# ② CELLULAR PHASE

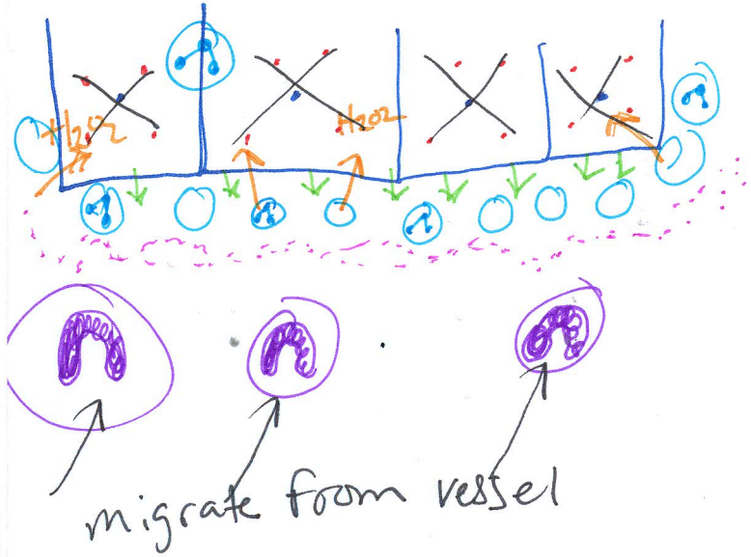
→ increase migration of WBC - neutrophils & move the cells towards the pathogen by chemotaxis

- Neutrophils follow cytokine gradient to reach the area

→ Neutrophils will aggressively try to destroy the pathogens at the site

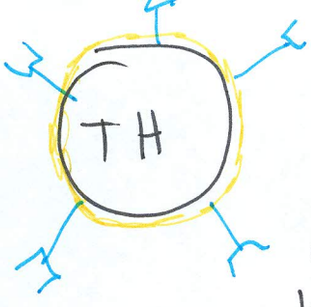
- degranulate & release superoxides ($H_2O_2$ hydrogen peroxide)
- phagocytose

this activity creates an area of destruction where healthy tissue, infected tissue, & WBC all are lysed

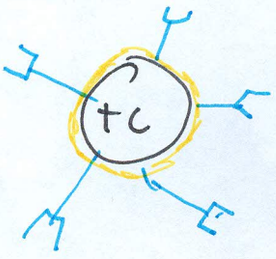



→ Monocytes are also attracted by cytokines released from neutrophils

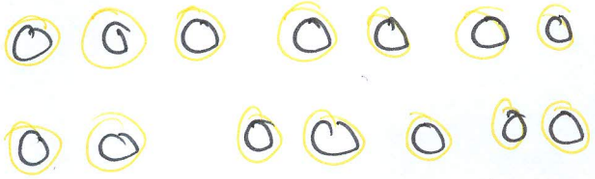
- monocytes must mature into Macrophages so they can participate in removing pathogen
- Macrophages are responsible for clearing the area by performing phagocytosis

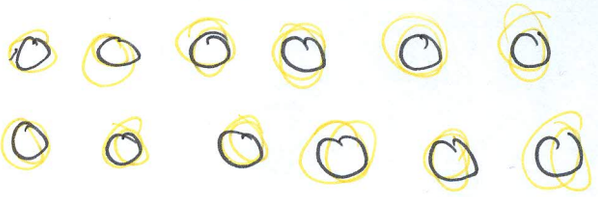



T cells Activate once the match is Verified




Next the Active cells Create An Army of Clones will perform Rapid mitosis





## Helper Clones

- Amplify the ~~Response~~

By Turning on Bcells & other inactive t cells

- Some of the Helper cells will Differentiate (mature) into Two Subtypes

$T_H$ memory

- will not participate in current infection, but will activate if pathogen returns.

~~Effector~~ Tregulatory

- Stop mitosis in T & B cells once pathogen is under control

## Cytotoxic Clones

- Directly attack Any cell with pathogen on Display, this limits death to only the infected cells & spares Healthy Tissue.
- secrete enzymes That lyse infected cells (performs/Defensins)
- Some cytotoxic cells will Become Tc-memory
  - will not participate in current infection, but activated in Future.

