

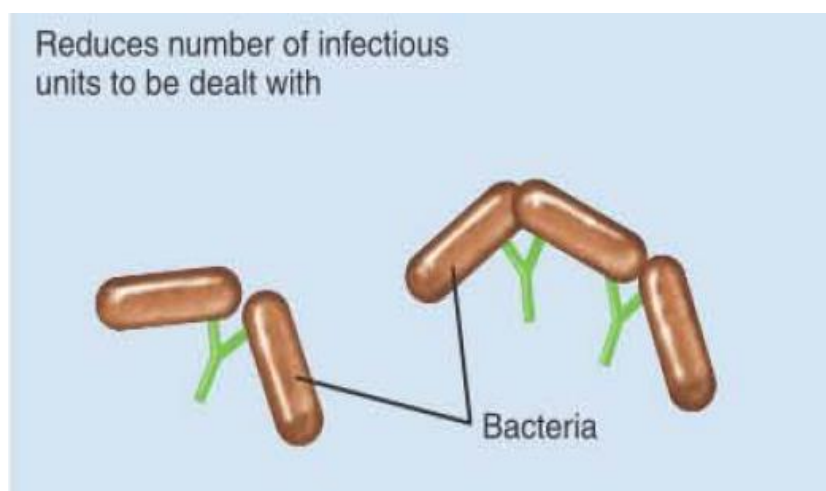
## **Antigen-Antibody Binding and Its Results**

The strength of the bond between an antigen and an antibody is called **affinity**. In general, the closer the physical fit between antigen and antibody, the higher the affinity. Therefore, antibodies can be used to differentiate between the viruses of chickenpox and measles and between bacteria of different species.

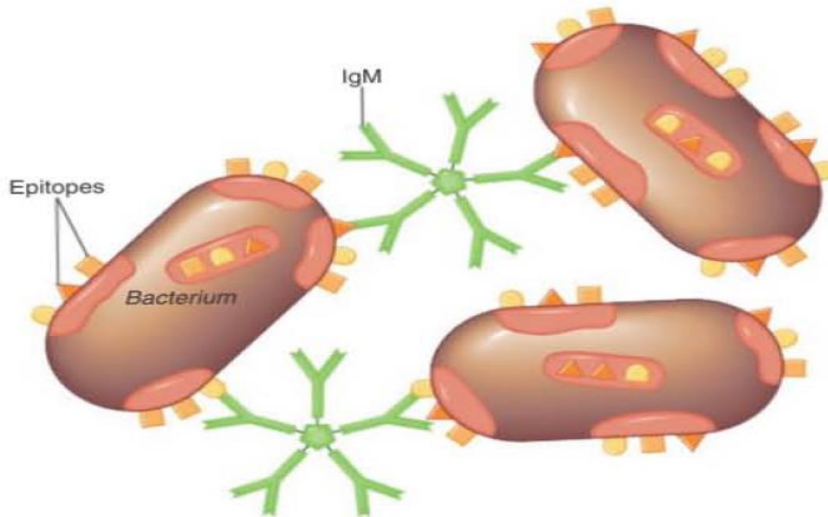
The antibody molecule itself is not damaging to the antigen. Foreign organisms and toxins are rendered harmless by only a few mechanisms: These are

- 1- Agglutination.
- 2- Opsonization.
- 3- Neutralization.
- 4- antibody-dependent cell-mediated cytotoxicity.
- 5- and the activation of complement leading to inflammation and cell lysis.

**Agglutination**, antibodies cause antigens to clump together. For example, the two antigen-binding sites of an IgG antibody can combine with epitopes on two different foreign cells, aggregating the cells into clumps that are more easily ingested by phagocytes.



