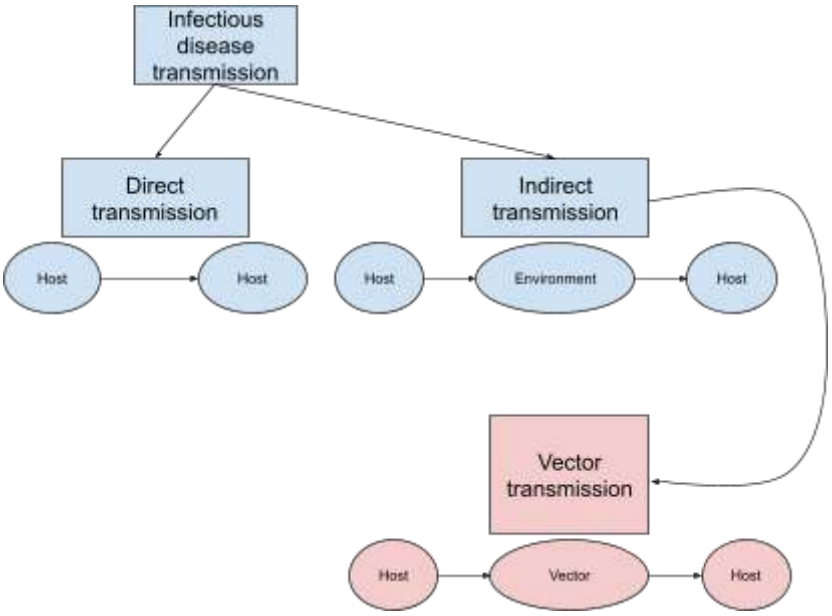
Module 7: Infectious Disease

7.1: Causes of Infectious Disease

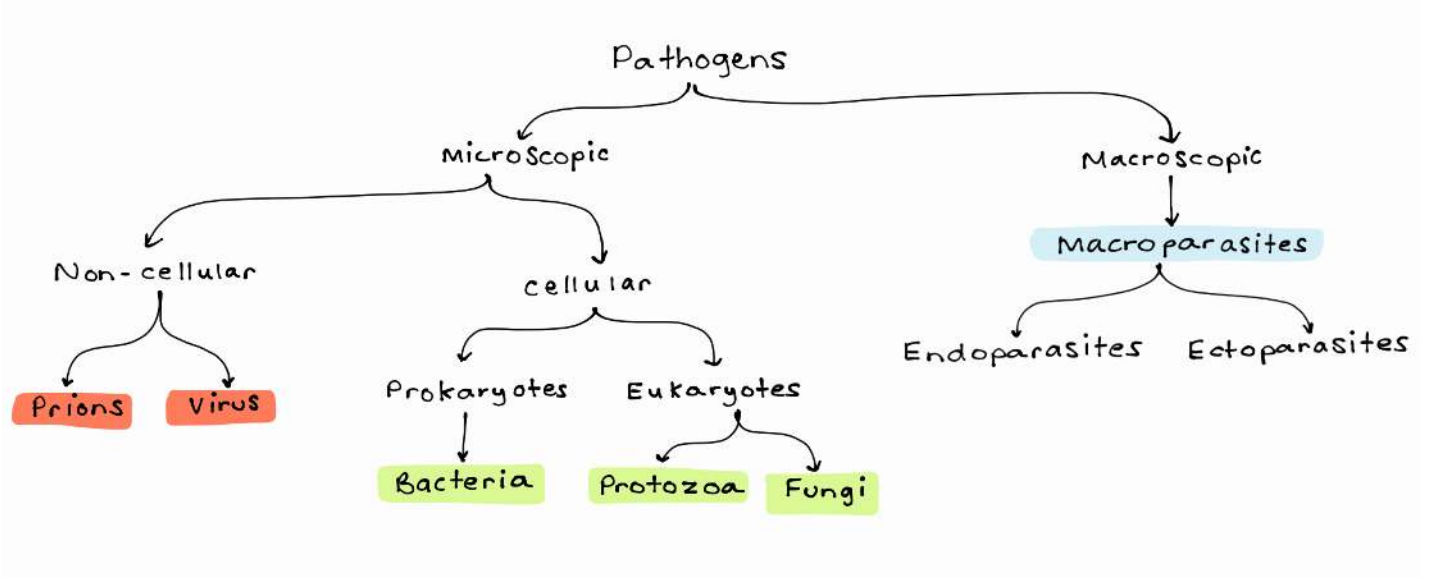
How are diseases transmitted?

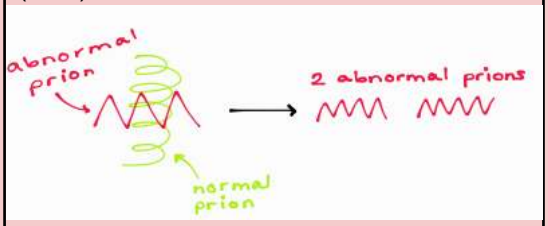
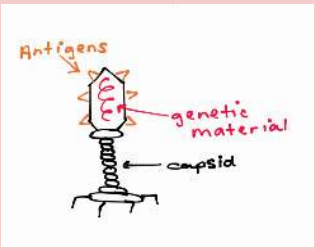
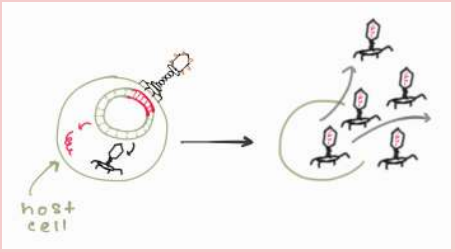
Infectious diseases are caused by pathogens, and are transmitted when a pathogen is moved from one host to another.

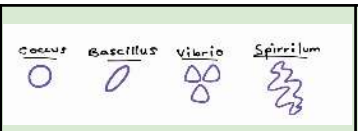
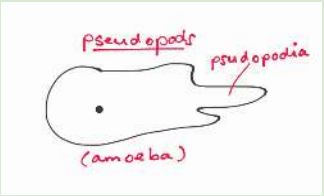
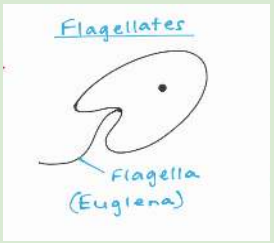
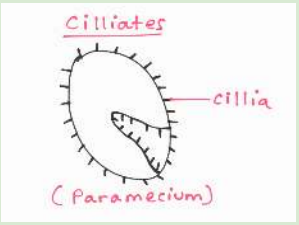


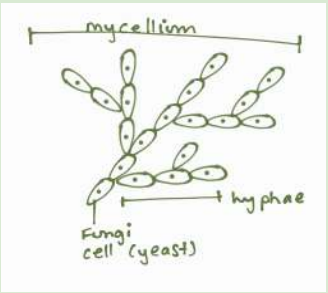
Both infectious and non-infectious diseases may be caused by mutagens, but only infectious diseases are caused by pathogens, which is why they can be transmitted between hosts.

