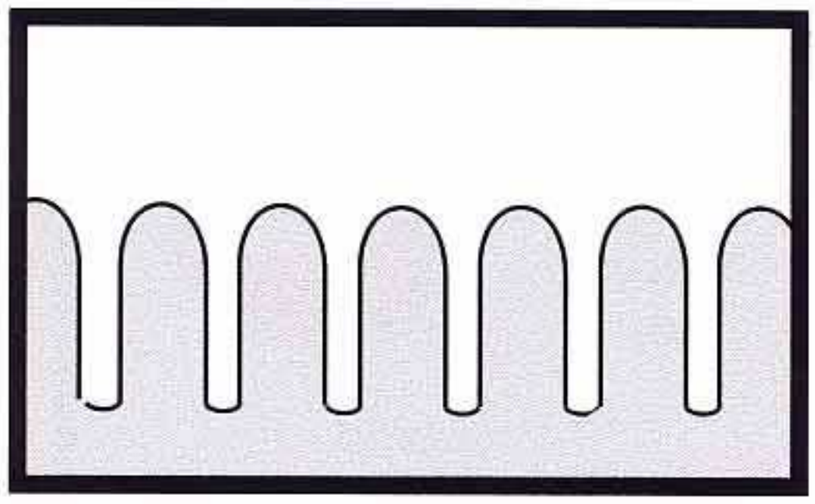
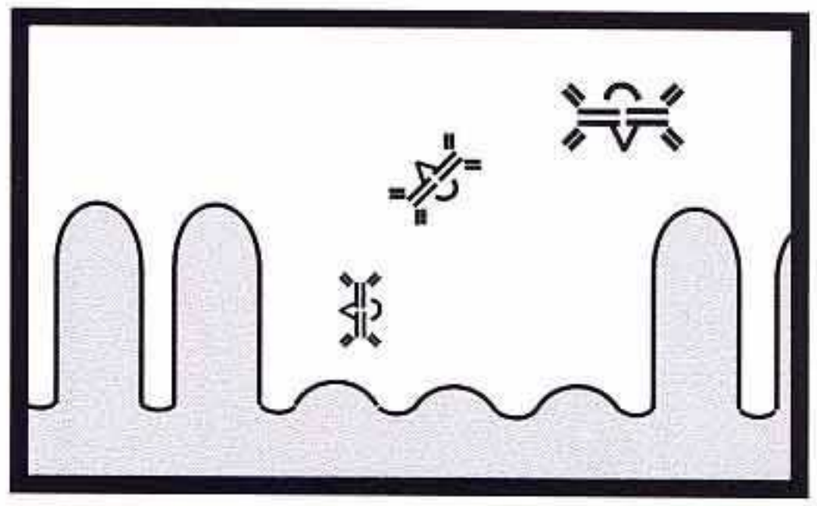
# CHAPTER SIXTEEN

