

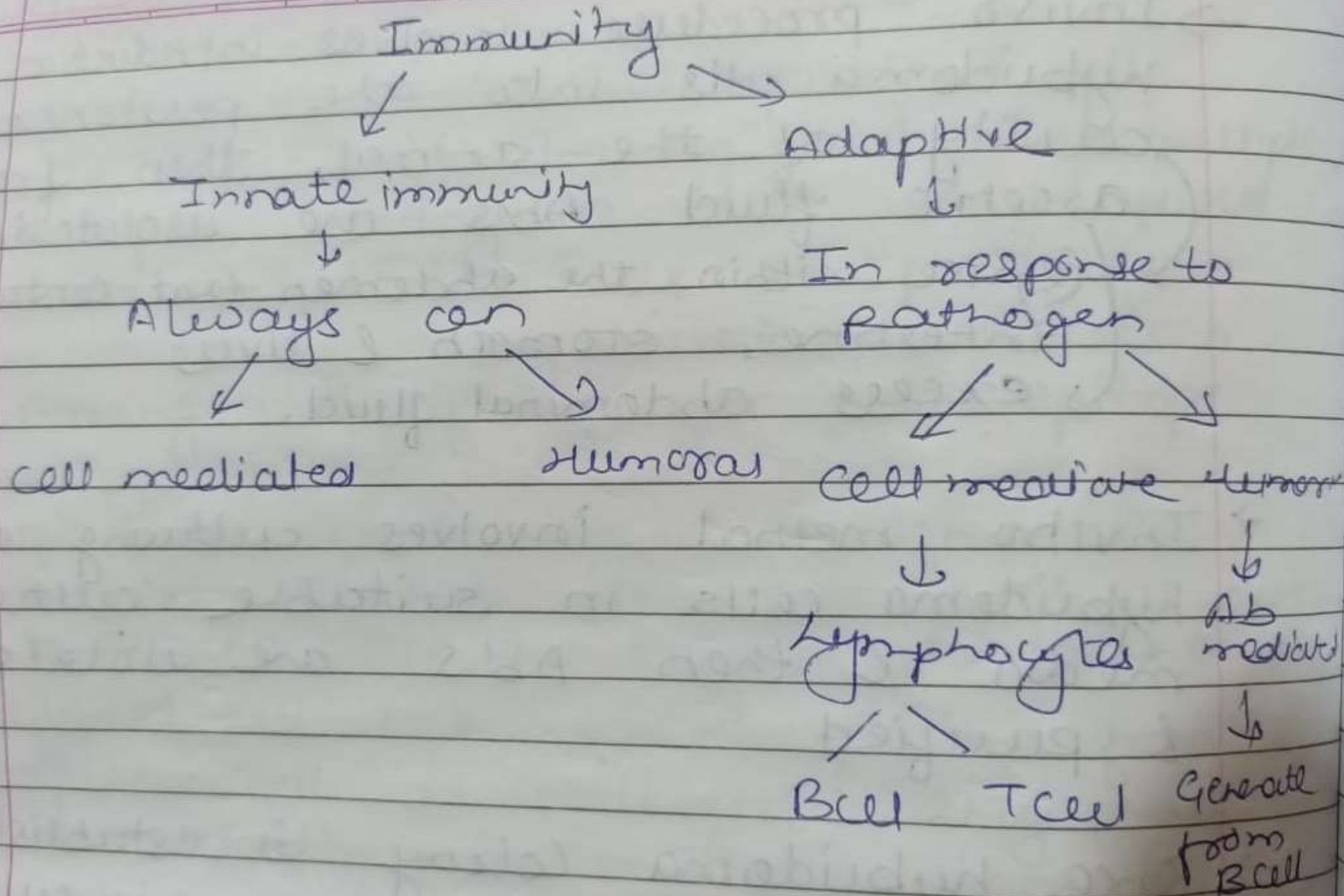
Department Of Biotechnology

Acquired or Adaptive Immunity

By

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Aquired cells Immunity



Innate immunity further classified

- Main Components of Innate and acquired Immunity that contribute to humoral (antibody-mediated) immunity and cell mediated immunity

	<u>Humoral Immunity</u>	<u>Cell mediated Immunity</u>
Innate	Complement Neutrophil	Macrophages Natural killer cells
Acquired	B cells Antibodies	Helper T cells Cytotoxic T cells

Adaptive immunity: second line of response

- Based upon resistance acquired during life
 - comes into action after innate immunity fails to get rid of microbe
- Relies on genetic events and cellular growth
- Responds more slowly, over few days

Three major functions

- Recognize nonself
- Respond to nonself
- Remember nonself

What is adaptive immunity?

Adaptive ('acquired') immune system **

B cells – mature in bone marrow; contribute to antibodies that bind directly with specific antigens; contribute to humoral immunity ^Δ

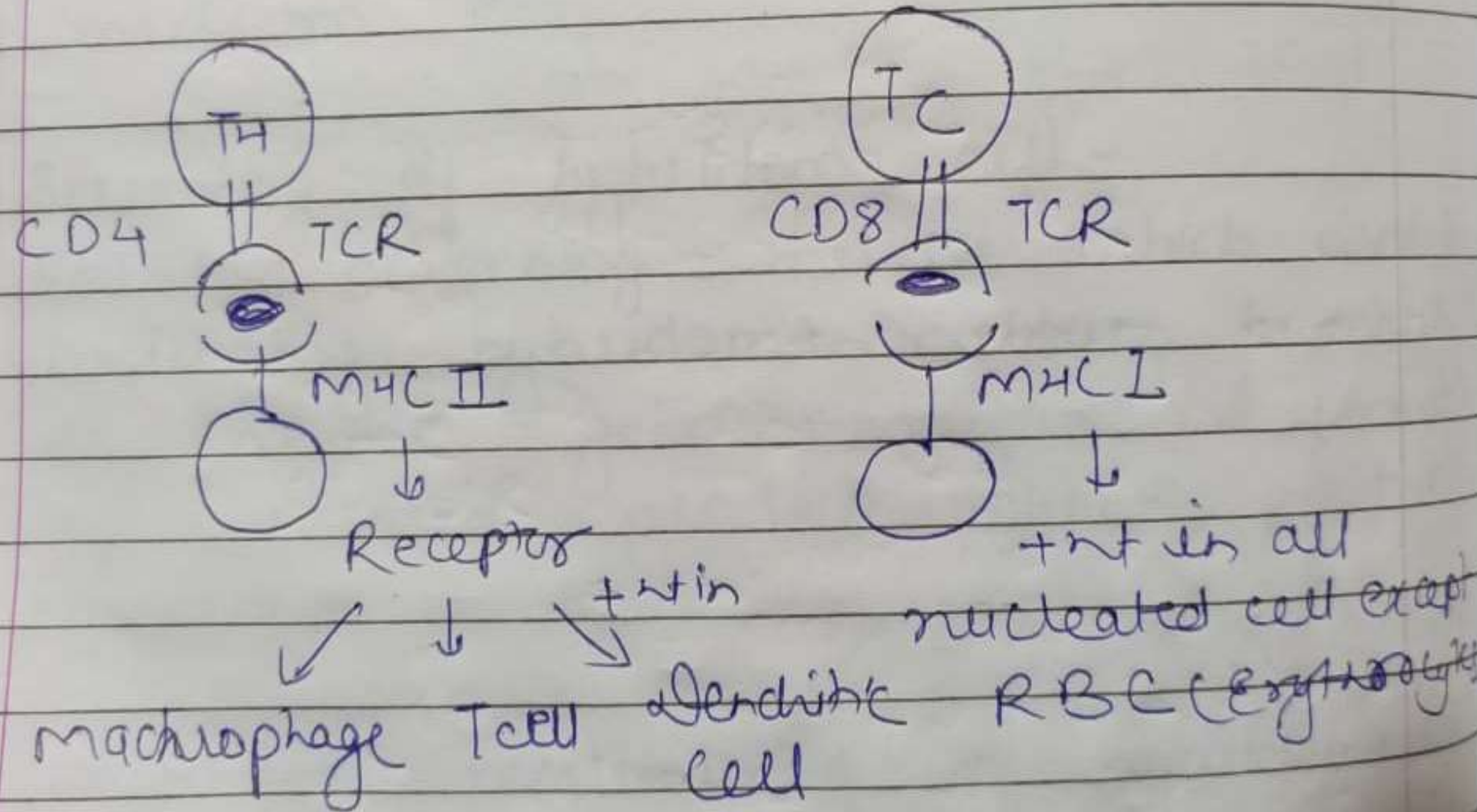
T cells – mature in the thymus; express T cell receptors and CD4 or CD8 (not both); contributes to cell-mediated immunity ^ε

T cell receptors only recognise antigens bound to certain receptor molecules (major histocompatibility complex [MHC] class I or II)

CD4 and CD8 contribute to T cell recognition and activation by binding to either MHC I or MHC II

