IMMUNE RESPONSE TO INFECTIOUS DISEASES

Immune Response to Bacterial Infection

Characteristics of Bacteria

① X

② X

③ X

④ X

⑤ X

⑥ X

⑦ X

Bacterial Diseases

Any part of the body can be infected by many Infectious agents and many bacterial diseases and causing a disease due to:

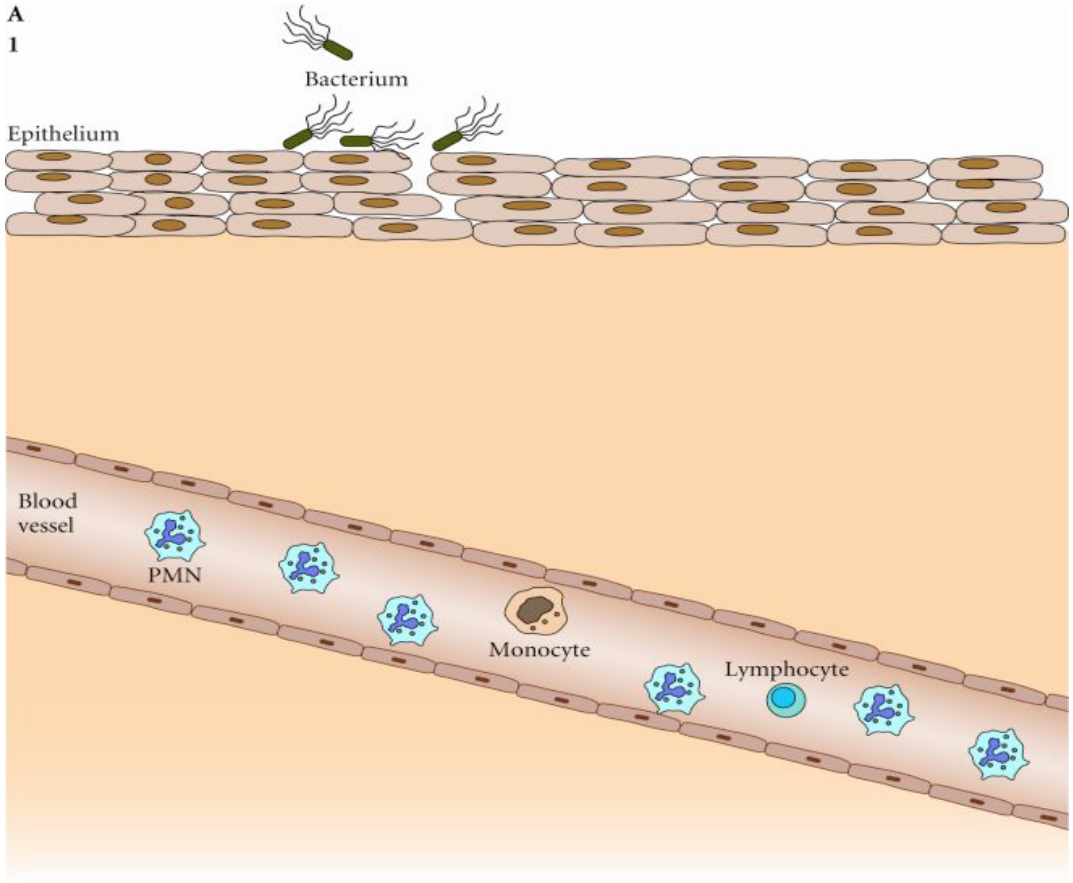
① Growth of the microbe in a tissue
② Produce Bacterial factors that are harmful to host
③ Elicite an inflammatory response that causes damage