PATHOGENS

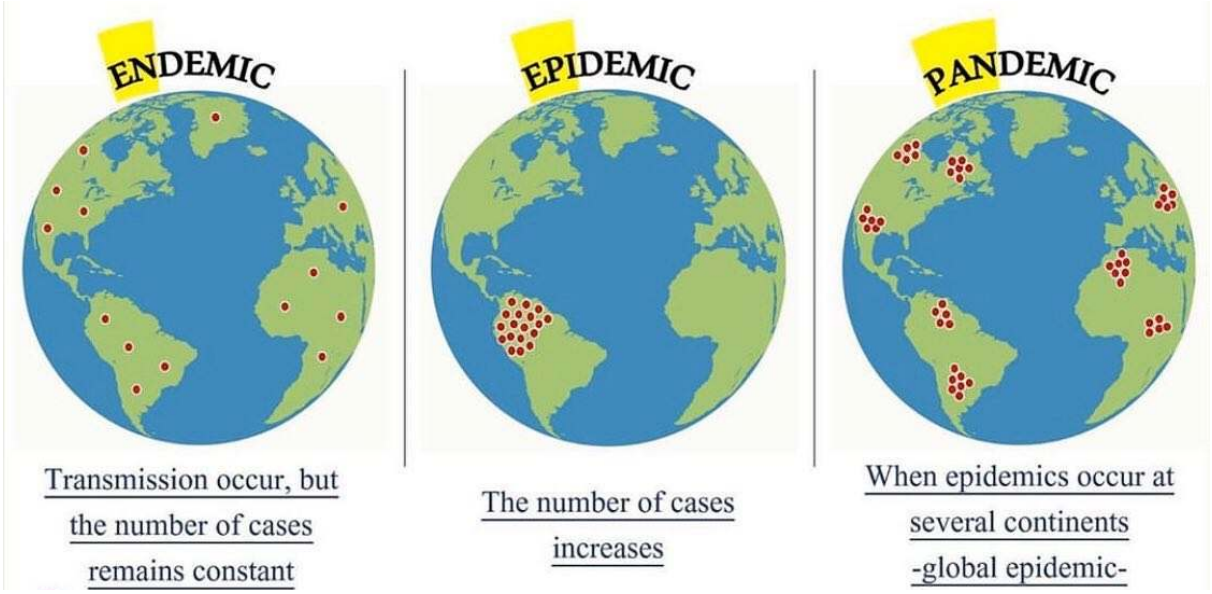


Pathogen	Features	Disease-causing mechanism	Transmission	Disease
Prion	Misfolded prion protein - nm - non-cellular - no genetic material	Abnormal prion induced misfolding in normal prions ↓ Damages the nervous tissue. ↓ Transmissible spongiform encephalopathy (TSE) 	Eating meat containing abnormal prions Contaminated surgical tools Contaminated transplanted organs Inheritance of a mutated gene coding for abnormal prion	Bovine spongiform encephalopathy (BSC) Creutzfeldt-Jakob's disease Kiru
Virus	Genetic material covered by a capsid protein - 30-300 nm - contains DNA or RNA (retrovirus) - antigens on surface of protein 	Each virus targets a specific cell ↓ Viral genetic material inserted into host cell ↓ Integrates into host's genome ↓ Redirects protein synthesis to form more capsids ↓ Genetic material replicates in DNA replication ↓ More proteins form in cell ↓ Cell bursts (lysis) ↓ Virus spreads to more cells 	Vector transmission - Insects Contact with infected surfaces/ materials Contaminated food/ water	AIDs ↳ HIV Hepatitis ↳ Hep-B Influenza ↳ Influenza virus COVID-19 ↳ Coronavirus
Bacteria	Unicellular prokaryotes covered in a capsule - µm - cell wall & membrane - Forms glossy colonies - Different shapes	Rapidly multiplied by binary fission ↓ Releases mycotoxins ↓ Toxins cause disease		Tuberculosis ↳ Mycobacterium Tuberculosis Tetanus Meningococcal

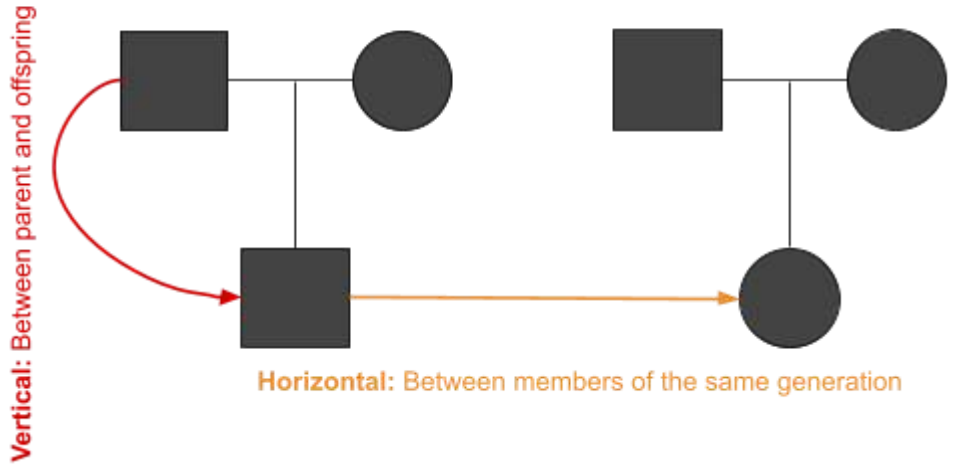
				
Protozoa	Unicellular eukaryotes - µm - cell membrane - organelles - classified based on movement   	Attaches to intestine ↓ Prevents nutrient absorption		Malaria ↳ Plasmodium
Fungi	Unicellular eukaryotes - µm - cell membrane, wall, organelles - live in moist environments - reproduce by spores - form fury colonies	Survive on surface of skin (dermatophytes) ↓ Release mycotoxins ↓ Cause disease		Athletes foot ↳ Tinia Thrush ↳ Candida albicants Plant rust

				
Macroparasite	Macroscopic, multicellular eukaryotes Endoparasites: Live in host Ectoparasites Live on host <u>Types</u> - Helminths (worms) - Arthropods (segmented body)	Can be: - Pathogens (cause disease) - Vectors (transmit disease)		Elephantiasis ↳ Helminths Tapeworm ↳ Fleas Anaemia ↳ Lice

PATHOGEN TRANSFER IN AN EPIDEMIC



Transmission can be direct or indirect. It can also be:



Example: Equine flu

Cause	Equine influenza virus
Host	Horses and donkeys
Symptoms	- Coughing - Nasal discharge - High fever - Cough - Appetite loss - Muscular pain - Breathing difficulties
Transmission	Direct: - Touching noses transfers virus in the nasal fluid Indirect: - Contaminated equipment
Management	● Quarantine of infected horses, suspected asymptomatic horses and horses entering the country ● Distancing horses ● Lockdown of all horse movement ● Sterilisation of equipment ● Awareness programmes for host breeders

7.1.2: Contributions of Koch and Pasteur

CONTRIBUTIONS TO DISEASE

Miasma theory exists

↓

Disproved by John Snow
- Disease must be caused by microbes

↓

Spontaneous generation theory suggested

↓

Pasteur disproves spontaneous generation
- Develops pasteurisation to prevent wine spoilage
- Develops vaccine for chicken cholera

↓

Pasteur suggests germ theory of disease

↓

Koch proves germ theory of disease

↓

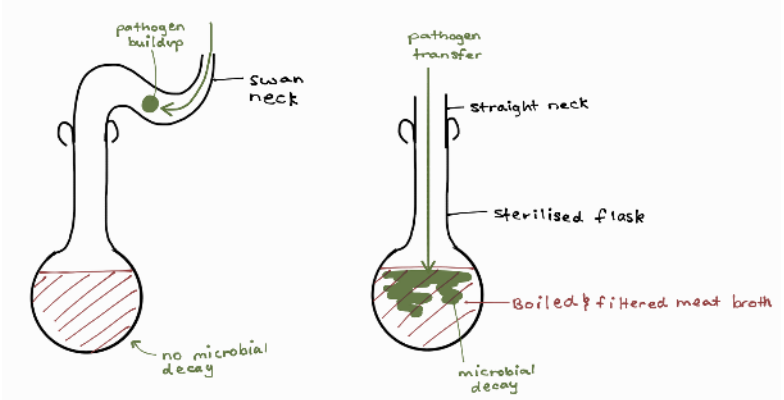
Koch expands germ theory of disease
- Identified mycobacterium tuberculosis as the cause of TB
- Developed the agar plate technique for culturing
- Developed vaccine for human cholera

Miasma theory
Disease is spread by miasma gas.

