- **Action Taken** → Immediate neutralisation (inducing apoptosis) of suspicious cells lacking proper identification.

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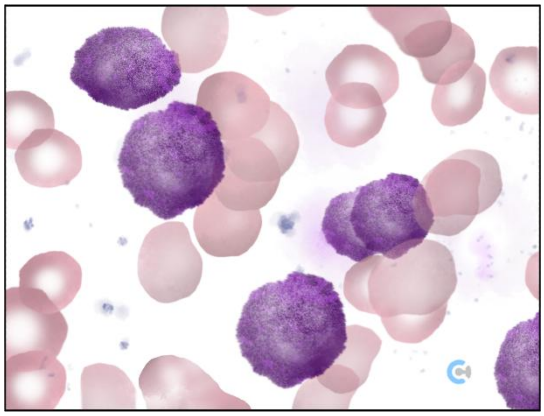
Sub-Section: Mast Cells

Mast Cells

- Immune cell that is primarily responsible for the release of histamine and other mediators that activate the immune system.
  - Not directly involved in the defence but rather stimulates other cells to do so – releases other kinds of cytokines as well.
- Remains in tissue.

↳ help coordinate immune response



What triggers the release of histamine?

Analogy: Messengers of the Castle

- Let's say you have someone attacking the walls – would they be allowed to just attack and break walls without that being reported?

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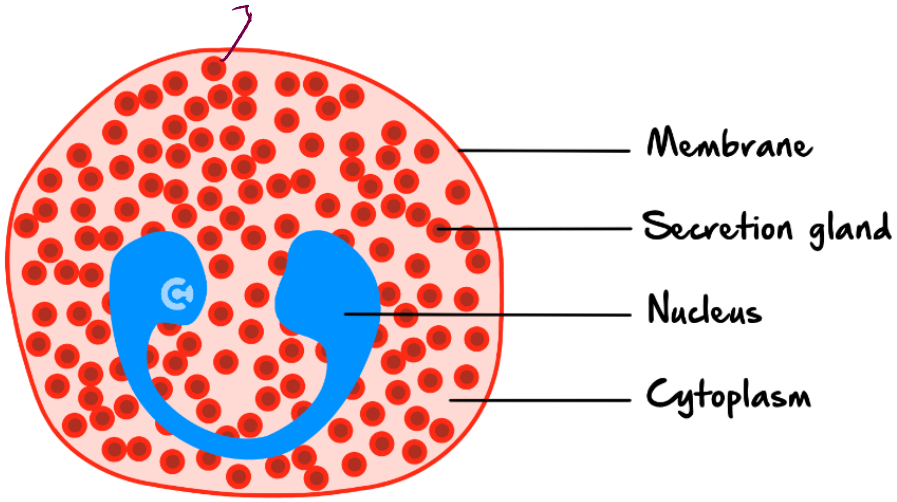
Sub-Section: Eosinophils

What pathogens do eosinophils target?

Eosinophils

- Immune cells which release toxic chemical mediators - impacting pathogens which cannot be ingested fully via phagocytosis.
  - Able to phagocytose, but to a very limited extent.
- Degranulate upon contact - what process would we call this ordinarily? exocytosis

Structure of an Eosinophil



Key Takeaways

- ☑ All these components have their own unique specific function - the first step should be to remember all the different things and then what they do.
- ☑ Answering questions is less systematic than what it was for something like transcription, but it still is repetitive and predictable enough that we can prepare.
- ☑ More questions will focus on the holistic impact – e.g., "What happens when a virus attacks us?"

Active Recall: Describe how NK cells function against pathogens.

Active Recall: Describe the role of mast cells against pathogens.

Active Recall: Distinguish between the 3 types of phagocytes.

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The Cells of the Innate Immune Response

Phagocytes

- Phagocytes engulf and digest pathogens through phagocytosis.
- Neutrophils are the most abundant white blood cells and act as rapid first responders.
- Neutrophils eliminate pathogens via engulfment, toxic granules, and extracellular traps (NETs).
- Macrophages are larger, longer-lived phagocytes that also release cytokines and perform antigen presentation.
- Dendritic cells act as key antigen-presenting cells, bridging innate and adaptive immunity by activating T-cells.

Natural Killer (NK) Cells

- NK cells identify and destroy infected or cancerous host cells.
- Recognition depends on detecting missing or abnormal MHC markers in host cells.
- NK cells kill by releasing perforin and granzymes, which induce apoptosis in target cells.

