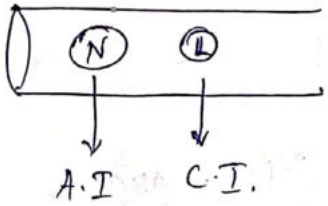
* Characterized by lymphocytes & plasma cells in Tissues.

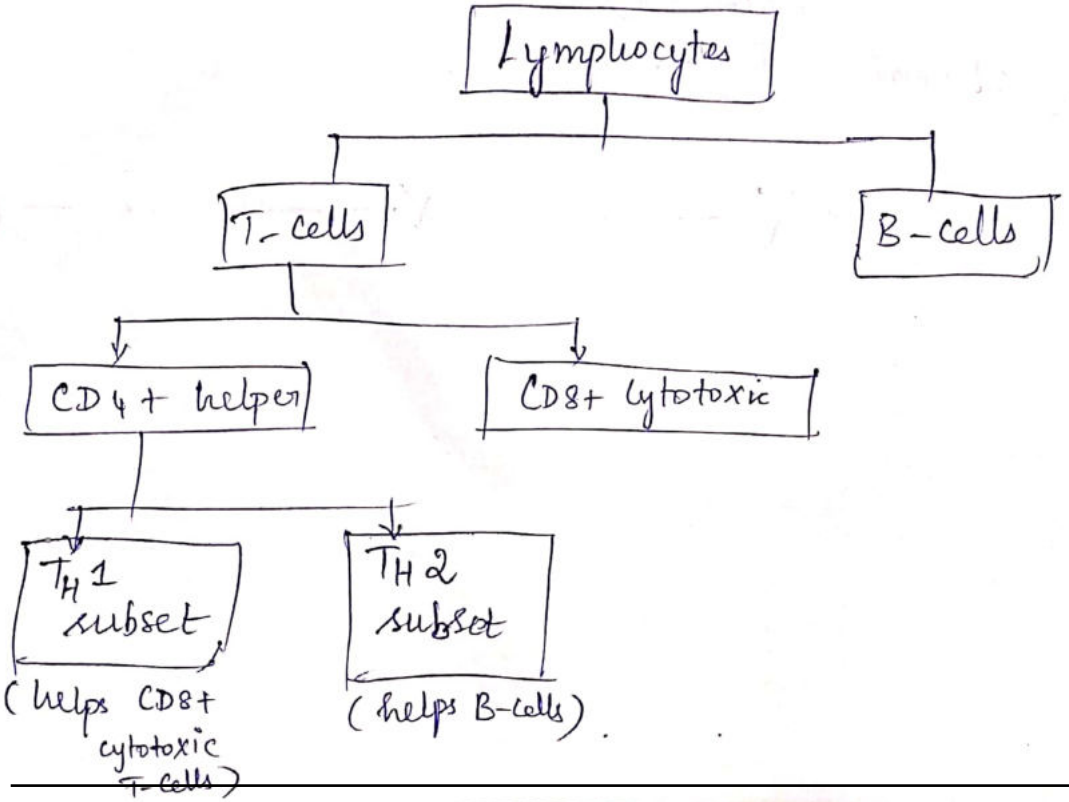
* Delayed response but More specific (Adaptive immunity).



* Lymphocytes → mononuclear (whereas Neutrophils are ....)
* plasma cells → " ⊕ glassy shiny pink cytoplasm

⊕ Stimuli for Chronic inflamm^n (compare with Acute infl.)

(i) Persistent infection (m/c).
(ii) infecn with viruses, mycobacteria, parasites & fungi.
(iii) Autoimmune ds.
(iv) foreign material
(v) Some Cancers.



* produced in Bone marrow ~~from~~ as progenitor Tcells

↓

further development in Thymus

↓

TCR undergoes rearrangement

↓

CD 4+ helper T-cells

CD 8+ cytotoxic T-cells

TCR + CD 3 = TCR complex

→ T-cells use this Ag surveillence.

→ They can recognize Ag only when Ag are present along with MHC.