Highly specific (identifies pathogens and differences in molecular structures) and **slow** (days)

CD4 - Helper T cell OR
 CD8 - cytotoxic T cell



Four Characteristics of Adaptive (Specific) Immunity

- **Discrimination between self and non-self**
 - usually responds selectively to non-self, producing specific responses against the stimulus
- **Diversity**
 - generates enormous diversity of molecules
- **Specificity**
 - can be directed against one specific pathogen or foreign substance among trillions
- **Memory**
 - response to a second exposure to a pathogen is so fast that there is no noticeable pathogenesis

Adaptive

Naturally acquired

Artificially acquired (nospiro)

Passive

Active

Passive

Active

we didn't do anything
↓
placenta

we did something
↓
infected

we didn't do anything
↓
Pre-formed

we did something
↓
vaccines (Ags)

Abs passing from
mom to baby
through placenta
or breast milk

↓
Ags enter
the body
naturally
↓ infects

↓
made Abs
from other
patient/lab

body forms
Abs

↓
Pre formed
Abs introduced
to your body
↓
Anti serum
X Y X

↓
a weekend
or killed
organism

↓
Then the
body
fights

↓
& develop
Abs
body

Adaptive Immunity

The resistance that an individual acquires during life

Active Immunity

Resistance developed as a result of antigenic stimulus

Natural active immunity

Type of specific immunity a host develops after exposure to foreign substance

Artificial active immunity

(vaccination)
Intentional exposure to a foreign material

Passive Immunity

Resistance transmitted passively in ready made form

Natural passive immunity

Transfer of antibodies, e.g., mother to fetus across

Artificial acquired passive immunity

Preformed antibodies or lymphocytes produced by one host are introduced into another host

Adaptive Immunity

Active immunity	Passive immunity
Produced actively by host immune system	Immunoglobulins received passively
Induced by <ul style="list-style-type: none">• clinical, sub-clinical Infection (natural)• Vaccination (artificial) Live, killed, purified antigen vaccine	Acquired by- <ul style="list-style-type: none">• Mother to fetus IgG transfer, breast milk, (natural)• Readymade antibody transfer immune serum, immune cells (artificial)
Long lasting	Lasts for short time

Types of Adaptive(Acquired)

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Acquired Immunity

Natural immunity

is acquired through the normal life experiences of a human and is not induced through medical means.

Active immunity

is the consequence of a person developing his or her own immune response to a microbe.

Passive immunity

is the consequence of one person receiving preformed immunity made by another person.

Artificial immunity

is that produced purposefully through medical procedures (also called immunization).

Active immunity

is the consequence of a person developing his or her own immune response to a microbe.

Passive immunity

is the consequence of one person receiving preformed immunity made by another person.



Infection



Maternal antibody












Vaccination



Immune globulin therapy

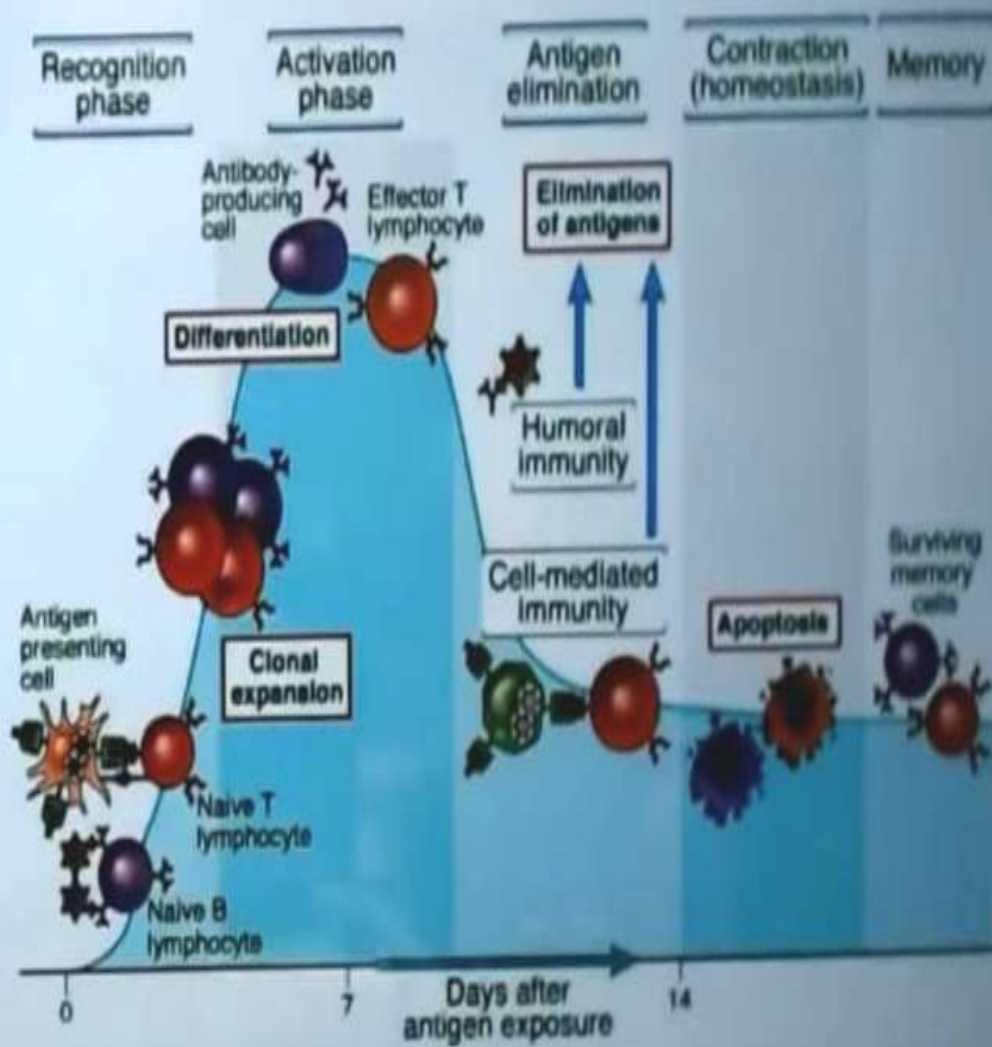
Type of adaptive immunity

- Different types of immune responses are mediated by different classes of lymphocytes and defend against different types of microbes

	<u>Humoral immunity</u>	<u>Cell-mediated immunity</u>	
<u>Microbe</u>	 Extracellular microbes	 Phagocytosed microbes in macrophage	 Intracellular microbes (e.g., viruses) replicating within infected cell
<u>Responding lymphocytes</u>	 B lymphocyte	 Helper T lymphocyte	 Cytotoxic T lymphocyte
<u>Effector mechanism</u>	 Secreted antibody		
<u>Functions</u>	Block infections and eliminate extracellular microbes	Activate macrophages to kill phagocytosed microbes	Kill infected cells and eliminate reservoirs of infection

Phases of adaptive immune response

- Need for proliferation and differentiation results in delay (typically 4-7 days) in the adaptive immune response



↓
Host expose to Ag

↓
engulf by macrophage

→ B cell

→ DC

↓

degrade in small p

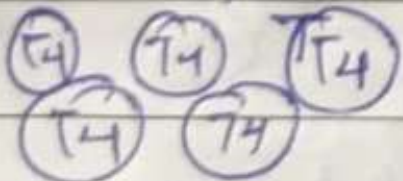
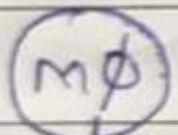
↓

present by APC

↓

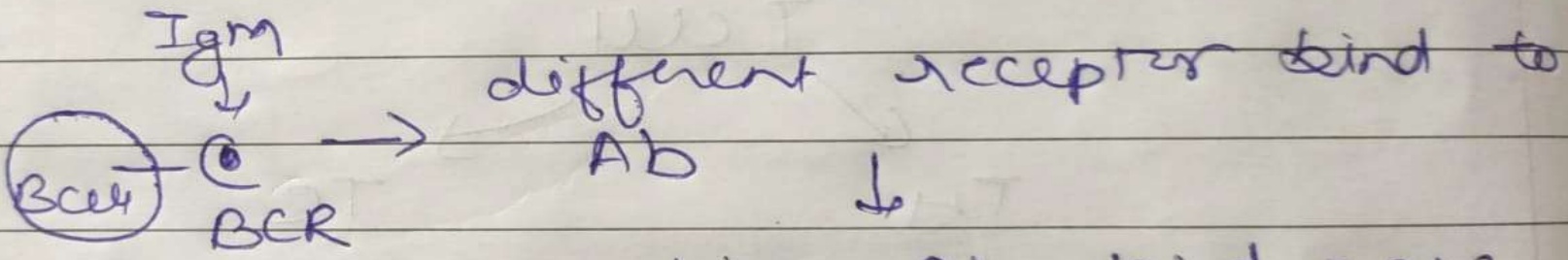
Recognize by naive

↓



Naive T cells propagation
↓
Release signalling molecule ← (clonal expansion) First time

that activate B cells
↓ naive B cell



↓
which Ab bind more accurately with Ag

↓
prepare that type of Ab



convert himself to plasma cell

② Activation phase

↓
Plasma cell → produce Ab's

Effector T_H cell + B cell

↓
Ag elimination

↓
Humoral Immunity

Ag level ↓ drop

Temp change, CRP → c reactive protein level high ← infection

↑ Opsonisation
→ complement fixation
→ direct lysis

& cell mediated cells

↓ NK cells ↓ Macrophages

↓
kill

④ Homeostasis

↓
Reach the previous situation

↓
After killing pathogen
Apoptosis — kill excess cells by
programme death

↓
that fight with pathogen

↓
cells left for

protein level high ← infection

④ Homeostasis

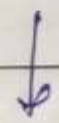


Reach the previous situation



After killing pathogen

Apoptosis — I kill excess cells by programme death



that fight with pathogen

⑤ Memory → some B cell & T cell left for memory



when again same Ag come



fight ~~with~~ fast

Adaptive immunity: mechanisms

▶ Cell-mediated immune response (CMIR)