But also leads to acquired immunity

Steps of Bacterial Infection

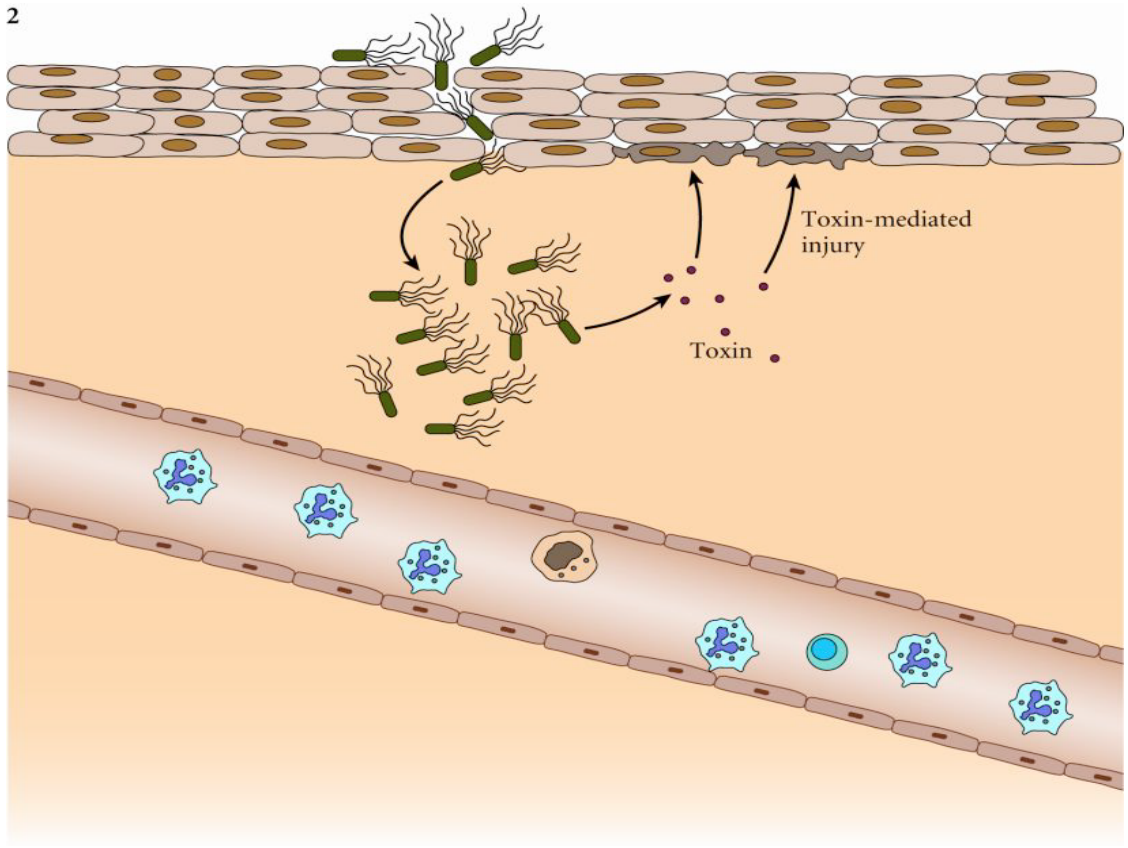
① Attachment of bacterium to host tissue

- Persistence and growth called colonization



Steps of Bacterial Infection 2

② Invasion into deeper host tissues and production of toxins and leads to injury to host cell and tissue