# MISCELLANEOUS

# PEYER'S PATCHES



**The surface of the small intestine is lined with 'finger-like' projections called "villi".**

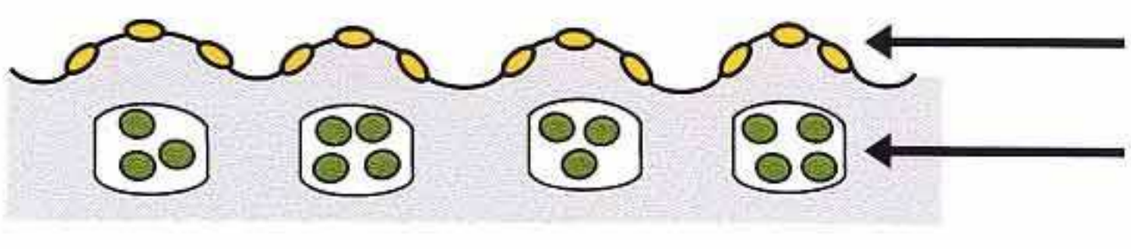


**But periodically these are replaced by Peyer's patches and it is from these that IgA are released.**

**Easy reading**

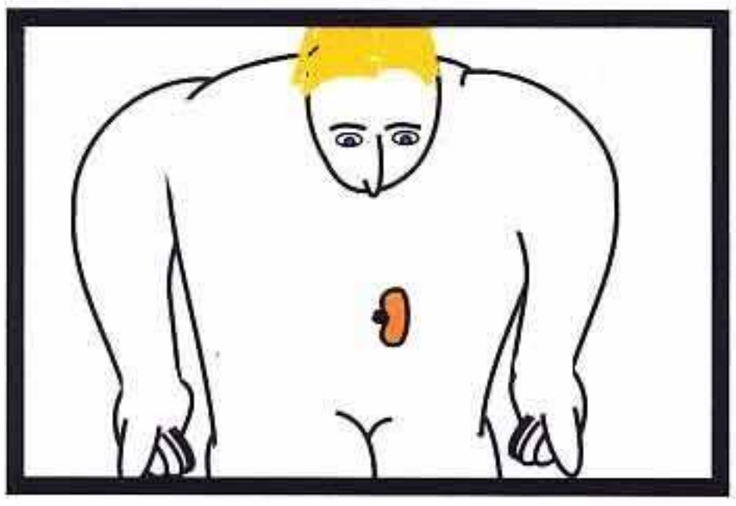
Technical information

A CLOSE-UP OF A PEYER'S PATCH

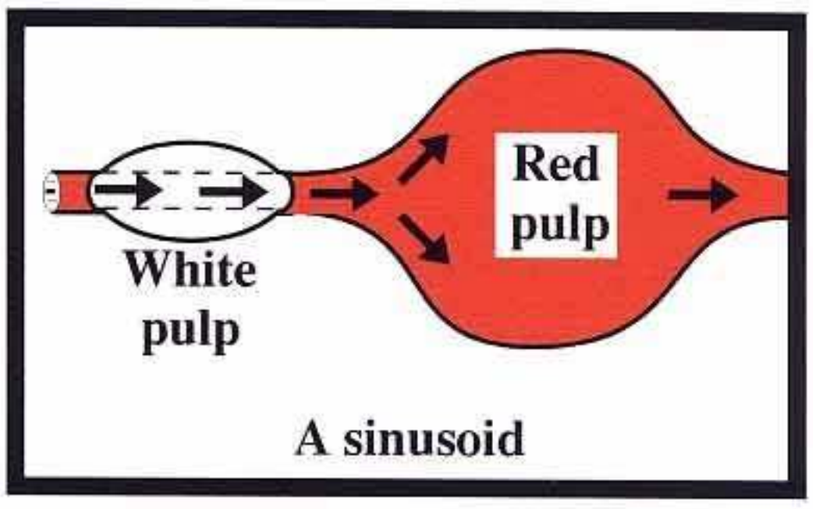



M cells allow microbes to easily penetrate the gut wall and so come into contact with the 'resting' B cells.

# THE SPLEEN



Inside the spleen, capillaries are replaced by sinusoids.

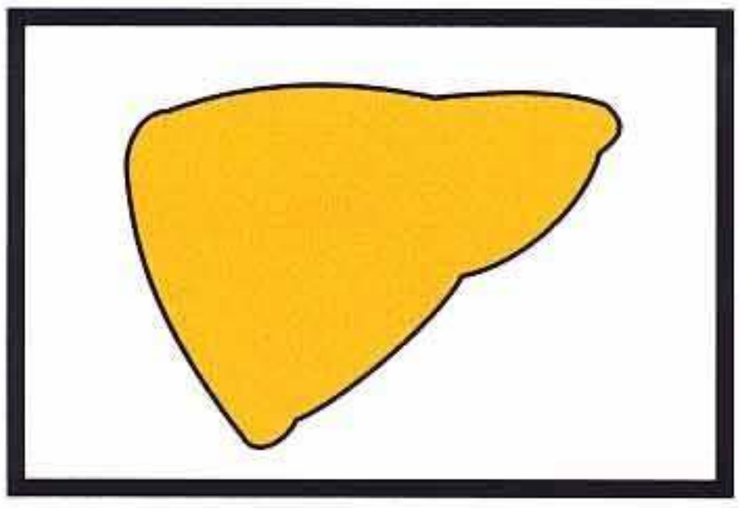



As the arteriole approaches the sinusoid, it passes through the white pulp.

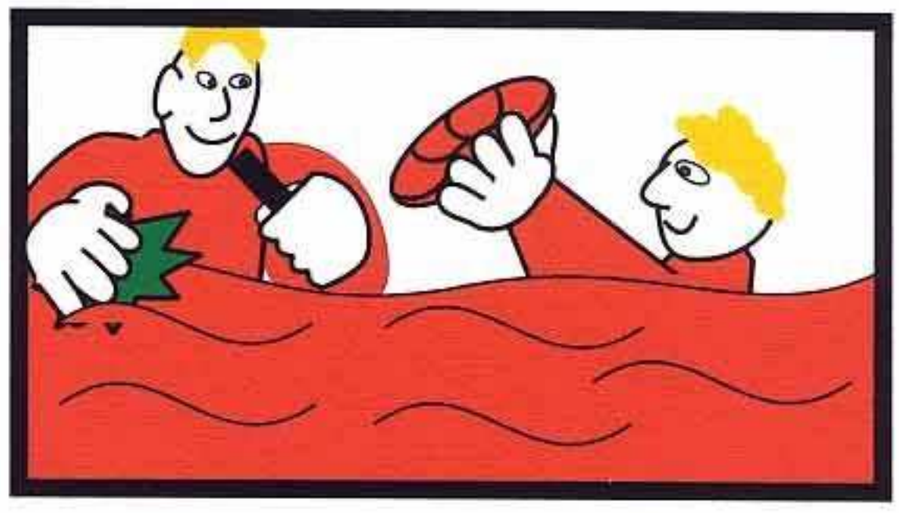
The white pulp contains many B cells, which can release antibodies straight into the blood.


Meanwhile, in the red pulp, resident macrophages remove microbes, waste material and worn out red blood cells