(B cells) (T cells)

# HUMORAL or CELLULAR IMMUNITY (or BOTH)

1. Can create antibodies B cells

   Humoral

2. Can destroy pathogens by secreting perforins $T_K$ cells

   Cellular

3. Is activated by a T-helper cell · Both

4. Undergoes clonal selection Both

   rapid mitosis

5. Has the ability to stop clonal selection

   Cellular - Treg

6. Must have an APC (antigen presenting cell) to be activated

   Cellular

7. Can be activated by pathogen in circulation

   Humoral - B

8. Migrates around the body in the lymph

   Both

9. Can create a tag of MHC proteins to alert body of infection

   Both

10. Creates memory cells to recognize the pathogen in the future

    Both

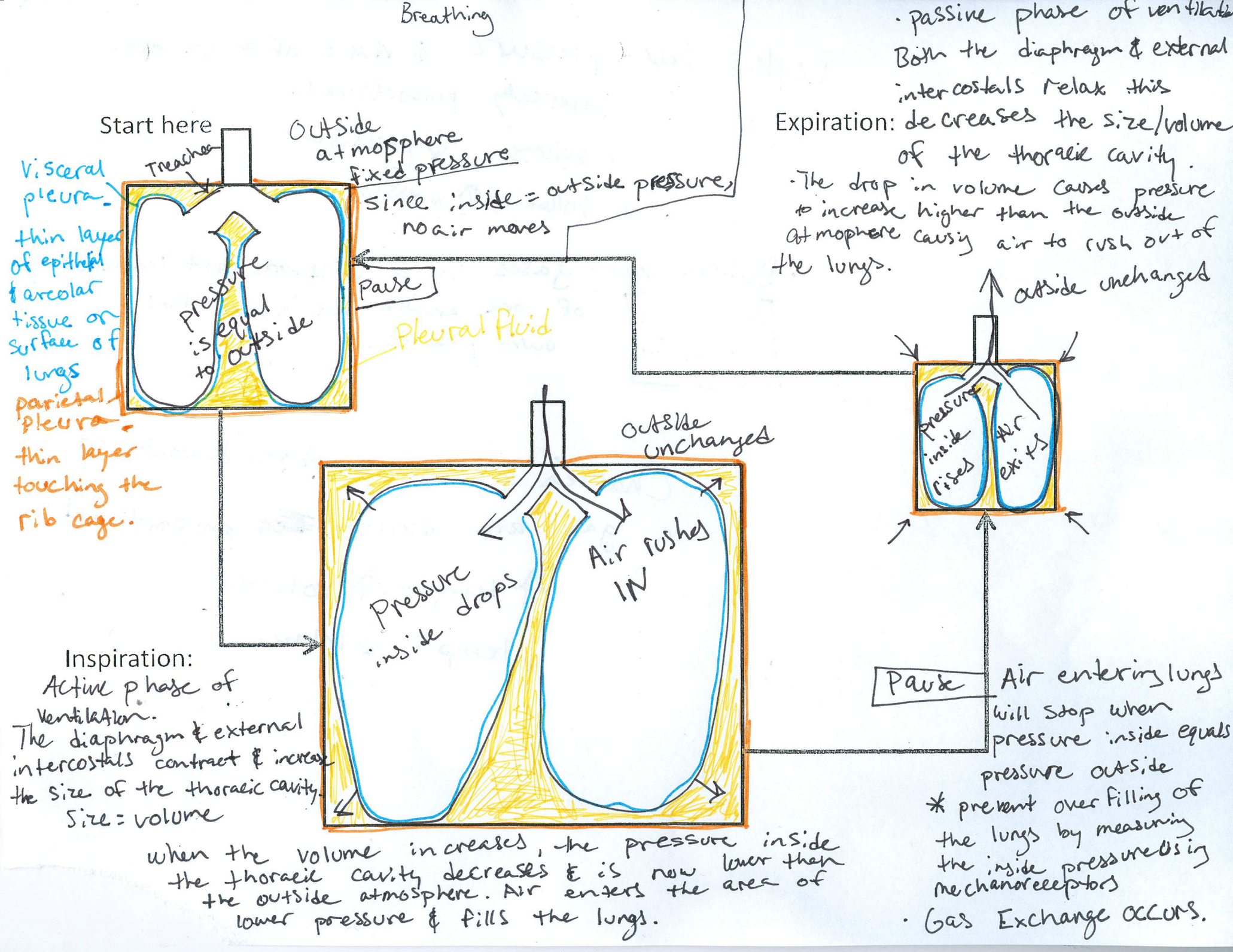
11. Stimulated by MHC class 2 APC cells only