Mast Cells

- Mast cells reside in tissues and are activated by injury or infection.
- Release histamine and other signalling molecules to increase blood flow and vessel permeability.
- Initiate inflammation and recruit other immune cells to the site of infection.

Eosinophils

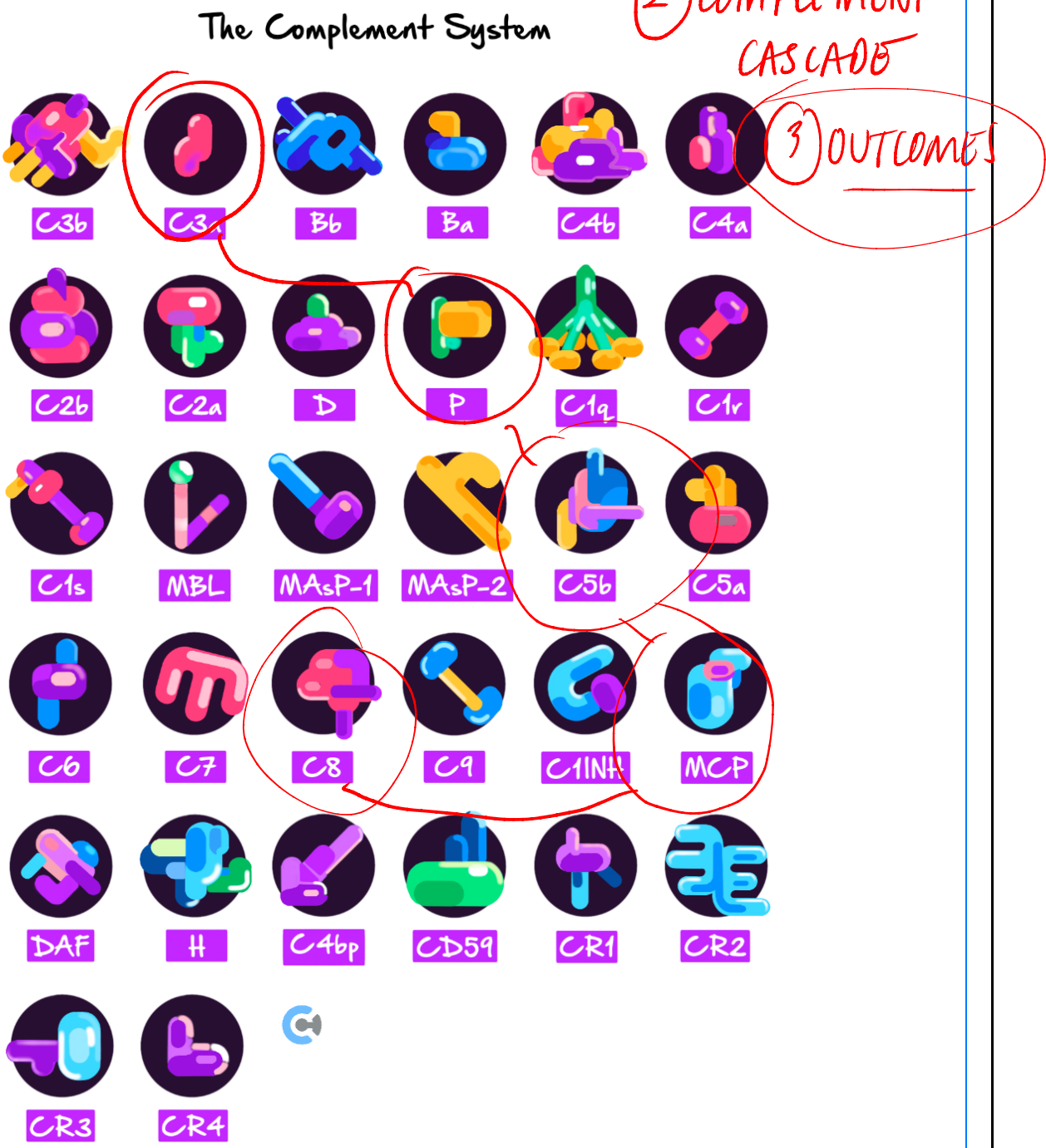
- Eosinophils target large parasites such as helminths and contribute to allergic responses.
- Release toxic granules that damage or kill pathogens too large for phagocytosis.
- Can perform limited phagocytosis and contribute to inflammation through degranulation.

Is the innate immune response ONLY made up of cells?

