

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

رَبِّ زِدْنِي عِلْمًا

اللَّهُمَّ أَرِنِي حَقِيقَةَ الْأَشْيَاءِ كَمَا هِيَ

“O Allah! Show me the reality of all things as it (really) is..”

IMMUNITY

Tayyab Hamid (MBBS, PhD)

LECTURER in Physiology

HEMATOPOIESIS

```
graph TD; A[HEMATOPOIESIS] --> B[LEUKOPOIESIS]; B --> C[Myelopoiesis<br/>(IL-1,3,GM-CSF, N-CSF)]; B --> D[Lymphopoiesis<br/>(IL-2,7,12,15)]; C --> E[Granulocytes]; C --> F[Monocytes]; D --> G[Lymphocytes];
```

LEUKOPOIESIS

Myelopoiesis

(IL-1,3,GM-CSF, N-CSF)

Granulocytes

Monocytes

Lymphopoiesis

(IL-2,7,12,15)

Lymphocytes

Why One has to wait for 48 to 72 Hrs after PPD skin test?

A 3 month old brought to ER with recurrent infections. Her brothers also died from disseminated mycobacterial infection during infancy?

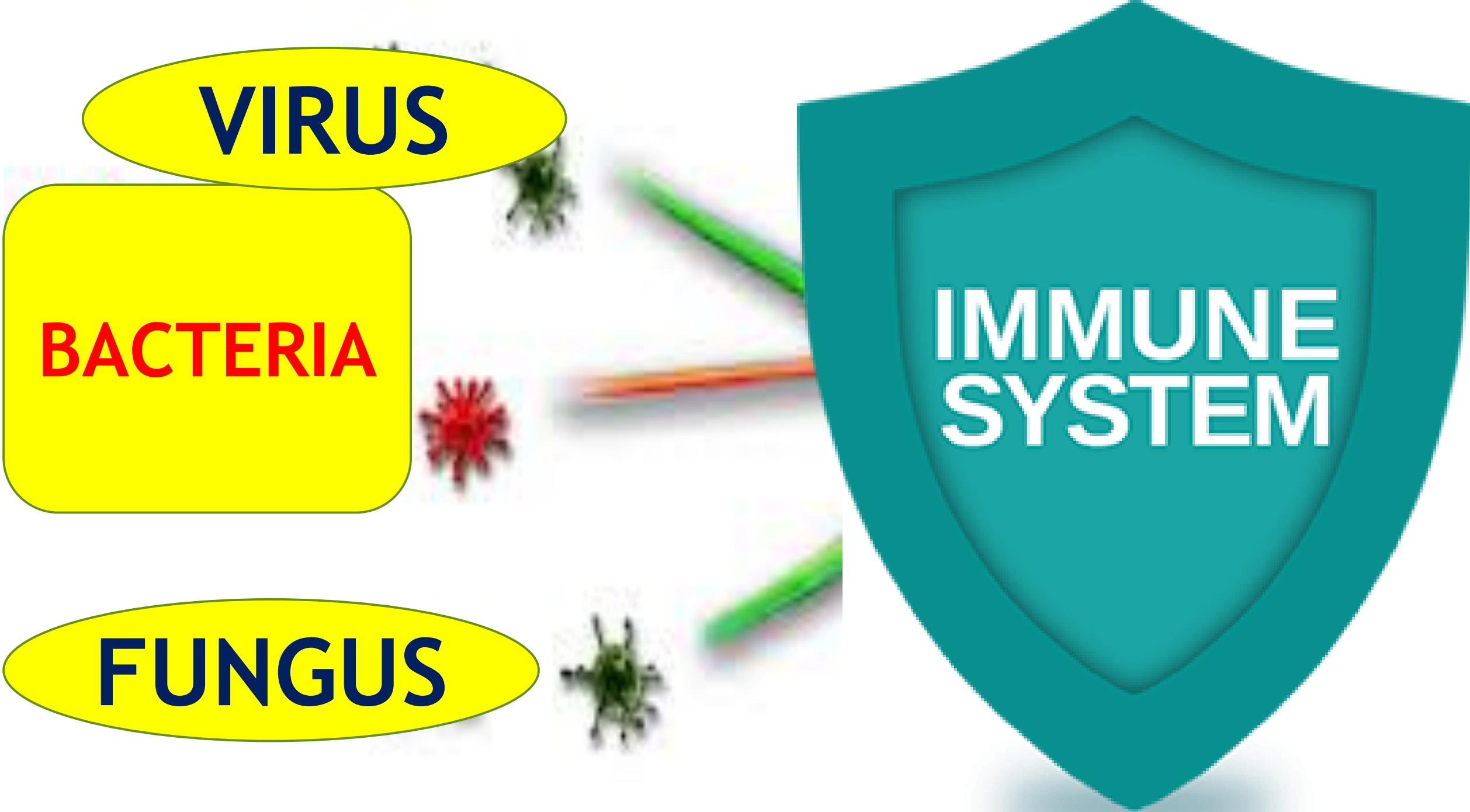
APCs show less interaction with T-lymphocyte. Observed effect due to less expression of which molecules?

