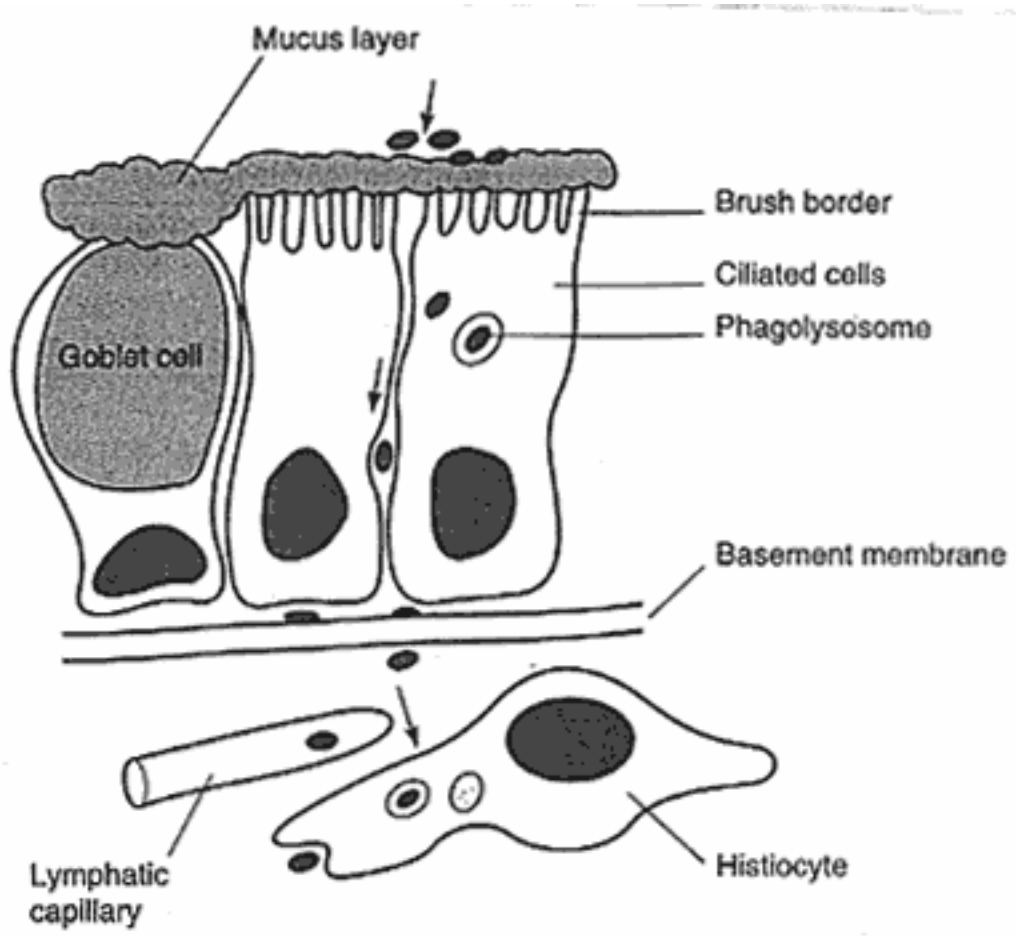



**Fig. 3.1** Microbial invasion across an epithelial surface.

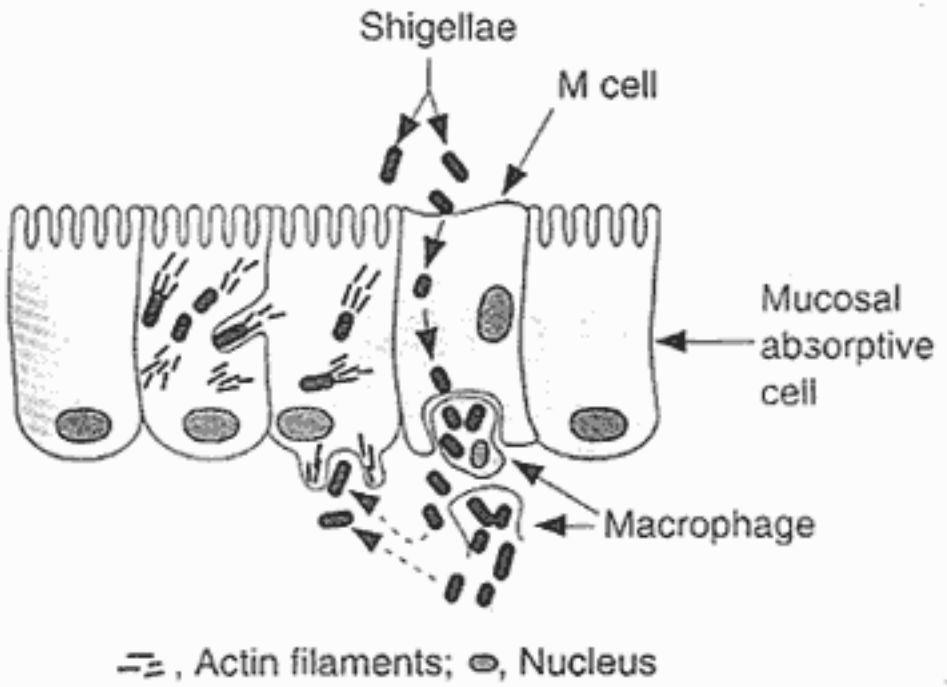
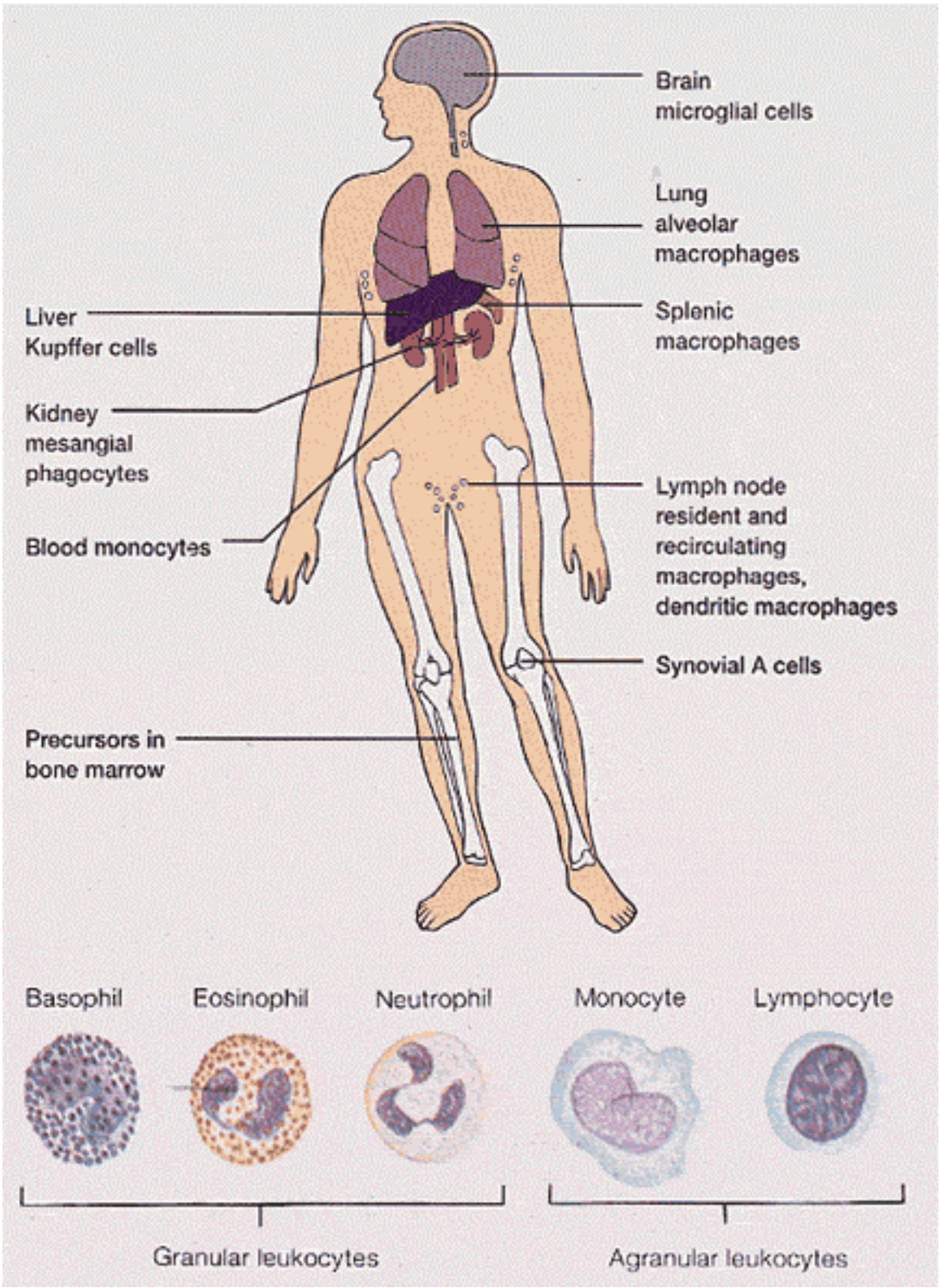



*Figure 14-4* Proposed model for *Shigella* invasion of the colonic mucosa. Invasion occurs in three steps: shigellae first transit the mucosa by passing through the M cells. They then use their invasins to invade mucosal cells from below, where integrins (putative host cell receptors for the Ipa proteins) are located. Shigellae then spread to adjacent cells, causing cell death and ultimately provoking an inflammatory response.