- Mediated by T cells via:
 - Direct lysis of target (infected) cells
 - Production of cytokines that activate infected cells to kill pathogens
- Eliminate intracellular microbes that survive within phagocytes or other infected cells

▶ Humoral immune response (HIR)

- Mediated by antibodies produced by B cells
 - Antibodies bind to whole or fractions of antigens outside cells
- Eliminate extra-cellular microbes and their toxins

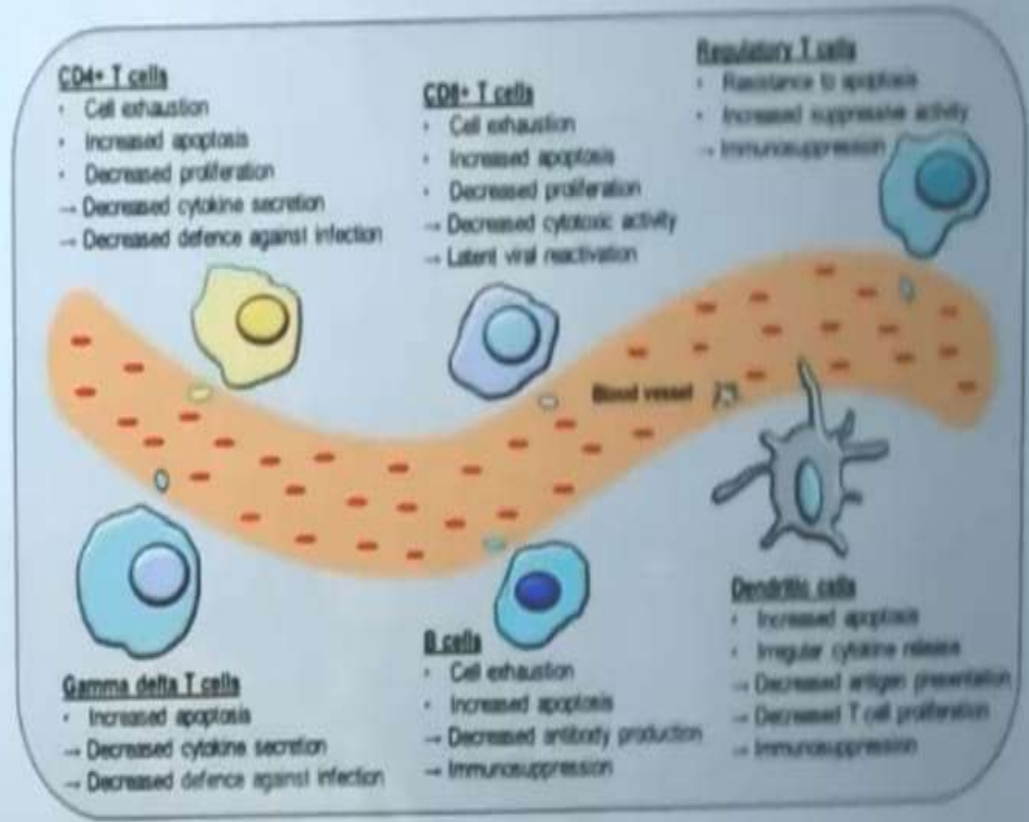
1. Cell mediated response

- **Primary response**

- production of specific clones of effector T cells and memory clones
- develops in several days
- does not limit the infection

- **Secondary response**

- more pronounced, faster
- more effective at limiting the infection
- **Example** - cytotoxic reactions against intracellular parasites, delayed hypersensitivity (e.g., Tuberculin test) and allograft rejection



- Humoral immunity is conferred by body (fluids) humors specifically by Igs secreted by terminally differentiated B cells (i.e plasma cells).
- If a foreign agent enters in to the body, immune system responds in different ways to get rid of it from the body.
- Response is not the same for all foreign agents. Response depends on the type of antigen or foreign substances which entered the body.
- Responses of immune system to any foreign agent are broadly classified in two main types one is humoral mediated response and the other is cell mediated.

- The destruction of antigens by producing antibodies is called antibody mediated immune response.
- Antibodies react with antigens (pathogens) present outside the cells. They cannot kill the pathogens present outside the cell.
- In humoral immunity, binding of antibodies to microorganism results in the formation of immune complex (Ag-Ab complex).