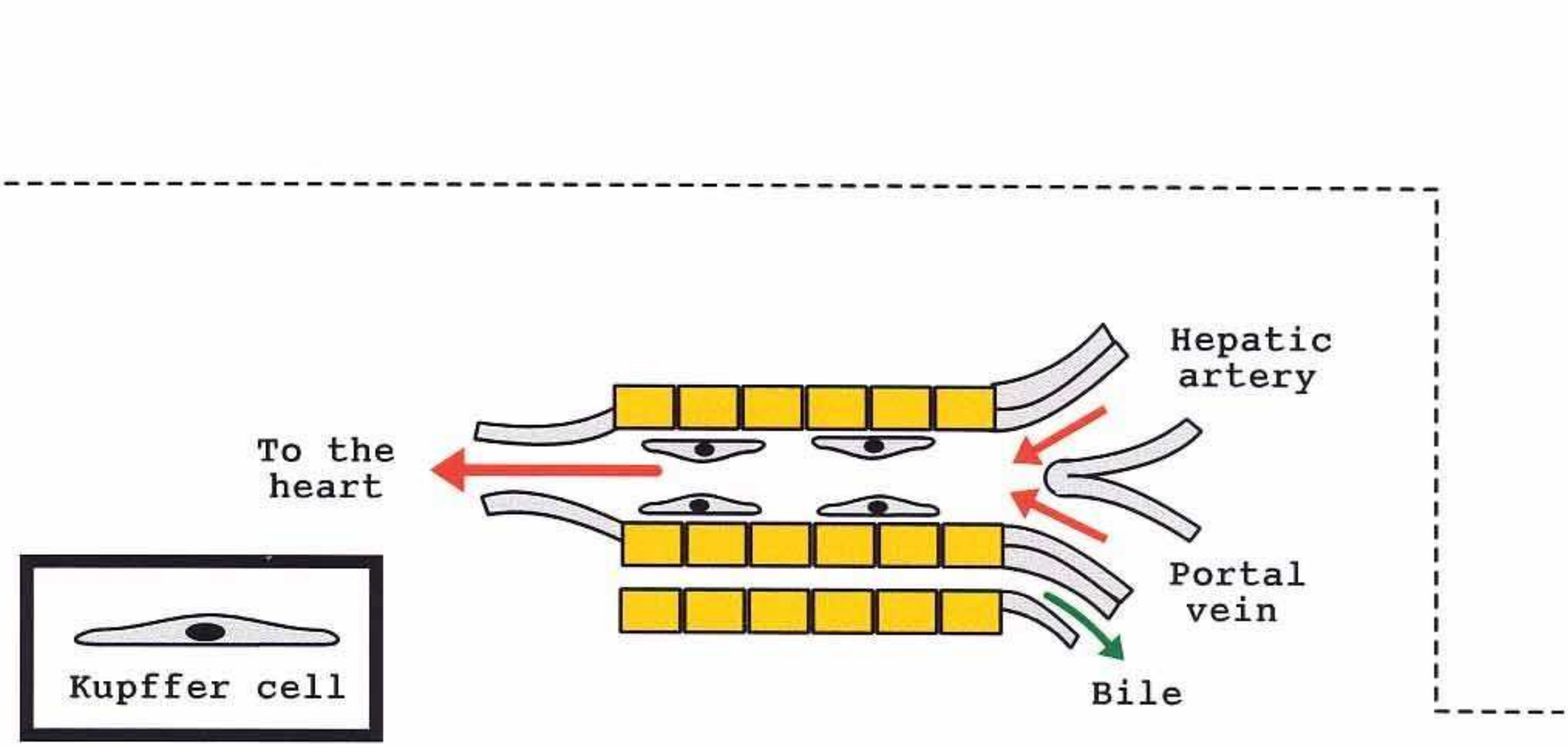
# THE LIVER



**The liver has many functions such as:- making bile, storing energy and detoxifying drugs.**

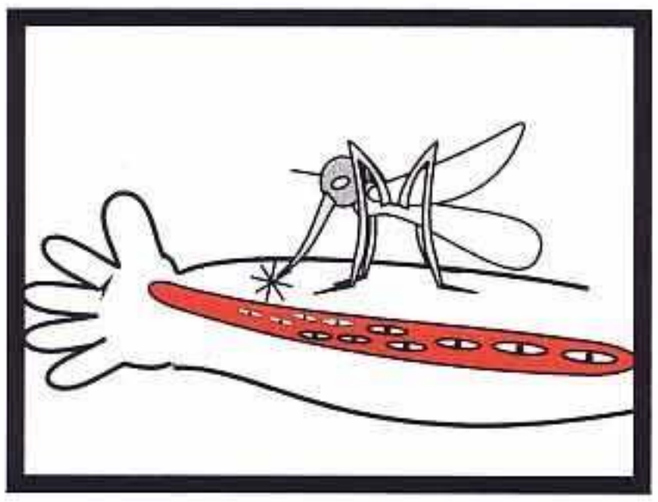


**Kupffer cells (a type of macrophage found in the liver), scavenge for worn out red blood cells, microbes and waste material.**

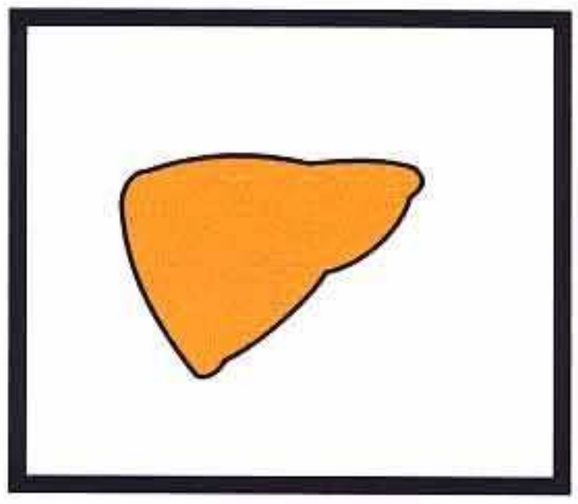



Inside the liver, capillaries are replaced by specialised sinusoids.