⇓

→ CD(4)+ T-cells —recog. Ag when present along with→ MHC class (II) (4 x 2 = 8)

CD(8)+ T-cells → MHC class (I) (8 x 1 = 8).

Mnemonic.

requires

(i) Binding of Ag-MHC Complex

(ii) Additional 2nd signal

CD 4+ T-cell activation:

* Extra cellular Ag is phagocytosed, processed by macrophages (APC) and presented via MHC class II } 1st signal

* Also B7 of APC bind with ... CD28 of CD4+ cells. (7 × 4 = 28) } 2nd signal

What will activated CD 4+ cells do?

* secrete "cytokines" that "help" inflammation

TH1 subset

* helps CD8+ Tcells
* secretes
  (i) IL-2 → T-cell growth factor
  → CD8+ T-cell activator
  (ii) IFN-γ → macrophage activator. Granuloma formation

TH2 subset

* helps B-cells.

secretes

(i) IL-4 (class switching to IgG & IgE)

(ii) IL-5 → eosinophil chemotaxis
→ B-cells → plasma cells
→ class switching to IgA

(iii) IL-10 (⊖ TH1 phenotype)

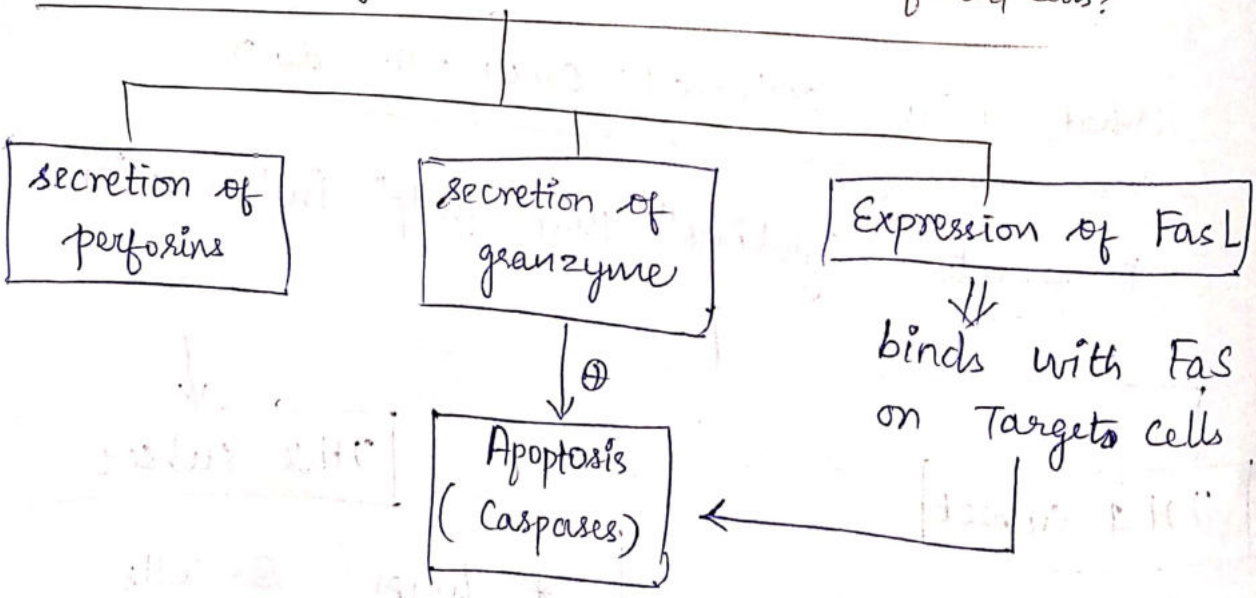
* CD8+ cells recognizes intracellular antigens after processing when presented on MHC-I. } 1st signal

* IL2 from CD4+ T_H1 cells } 2nd signal.

Note: Why intracellular Ag.?