VIRUS

BACTERIA

FUNGUS

**IMMUNE
SYSTEM**



1. BARRIERS
2. INFLAMMATORY CELLS
3. NATURAL KILLER CELLS

Innate
Immunity

Adaptive
Immunity

1. Humoral Immunity
(Active , Passive)

2. CMI

Innate
Immunity

Adaptive
Immunity

TISSUE DAMAGE - CELLULAR RESPONSE

1

ENDOC

PHAG



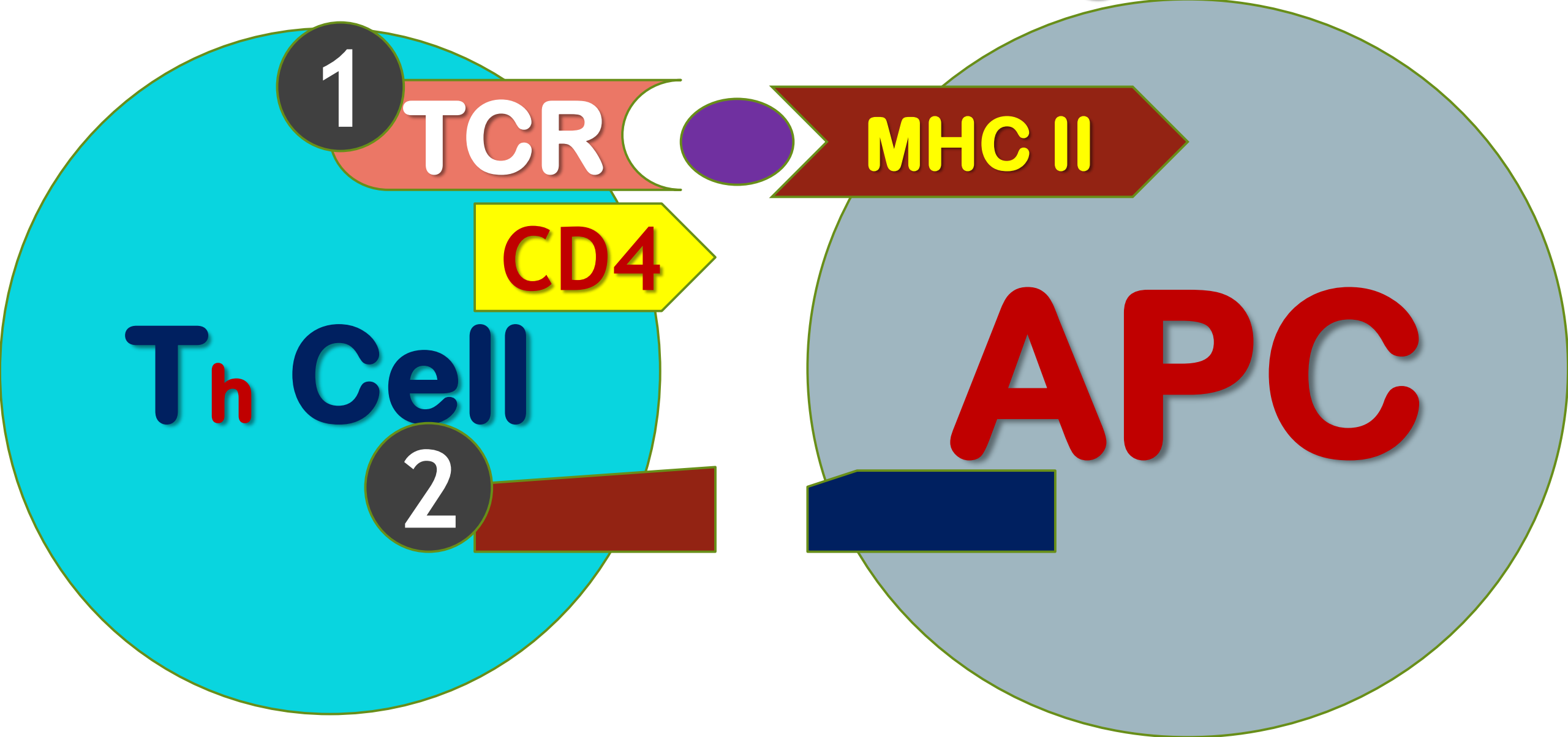
Antigen Presentation