# MALARIA



**When an infected mosquito bites someone, sporozoites can enter their blood.**

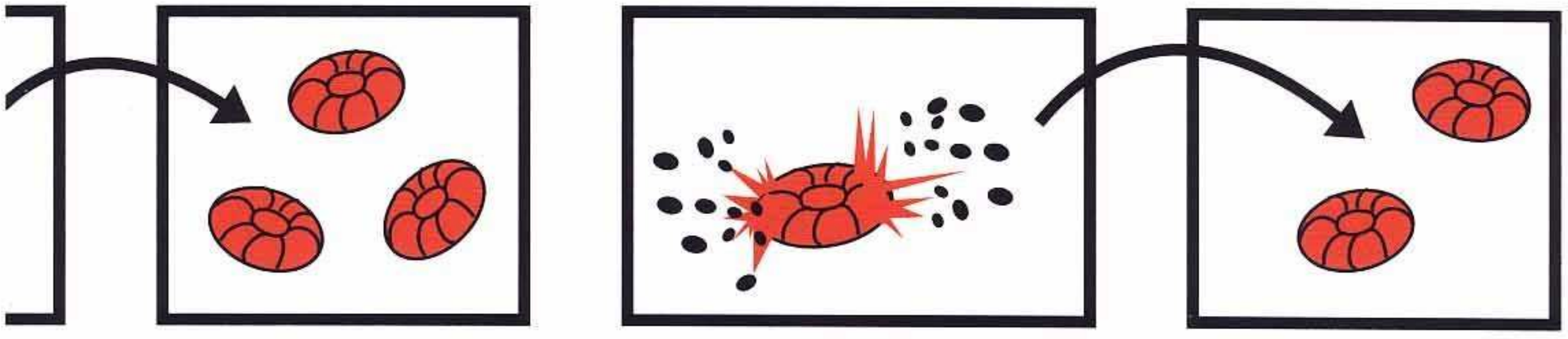


**The sporozoites must now reach and infect cells inside the liver.**

**A couple of weeks later and merozoites burst out into the blood.**

It is the anopheles mosquito which harbours the parasite responsible for causing malaria.

The parasite has a complicated life-cycle, part of which must be completed in man.



**The 'exposed' merozoites, must now quickly infect other red blood cells.**

**After a couple of days the infected cell is filled to bursting point with many merozoites.**

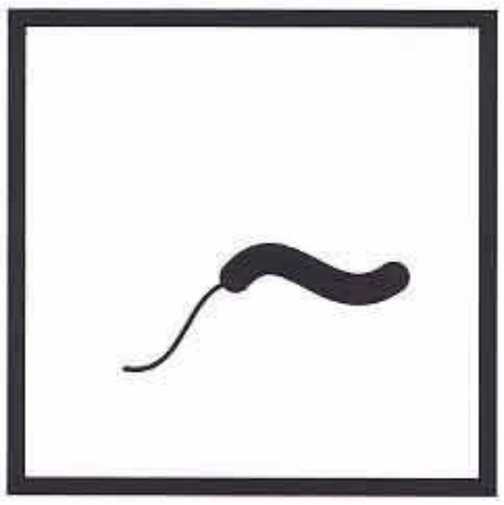
**On entering the blood, the merozoites will infect other red blood cells.**

**This cycle will now continue many times inside the patient.**


Cold stage Hot stage Sweating stage

Each time the merozoites burst out into the blood, the patient will for a few hours, experience the classic malarial symptoms.

# CHOLERA

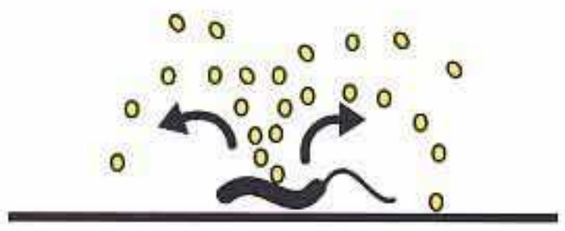


This disease is caused by the cholera vibrio.

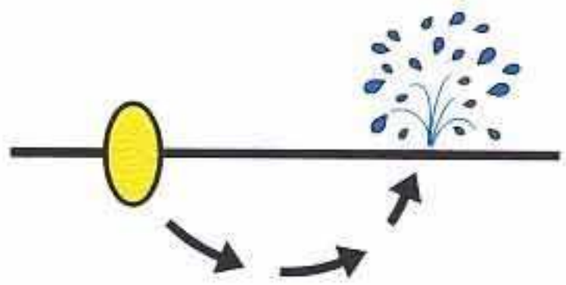
Infection usually occurs by drinking contaminated water.

Excessive 'rice water' diarrhoea now follows.

**After replicating in the gut, millions of vibrios are flushed out to infect new hosts when the patient has violent diarrhoea. This is due to an excessive fluid build up, inside the bowel.**

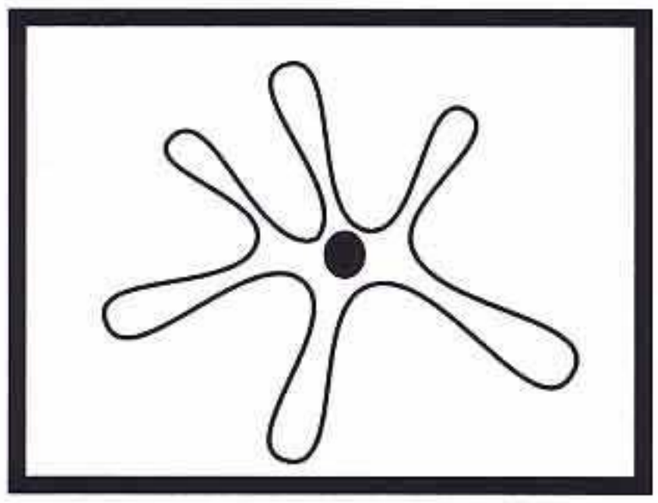


As well as replicating in the small intestine, each cholera vibrio releases enterotoxins.

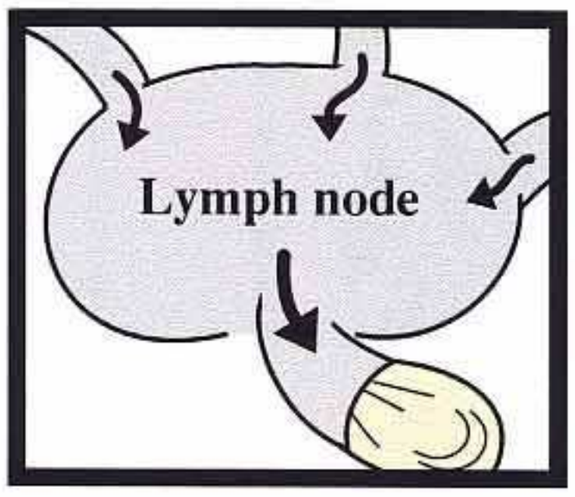


The enterotoxins pierce the gut epithelium and trigger it into releasing water and salts.