John Snow's epidemiology
Traced cases of cholera and found that it occurred in areas with no miasma.

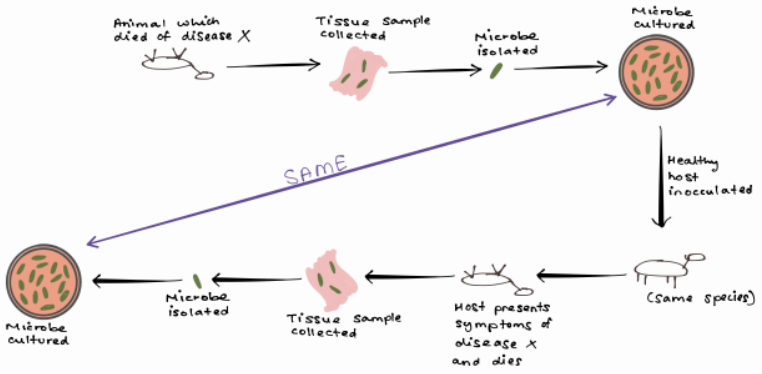
Spontaneous generation
Microbes spontaneously generate.

Pasteur's swan neck flask experiment:
Proved that microbes could not spontaneously generate, but had to be transferred.



Initial germ theory
Disease occurs due to pre-exisiting microbes that are transferred into the host.

Koch's postulates
Proved that microbes cause disease



Current germ theory
A specific microbe causes a specific disease when it is *transferred* into a host.

7.1.3: Diseases in Agriculture

For a disease to be prevalent in agriculture, it must have:

1. A pathogen present
2. An immunocompromised host
3. A favourable environment

Factors contributing to disease spread

- Overuse of antibiotics & pesticides
- Loss of genetic diversity
- Overcrowding of animals and plants
- Mobility of crops and animals between regions
- Displacement of insect vectors (deforestation & natural disasters)

ANIMAL DISEASES

Case studies

Footrot in sheep and cattle
Cause: Bacteria (*Dichelobacter nodosus*)

Effect:

- Inflammation and pus in the hooves
- Lameness
- Weight loss

Transmission:

- Reservoirs in moist soil
- Overcrowding assists in spread
- Pre-existing cracks in hooves allow entrance into animal

Newcastle disease in poultry
Cause: Virus (*ND virus*)

Effect:

- Diarrhea
- Coughing/ sneezing
- Tissue swelling
- Discharge

Transmission:

- Contact with host
- Waste

PLANT DISEASES

Pathogen	Transmission	Symptoms
Fungi (*most common*)	Spores contaminate soil and gardening equipment. Transmitted by wind, water, farming practises and root contact. Enter the plant through stomatal pores and damage xylem and phloem.	- Mildew on leaves - Leaf rust - Blight (marks on plant) - Wood rot (damage to vascular tissue)
Viruses	Viral reservoirs on contaminated equipment. Transmitted by frequent handling of the plant.	- Yellowing of leaves - Mosaic patterns - Streaking patterns
Bacteria	Bacterial reservoirs in soil, weeds, seeds. Transferred through contaminated equipment and handling. Assisted by environmental conditions: - Warm, humid weather - Optimal pH - Overcrowding of plants	- Blight
Macroparasites	<u>Endoparasites</u> - Nematodes (worms) Reservoirs in soil.	- Gall Lumps on leaves which cause them to fall off
	<u>Ectoparasites</u> - Locusts - Beetles - Ants - Flies Suck sap of the plants, damaging the phloem and restricting nutrient uptake.	- Tunnels in the leaves As the pathogen burrows through the plant - Twisting and curling of leaves

Case studies

Panama disease in banana trees
Cause: Fungi (*fusarium oxysporum*)

Effect:
- Yellowing and wilting of leaves
- Stem splitting
- Damage to vascular tissue → nutrient deprivation

Transmission:
- Root-to-root contact between plants
- Contaminated equipment

Fire blight in pears and apples
Cause: Bacteria (*erwinia amylovora*)

Effect:
- Plant browning
- Mushiness of fruit

Transmission:
- Contaminated equipment

7.1.4: Pathogen adaptations

PRIONS

Transmission *Reaches host*	Small, and can enter the body through contaminated food or surgical instruments.
Adhesion *Attaches to host*	/
Entry *Enters host*	B-lymphocytes → lymph nodes → dendritic cells → nerve tissue → brain OR Gut → villi → lacteal tube → lymphatic system → dendritic cells → brain
Neutralisation *Weakens immune system*	Small proteins in brain issue, therefore the immune system can't reach it once it has entered
Disease-causing mechanism *Causes disease*	Induces abnormal folding in normal prions, leading to spongiform disease.

VIRUSES

Transmission	Airborne viruses: - Have long suspension time in air - Cause sneezing and coughing to be transferred
Adhesion	Viral antigens lock into cell surface receptors of host cells
Entry	Receptor-mediated endocytosis
Neutralisation	High mutation rate - Can rapidly adapt to host's internal environment - Antibodies are not permanently effective due to changing antigens
Disease-causing mechanism	Virus replicates in cell, causing lysis. Virus is then spread to other cells.

BACTERIA

Transmission	Waterborne bacteria: - Flagella to move in water
Adhesion	Pili and fimbriae allow bacteria to attach to host cell
Entry	Proteins in bacteria stimulate the host to engulf it. The cell wall & capsule protect the bacteria from the digestive enzymes until it releases haemolysin. Haemolysin destroys the vesicle, allowing the bacteria to enter the cell.
Neutralisation	Releases toxins to damage the immune cells.
Disease-causing mechanism	Releases toxins to destroy host cell.

PROTOZOANS

Transmission	Waterborne bacteria: - Flagella to move in water Insect vector: - Protozoa doesn't kill the insect - Entry is synchronised to the insect's life cycle
Adhesion	/
Entry	Microtubule protrusion is used to enter the host cell and forms a vacuolar membrane around it. Once inside, it deactivates the lysosome enzymes to prevent digestion.
Neutralisation	The saliva of the mosquito is an anticoagulant. It stops the blood from clotting. Kills liver cells, which causes accumulated calcium ions to block antigens. The immune system can no longer recept these.
Disease-causing mechanism	Releases toxins which damage cells. Prevents nutrient absorption in digestive system, weakening the host.

FUNGI

Transmission	Through soil - Release spores, which aren't damaged when soil dries up
Adhesion	Cell wall and capsule help fungi to adhere.
Entry	Most fungal infections in animals are dermal. Saprophytic nature of fungi helps it to access nutrients from skin surface.
Neutralisation	Capsule and cell wall suppress cytokines (complement proteins).
Disease-causing mechanism	Hydrolytic enzymes damage the host cell.

MACROPARASITES: endoparasites (inside)

Transmission	Through fecal matter: - Resistant to pH change - Resistant to low-oxygen environments
Adhesion	Hooks in mouth help attach to host cells.
Entry	Move from soil into body through the hair follicles. Transported into digestive system through blood stream. Eggs laid in the intestine and excreted back into soil, continuing the cycle of infection.
Neutralisation	Produce proteins to reduce immune response.
Disease-causing mechanism	Reduce nutrients available for host.

MACROPARASITES: ectoparasites (outside)

Adhesion	Mouthparts which are inserted into the skin to attach. Most are temporary.
Entry	/
Neutralisation	/
Disease-causing mechanism	Anticoagulants in saliva prevent clotting until the parasite has taken the blood meal.

7.2: Response to Pathogens

How does a plant or animals respond to infection?

	Blocks pathogen entry	→	*Kills entered pathogen*	→	*Prevents secondary infection*
Plant	Passive response		Active response		Delayed response
Animal	1st line		2nd & 3rd line		3rd line

[7.2.1] Plant responses

1st LINE: PASSIVE RESPONSE

To prevent the pathogen from entering.

