Host Defenses Systems

#1

- **Innate Immunity (Non-specific)**
  - **Physical Barriers**
    - e.g. skin & mucous membranes
    - * book 52-53 *
  - **Phagocytic Cells & Inflammation** #2-#4 (cellular)
    - Neutrophils
    - Macrophages
    - NK Cells (lymphocytes)
      - slides
      - for virally-infected & tumor cells
  - **Proteins** (Humoral) "soluble"
    - Complement chapter 63
    - Lysosome
    - Interferon slides
- **Acquired Immunity (specific)** #4 & #5
  - **Active**
    - Primary
    - Secondary
  - **Passive**

White Blood Cells:

- PMNs & Monocytes ⇒ phagocytes
- Lymphocytes
  - B lymphocytes → produce antibodies (humoral)
  - T lymphocytes → cell-mediated responces.
  - Natural Killer Cells → against viral infections & tumor cells (non-specific)
- Eosinophils → Parasitic infections
- Basophils ⇒ Allergic infections

→ Granulocytes

Inflammatory Responce & Phagocytosis..

Levinson: 54-56 #2

* Presence of foreign bodies * lead to → Inflammatory Responce characterized by

- Redness
- Swelling
- Warmth
- Pain * Bradykinin * mediator of pain

Swelling & Warmth ⇝ due to ↑ capillary permeability & ↑ Blood flow

Complement components: C_{3a} & C_{5a} contribute to ↑ capillary permeability & ↑ Blood flow

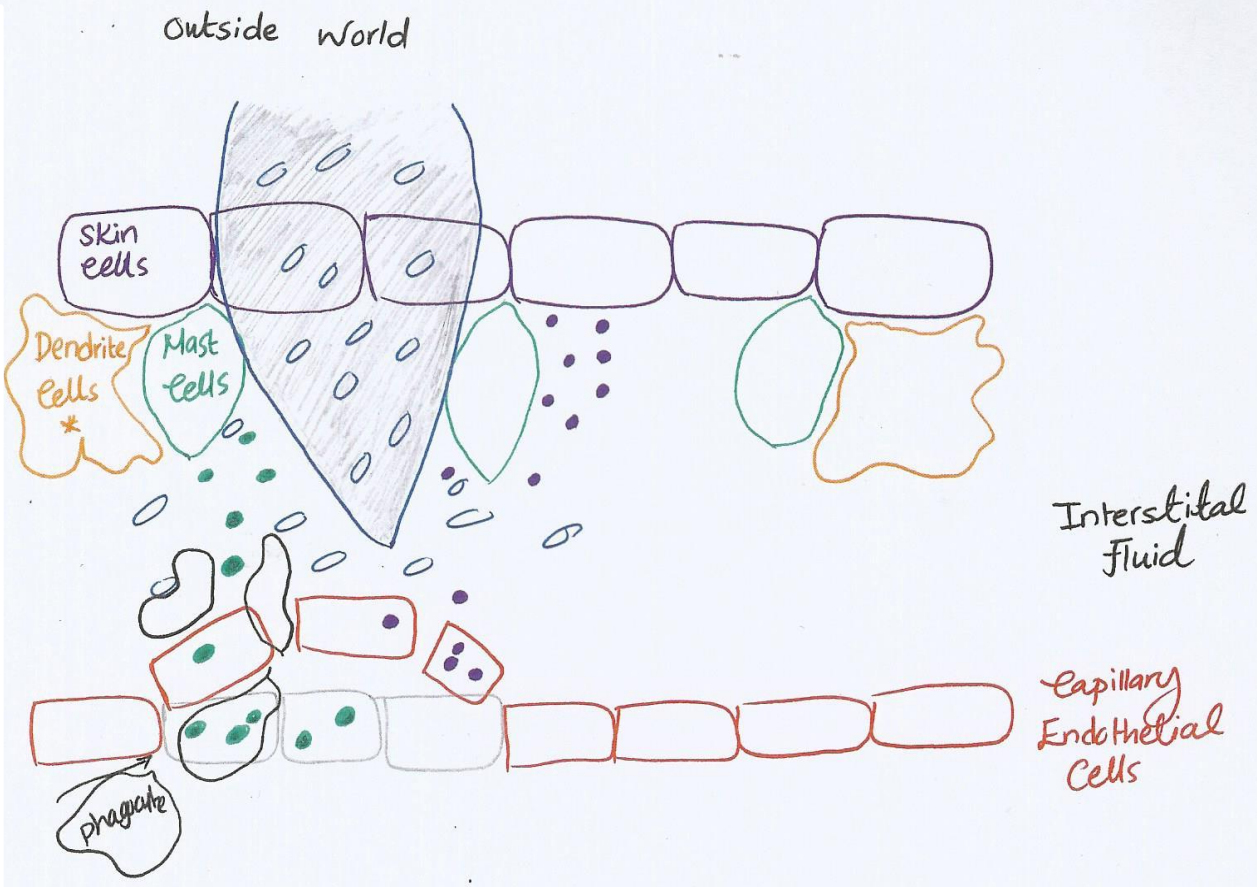
↑ capillary permeability & ↑ Blood flow ⇝ in responce to chemical mediators: Histamine, prostaglandins, & leukotrienes.