Section D: The Non-Cellular Components of the Innate Immune System

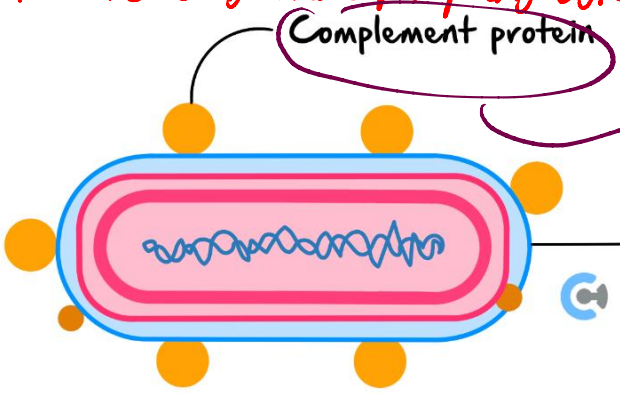
The Complement System

- The complement system is a set of proteins that float around the body in the blood and act as an innate defence against pathogens.
  - Primarily focused on bacteria.



- Not very well understood, but there are 3/4 main methods of attack against pathogens.

- Opsonisation – tagging pathogens for destruction by phagocytosis
  complement proteins will bind to surface of a pathogen and make it more recognisable for phagocytes to engulf + destroy

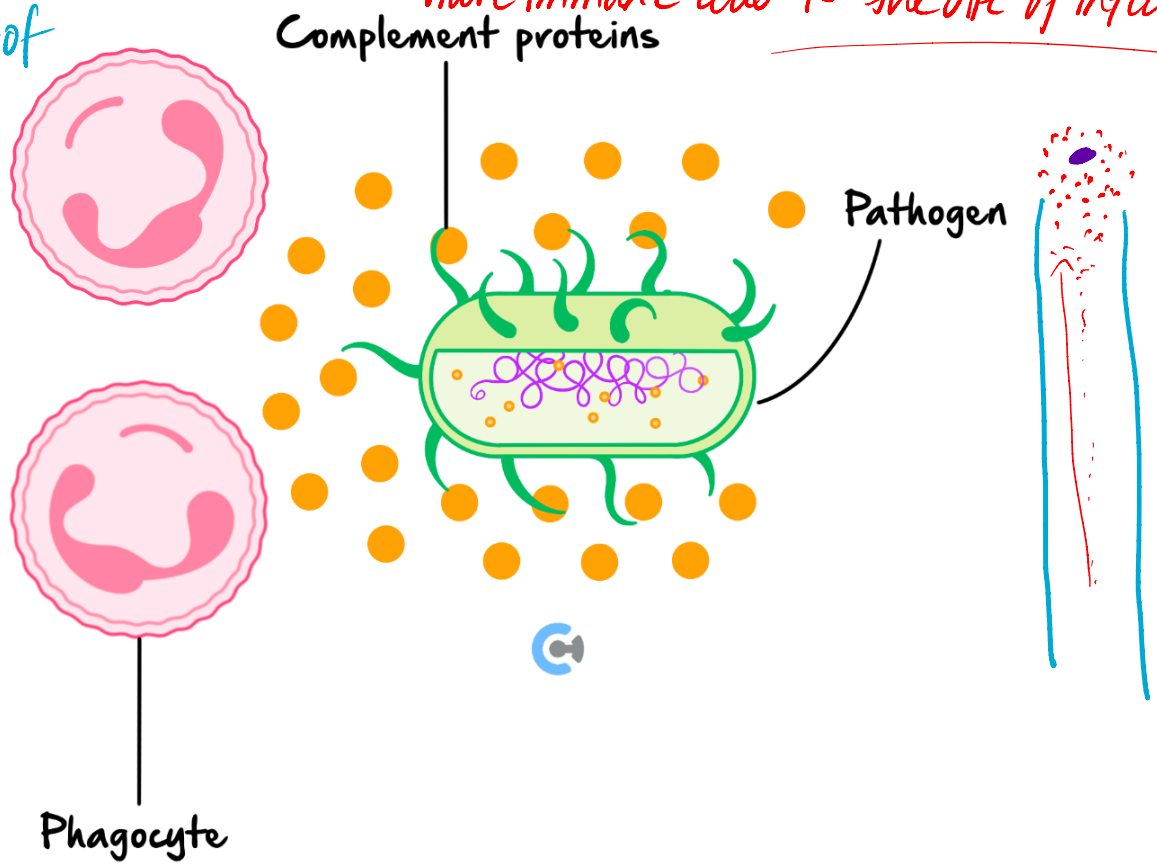


↳ multiple complement proteins → phagocyte can bind to the complement proteins

Opsonisation of a pathogen by complement proteins

- Chemotaxis – complement proteins gather around pathogens and attract more immune cells to the site of infection