Bacteria



Extracellular Spaces of the Body



Intracellular Space



Cause Infectious Disease



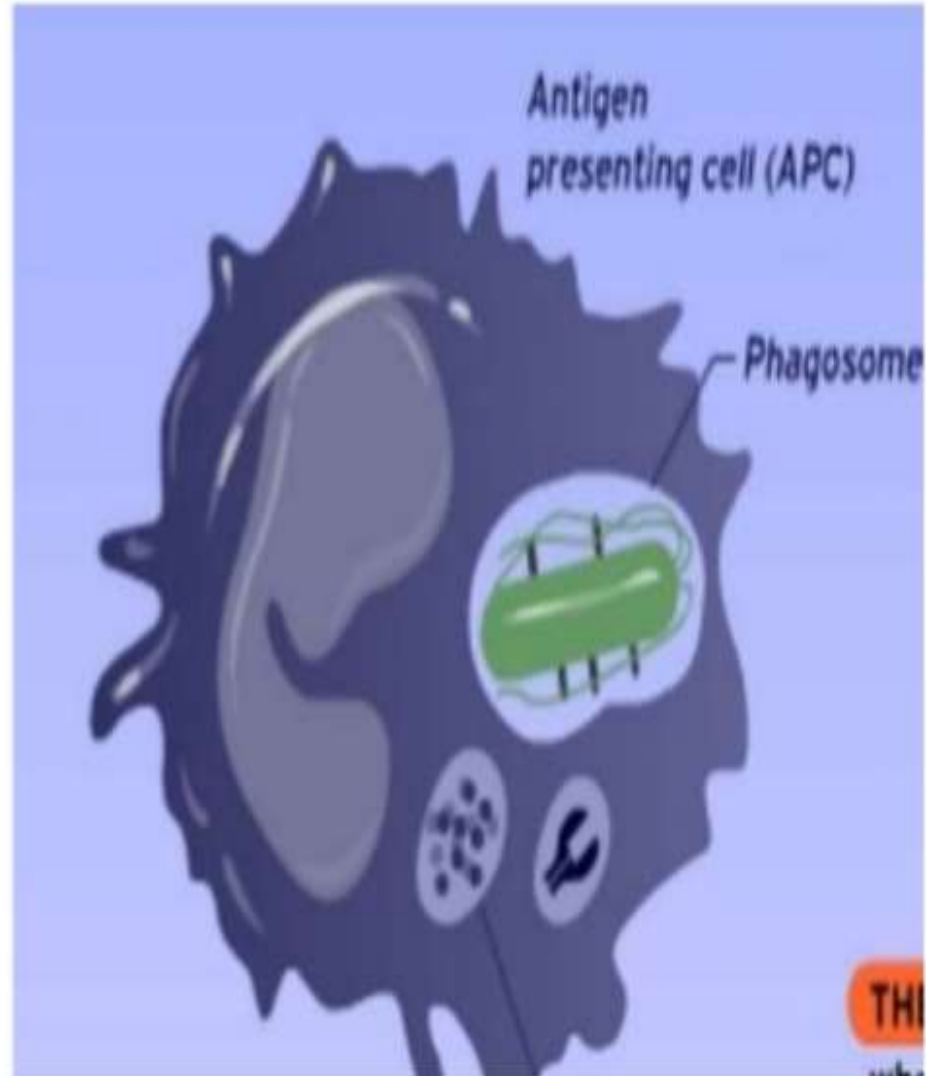
Protected



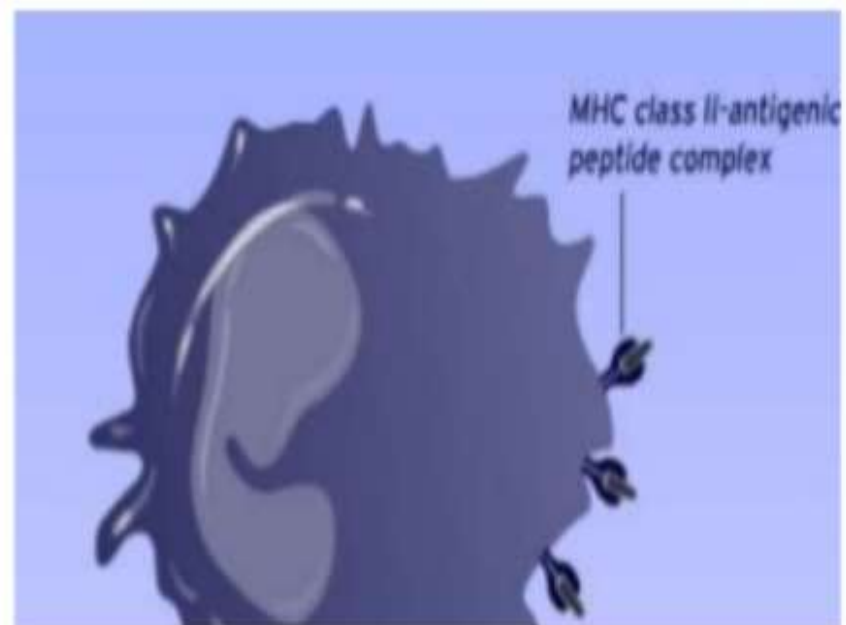
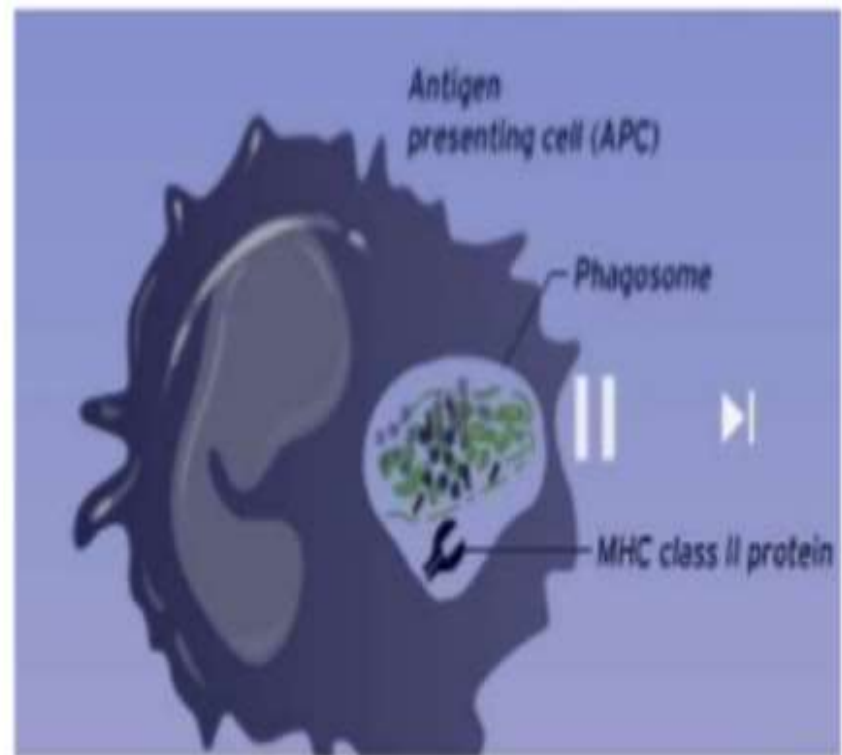
Humoral Immune Response

➤ Activation phase;

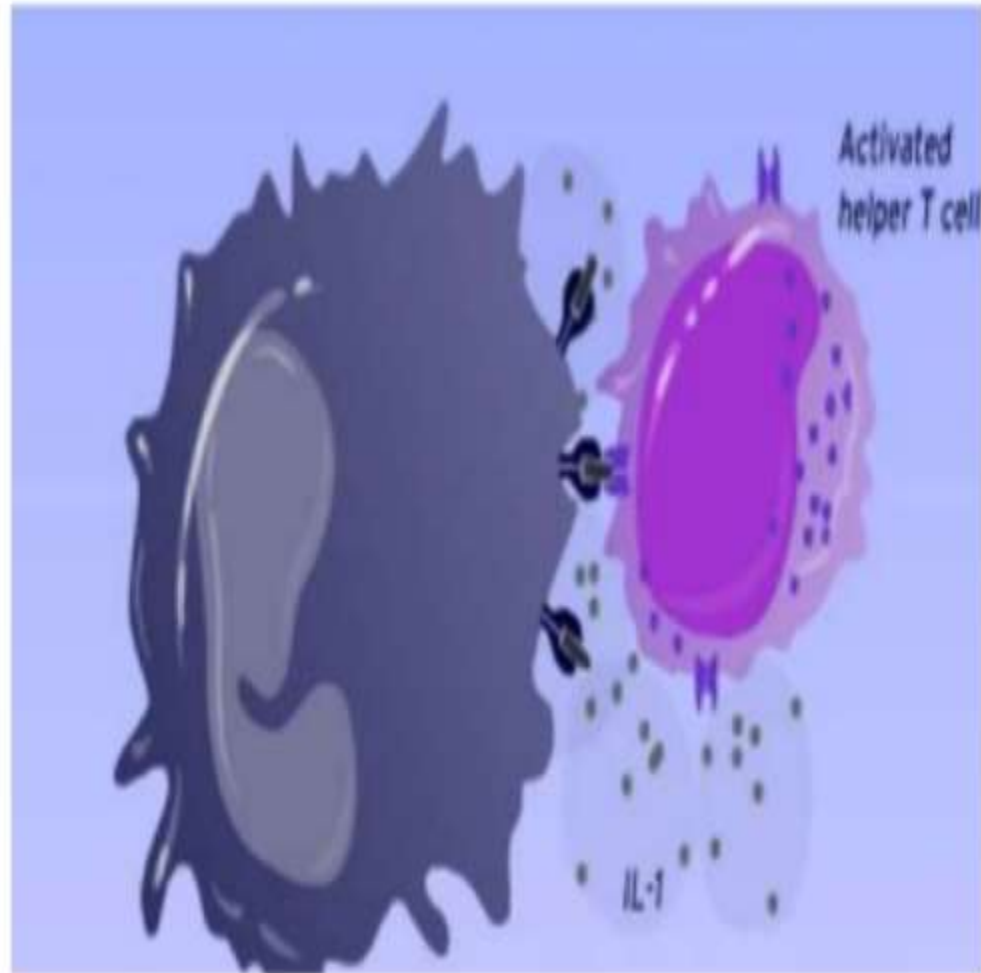
- The activation phase begins with an invading bacteria is phagocitized [engulfed] by an antigen presenting cell [APC].
- A lysosome containing digestive enzymes combines with the phagosome to process the antigen.



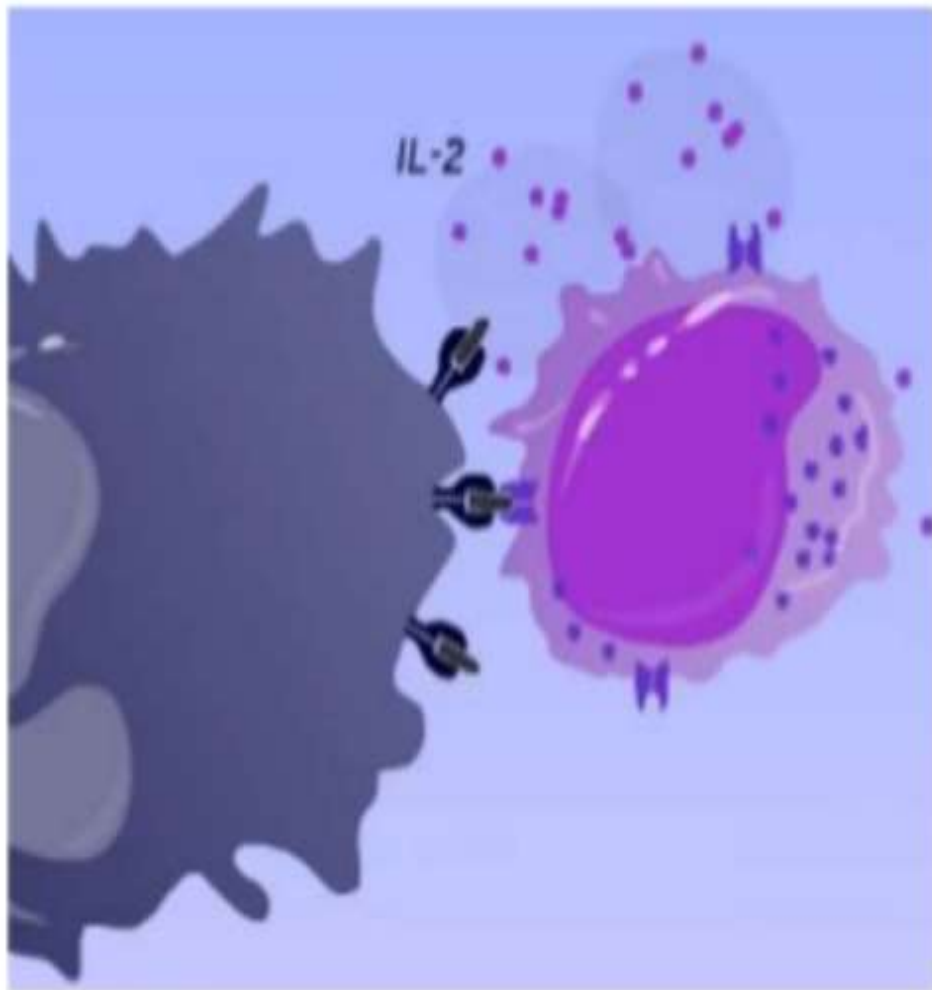
- The processed antigens combine with the MHC class II proteins and are presented on the surface of the APC.