Physical Barriers

- Bark
- Thick, waxy cuticles
- Closable stomata
- Cell walls reinforced by lignin and cellulose
- Vertically hanging leaves

Chemical barriers

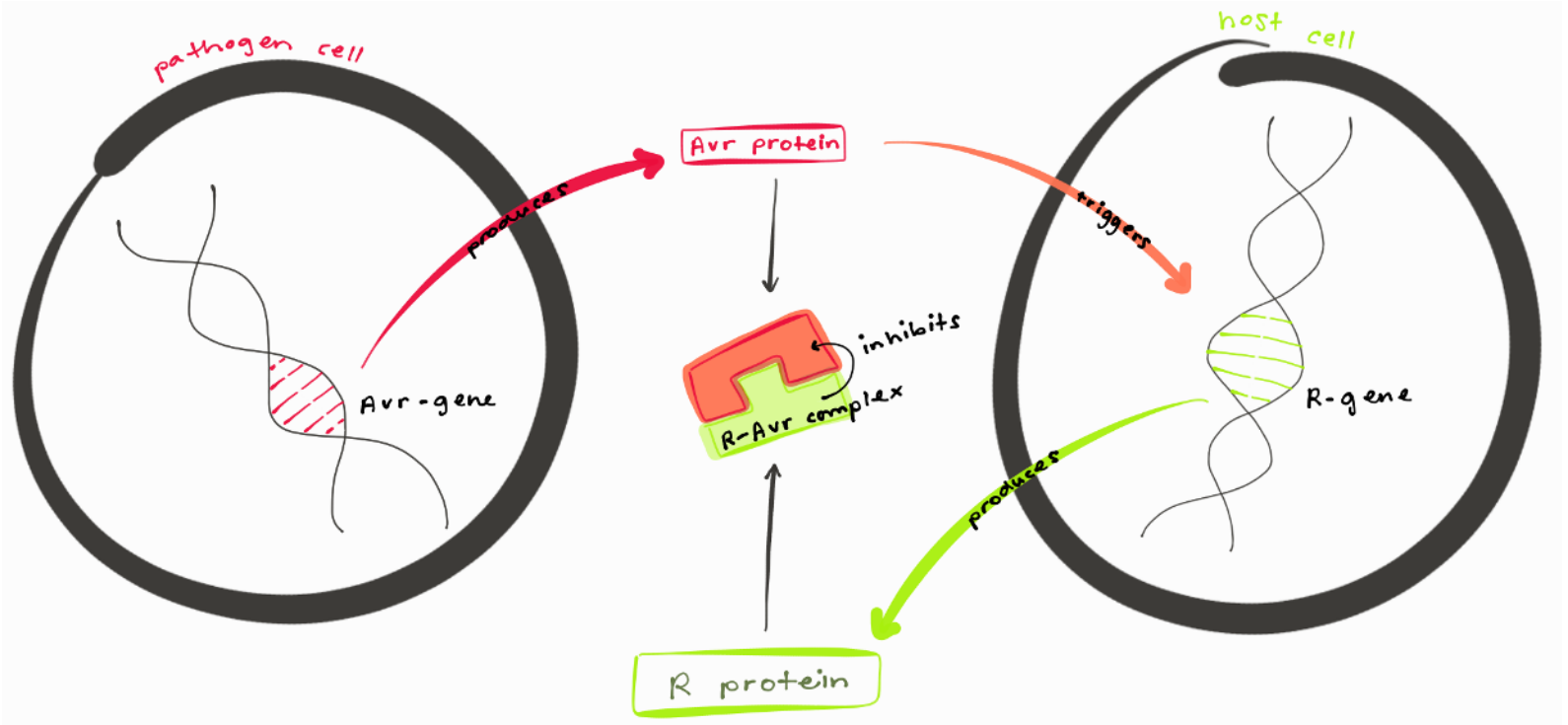
Pathogen suppressors

Plants secrete chemicals like glucosides and saponins to suppress fungal/ bacterial growth.

2nd LINE: ACTIVE RESPONSE

To kill the pathogen.

Gene-for-gene resistance



Basal resistance

The plant recognises the pathogen's PAMPs. Basal resistance is activated, causing the tissue to fortify so that the pathogen can't move deeper into the plant.

Hypersensitive localised response

Plants in the infected area release chemicals to make the cell walls impermeable. The infected cells then commit apoptosis, killing the pathogens in them.

Oxidative burst

The plant releases hydrogen peroxide (H_2O_2) which kills pathogens.

3rd LINE: DELAYED RESPONSE

To prevent the pathogen spreading or re-infecting

Systematic acquired resistance

Follows the HR response.

Salicylic acid is released and spreads to other parts of the plant. It triggers the production of R-proteins (from gene-for-gene resistance) in other parts of the plant for if the pathogen arrives.

This gives the entire plant immunity.

Apposition & new barriers

Substances in the cytoplasm cover breaches in the cell wall, preventing more pathogen entry.

Lignin, resins and cork cells are produced which solidify entrance points for the pathogen.

Abscission

Infected parts of the plant fall off to prevent the rest of the plant being infected.