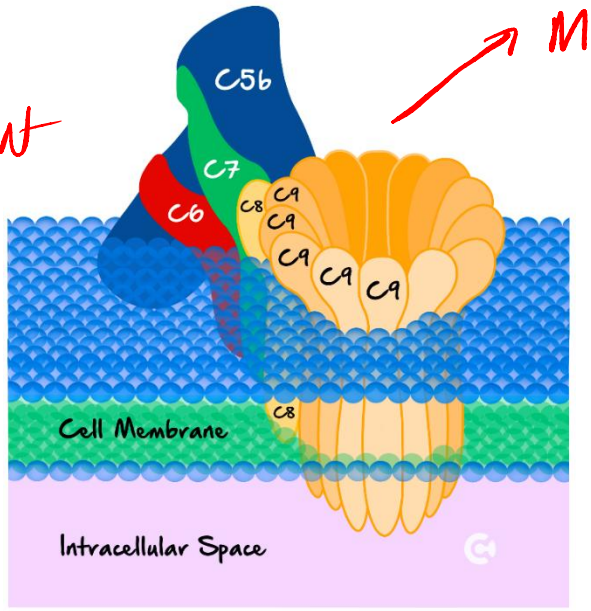
movement of immune cells



Chemotaxis of phagocytes towards a pathogen

- Lysis - complement proteins to form membrane attack complex on surface of pathogen → lysis through perforation of membrane

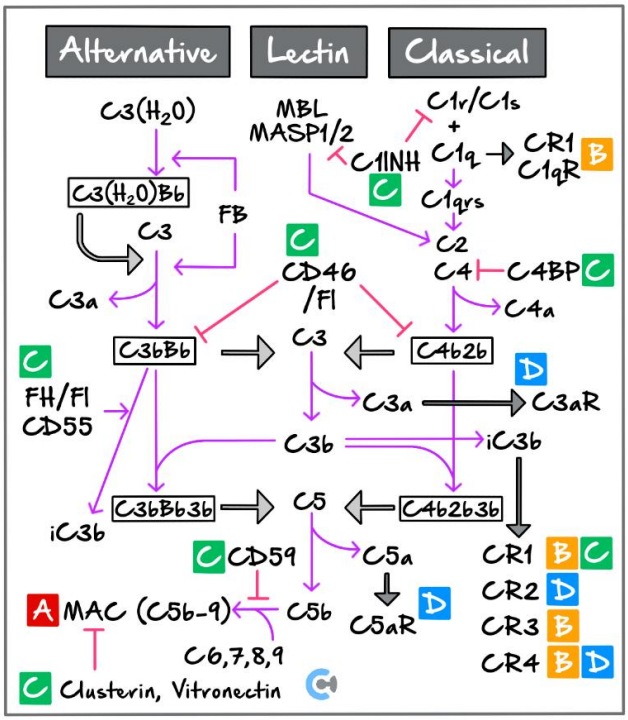
directly killing pathogen via complement system



MEMBRANE ATTACK COMPLEX

→ poke a hole in membrane

Exploration: If you thought it was that easy.....



Functions

A Cell lysis
Killing of pathogens

B Opsonization / Phagocytosis
- Clearance of apoptotic cells and debris (CR1/CR3/CR4/C1qR)
- Synaptic pruning (CR3)