    Cellular

12. Can activate the second line of defense by secreting antibodies

    Humoral

# Breathing

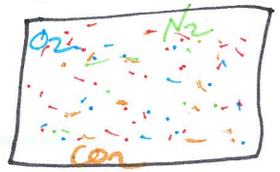

Start here
Outside atmosphere fixed pressure
Trachea
Visceral pleura - thin layer of epithelial & areolar tissue on surface of lungs
parietal pleura - thin layer touching the rib cage.
since inside = outside pressure, no air moves
Pause
Pleural fluid
pressure is equal to outside
Inspiration:
Active phase of ventilation.
The diaphragm & external intercostals contract & increase the size of the thoracic cavity.
Size = volume
Pressure inside drops
Air rushes IN
outside unchanged
when the volume increases, the pressure inside the thoracic cavity decreases & is now lower than the outside atmosphere. Air enters the area of lower pressure & fills the lungs.
Expiration: decreases the size/volume of the thoracic cavity.
· The drop in volume causes pressure to increase higher than the outside atmosphere causing air to rush out of the lungs.
outside unchanged
pressure inside rises
Air exits
Pause
Air entering lungs will stop when pressure inside equals pressure outside
* prevent over filling of the lungs by measuring the inside pressure using mechanoreceptors
· Gas Exchange occurs.

Boyle's law - pressure & volume of a gas are inversely proportional.

↑ volume, ↓ pressure

↓ volume, ↑ pressure

Dalton's law - gases in a mixture act independently of one another but exert their own pressure.




Charle's law - temperature & volume of a gas are directly proportional

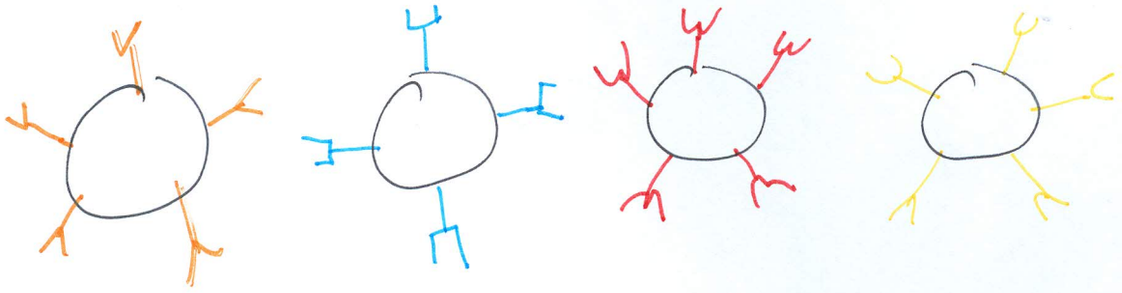
↑ temp = ↑ volume

↓ temp = ↓ volume

# 3rd line : Cellular Immunity - Tcells

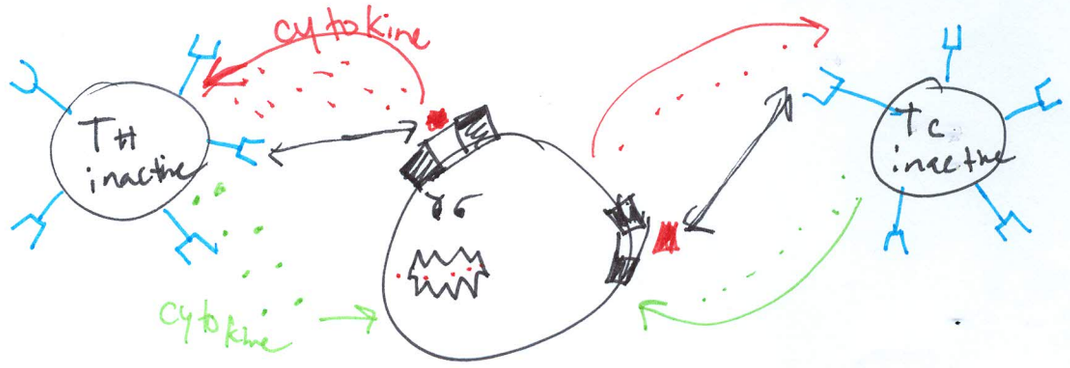
Tcells are formed in bone marrow, but travel to the thymus prior to being released inactive

In the thymus, hormones are secreted, stimulating T cells to fully mature & to test for reactivity to self antigens. If a Tcell reacts to self, it will be destroyed; if no reaction, the T cell is permitted back in blood stream.