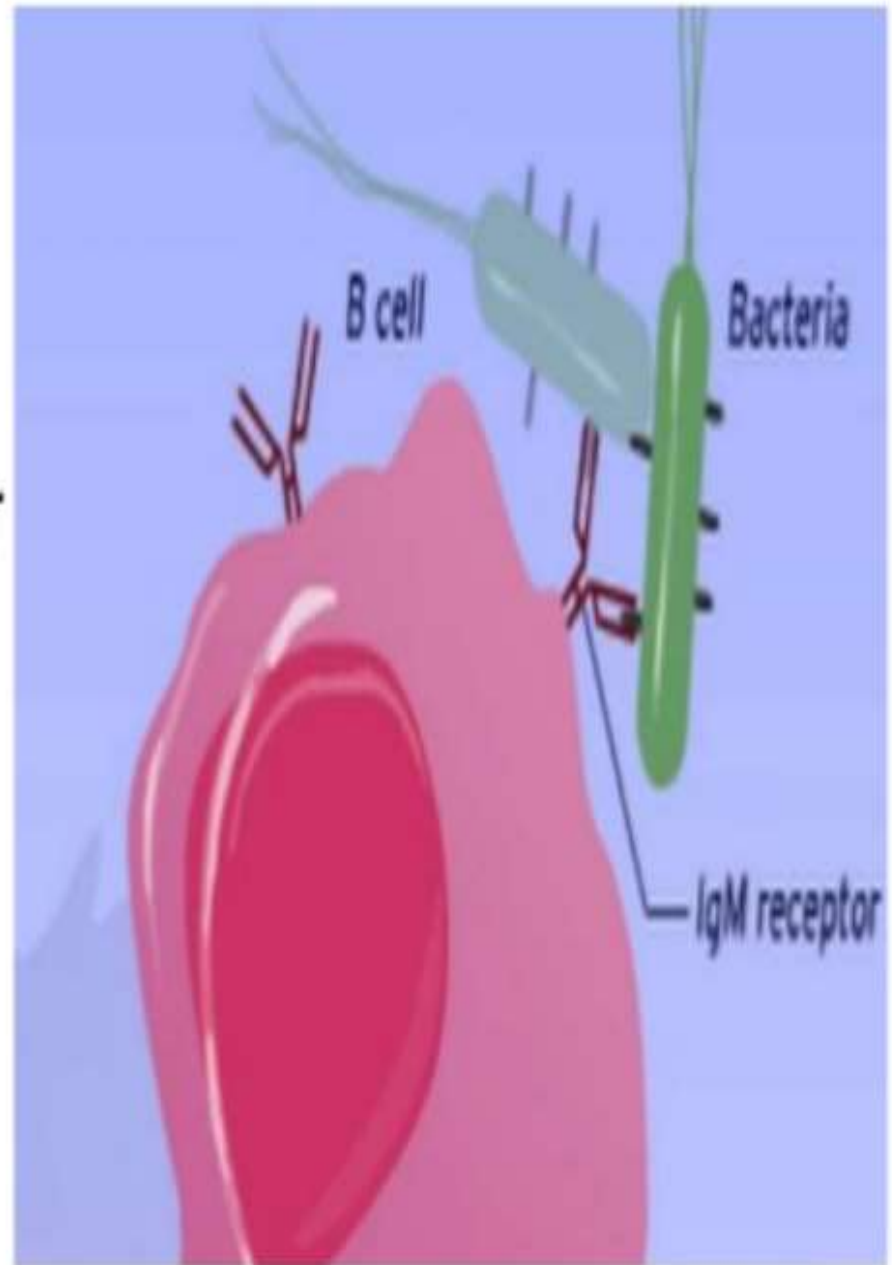
- Helper T cells [CD4+] recognize the displayed antigen on the APC and bind to the MHC class II-antigenic peptide complex.
- The binding triggers the APC to release the cytokine IL-1, which activates the Helper T cell.



- The activated Helper T cell releases the cytokine IL-2, which stimulates the helper T cell to proliferate.
- Thus, producing many Helper T cells, each with receptor specific for the original processed antigens.

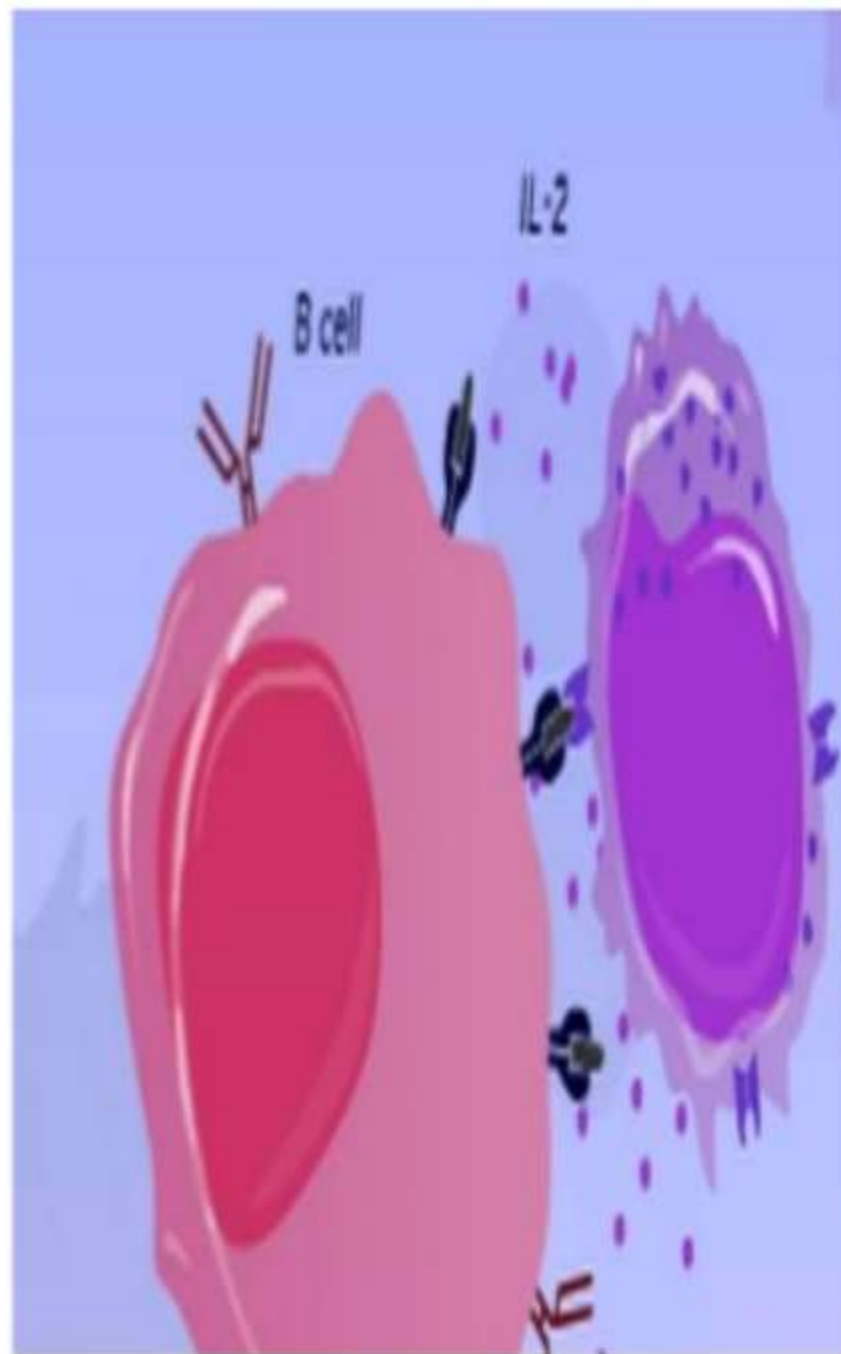


- The effector phase begins when a B cell that exhibits on its surface an IgM receptor specific for the same antigen originally engulfed by the APC encounters and binds the antigens.