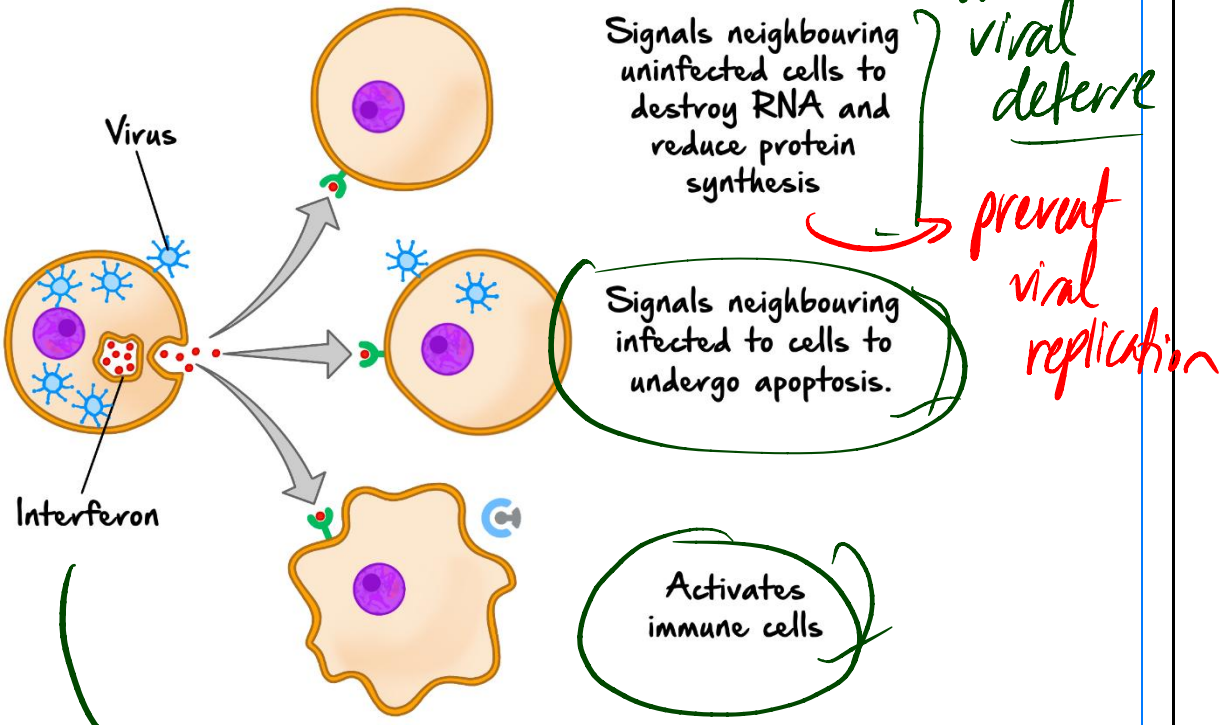
C Regulation
Inhibition of the complement cascade

D Other functions
- Promotion of cell differentiation and recruitment (C3aR/C5aR)
- Modulation of immune cell migration (C3aR)
- Regulation of B-cell functions (CR2)
- Leukocyte adhesion (CR4)

- The immune system gets much more complicated!

Interferons

- These are signalling molecules that are released by virally infected cells – warn surrounding cells of impending infection.
  - What could a cell do to reduce its risk of viral infection? ↳ & heighten anti-viral defense



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The Non-Cellular Components of the Innate Immune System

Complement Proteins

- Complement proteins are inactive in the bloodstream until triggered by pathogens.
- Activation occurs via multiple pathways but always results in a targeted innate response.
- Opsonisation tags pathogen surfaces to enhance recognition and engulfment by phagocytes.
- Chemotaxis involves the recruitment of additional immune cells to the site of infection.
- Lysis is achieved by forming a membrane attack complex (MAC) that creates pores in the pathogen membrane, leading to cell death.
- Most effective against bacterial pathogens.

Interferons

- Interferons are released by cells infected with viruses.
- These signalling molecules act locally to prepare neighbouring cells for potential viral invasion.
- Stimulate the production of antiviral proteins in surrounding cells.
- Slow down viral replication and signal for immune responses like NK cell activation.
- Do not directly destroy viruses but enhance resistance across nearby tissues.

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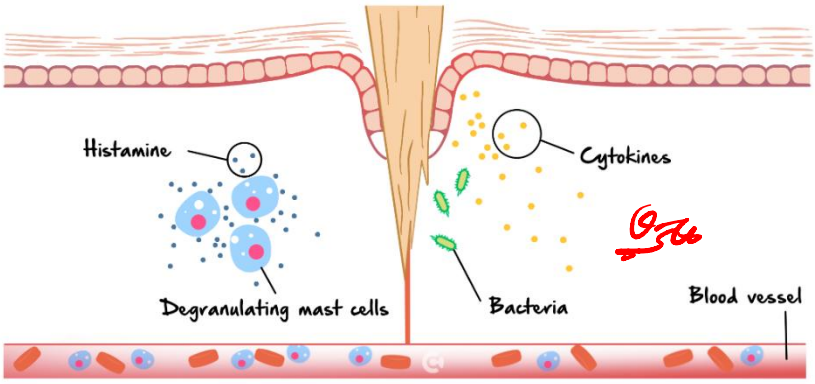
Section E: The Inflammatory Response