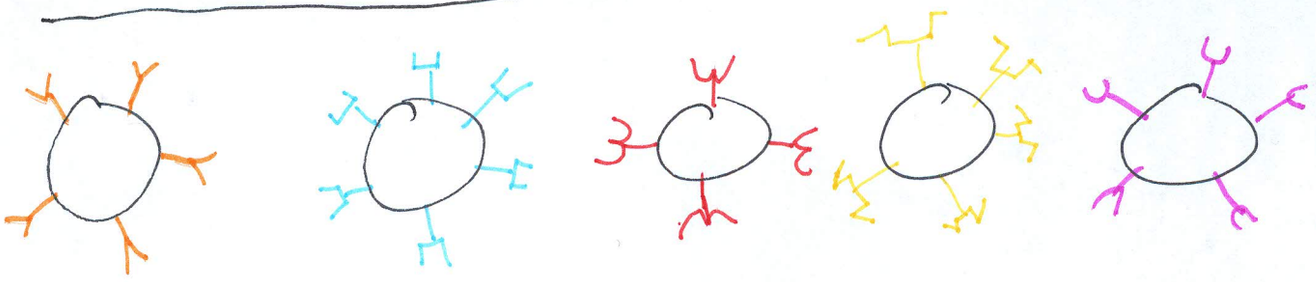
Inactive T cells are pre-programmed to identify One pathogen. Two main populations of Tcells exist:

T helper (CD4) T cytotoxic (CD8)



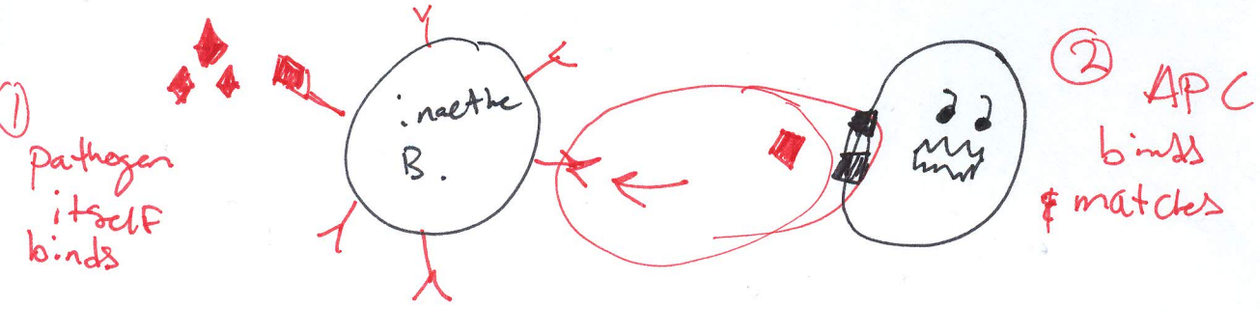

To activate T cells we first need an APC. ①
to match the receptor on Tcell.
② verify the match by cytokine release from Tcell to APC, asking if it is correct. The APC will signal back a cytokine if "yes". No return signal means no match.

# 3rd line: Humoral Immunity - B cells

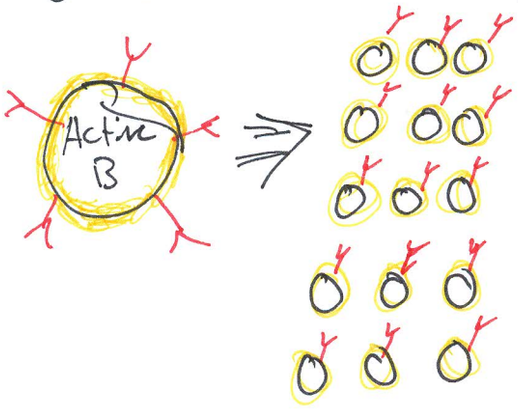


B cells are released from the bone marrow Inactive. There are hundreds of thousands of different displays on the various B cells Each one is pre-programmed to identify one (foreign / non-self) material.