Example: The Marri gum

Water mold protozoa enters

↓

Plant stress trigger the PAL enzyme

↓

Produces phenolics chemicals

↓

Triggers lignin production

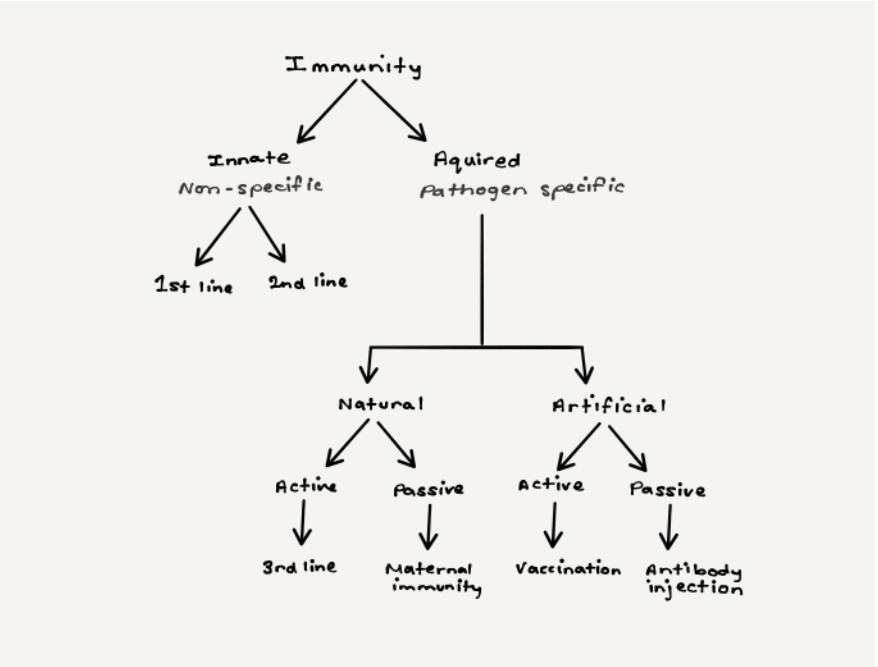
↓

No more of the protozoa can enter

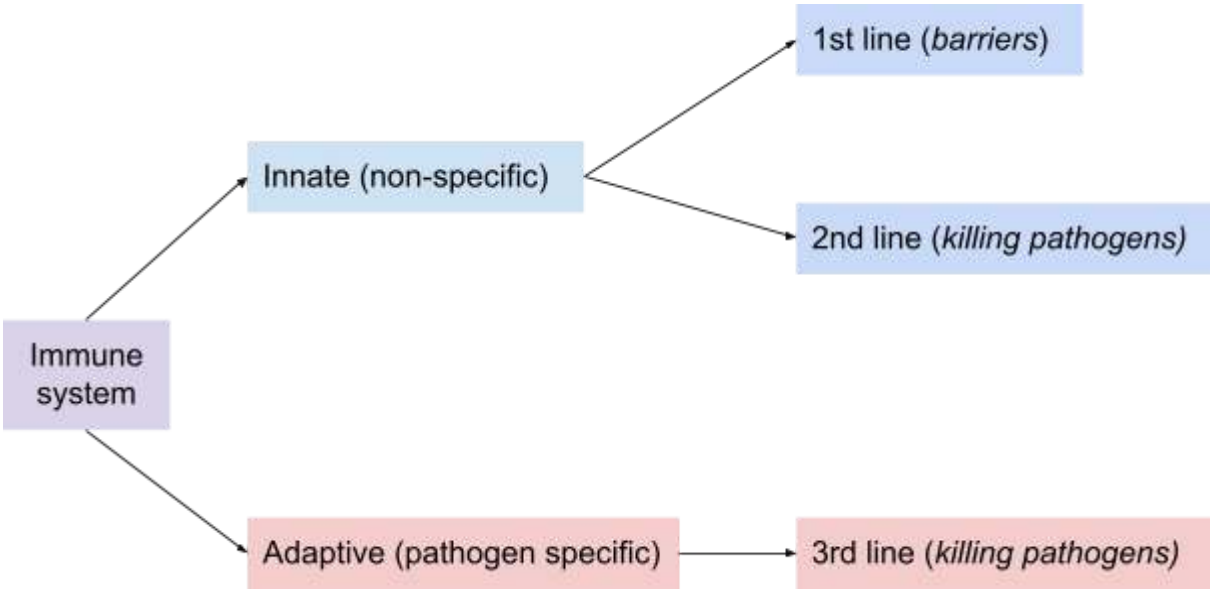
This makes the marri gum more resistant to water mold infection than the Jarrah gum.

7.3: Immunity

How does the human immune system respond to exposure to a pathogen?



[7.3.1] Innate and Adaptive Immunity



1st LINE OF DEFENCE

Non-specific barriers to prevent pathogen entry.

Skin

Physical	Chemical	Biological
<u>Tightly packed cells</u> Prevents pathogens passing. <u>Dead layer</u> No nutrients for pathogens. <u>Dry</u> Prevents growth of water-dwelling pathogens (fungi).	<u>Sweat & skin oil</u> Produces an acidic microenvironment which inhibits pathogens. Prevents growth of colonies.	<u>Beneficial bacteria</u> On the surface which outcompete pathogenic bacteria.

Mucous membrane & cilia

Physical	Chemical	Biological
Produces mucus which lines the body openings to entrap pathogens. Cilia on the mucous beat towards the openings, moving pathogens to be expelled (*eg. by coughing*). This creates the mucociliary elevator.	Mucus contains lysozymes which destroy the pathogen.	Beneficial bacteria in nutrient-rich mucous outcompete pathogenic bacteria.

Other

Physical	Chemical
Peristalsis moves the digestive tract to disrupt pathogen buildup.	Stomach is acidic due to HCl and the small intestine is basic. Rapid pH change denatures pathogen's enzymes. Tears and saliva contain digestive lysozymes Urinary and vaginal tracts are acidic Urine is acidic, which prevents growth in the urinary tract

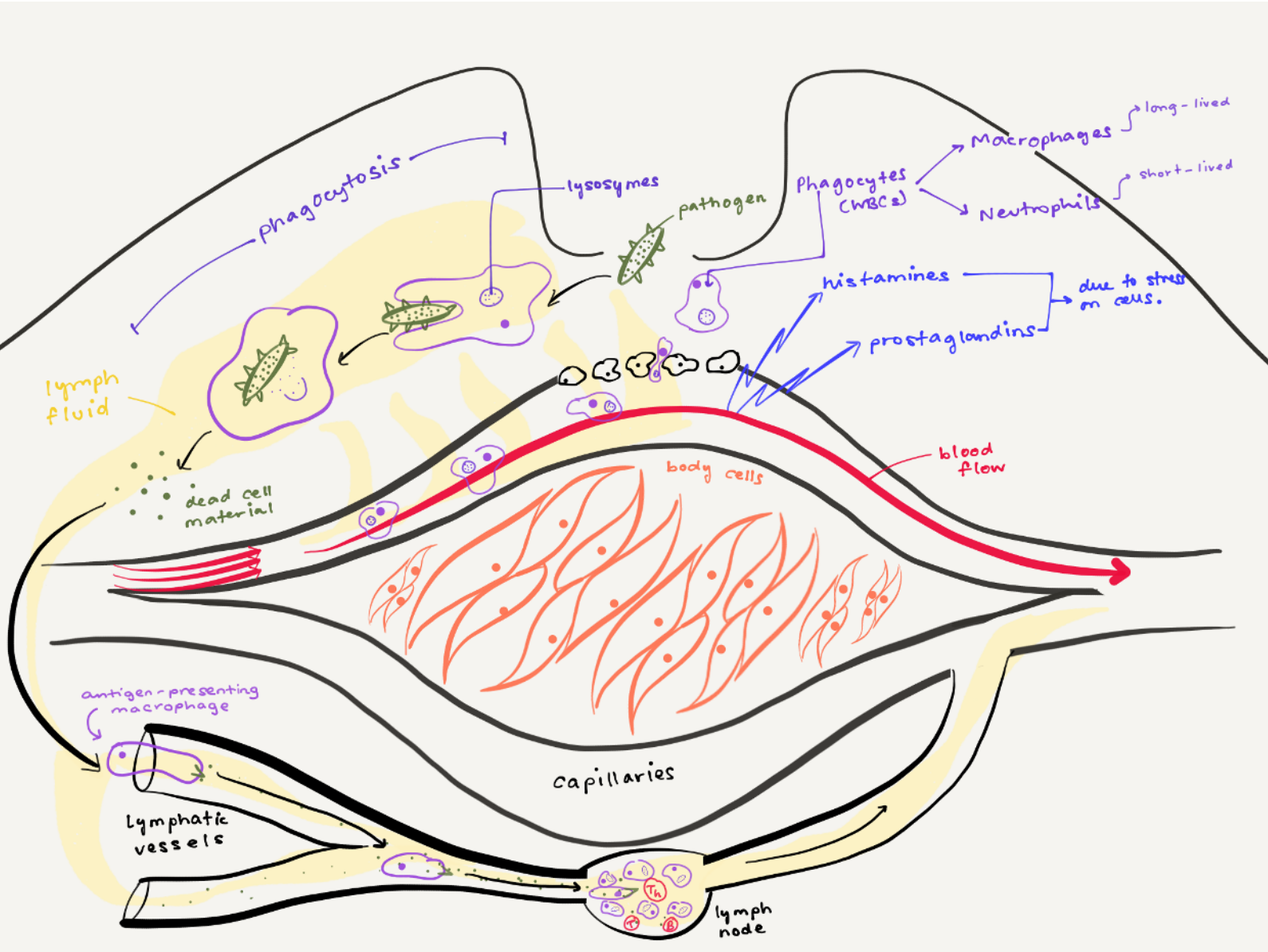
2nd LINE OF DEFENCE

Non-specific responses to kill pathogens.

The **inflammatory response** is triggered when cells under stress from a pathogen release **histamines and prostaglandins**. They cause **vasodilation**, increasing blood flow to the site of infection.
This increases the temperature and the movement of phagocytes to the infected site.
Lymph fluid also moves into the cavity.