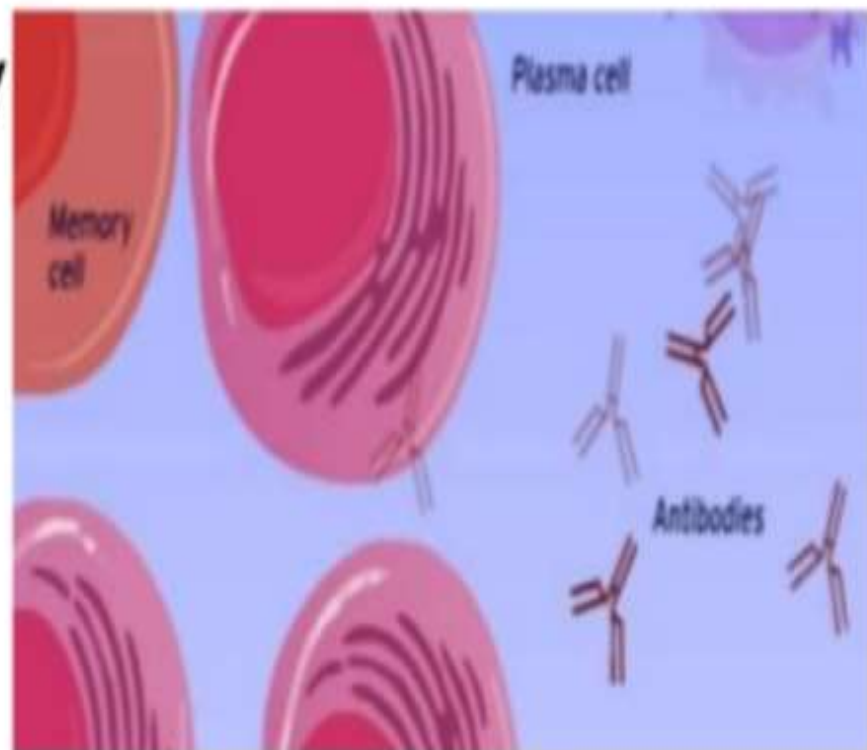


- The B cell engulfs the complex by receptor mediated endocytosis. The phagosome containing the antigens fuses with a lysosome. The antigen is processed.
- The processed antigens binds MHC class II protein and is displayed on the surface of the B Cell.

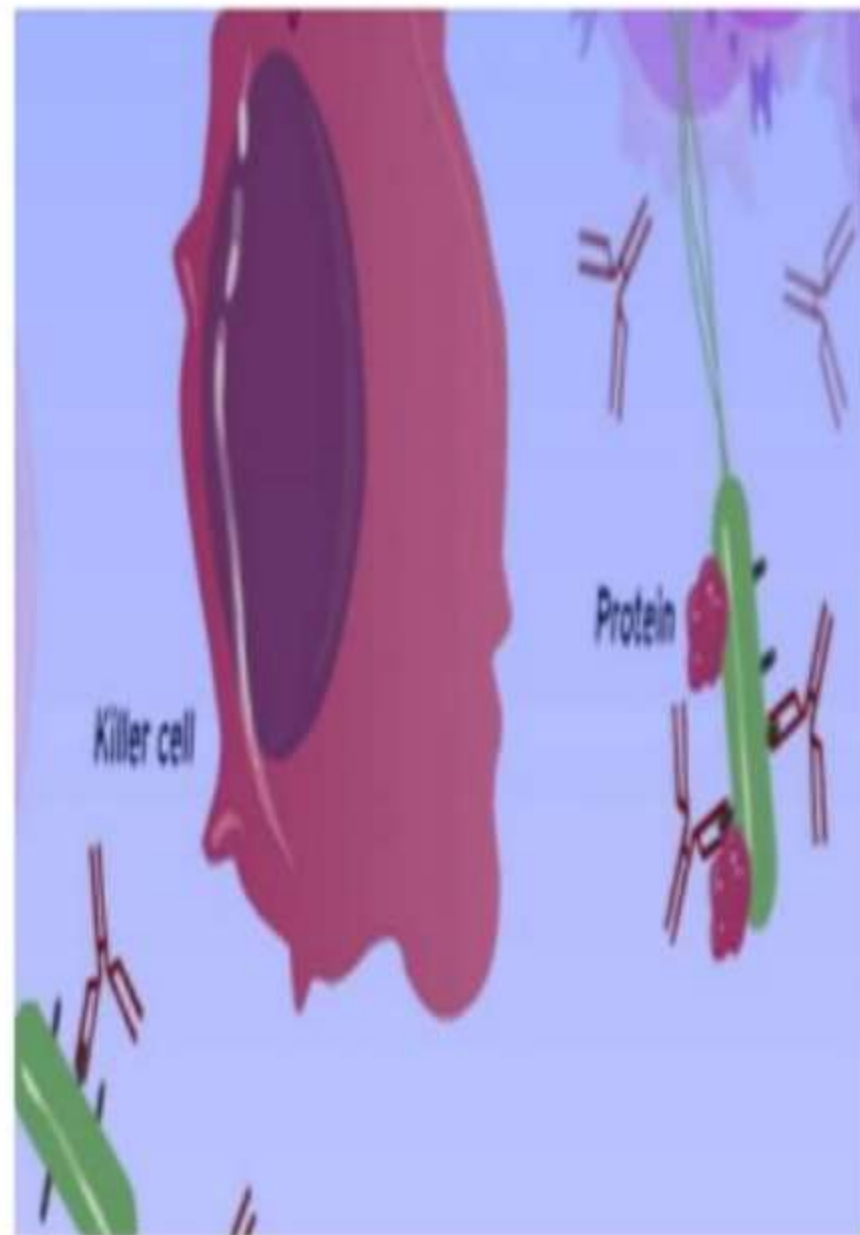
- Helper T cells now bind to the displayed antigens on the surface of the B cell causing the Helper T cell to release Cytokines.
- The cytokines stimulate the B cell to divide and proliferate into identical B cell copies.



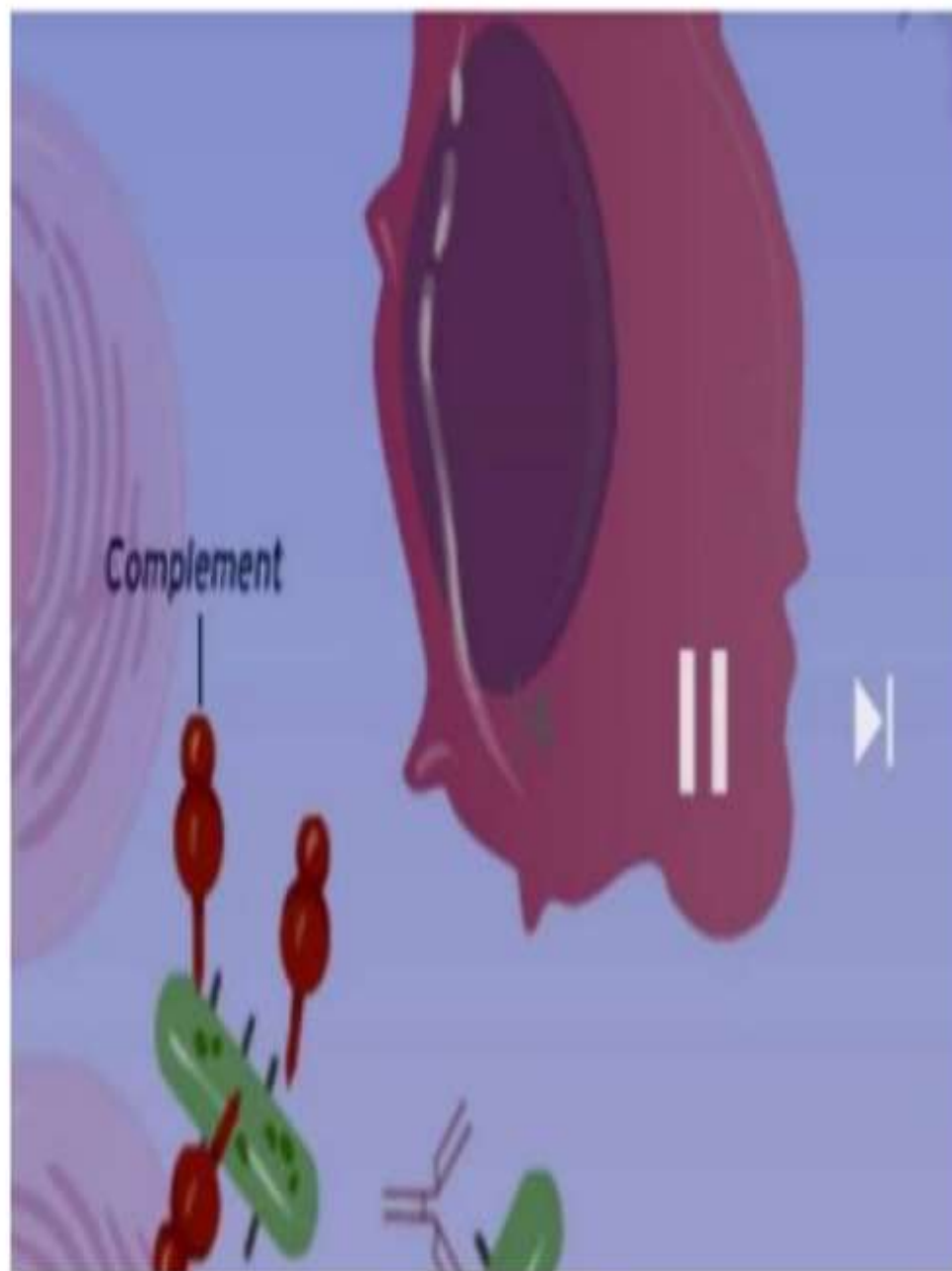
- The B cells differentiate into antibody producing plasma cells and memory cells.
- The plasma cells release antibodies with a specificity identical to that of the surface receptor on the parent B cell. These carry out the ultimate goal of fighting the foreign invaders.