In order to activate (1 or 2) B cells, they must bind to the appropriate match



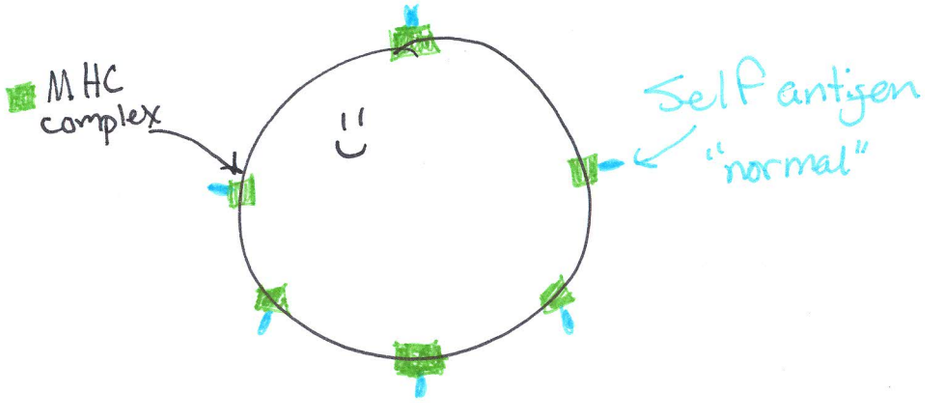

Once a match is found, B cell Activates & immediately begins to rapidly divide & create an army of clones. (Clonal selection)

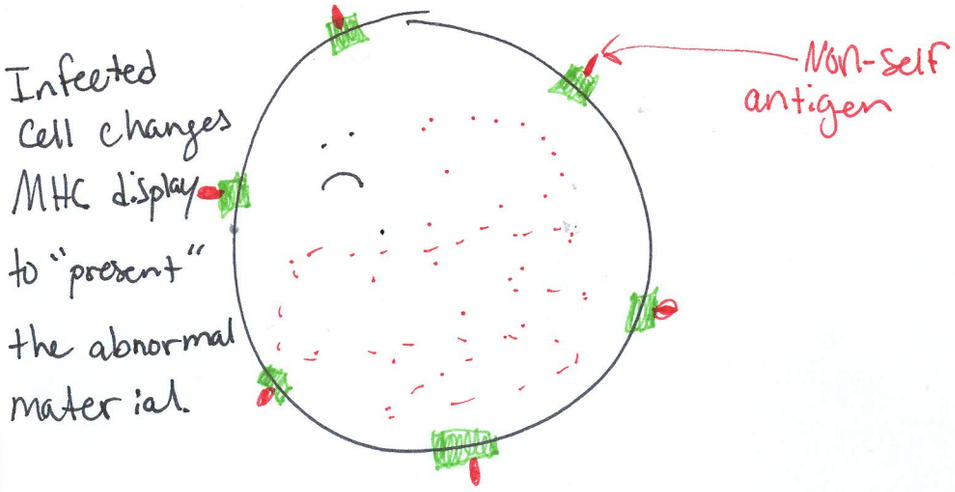


# Major Histocompatability complex (MHC)

## MHC class I

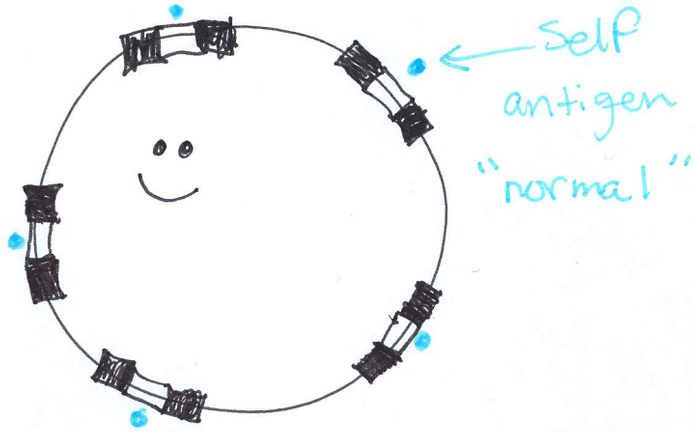
- present on all nucleated cells except blood cells