- **what is Antigen?**
- **Which cells present Antigen?**
(**Dendritic Cells,**
Macrophages, B cells)
- **To Whom is Antigen Presented?**

T - Lymphocyte

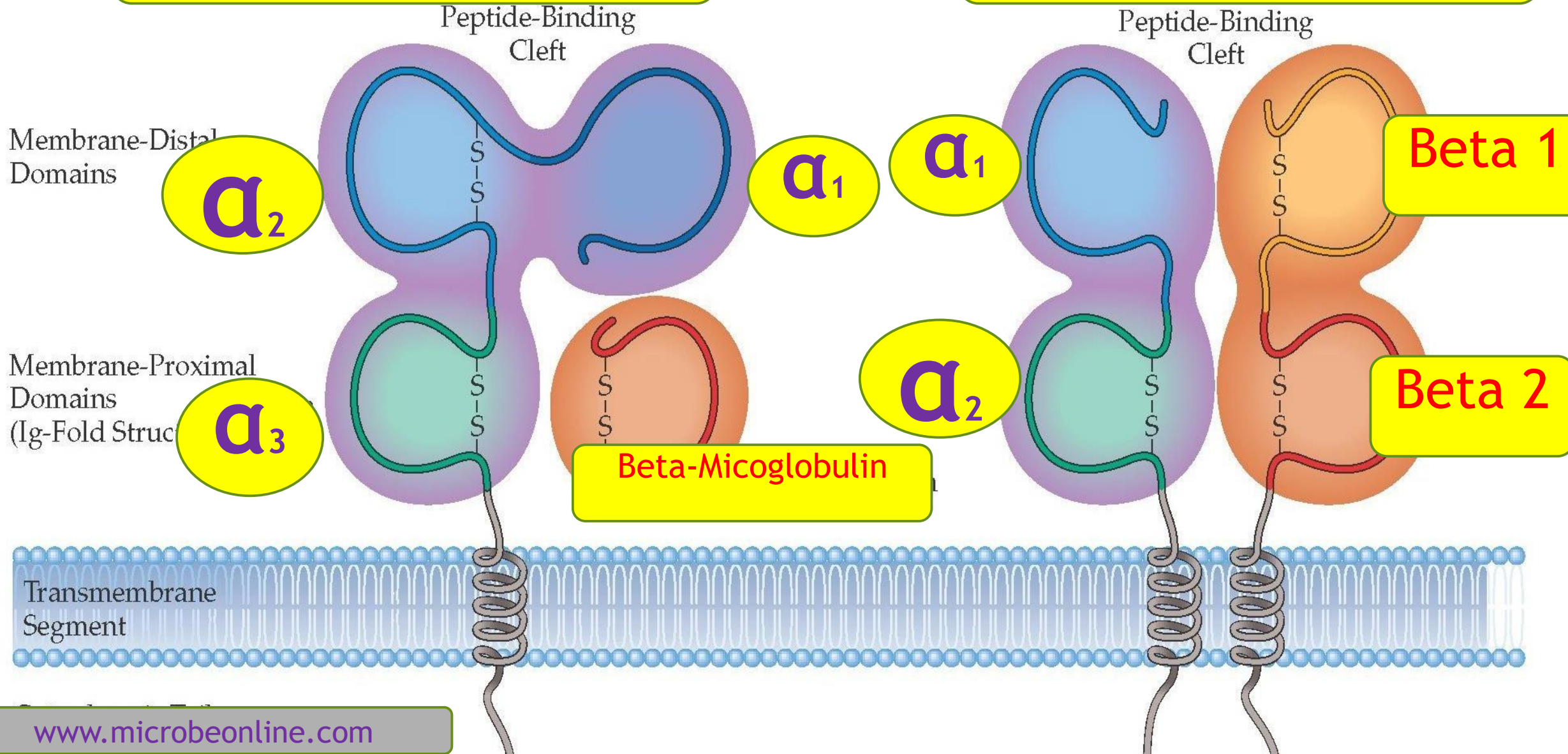
- T – cell Matures in *Thymus*, only those survive which differentiate “self from nonself”
- HelperT cells CD4+ve (Th1, Th2, Th17, Treg)
- Cytotoxic T cells CD8+ve