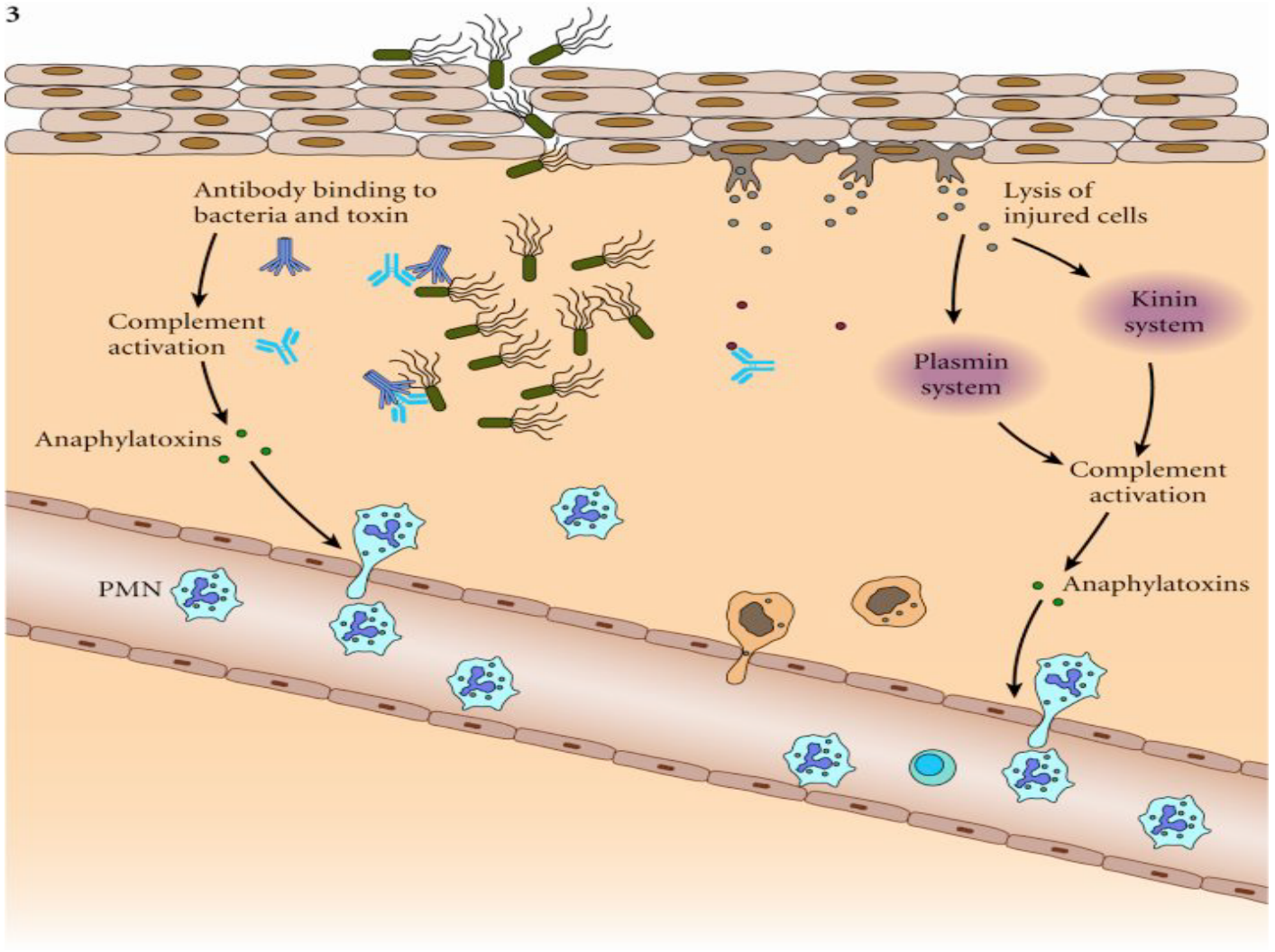


Steps of Bacterial Infection 3

③ Inflammation at site of invasion

- Initiated by antibody binding to bacterium
- Initiated by complement activation at bacterial surface
- Initiated by wound healing mechanisms
- All can activate complement pathways that alters vascular permeability and activates local macrophage and neutrophils (PMNs)