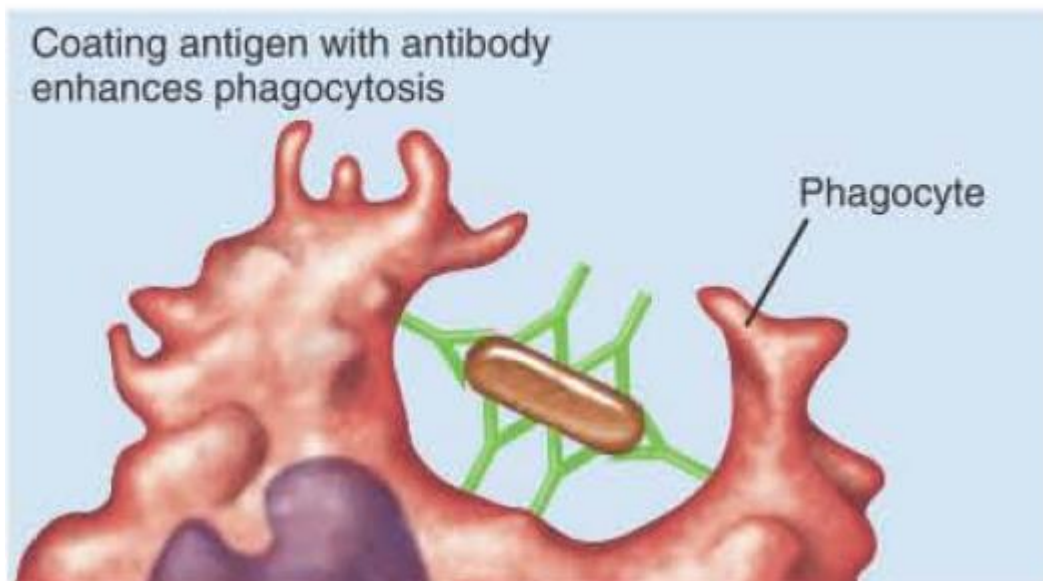
Because of its more numerous binding sites, IgM is more effective at cross-linking and aggregating particulate antigens



**opsonization**

the antigen, such as a bacterium, is coated with antibodies that enhance its ingestion and lysis by phagocytic cells.

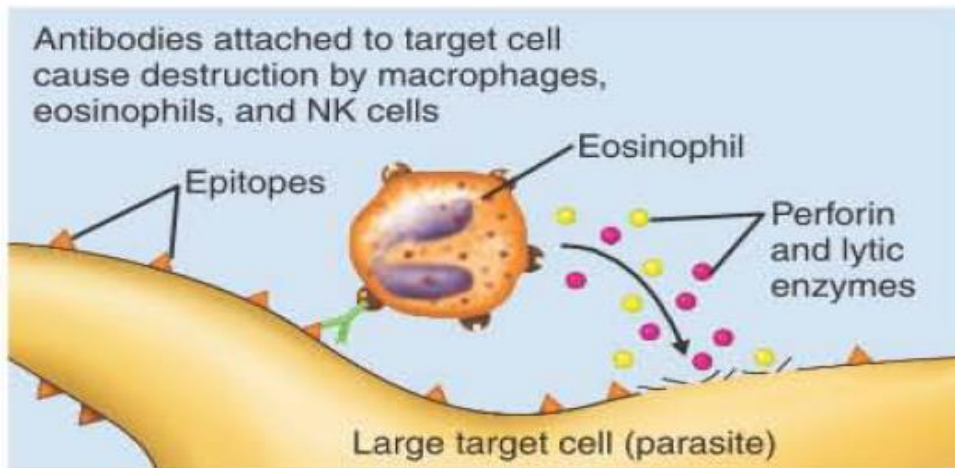
**Opsonization**  
(see also Figure 16.9)



**Antibody-dependent cell-mediated cytotoxicity**

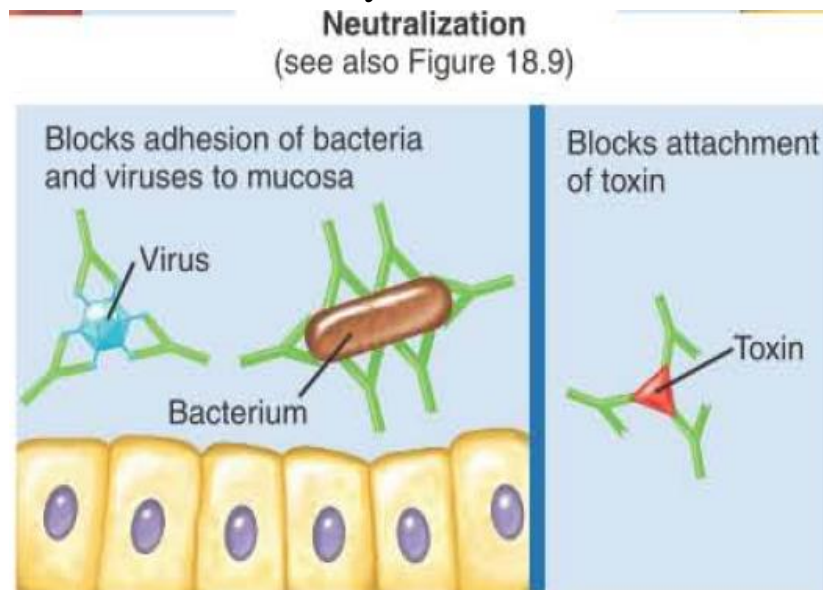
resembles opsonization in that the target organism becomes coated with antibodies; however, destruction of the target cell is by immune system cells that remain external to the target cell.