T CELL ACTIVAION – Two Signals

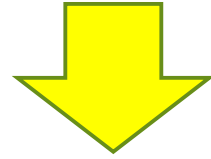


MHC Class I

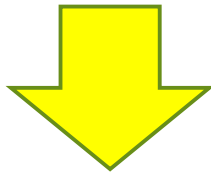
MHC Class II



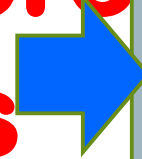
**APC activates
Helper T cell**



Th1/Th2 cell secrete cytokines

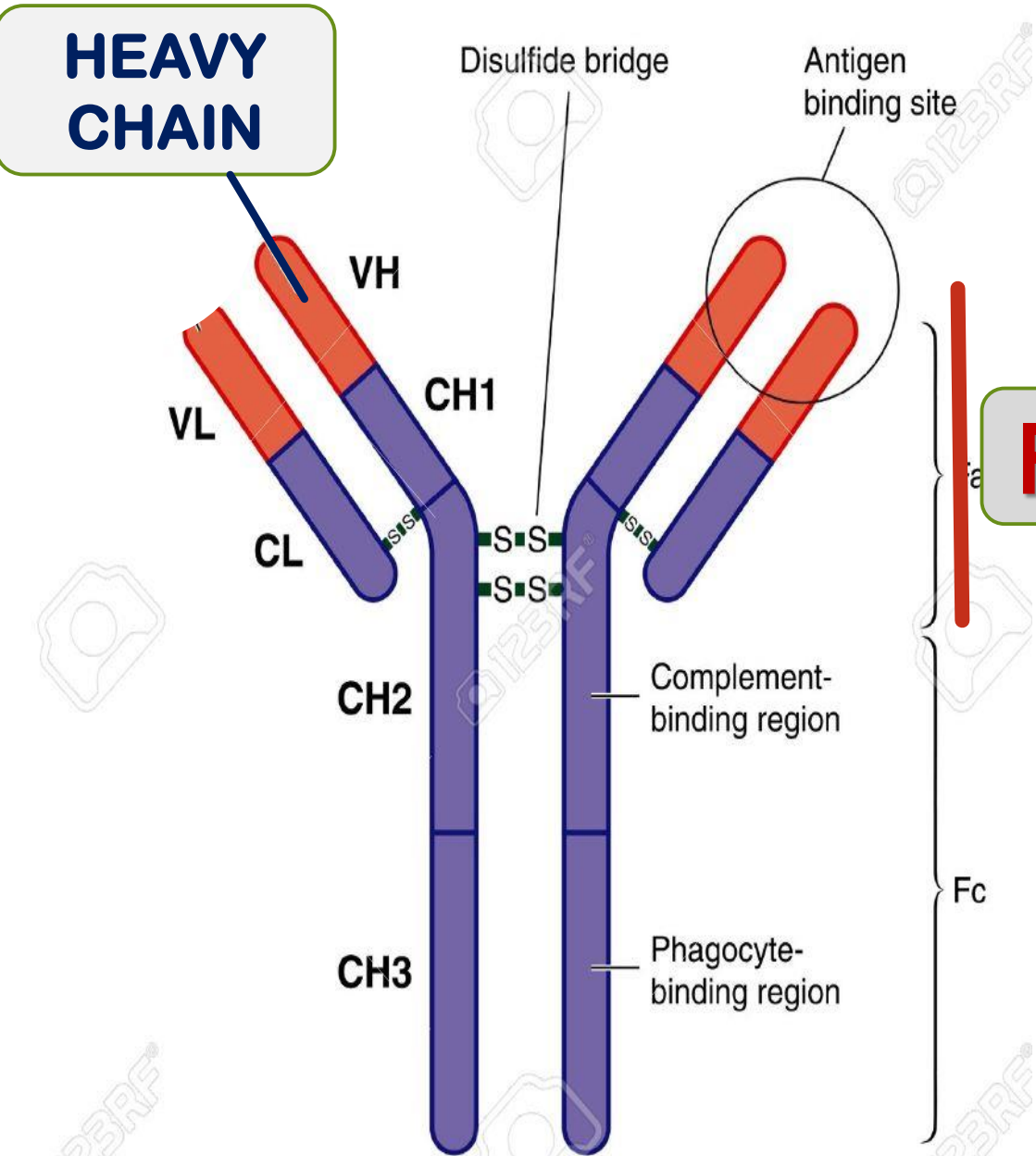


- 1. Stimulate B cells (transform into Plasma cells) to produce antibodies**
- 2. Activate Macrophages**



- **Complement Pathway Activation**
- **Opsonization**
- **Neutralization**

BLOOD GROUPS



➤ **Antibodies** are immune system-related proteins consisting of **two heavy chains & two light chains**.

➤ The chains join to form a **“Y” shaped molecule**

➤ **IgM, IgG, IgA, IgD, and IgE** are five classes

Complement Pathways

Classical
(IC me)