Inflammation at site of invasion



Immunity to bacteria

The defence mechanisms used depend on:-

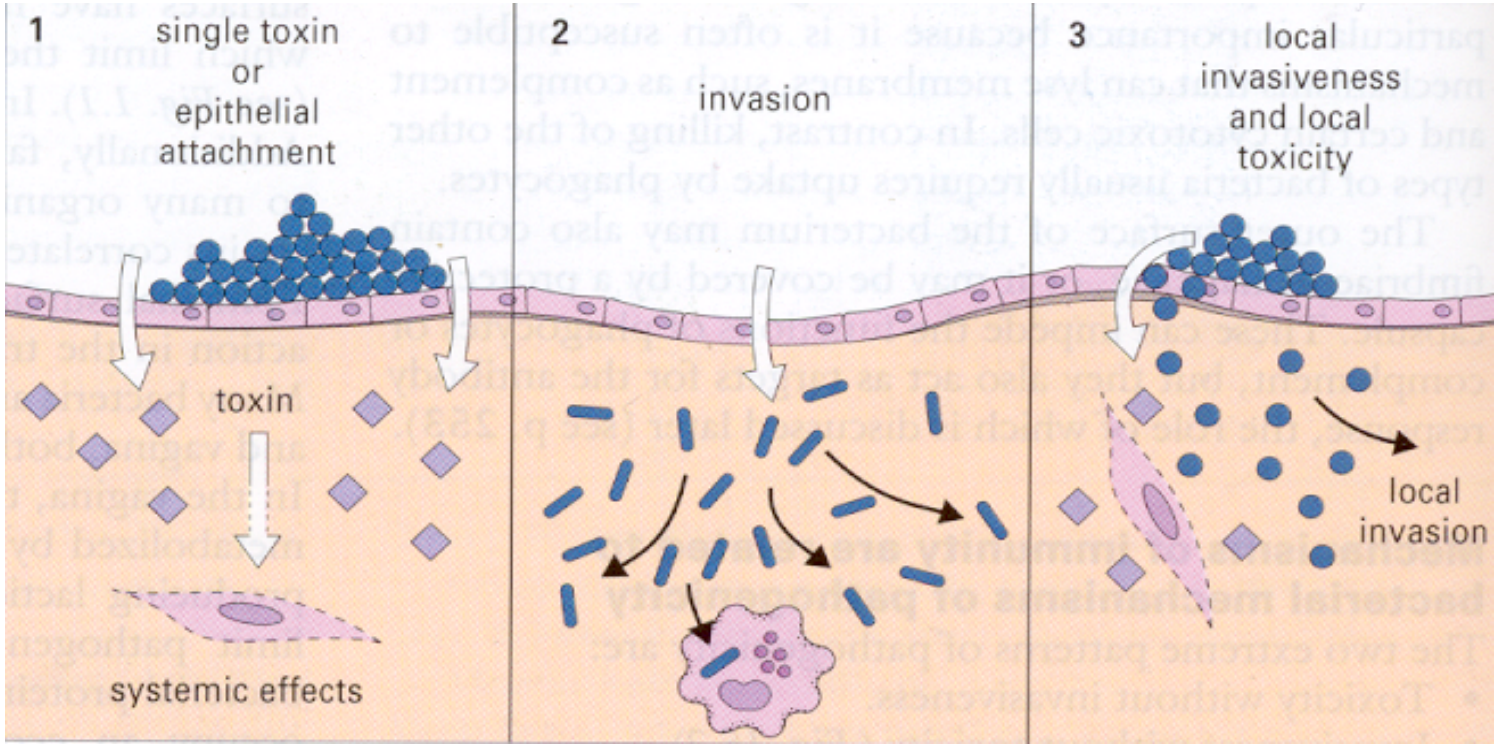
- Site of infection
- Structure of the invading bacteria
- How they cause damage
- Intracellular vs. extracellular location

How Can Bacteria Cause Damage?

Toxin production

Tissue invasion

Both



Clostridium tetani

Mycobacteria

Most bacteria

Immunity to bacteria

- Innate and adaptive immune systems work together.
- In general, the innate response is important in preventing an infection becoming established, the adaptive subsequently in combating an established infection.