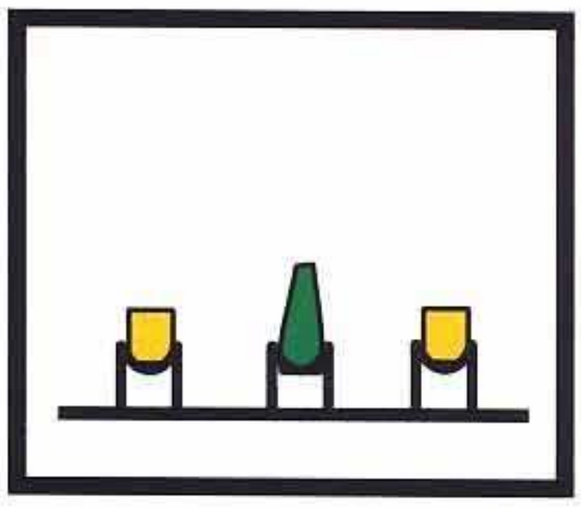
# THE FOLLICULAR DENDRITIC CELL



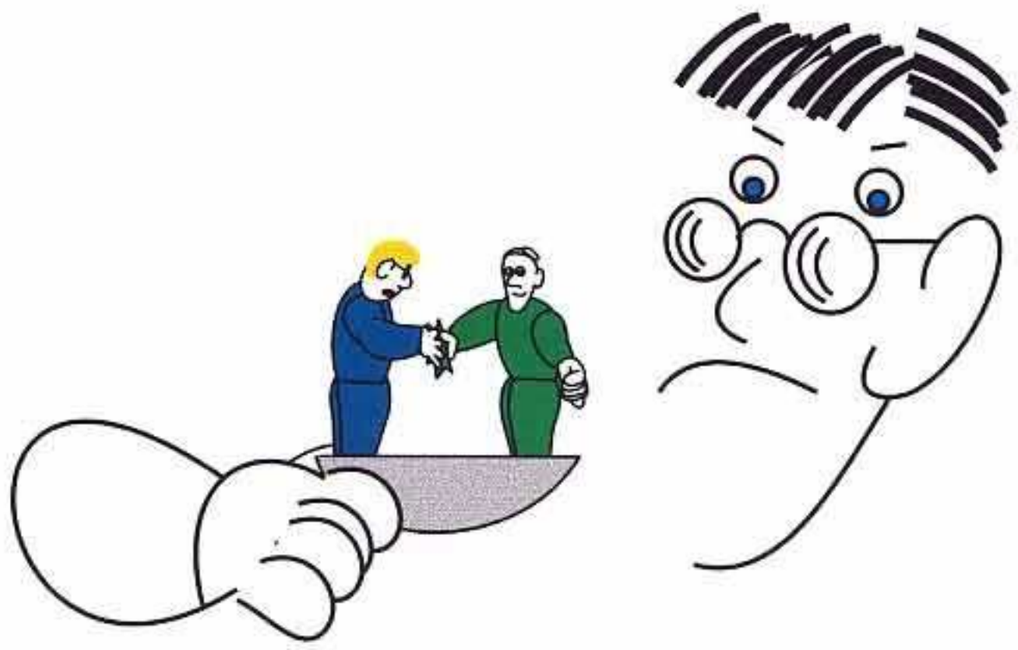
**The follicular dendritic cell is found in the cortex of the lymph nodes.**