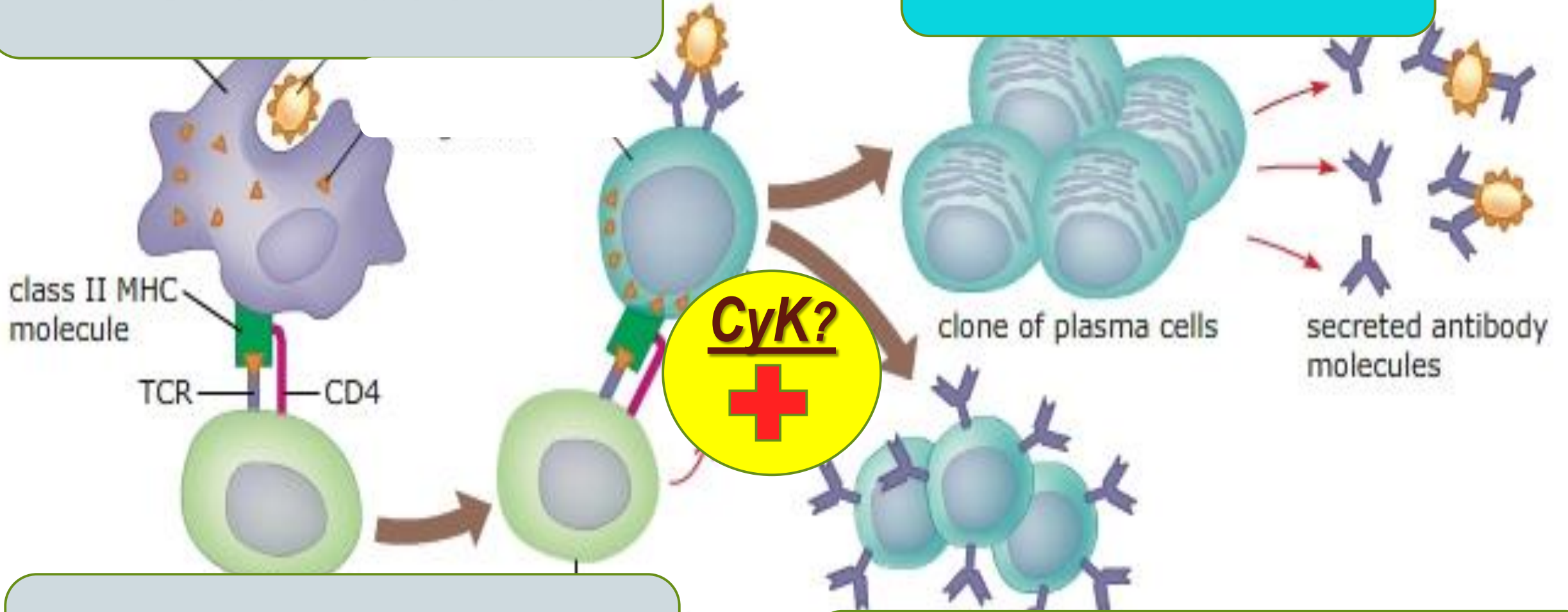
Leuc
Path

- **MAC (C5b,C6-9)**
- **Chemotaxis (C3a, C5a)**
- **Opsonization and Phagocytosis (C3b)**
- **Degranulation of mast cells (C3a, C5a)**

MAC
(C5b,
C6-9)

APC & Bacteria

PLASMA CELLS



Helper T cell

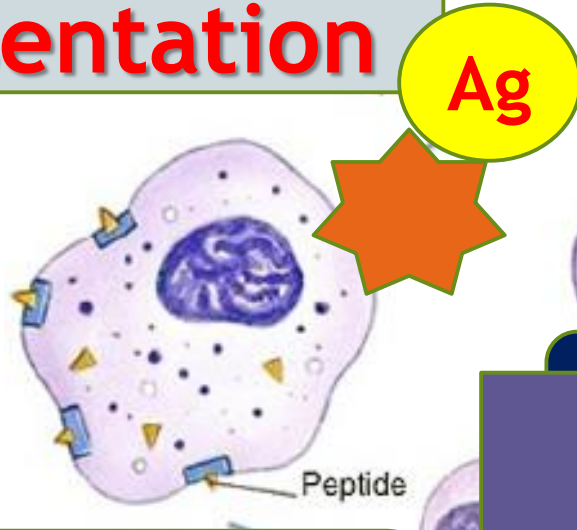
MEMORY B CELLS

T - Lymphocyte

- T – cell Matures in *Thymus*, only those survive which differentiate “self from nonself”
- HelperT cells CD4+ve (Th1, Th2, Th17, Treg)
- Cytotoxic T cells CD8+ve

Type I Hypersensitivity

APC & Ag
Presentation



1st
Exposure

- Anaphylaxis
- Allergic Rhinitis
- Asthma

IgE

Type II Hypersensitivity

- Attachment of Ab (IgG or IgM) against antigen or receptor on target cell (HDN, myasthenia gravis).
- Activation of complement system. Macrophages have receptors for IgG & C3b – more susceptible for phagocytosis (Opsonization)
- ADCC involves of death of target cell without complement activation.

Peforins secreted by Effector cells (NK) as they have Fc-receptors and get activated

Type III and type IV Hypersensitivity

➤ **IC deposit in target tissue (complement activation and phagocyte recruitment)**

(glomerulus – glomerulonephritis

Synovial membrane – rheumatoid arthritis)

➤ **Type IV – Delayed reaction hypersensitivity**

NO antibodies involved; CD4 and CD8 T cells involved

Immunity

➤ **Loss of self-tolerance and autoimmune diseases**