- The released antibodies bind in a lock and key fashion to the antigen on the surface of the original invaders.
- These makes it easier for killer cells to attack and destroy the bacteria by phagocytosis and release of proteins causes the direct lyses of the bacteria.

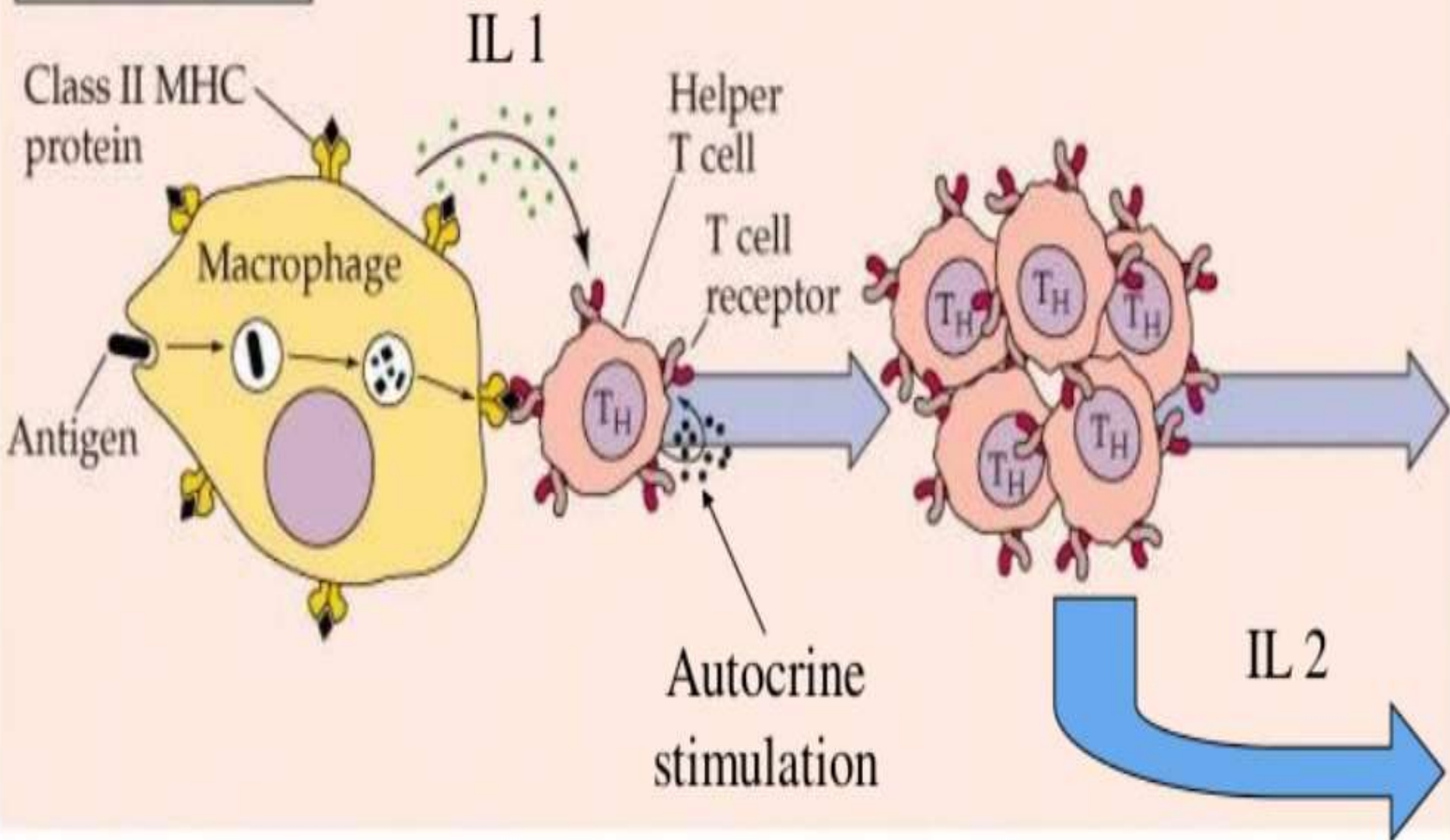


- Simultaneously a blood component known as complement is signaled to attack and puncture holes in the bacteria.

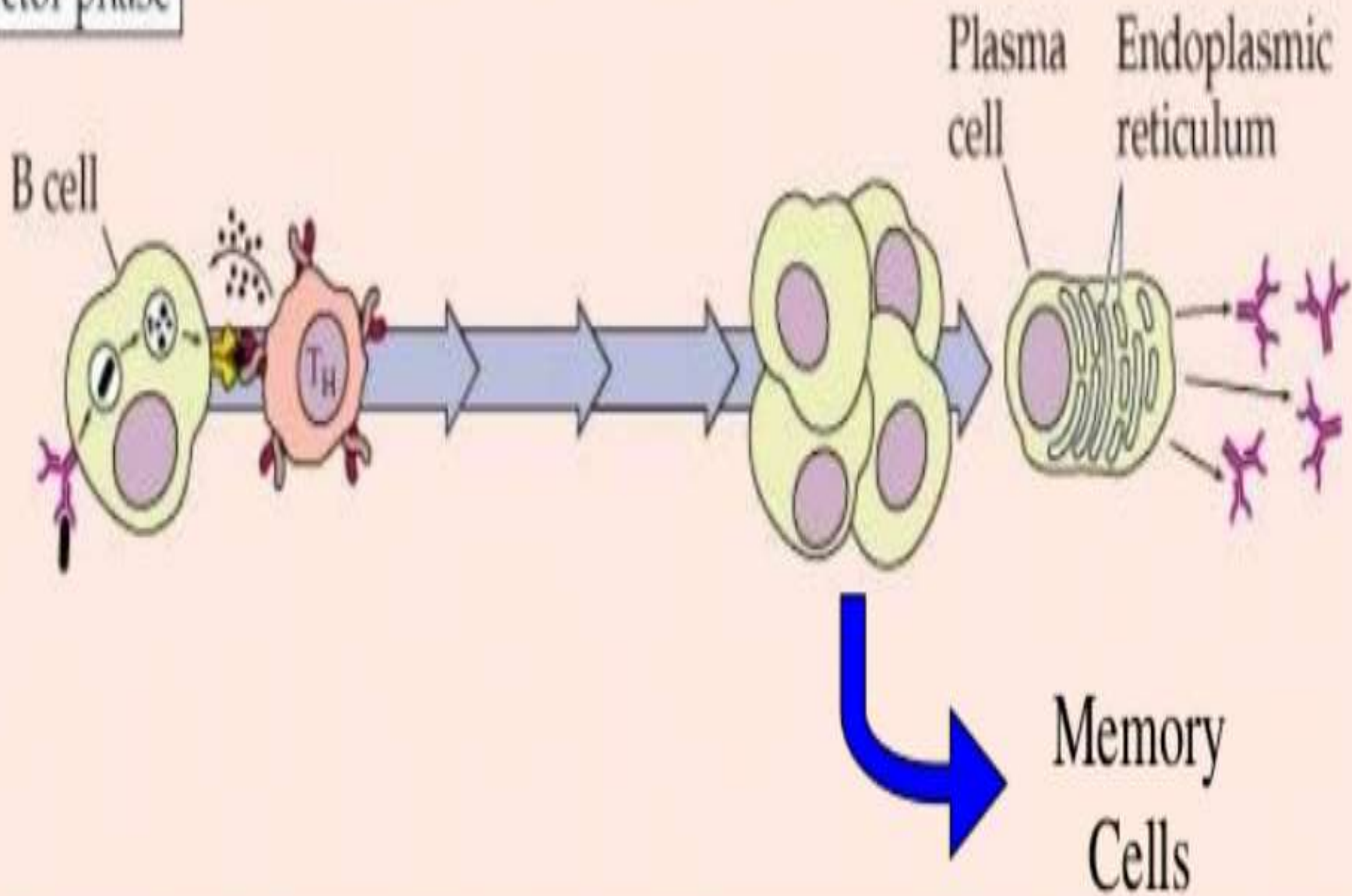


HUMORAL RESPONSE

Activation phase



Effector phase



➤ Functions and Properties of Antibody

- **Neutralization**

- Direct inactivation of pathogen or toxin thereby preventing its interaction with human cells