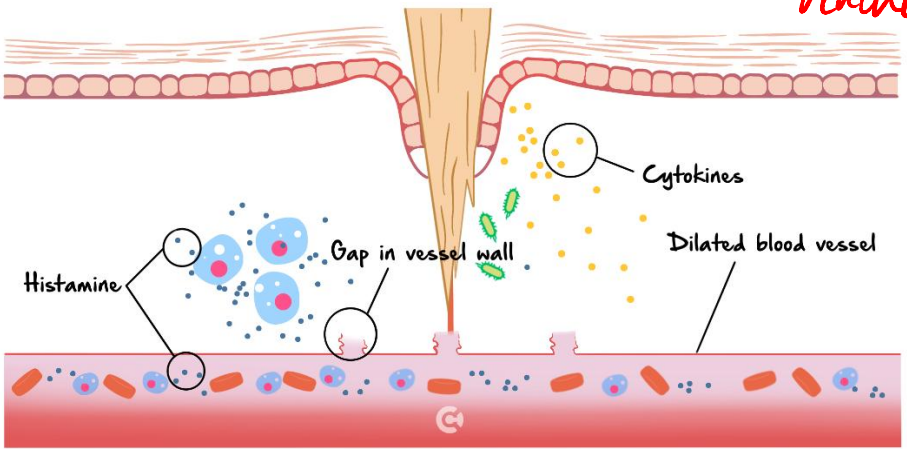
Overview of the Inflammatory Response

- The inflammatory response is an innate response to pathogenic infection and injury which serves to rapidly shut down further infections and damage by ~~increasing blood flow~~ to the affected tissue.
  - There are three main aspects to it – initiation, vasodilation, migration
- A classic example is a splinter.
- *Initiation* – When the splinter pierces the protective layer of the skin, it damages skin cells and introduces pathogens that may have been present in the splinter to the body. Damaged cells release cytokines, mast cells degranulate releasing histamine.



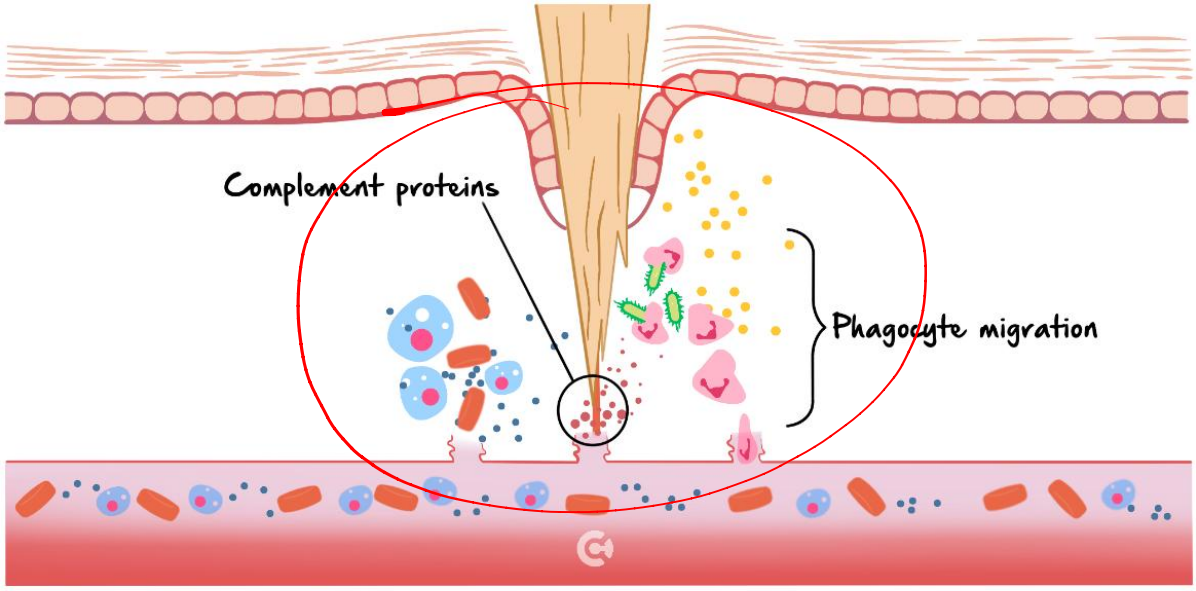
Initiation of inflammation involves cytokines being secreted from damaged cells and activation of mast cells, causing the release of histamine.

- *Vasodilation* – Histamine travels from the mast cells to the nearby blood vessels, binding to specific receptors which trigger vasodilation – widening blood vessels to increase blood flow. The permeability of the vessel wall also increases – blood vessels become more leaky and more fluid (blood) reaches the tissue ↓ site of infection



Histamine signals the blood vessel to dilate and become leaky.