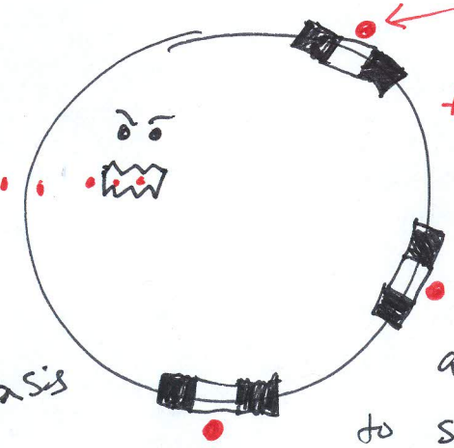




## MHC class II

- present on only WBC




Macrophage ingests pathogen & destroys it by phagocytosis

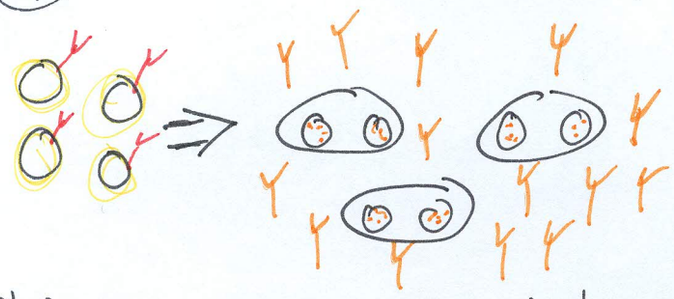



Antigen presenting Cell (APC)

- presents the abnormal material to stimulate B-cells & T-cells

from the created clones, Active B cells can transform into one of two subtypes.

## Plasma cell



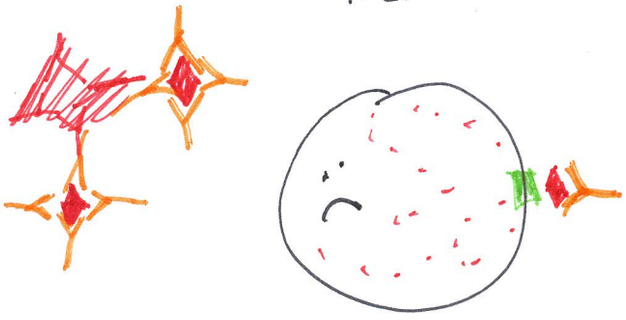
Plasma cells only job is to produce Antibodies against the pathogen.

Antibodies are an indirect form of attack.

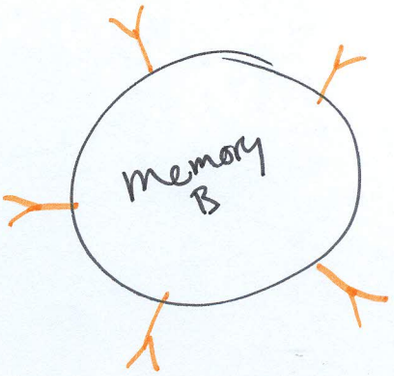
These will stimulate 2nd line of defense by marking the pathogen for destruction.

→ macrophages & neutrophils can "see" Antibodies & induce phagocytosis

→ Antibodies can coat the pathogen renders it ineffective



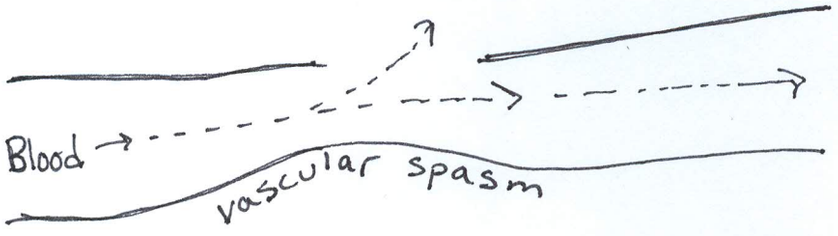
## Memory B cell




This cell deactivates and transforms into a memory B cell, which does not participate in the current infection instead, it exists in circulation in case the pathogen returns in the future.

# Steps of Hemostasis


- During the first phase of hemostasis, damage has occured to the vessel wall and blood leaks out into the surrounding tissues.
- Immediately upon damage Endothelial cells Vasoconstrictors at the damage site.




- This action will result in a decrease in blood flow to the location. Therefore less blood ends up in the tissue.

- The next step of the process involves platelets. Formation of a platelet plug is an example of a positive feedback loop.


Platelets in circulation arrive at the site of damage & adhere to the exposed collagen.

→

→ Once they stick, the platelets degranulate + release chemicals:

1. Secrotonin – potent vasoconstrictor
2. ADP
3. ThromBoxane A2

→ Stimulate platelet aggregation & adherence (sticky)

- The more platelets that arrive, the more will adhere & the "plug" will eventually occupy all the open space at the damaged site. Endothelial cells will begin to secrete chemicals upstream & downstream of the "plug" to prevent platelets from adhering to normal tissue.

- The final step of the process is coagulation, or formation of a fibrinelot. common pathway series of biochemical reactions will ultimately lead to a formalized clot securing the damaged site so regeneration/repair can occur.

Fibrin monomers deposit into plug
factor XII & calcium cross-linking to form fibrin polymer (mesh)