- **Opsonization**

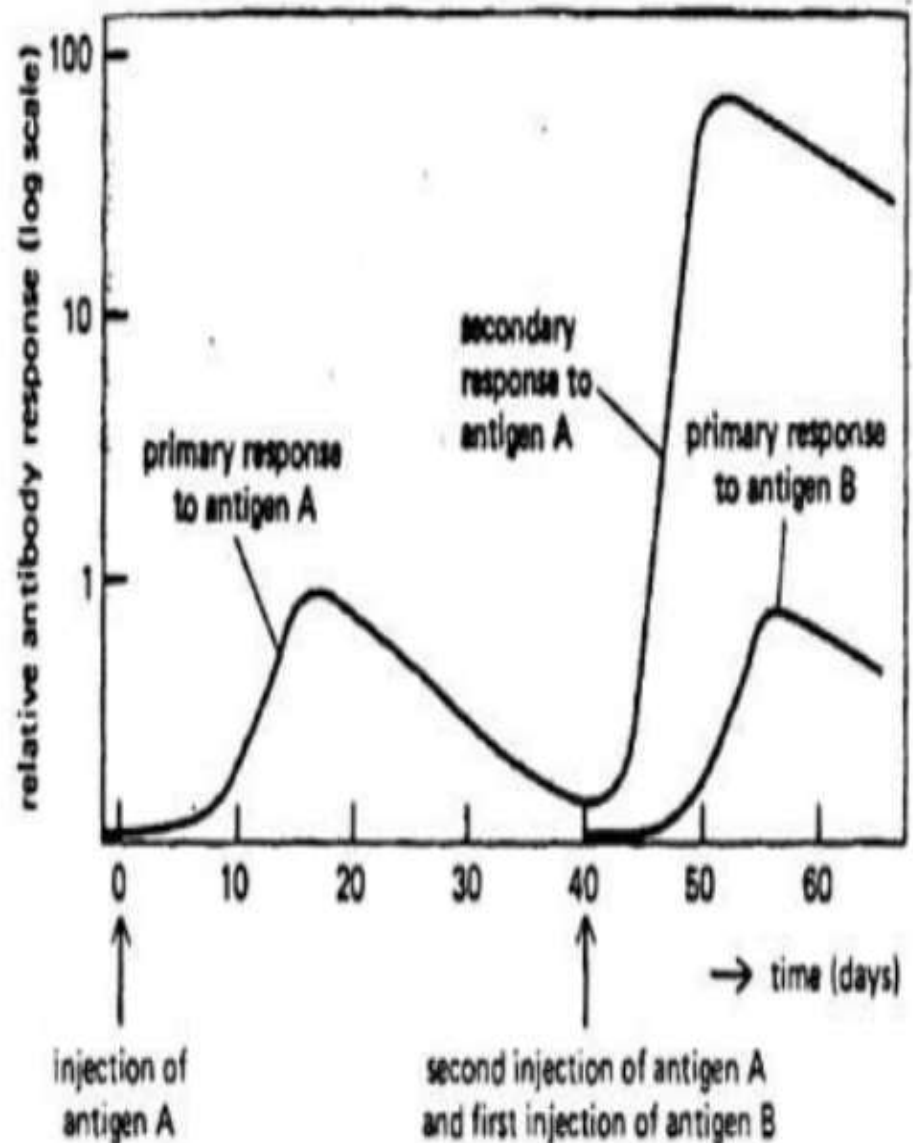
- Coating of pathogens for more efficient phagocytosis

- **Activation of complement**

- More efficient phagocytosis
- Direct killing

Dynamics of Antibody Production

- **Primary immune response**
 - Latent period
 - Gradual rise in antibody production taking days to weeks
 - Plateau reached
 - Antibody level declines



- The preparation period that is the period between entry of pathogen and expression of immune response is known as “latent period”.
- Latent period in humoral immunity indicates activation and proliferation of naïve B cells to produce plasma cells and memory cells.

The immune response curve has four phases namely:

- Lag phase:

- Antibodies are almost absent.
- Duration of this period varies from several hours to days depending on many factors such as type and amount of antigen given, route of administration of antigen, species and health of the animal to which the antigen is injected and so on.
- For example, the lag phase is 2-3 weeks for Diphtheria toxoid and it is only a few hours with Pneumococcal antigen.

- Log phase: Antibodies gradually rise from zero stage to a maximum and this raising period is referred to as log phase.

- Stationary phase: After attaining maximum, antibody levels remain constant for some time and the period with constant antibody levels is referred as “plateau phase” .

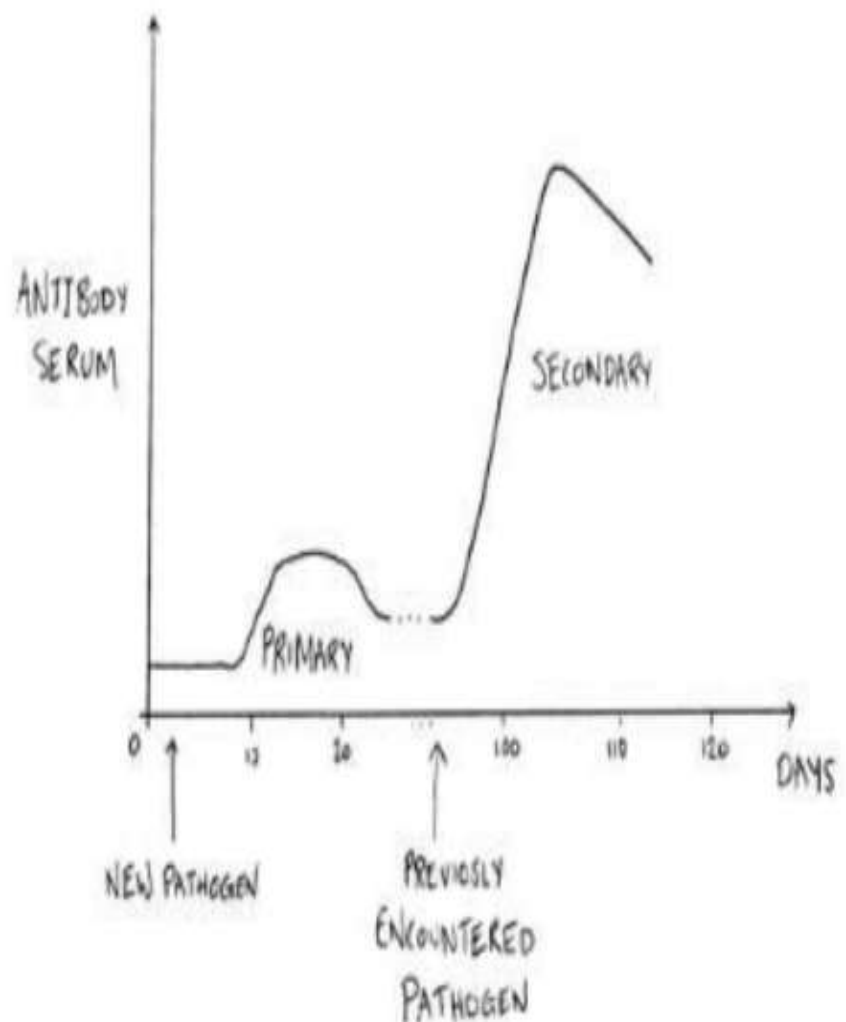
- Decline phase:
 - indicates reduction of antibodies.

- Presence of antigens is necessary for the production of plasma cells and since the antigens are removed by the action of antibodies B cells failed to get stimulation from antigens and stops producing plasma cells.

- Drop in the formation of plasma cells producing antibodies leads to reduced antibodies level in the decline phase.

Secondary Response

- Second exposure to SAME antigen.
- Memory cells are a beautiful thing.
- Recognition of antigen is immediate.
- Results in immediate production of protective antibody, mainly IgG but may see some IgM



THANK YOU