- *Migration* - Signals and permeability increase allow innate immune cells to travel to the site of infection. What cells and components would be present and what would they do? Pus?



Various components of the innate immune system are able to access the site and destroy pathogens and remove debris.

Discussion: How does this process relate to the symptoms of inflammation?

- Ali, the soldier has sprained his ankle running to the battlefield! What symptoms might he get?
- SWELLING → increased blood flow → blood vessels are more leaky → INCREASED PERMEABILITY → fluid build up in tissue
- WARMTH → increased blood flow / more immune activity
- REDNESS → increased blood flow
- PAIN
- LOSS OF FUNCTION

Space for Personal Notes

Fever

- Increasing central body temperature – to disrupt the function of pathogens. How?
  - Can fevers be bad for us too?

NOTE: Technically speaking fevers are not part of the inflammatory response, I have grouped them together as they often can go hand in hand.

Space for Personal Notes

Key Takeaways

- **The Inflammatory Response:**
  - Inflammation is a rapid innate immune reaction to tissue damage or microbial invasion.
  - Aims to isolate pathogens, limit spread, and begin tissue repair.
- **Initiation:**
  - Triggered by physical injury or pathogen entry.
  - Damaged cells release cytokines to signal immune activity.
  - Mast cells in local tissues degranulate and release histamine.
- **Vasodilation:**
  - Histamine causes nearby blood vessels to widen (vasodilation).
  - Increased blood flow delivers more immune cells and nutrients to the site of damage.
  - Vessel walls become more permeable, allowing plasma and immune cells to exit into tissues.
- **Migration:**
  - Increased permeability allows immune cells like neutrophils and macrophages to move from the bloodstream into the affected area.
  - These cells begin engulfing pathogens and releasing further signalling molecules.
  - Accumulation of dead cells and debris forms pus at the site.
- **Fever:**
  - Cytokines released during inflammation may travel to the brain, resetting the hypothalamus to a higher temperature.
  - Fever slows down pathogen replication and boosts immune efficiency.
  - Excessively high fevers can be harmful and need to be regulated.

- **Symptoms:**
  - Redness and heat occur due to increased blood flow.
  - Swelling results from fluid leaking into tissues.
  - Pain is caused by pressure on nerve endings and inflammatory signals.
  - Loss of function can occur due to swelling, pain, or tissue damage.
- **Overall Role:**
  - Localises infection, recruits immune cells and initiates the repair process.
  - If unregulated, inflammation can damage healthy tissue or lead to chronic inflammatory conditions.

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Section F: Examples

COVID Infection

- What type of pathogen is COVID-19?
  - How does this infect cells?
- Where in the body does COVID-19 target?
- What are the first line of defence barriers?
- What are some of the second line of defence barriers?

- How does the immune system recognise the virus?

- What overall response might occur? Relate this to symptoms.

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Contour Check

Learning Objective: [3.2.1] - Explain how the innate response is triggered in response to infection & its defining characteristics

Key Takeaways

Introduction to the Innate Immune System

- Innate immunity acts immediately after pathogen entry.
- Response _____ and does _____ based on pathogen type.
- Innate immunity lacks long-term _____ of pathogens.
- Main functions include preventing pathogen entry, eliminating invaders, activating _____ , and recruiting _____.
- Non-specificity alone may lead to future infections if pathogens adapt.
- Fast response prevents pathogens from _____ and multiplying.

Recognition of Pathogens

- Innate system identifies threats using general molecular patterns.
- _____ _____ are found on microbial surfaces.
- _____ _____ originate from stressed or injured host cells.
- Pattern recognition receptors (PRRs) on immune cells detect these patterns.
- Recognition relies on conserved structural features rather than _____.

Learning Objective: [3.2.2] - Identify & describe the function of the cellular components of the innate immune response - macrophages, neutrophils, dendritic cells, mast cells, eosinophils

Key Takeaways

- The Cells of the Innate Immune Response
  - Phagocytes
    - Phagocytes engulf and digest pathogens through phagocytosis.
    - Neutrophils are the _____ white blood cells and act as _____.
    - Neutrophils eliminate pathogens via _____ , toxic granules, and extracellular traps (NETs).
    - Macrophages are _____ , _____ phagocytes that also release _____ and perform antigen presentation.
    - Dendritic cells act as key _____ , bridging innate and adaptive immunity by activating T cells.
  - Natural Killer (NK) Cells
    - NK cells identify and destroy infected or cancerous host cells.
    - Recognition depends on detecting _____ on host cells.
    - NK cells kill by releasing _____ and _____ , which induce _____ in target cells.
  - Mast Cells
    - Mast cells reside in tissues and are activated by injury or infection.
    - Release _____ and other signalling molecules to _____ and _____.
    - Initiate inflammation and recruit other immune cells to the site of infection.