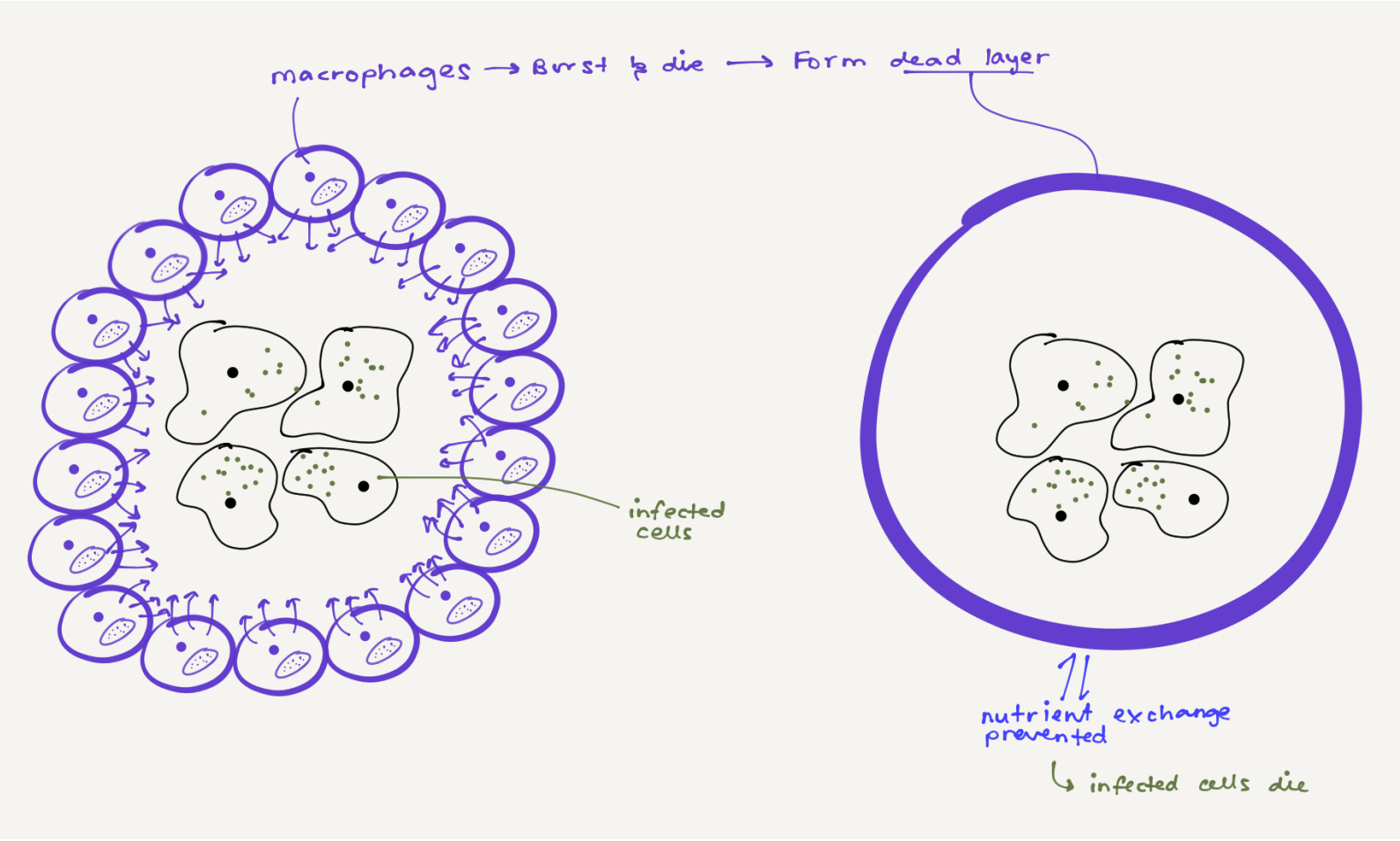
WBC in the 2nd line are **phagocytes** (neutrophils and monocytes). They squeeze through the capillary walls towards the pathogen. They engulf it and destroy it with digestive lysosomes in **phagocytosis**.

The lymph fluid and cell debris are filtered by the **lymphatic system**. They pass through the lymph nodes which are filled with phagocytes. These remove any remaining pathogens, and the lymph fluid is returned to the blood.



Antigen-presenting macrophages can also present antigens to **T-helper cells** in the lymph nodes.
The lymph nodes also contain **memory B and T cells** which can be triggered if the infection is secondary.

Macrophages surround the pathogens and die, creating a dead layer. This is **apoptosis**, and prevents the nutrient exchange, killing infected cells if the pathogen evades the first three 2nd-line defences.



Pyrogens are chemicals sent to the hypothalamus that increase body temp, causing **fever**. This can kill pathogens by inhibiting enzymes.

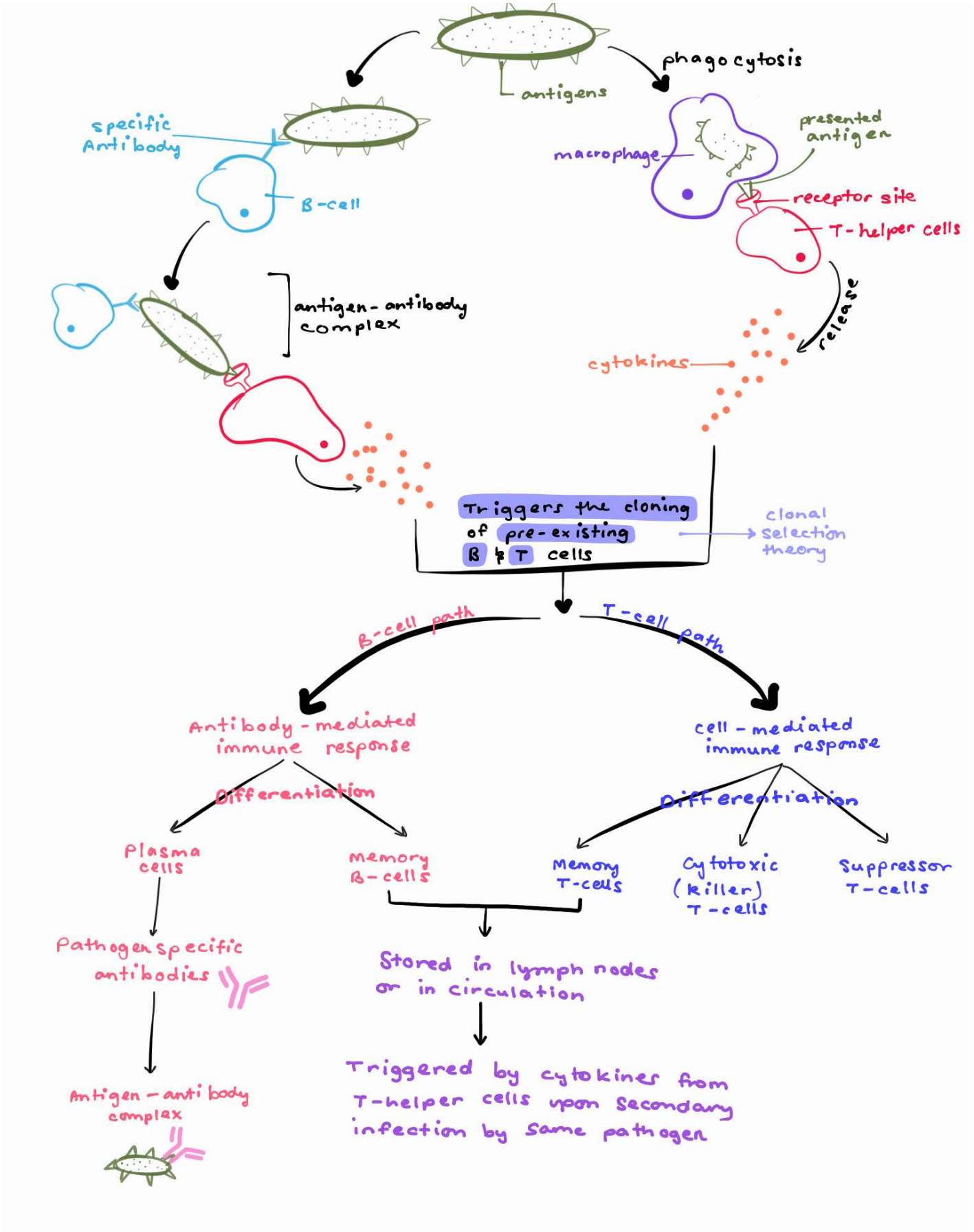
Cytokine proteins released by infected cells communicate with surrounding cells to:

- Tell infected cells to undergo apoptosis
- Tell non-infected cells to stop protein synthesis for viral replication
- Link the 2nd and 3rd line

3rd LINE OF DEFENCE

All pathogens and host cells have identifying **surface proteins**. When foreign surface proteins enter a host, they become **antigens**.