**Hence they come into contact with material passing through the lymph node.**

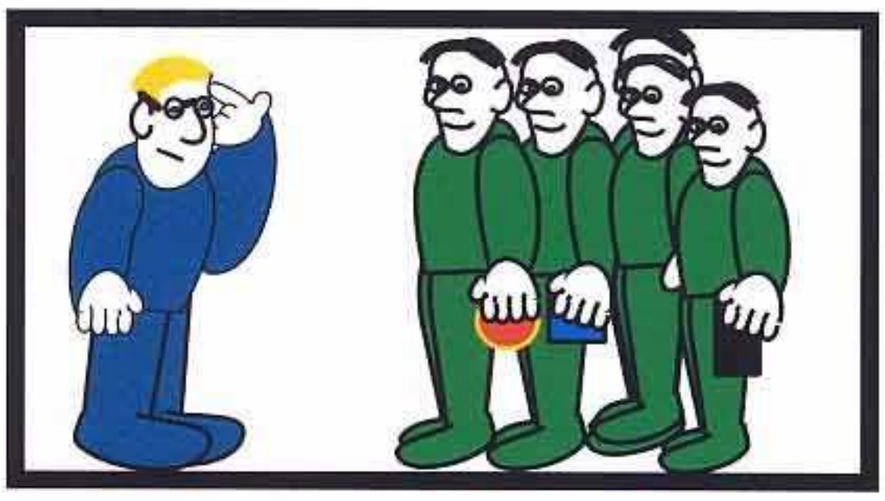


**At their surface, matter will be presented, attached to 'attack' proteins.**

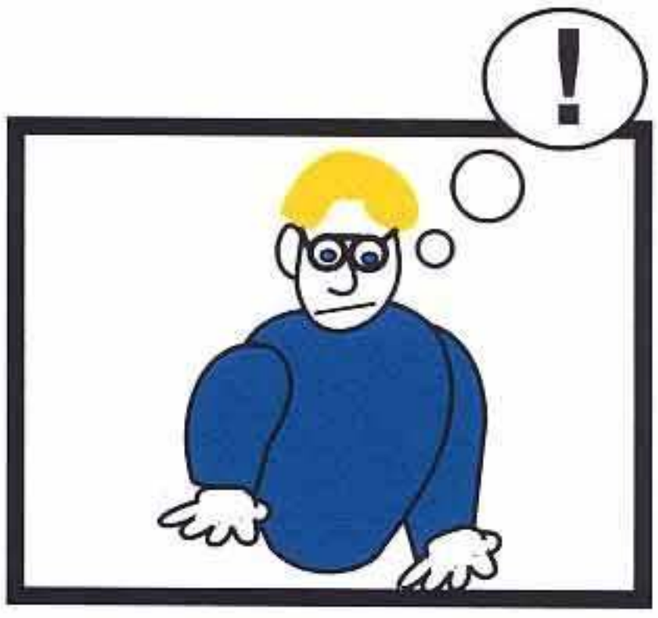


In the laboritory it is difficult to get T helper cells to stimulate B cells into producing antibodies. It appears that the follicular dendritic cells need to be present.

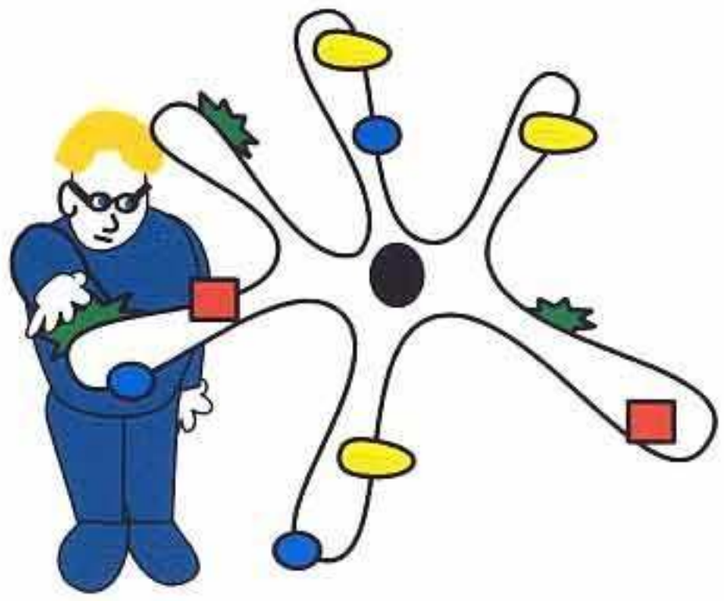
# INSIDE A LYMPH NODE



**When a T helper entres a lymph node, he has to see if any of the resident B cells are presenting foreign material that 'fits' his 'hands'.**

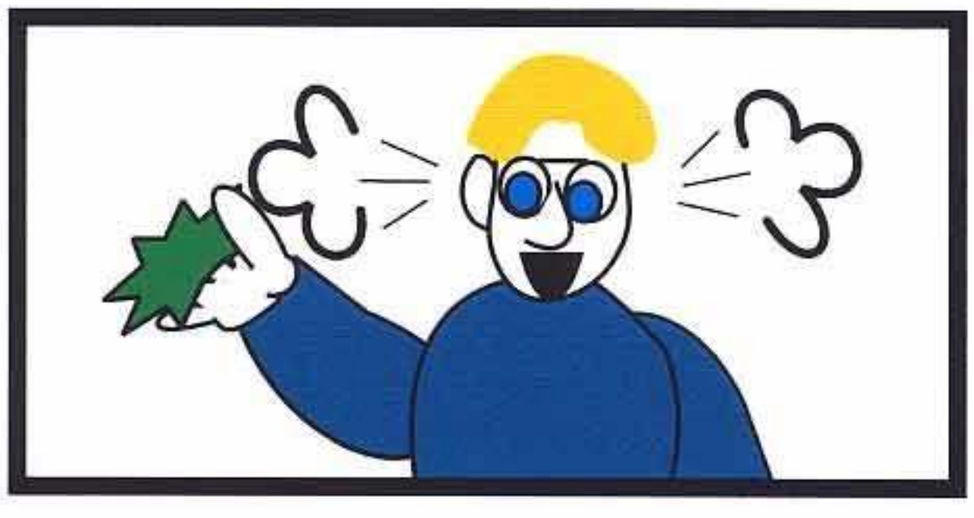


**But it is difficult to see how a T helper cell can single handly check all the resident B cells.**

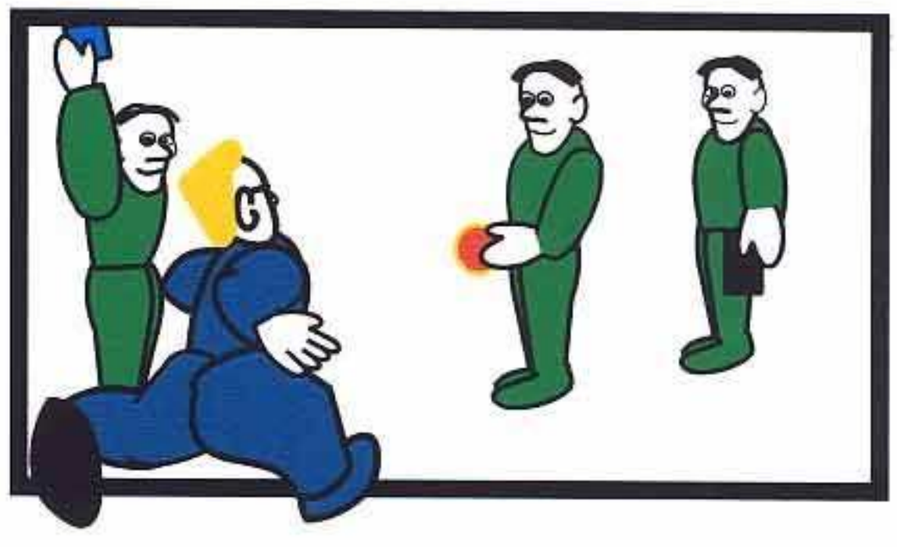


So what could happen is that when the T helper enters the a lymph node it samples all the material being expressed by the follicular dendritic cells.