- Eosinophils
  - Eosinophils target large parasites such as helminths and contribute to allergic responses.
  - Release toxic granules that damage or kill pathogens too large for phagocytosis.
  - Can perform limited phagocytosis and contribute to inflammation through degranulation.

Learning Objective: [3.2.3] - Identify & describe the role of non-cellular components of the innate immune response - complement proteins & interferons

Key Takeaways

- The Non-Cellular Components of the Innate Immune System
  - Complement Proteins
    - Complement proteins are inactive in the bloodstream until triggered by pathogens.
    - Activation occurs via multiple pathways but always results in a targeted innate response.
    - _____ tags pathogen surfaces to enhance recognition and engulfment by phagocytes.
    - _____ involves the recruitment of additional immune cells to the site of infection.
    - _____ is achieved by forming a _____ (MAC) that creates pores in the pathogen membrane, leading to cell death.
    - Most effective against bacterial pathogens.

- **Interferons**
  - Interferons are released by cells infected with _____.
  - These signalling molecules act locally to prepare neighbouring cells for potential viral invasion.
  - Stimulate the production of antiviral proteins in surrounding cells.
  - Slow down viral replication and signal for immune responses like NK cell activation.
  - Do not directly destroy viruses but enhance resistance across nearby tissues.

Learning Objective: [3.2.4] - Describe key innate immune processes - phagocytosis, inflammatory response

Key Takeaways

- **The Inflammatory Response**
  - **Type of Response:** Inflammation is a [rapid] / [delayed] innate immune reaction to tissue damage or microbial invasion.
  - **Primary Aim:** Aims to [isolate pathogens] / [spread infection], limit spread, and begin tissue repair.
- **Initiation**
  - **Triggering Event:** Triggered by [physical injury] / [adaptive response] or pathogen entry.
  - **Cytokine Source:** Damaged cells release [cytokines] / [antibodies] to signal immune activity.
  - **Histamine Source:** Mast cells in local tissues degranulate and release [histamine] / [interferons].

- **Vasodilation**
  - **Effect of Histamine:** Histamine causes nearby blood vessels to [widen] / [constrict].
  - **Blood Flow Role:** Increased blood flow delivers more [immune cells and nutrients] / [waste products] to the site of damage.
  - **Vessel Permeability:** Vessel walls become more [permeable] / [rigid], allowing plasma and immune cells to exit into tissues.
- **Migration**
  - **Movement Pathway:** Increased permeability allows immune cells like neutrophils and macrophages to move from the [bloodstream] / [lymph] into the affected area.
  - **Function at Site:** These cells begin engulfing pathogens and releasing further [signalling molecules] / [antibodies].
  - **Byproduct Formation:** Accumulation of dead cells and debris forms [pus] / [lymph] at the site.
- **Fever**
  - **Fever Mechanism:** Cytokines released during inflammation may travel to the brain, resetting the [hypothalamus] / [amygdala] to a higher temperature.
  - **Effect on Pathogens:** Fever slows down [pathogen replication] / [immune cell production] and boosts immune efficiency.
  - **Risk of Fever:** Excessively high fevers can be [harmful] / [beneficial] and need to be regulated.
- **Overall Role**
  - **System Goal:** Localises infection, recruits immune cells, and initiates the [repair process] / [adaptive memory response].
  - **Risk of Dysregulation:** If unregulated, inflammation can damage [healthy tissue] / [bacterial colonies] or lead to chronic inflammatory conditions.

Learning Objective: [3.2.6] - Explain the symptoms associated with the inflammatory response

Key Takeaways

- **Symptoms of Inflammation**
  - **Cause of Redness and Heat:** Redness and heat occur due to increased [blood flow] / [white blood cell count].
  - **Cause of Swelling:** Swelling results from fluid leaking into [tissues] / [vessels].
  - **Cause of Pain:** Pain is caused by pressure on [nerve endings] / [antibodies] and inflammatory signals.
  - **Cause of Loss of Function:** Loss of function can occur due to swelling, pain, or [tissue damage] / [fever].

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