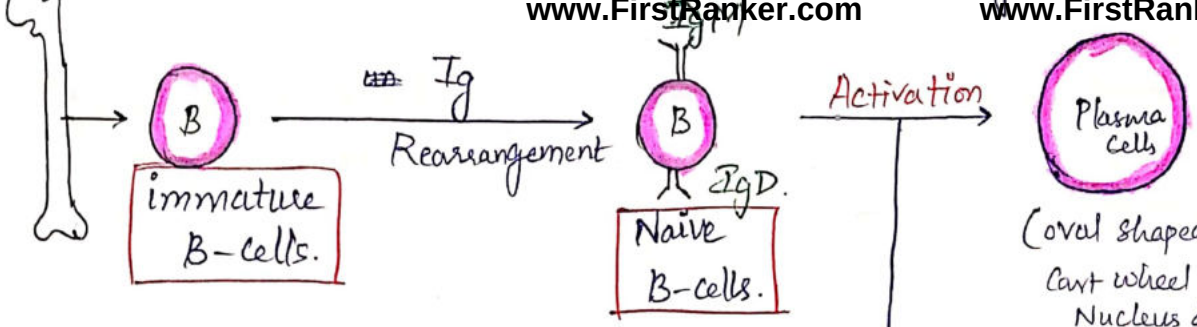
* Virus infected cells produce viral proteins intracellularly which can't processed by APC.

How CD8+ Cytotoxic T-cells kill infected cells?



(R) - Receptor
(L) - Ligand

Refer Hyper IgM syn. ↳ Pg 97.



② methods of Activation.

(i) Ag binding by surface Ig M (or) Ig D

↓

Now plasma cells can produce -IgM-

After internalizing Ag,

(ii) B-cell Ag presentat to CD4+ Helper T-cells via MHC II (1st signal) & CD 40 (R) on B-cell binds CD 40 (L) on Helper T-cell (2nd signal)

⇐

These ② signals activate ~~B-cell~~ CD4+ Helper T-cell which in turn activates B-cell. to become plasma cell.

↳ Now plasma cells can produce Ig A, G, E

In short:

B-cell ⊕ activates → T-helper cells secrete → IL-④ & IL-⑤

⇓

(i) B-cell isotype switching
(ii) Hypermaturation
(iii) B cells → plasma Cells.

⇐

IgM (or) Ig D secreting B-cell.

↓

Ig A, IgG, IgE secreting B-cell.

Granulomatous inflammatn.

Non Granulomatous inflammatn.

(i) Non caseating subtype (No central Necrosis in granuloma).

(ii) Caseating subtype (central Necrosis in granuloma).

Granulomatous inflammn.

* chacacterized by Granuloma
* key cell of a granuloma – epitheloid Histiocytes.
(Macrophages with Abundant pink -cytoplasm).
* These cells are surrounded by Giant cells and rim of lymphocytes

(i) NON CASEATING GRANULOMA (lack central Necrosis)

a) Reaction to foreign material
b) Sarcoidosis
c) Beryllium exposure
d) Crohn's ds
e) Cat scratch ds.

Kim → foreign material.

* Suppose a person after Breast Ca with Implant after surgery comes to the OPD, with enlarged Axillary L.N.

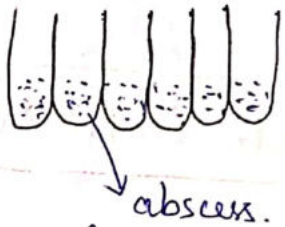
* There are (2) possibilities, (i) Spread of Ca to Axillary L.N
(ii) Leaking of foreign material into L.N.

* Leakage of foreign material produces Non caseating granuloma.

b) Sarcodosis.
c) Beryllium exposure. } → Resp. pathology

d) Crohn's ds:

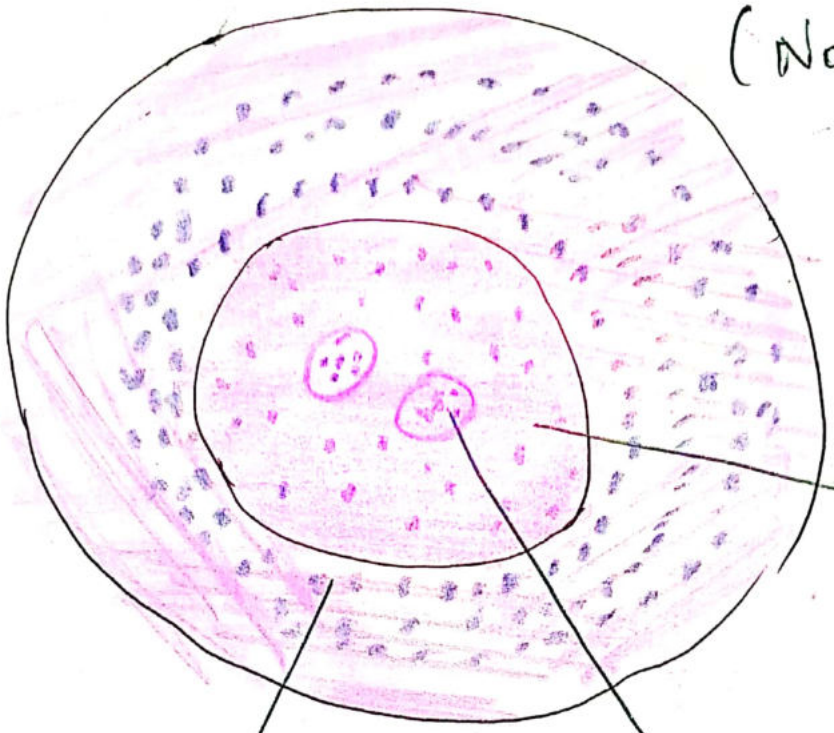
* Histological Hallmark of
(i) Crohn's ds → Non caseating granuloma
(ii) Ulcerative Colitis → Crypt abscess.



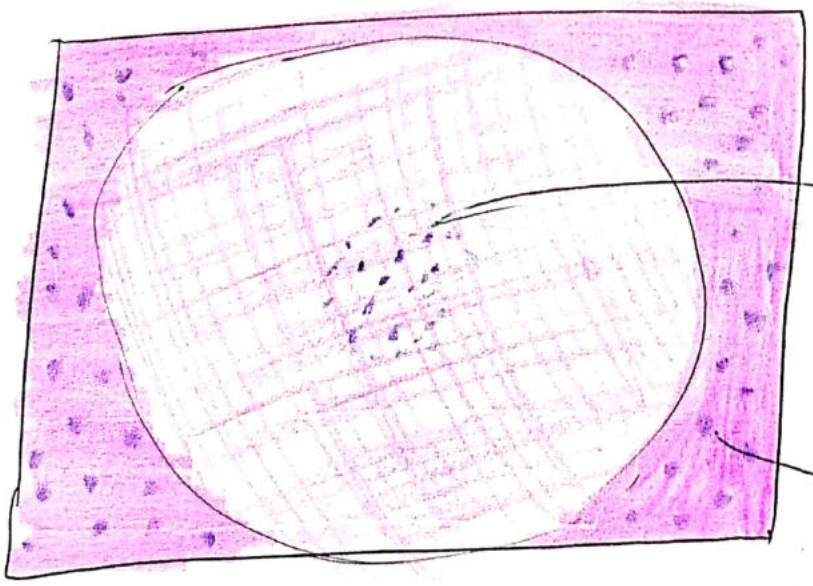
e) Cat scratch ds:

* geanuloma in Neck region.
* ☆ Stellate shaped geanuloma

Non caseating granuloma



Caseating granuloma



Caseating Granuloma

Characteristic

TB

Stain used for Δ^{sis}

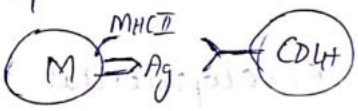
AFB.

Fungal infections

Stain: GMS.
(silver stain).

STEPS INVOLVED GRANULOMA FORMATION

Step①: Macrophages present Ag via MHC II to activate CD4+ Helper T-cells.



Step②:

Macrophages secretes IL-12

CD4+ Helper T-cells → IL-12 → TH1 subtype

Step③:

TH1 cells. secrete IFN-γ

"secretes"

macrophages → IFN-γ → epithelioid histiocytes & Giant cells