# THE TURNED ON T HELPER!!

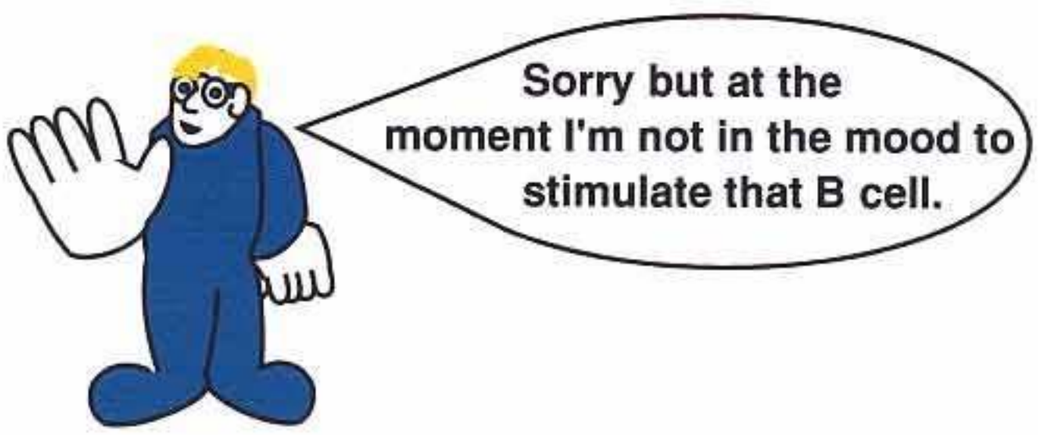


If the T helper's 'hands' fit material being expressed by a follicular dendritic cell, then this could turn the T helper on.



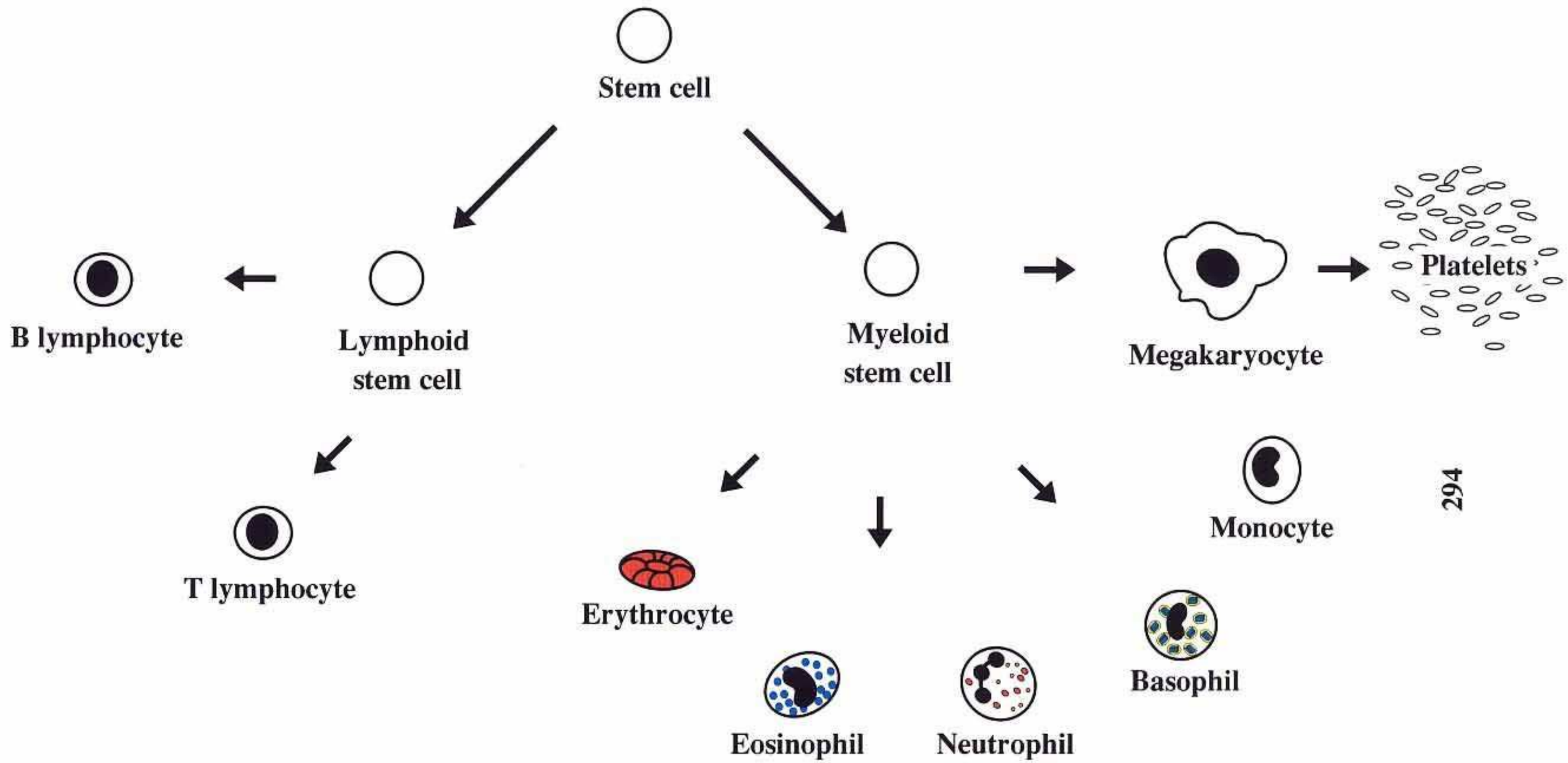
He might then search that lymph node for a B cell who is also presenting material that fits his 'hands'.