Innate immune system can recognise and respond to common bacterial components

① X
② X
③ X
④ X
⑤ X
⑥ X
⑦ X

Innate Immune Effectors and Bacterial Infection

Table 18.1 Innate immune effectors mediating resistance to bacterial infection

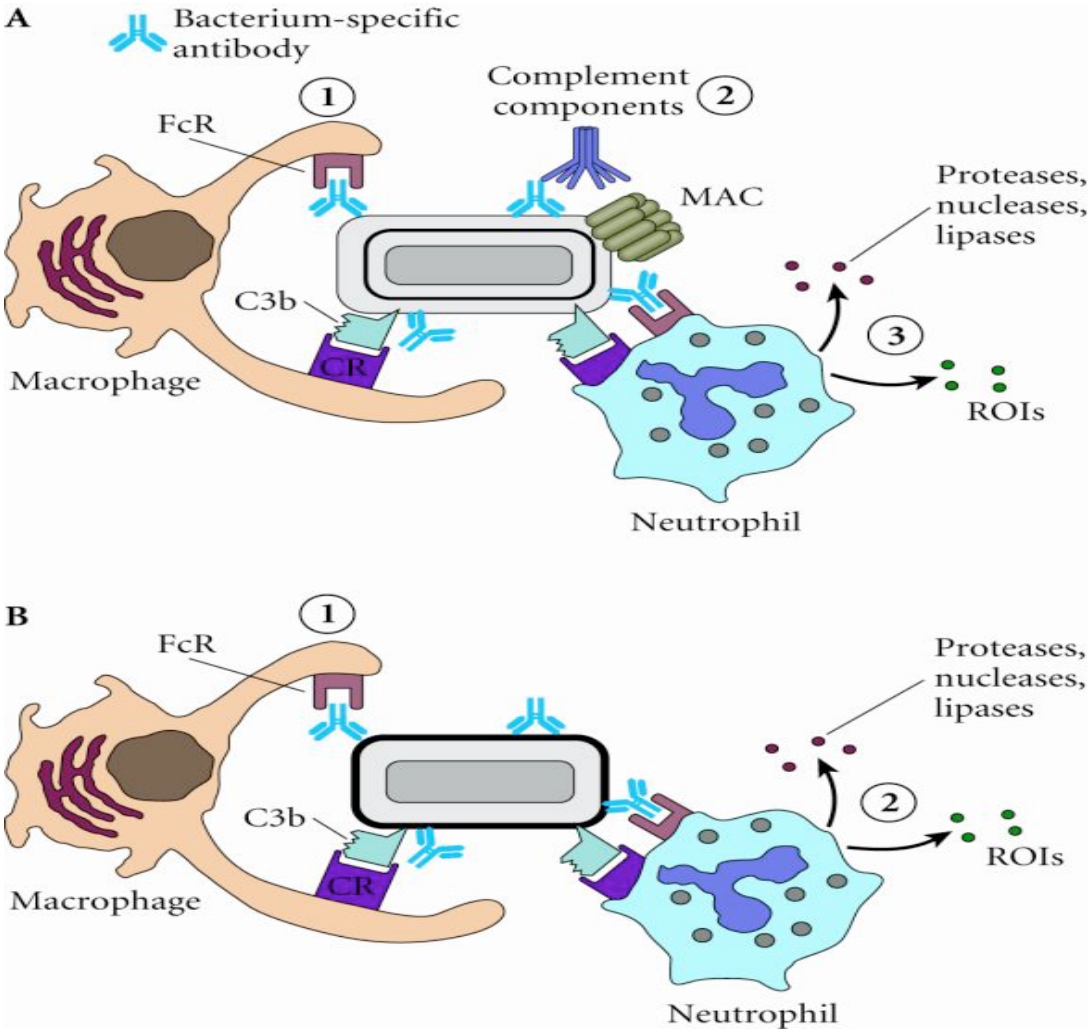
Type of mechanism and examples
Physical and physiologic barriers
Skin, sebum, mucosal epithelial cells, mucus, and mucous flow
Enzymatic and protein effectors
Lysozyme, proteases, antimicrobial peptides, iron-sequestering proteins, complement
Recognition of PAMP
TLRs
TLR2 and peptidoglycan and glycopeptides
TLR4 and LPS
TLR5 and bacterial flagella
TLR9 and CpG DNA
Endocytic pattern recognition molecules
Mannose receptor/scavenger protein
CR3
Soluble collectins
Conglutinin
Mannose-binding lectin
Surfactant proteins A and D

Adaptive Immunity:

I. Antibody mediated response

Action	How
Block adhesion	Ab to surface proteins
Block proliferation	Ab to surface receptors eg. iron
Cause phagocytosis	Bind bacterial surface and allow phagocytes to bind bacteria
Lysis, phagocytosis and inflammation	Activate complement
Neutralise toxic products	Bind toxins
Prevent tissue invasion and damage	Neutralise bacterial products and proteases

Immunity to extracellular bacteria by antibodies:



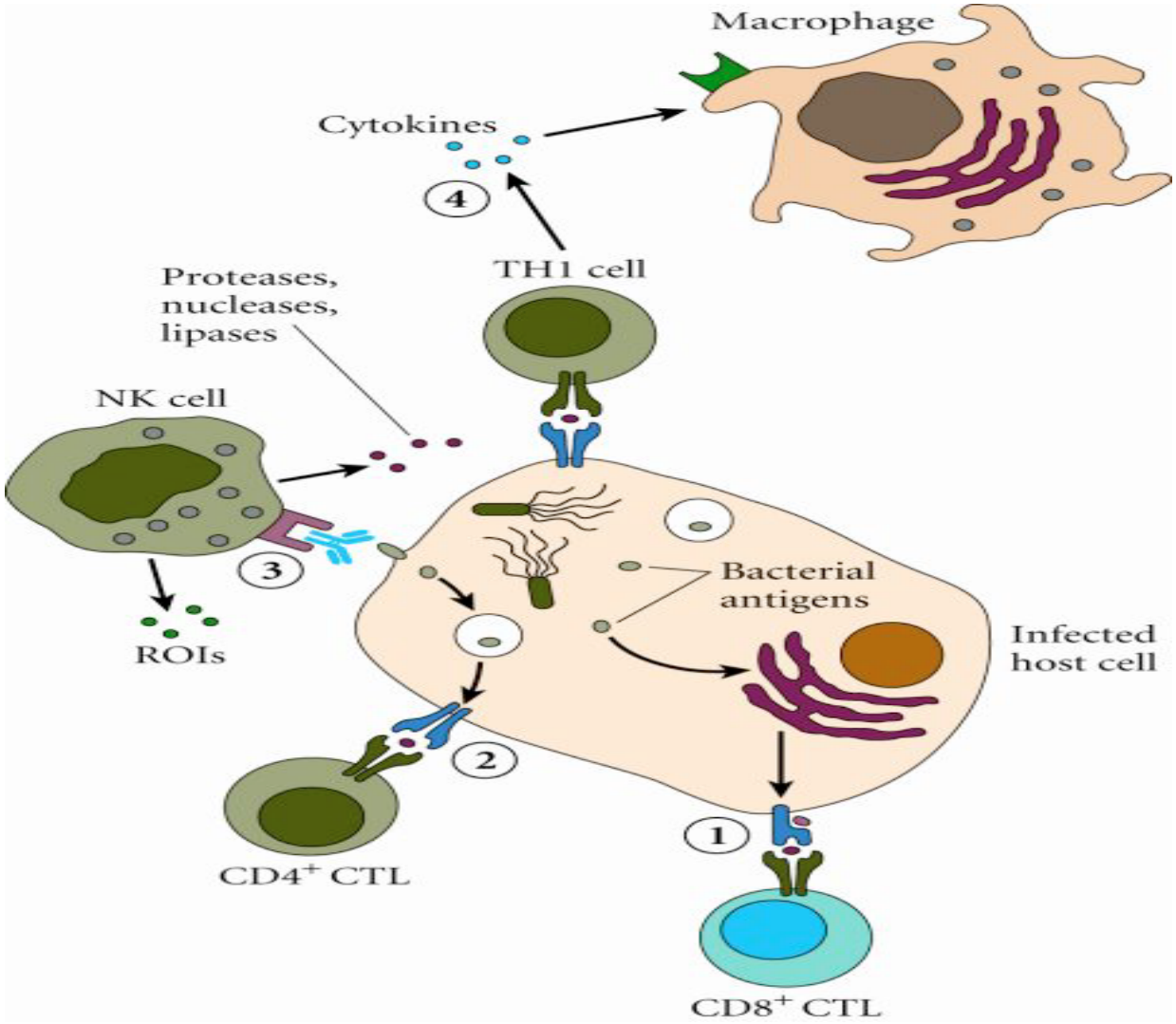
Immunity to extracellular bacteria by antibodies:

①**Antibodies and complement** result in opsonisation via FcR or CR on MØ and PMN.

②**Antibody** can activate classical complement pathway resulting MAC formation and opsonisation.

③**Antibodies** can also trigger antibody-dependent cell mediated cytotoxicity (ADCC) by PMN with FcR and CR and that leads to release proteases, nucleases, lipases.

Mechanisms of Immunity to intracellular bacteria by CMI



II. Cell mediated immunity vs intracellular bacteria

1. CD8+ T cells (Tc) cytotoxic T lymphocytes (CTLs).
2. CD4+ helper T cells (T_H1).
3. NK cells using ADCC

Do T Lymphocytes Have a Role in the Response to Bacteria?

① **T cytotoxic lymphocytes** can kill infected cells

(intracellular pathogens eg Mycobacterium tuberculosis)

② **T helper cells** produce cytokines or interleukins

A. more **antibody production**
B. **Interferon γ** activates macrophages and increases bacterial killing, increases production of IgG used by phagocytes, increases cytotoxicity and CD8 T cell activity

How bacteria evade Host Immunity?

① Avoiding antibody
② Prevent phagocytosis
③ Avoiding complement
④ Down-regulate the expression of MHC-I and MHC-II.
⑤ Impede the function of lymphocytes and granulocytes (toxin ,enzyme and proteins).
⑥ Overstimulate inflammation