Detecting these antigens allows the 3rd line to launch an **antigen-specific immune response**.



WBCs in the third line are **lymphocytes**. These are:

- B-cells (for extracellular pathogens)
- T-cells (for intracellular pathogens)

Antigens are presented (by either macrophages or pre-existing B-cells) to the **helper T-cells**, which release **cytokine proteins** such as **interleukin**.
This triggers cloning of existing B and T cells.

In the **antibody-mediated immune response**, antibodies attach to the antigens of the pathogen. They **agglutinate** (clump) or **neutralise** the pathogen, and **attract cytokine** proteins.
These proteins **attract phagocytes**, which engulf the pathogen.

In the **cell-mediated immune response**, **cytotoxic T-cells** release perforin which penetrates the cell membrane, causing the cell to burst.

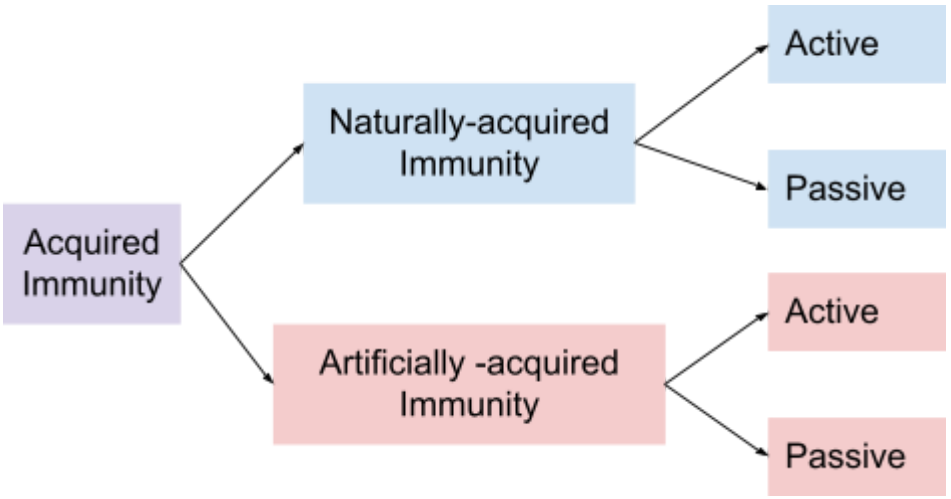
Suppressor T-cells inhibit the helper T-cells, stopping the immune response after the pathogen is removed.

Memory B and T cells remain in circulation or in the lymph nodes in preparation for a secondary infection by the same pathogen.

[7.3.2] Innate and Acquired Immunity

TYPES OF IMMUNITY

Immunity to a pathogen means that the body can produce the antibodies required to inhibit it. This only occurs from antigen-specific adaptive/ acquired immunity.

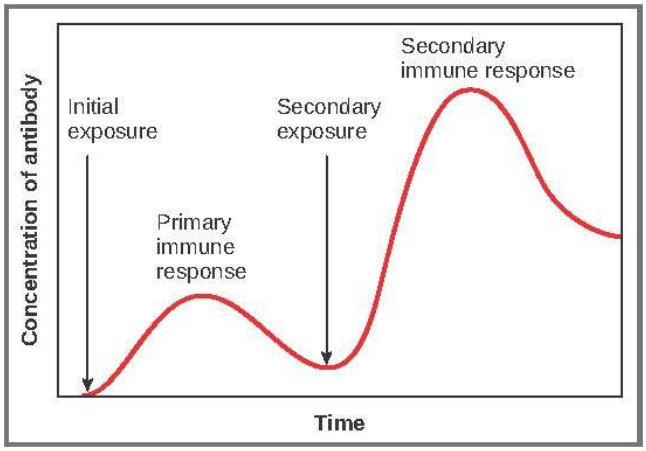
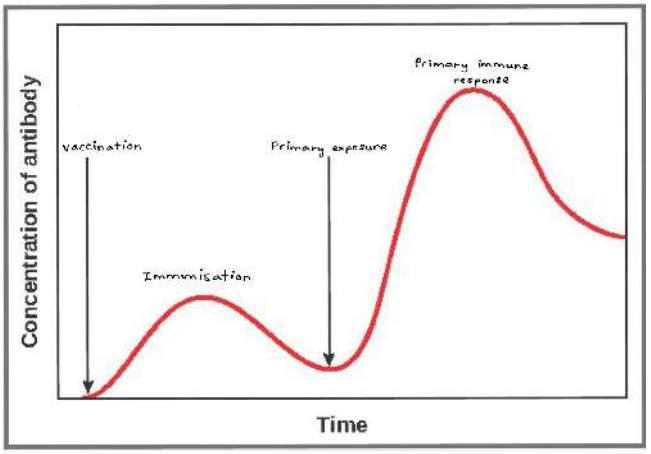


Naturally acquired	Active *The body has to produce antibodies*	Body is infected and the adaptive immune system produces memory cells
	Passive *Antibodies are provided*	Antibodies passed from mother to child
Artificially acquired	Active	Vaccination
	Passive	Injecting antibodies directly

VACCINATIONS

Vaccination is the process of injecting antibodies OR the mRNA to produce antibodies into the host.

Immunisation is the host's response to this.

Unvaccinated host	Vaccinated host
	

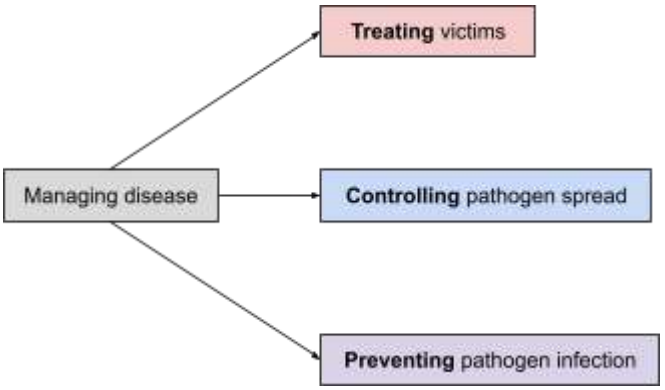
Vaccines provide the antigens to produce memory B and T-cells without symptoms of the infection.

Then, if a primary infection occurs, antibodies can be produced rapidly.

7.4: Prevention, Treatment and Control

How can the spread of infectious diseases be controlled?

Health: A state of physical, mental and social well being.
Disease: A condition that adversely affects the normal functioning of a body part/ system



The focus has shifted from treating disease and controlling the spread of pathogens, to preventing the disease by stopping pathogens entering the body.