↓ **Antibody-dependent cell-mediated cytotoxicity**  
(see also Figure 17.15)



**Neutralization**

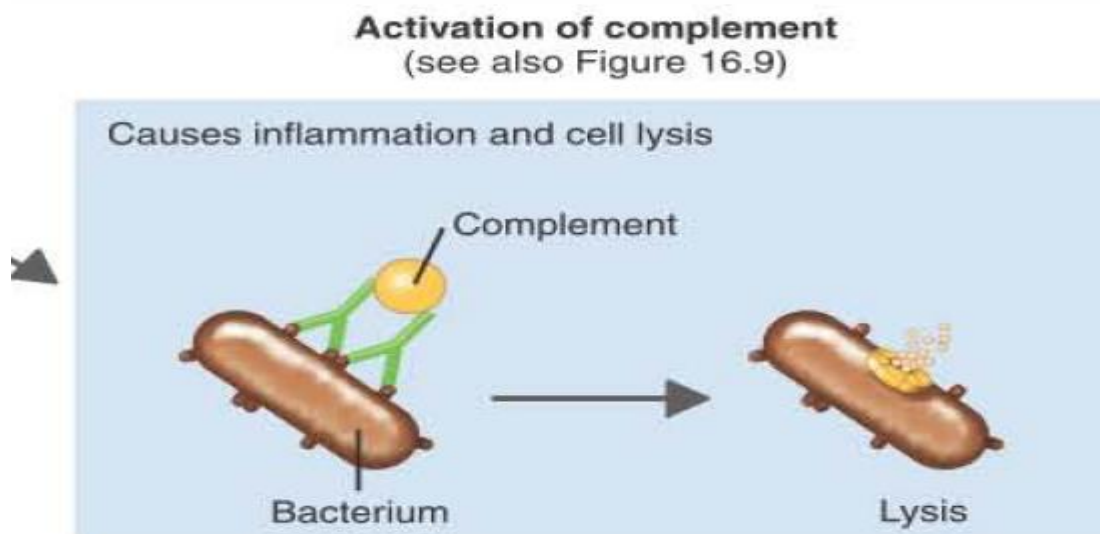
IgG antibodies inactivate microbes by blocking their attachment to host cells, and they neutralize toxins in a similar