* Neutrophils & Macrophages ⇒ part of inflammatory responce.
  - ⇓ in acute pyogenic infections.
  - ⇓ in chronic or granulomatous infections.

* Macrophages ⇒ 1- phagocytic
  2- produce cytokines → tumur necrosis factor (TNF)
  → Interleukin-1 (IL-1) synthesized from precursor mediated by proteolytic enzymes in the inflammasome. (found in cytoplasm)

* Inflammatory Responce Process:-

* Dendrite cells activate T helper cells. #3



* when Bacteria invade our body (e.g with the help of a nail), they spread in the ISF.

* Skin cells start to release chemokines (chemical protein molecules released for signaling purposes) & to attract WBCs.

Neutrophils ⇒ Interleukin-8

Macrophages ⇒ Monocyte chemotatic protein-1 (MCP-1) & Macrophage inflammatory protein (MIP)

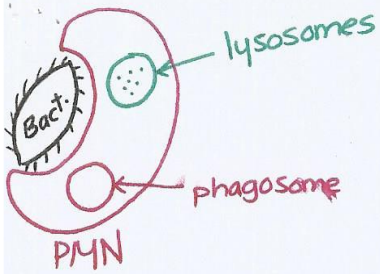
* Mast cells gets activated by direct contact w/nail, chemokines or Bacteria & then they will release Histamine which will go to the Endothelial cells of the capillary & make them further apart * Vasodilation *

* This will allow the phagocytes to reach & Kill the Bacteria by Phagocytosis:-

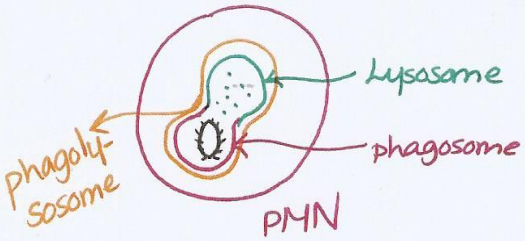
① Migration (aka diapedesis) ⇒ they migrate to the infection site due to production of chemokines.

② Ingestion

③ Killing (see next page)



the PMN cell membrane invaginate the Bacteria to form a phagosome (enhanced by binding IgG & C_{3b})



the lysosome fuses with the phagosome & it releases its components by degranulation.

* Fever :-

Infection —cause→ ↑. body Temp. —due to→ presence of endogenous pyrogen (IL-1) —released from→ Macrophages.

- Fever may be protective as: many viruses & Bacteria grow ↓ (slowly) when the Temp. is ↑.

Adaptive (Specific) Immunity

- Active: exposure to organism
  - Primary
  - Secondary
- Passive: performed Ab from another host

* Comparision between Active & Passive immunity:-

	Active	Passive
Definition	protection based on exposure to the organism in disease/subclinical infection.	temporary protection against an organism & is acquired by recieving serum containing antibodies
Example	vaccination ...	through placenta (IgG) or breast milk (IgA)
Duration	- protective abilities are delayed (few day - few weeks) - [Ab] ↑ duration	- protective abilities are present immediately. - [Ab] ↓ duration as they get degraded ⇝ protection is only 1/2 month.
Types of Responces	* Primary: takes 7 to 10 days for the Ab to become detectable. * Secondary: rapid (3 day) responce to an antigen that was exposed previously.	—
Mediated by	Antibodies & T cells. ↓ cytotoxic destruction of infected cells.	