HISTORICAL AND CULTURAL DISEASE MANAGEMENT

Case study: *Malaria*

Time period	Management
Greeks	Thought the source was bad air
Romans	Found more infections in settlements near stagnant water. Thought the source was water and drained sources. *Even though the cause was not known, they still found ways to control the spread based on trends.*
1600s	First treatment tested. It was produced from the bark of the Quinine tree
1880	Plasmodium protozoa discovered as cause of malaria. Mosquito vector and steps in transmission discovered.
1898	DDT pesticide was developed to kill malaria vectors, but was banned as it was not biodegradable
1944	Synthetic quinine and chloroquine developed to treat malaria. Chloroquine is the currently used treatment.
1950	As malaria began re-spreading, the W.H.O input preventative methods: - Protective clothing - Insecticides - A fish species introduced to eat the vectors in waterways
1970	Prophylactics developed as preventative drug

FACTORS IMPACTING DISEASE TRANSMISSION

Pathogen	- High virulence (rate of spread) - Incubation period (shorter incubation, higher virulence) - Resilience to disinfectants
Host	Immunodeficiency due to: - Malnutrition - Immunosuppressants - Antibiotics/ corticosteroids which produce resistant pathogens or kill beneficial microbes
Environmental	- Natural pathogen reservoirs - Deforestation/ natural disasters (which displace vectors and move them closer to settlements) - Geographical isolation (reduces pathogen transfer)
Societal	Local: - Miseducation campaigns - Poverty (can't afford medicine) - Religious obligations to modern treatment - Crowding in low socioeconomic regions - Portion of population immunised Regional: - Isolated gene pools with less variation - Cultural practises (eg. burial rituals which contact the corpse) Global: - Mass migration (due to war, natural disasters) leads to movement of pathogens into new regions

PROCEDURES LIMITING DISEASE SPREAD

Sanitation	Personal hygiene: - Washing hands w/ disinfectant - Keeping body openings clean - Washing hair Community hygiene: - Controlled sneezing/ coughing - Preventing overcrowding (controlled urbanisation) Food hygiene: - Washing hands and food before cooking - Cooking fast at high temp - Prevent exposure to open air - Allow air flow in storage Water hygiene: - Treat water before distributing - Boil/ filter water before drinking
Facilities	Community hygiene: - Garbage & sewage disposal - Regular sanitation - Sterilisation of surgical equipment in hospitals - Maintaining hygiene standards in restaurants Farming practises: - Must be carried out away from houses
Quarantine	Preventing pathogens from entering into a country (exotic) or between states (endemic) by limiting the movement of its host. **Border control** Passengers have to declare/ discard animal products, plant products or soil before entering Aus. **Animal quarantine** Animals are kept at quarantine stations and monitored for disease symptoms before entering Aus. **Plant quarantine** Most plants refused entry. Some are fumigated (sprayed with insecticides) before entering Aus. **Human quarantine** Authorities required to notify if anyone entering Aus shows symptoms of a disease. Humans may self-isolate to prevent disease spread. **North Australian quarantine** Animals enter Aus from the asian region through North Aus. An early warning system alerts if pests enter. Cattle and sheep regularly checked for disease.

Vaccinations	Provides antigens so that memory B and T cells exist before primary infection. When the pathogen enters, it is inhibited, reducing the time that it can remain in the host and spread.
Public health campaigns	To teach people how to limit disease spread. **Government regulations** ● Hygiene regulations in restaurants and health clinics ● Provides infrastructure and services to maintain community hygiene ● Requires prohibited disease to be reported **Regular screening** ● Early detection of breast, cervical or prostate cancer ● Provides free packages to detect bowel cancer **Immunisation programmes** ● Childhood immunisation ● Mass immunisation for herd immunity **Public education** ● Educating on any media platform ● Encouraging a health benefit - QUIT (prevention of lung cancer by quitting smoking) - Slip, Slop, Slap (preventing skin cancer by limiting UV exposure)
Pesticides	Use of chemicals to kill pests that may be macroparasites or vectors. Overuse leads to rapid natural selection favoring resistant varieties. May lead to rise of a pesticide-resistant generation.
Genetic engineering	The genome of a species may be altered to give immunity from a pest/ disease (*eg. Bt-cotton*). However, this can: - Decrease biodiversity as modified alleles will be favoured and inherited - Alters evolutionary path - May cause resistant disease/pest variety to emerge as the lack of a host becomes a new selection pressure

EFFECTIVENESS OF PHARMACEUTICALS

Antibiotics

Function	Effectiveness
Can treat bacterial infections ONLY.	• Not effective on any other infection
Can only kill pathogens *after* infection is established.	• Not effective as prevention • Only effective as treatment
Kills/ inhibits bacteria by: - Preventing cell wall forming - Destroying cell membrane - Interfering with bacteria protein synthesis	• Overuse can cause rapid natural selection • Favours resistant varieties which re-infect the host • Antibiotic effectiveness decreases
Broad-spectrum antibiotics can kill a wide range of bacteria	• Effective when bacterium is unknown • Can also kill beneficial bacteria that suppress pathogenic bacteria
Narrow-spectrum antibiotics kill specific bacterial pathogens	• Very effective against specific bacterium • Not effective if predicted bacterium is incorrect

Antivirals

Function	Effectiveness
Work on viral infections ONLY	• Not effective on any other infection
Slow down replication of viral DNA/RNA when it is inserted into the host genome. Once the spread of virus is slowed, the immune system can kill it more effectively	• Can not cure the disease alone
Can assist before symptoms appear	• Most effective in early disease-onset • Can be a preventative measure • Efficacy decreases after disease onset
Lowers chance of secondary bacterial infection as the immune system is not redirected long enough to suppress bacterial growth	• Not effective if secondary infection occurs • Antibiotics have to be taken instead

ENVIRONMENTAL MANAGEMENT AND QUARANTINE

Case study: *Ebola*

Caused by ebola virus. Symptoms start as fever, tiredness, headache and then progress to vomiting, diarrhea, liver damage and rapid death.

Factors contributing to disease	Management strategies
Lack of education - Direct transmission during sex via fluids - No hygiene practises	• Education on protected sex • Safe cooking • Safe waste management • Safe burial practises • Disinfection & sterilisation • Personal protective equipment • Quarantine of patients
Cultural/ religious beliefs - Refusal to take modern medicine - Burial rituals involving contact with diseased corpse	• Convince tribal leaders first. Then they will convince the rest of the society
Poverty - People hunt meat instead of buying, but meat is a reservoir for ebola virus	• Humanitarian aid to provide clean food
Lack of biosecurity No plan to cope/ counteract the impacts of disease spread on the economy, education, society, etc. due to lack of reserve funds.	• International financial aid

INCIDENCE AND PREVALENCE OF INFECTIOUS DISEASE

Incidence	Prevalence
No. of new cases in a time period Can be taken as a *rate* to show how fast the disease is spreading: ↪ $ind = new\ cases/\ 100,000$ OR as a *percentage* to show how many in a population are new patients. ↪ $\%\ inc. = \frac{new\ cases}{total\ population} \times 100$ Incidence ∝ rate of spread	No. of total existing cases $Prev = Existing\ cases + new\ cases\ (ind) - cured\ cases - mo$ It is taken as a *percentage* $prev = \frac{total\ cases}{total\ population} \times 100$

INDIGENOUS AUSTRALIAN DISEASE PROTOCOLS

Indigenous Australians have used compounds bush medicine to treat or prevent disease for generations before white settlement:

Compound	Source	Use
Tanin	Tea tree (extract from crushed leaves)	- Inhaled to reduce fever and sore throats - Antimicrobial properties prevent wound infection
Latex	Tree milk	- Contains enzymes which clean wound and removes warts
Mucilage	Cacti (jelly-like substance on the inside)	- Used as skin moisturiser to prevent cracks, which pathogens can enter through
Alkaloids - Caffeine - Morphine - Codeine	Plant material	- Pain relief - Stress relief

Furthermore, scientists have discovered the potential of bush medicine to manage/ treat currently incurable diseases

- Black bean plant for AIDs
- Smoke bush for cancer and AIDs

However, rights to the intellectual property of these medicines is questionable because:

➔ Australian intellectual property law allows any Australian person/ company rights to these medicines
BUT
➔ Australian-Aboriginal customary law connects Indigenous people to their land, giving right over traditional practises and medicines within it

Either every Australian is equal OR Indigenous Australians receive proper recognition for their properties and practises.

Whatever the decision:

1. Indigenous elders must be consulted first
2. It must align with Aboriginal customary law