However, if the dendritic cells in the lymph node have not got anything for the T helper, then he will probably move quickly onto the next lymph node as it is unlikely that any B cell here would be presenting suitable foreign material.




So it could be that the reason the B and T helper cells will not react in the laboritory is because the T helper cell must first be turned on by a follicular dendritic cell.

## THE BLOOD CELL FAMILY TREE




The bone marrow contains a pool of stem cells, each of which is capable of developing into any blood cell.

# USEFUL TERMS FOR A BUDDING IMMUNOLOGIST

***Accessory cell:*** **Any cell which expresses material attached to 'attack' proteins.**

***Adjuvant:*** **Something which enhances an immune response.**

***Agglutination:*** **When particulate matter is clumped together by antibodies.**

***Allergen:*** **Any substance which triggers the release of IgE antibodies.**

***Alpha interferon:*** **Released by virally infected cells, it appears to 'shut down' neighbouring cells in an attempt to contain the viral infection.**

***Anergy:*** **When a B or T lymphocyte is no longer able to respond to a particular antigen.**

***Antigen:*** **Material that is capable of triggering an antibody or T cell response.**

***Antigen presenting cell:*** **Another way of describing an accessory cell.**

***Autocrine growth factors:*** **When these are released (ie interleukin-2), they will trigger the cell to clone itself.**

***Basophil:*** **This white blood cell has an S shaped nucleus, contains histamine granules and is lined with surface IgE receptors.**

***Cluster determinant (CD):*** **A cluster of antibodies which are specific for a particular cell surface marker.**

***Cytokines:*** **These are factors released by cells which will affect other cells.**

***Delayed-type hypersensitivity:*** **Otherwise known as a type 4 hypersensitive reaction. Because it results from T cell activation and not does not involve antibodies, this type of reaction takes at least 24 hours to occur, following an antigenic challenge.**

***Enterotoxin:*** **A form of exotoxin, which affects the lining of the digestive tract.**

***Exon:*** **A length of DNA which codes for a protein.**

***Farmer's lung:*** **This and other local type 3 hypersensitive reactions, are often associated with people who work in dirty environments.**

***Gamma interferon:*** **Released by T helper cells, this cytokine will overtly stimulate macrophages and neutrophils. An affected macrophage acquires a characteristic roughened membrane and may now be referred to as an 'angry' macrophage.**

***Granulocytes:*** **White blood cells which contain granules (ie neutrophils and eosinophils).**

***'Hands':*** **Receptors found on lymphocytes and at the ends of antibodies, which have a unique fixed shape.**

***Histocompatibility:*** **A transplants ability to survive inside a recipient.**

***Interleukins:*** **Factors which allow white blood cells to 'communicate' with each other.**

***Interleukin-1:*** **Released by macrophages, it has a wide range of actions, one of which is to act as a second signal to T helper cells (see page 88).**

***Interleukin-2:*** **Released by T helper cells, this cytokine will affect other immune cells in the vacinity and also act as an autocrine growth factor.**

***Intron:*** **A non coding length of DNA.**

***Isotype:*** **This word is referring to an antibody's class (ie IgA, IgE etc).**

***Leucocyte:*** **A white blood cell.**

***Lymphokine-activated killer cells (LAKs):*** **When NK cells are exposed to a high concentration of interleukin-2, they transform into LAK cells, which appear to possess enhanced anti-cancer and anti-viral properties.**

***Macrophage-activating factor (MAF):*** **Released by T helper cells, this cytokine enhances a macrophage's ability to breakdown phagocytosed material and to upgrade its release of inflammatory factors.**

***Migration inhibition factor (MIF):*** **Released by T helper cells, this cytokine encourages macrophages to remain at a particular location.**

***Mucosa-associated lymphoid tissue (MALT):*** **Peyer's patches, the adenoids and tonsils and mesenteric lymph nodes are all part of MALT. From these, IgA are released into the respiratory and gastrointestinal tracts.**

***Natural killer cell (NK cell):*** **A type of lymphocyte that has surface IgG receptors but lacks T cell receptors. Although their function in health remains obscure, they appear to possess anti-cancer and anti-viral properties.**

***Opsonins:*** **When antibodies or complement C3b coat the surface of a microbe to facilitate phagocytosis, they are called "opsonins".**

***Perforin:*** **Released by NK cells onto the surface of a target cell, perforin molecules will insert into the membrane before polymerising into MAC-like complexes.**

***Polymorphonuclear leucocytes:*** **Otherwise known as neutrophils, due to their multi-lobed nucleus.**

***Transforming growth factor:*** **Released by macrophages and T helper cells, this cytokine switches off an immune assault and initiates tissue repair.**

***T cell Receptor (TcR):*** **This specifically refers to the CD3 and Ti molecules.**

***Thymus gland:*** **Although this organ is vital for T cell maturation, it starts to undergo involution once an individual reaches adolescence.**

***Vascular cell adhesion molecules:*** **Endothelial cells can be triggered into expressing a range of adhesion molecules. Passing immune cells will attach onto these, before then migrating from the blood vessel into the tissues.**