manner

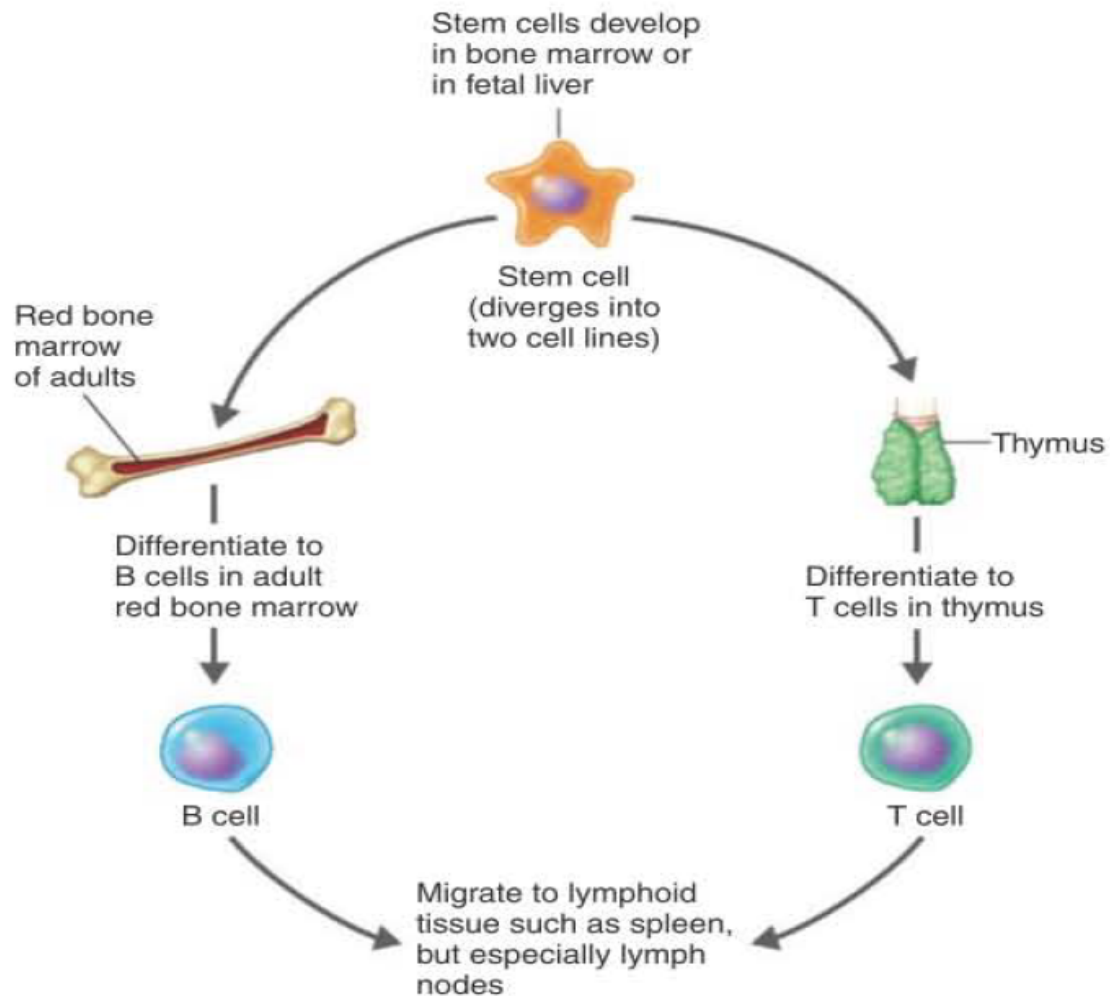
### **activation of the complement system**

For example, inflammation is caused by infection or tissue injury. One aspect of inflammation is that it will often cause microbes in the inflamed area to become coated with certain proteins. This, in turn, leads to the attachment to the microbe of an antibody-complement complex. This complex lyses the microbe, which then attracts phagocytes and other defensive immune system cells to the area.



### **T Cells and Cellular Immunity**

Humoral antibodies are effective against pathogens such as viruses and bacteria that are circulating freely, where the antibodies can contact them. Intracellular antigens, such as a virus within an infected cell, are not exposed to circulating antibodies. Some bacteria and parasites can also invade and live within cells. T cells probably evolved in response to this aspect of pathogenicity. They are also the way in which the immune system recognizes cells that are nonself, especially cancer cells. Like B cells, each T cell is specific for only a certain antigen. T cells have TCRs. T cells develop from stem cells in the fed bone marrow (Figure 17.8).



**Figure 17.8 Differentiation of T cells and B cells.** Both B cells and

The recognition of antigens by a T cell requires that they be first processed by specialized antigen-presenting cells (APCs). This resembles the situation previously discussed in humoral immunity in which a B cell served as the APC (see Figure 17.4). After processing, an antigenic fragment is presented on the APC surface together with a molecule of the MHC. They include activated macrophages and, most important, dendritic cells.

The body's ability to make new T cells decreases with age, beginning in late adolescence. Eventually, the T-cell producing thymus becomes less active, and red bone marrow produces fewer B cells. As a result, the immune system is relatively weak

in older adults. However, sufficient long-lived T and B memory cells survive to make immunization of older adults effective for such diseases as influenza and pneumococcal pneumonia.