Brain
microglial cells
Lung
alveolar
macrophages
Liver
Kupffer cells
Splenic
macrophages
Kidney
mesangial
phagocytes
Lymph node
resident and
recirculating
macrophages,
dendritic macrophages
Blood monocytes
Synovial A cells
Precursors in
bone marrow
Basophil
Eosinophil
Neutrophil
Monocyte
Lymphocyte
Granular leukocytes
Agranular leukocytes

7c

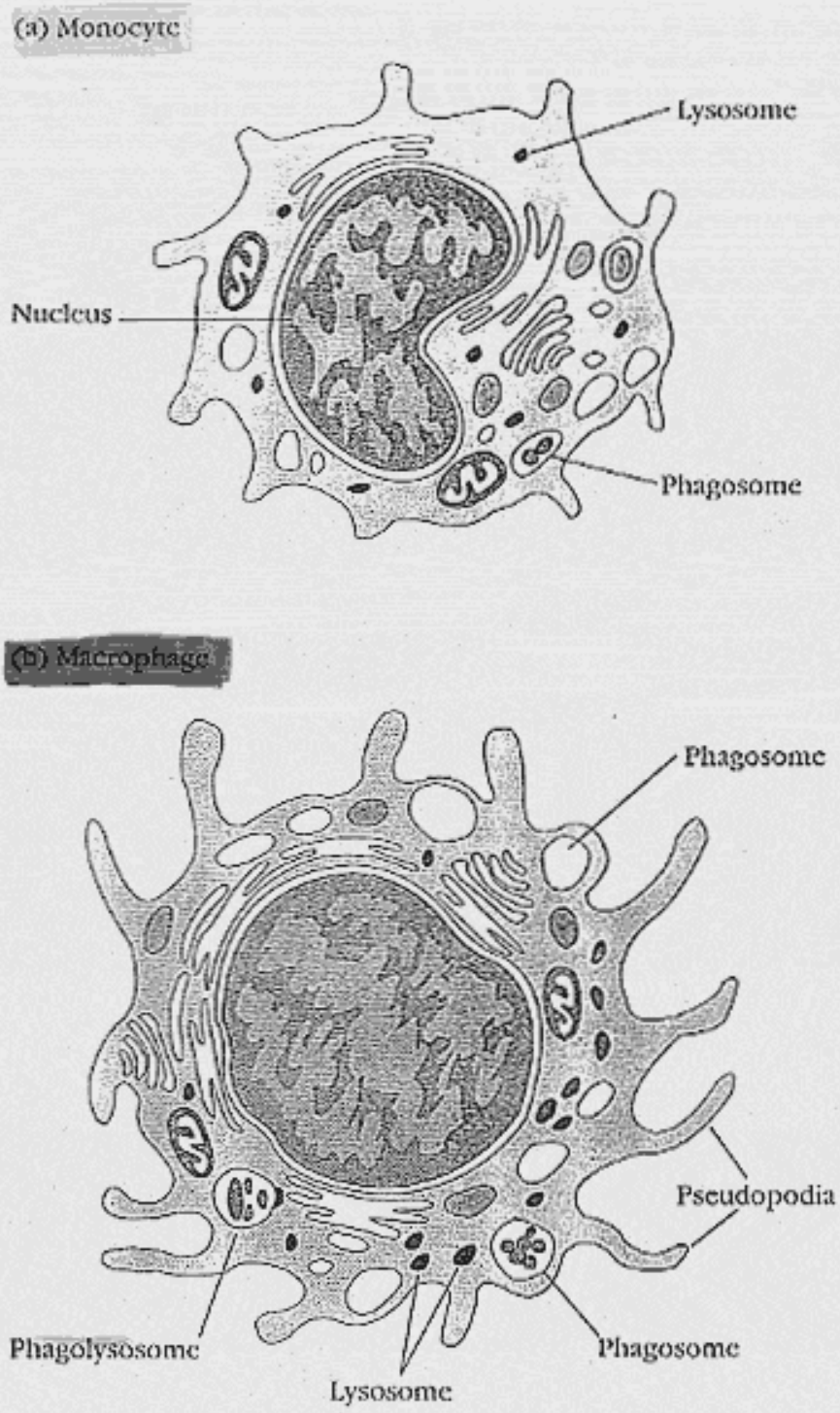
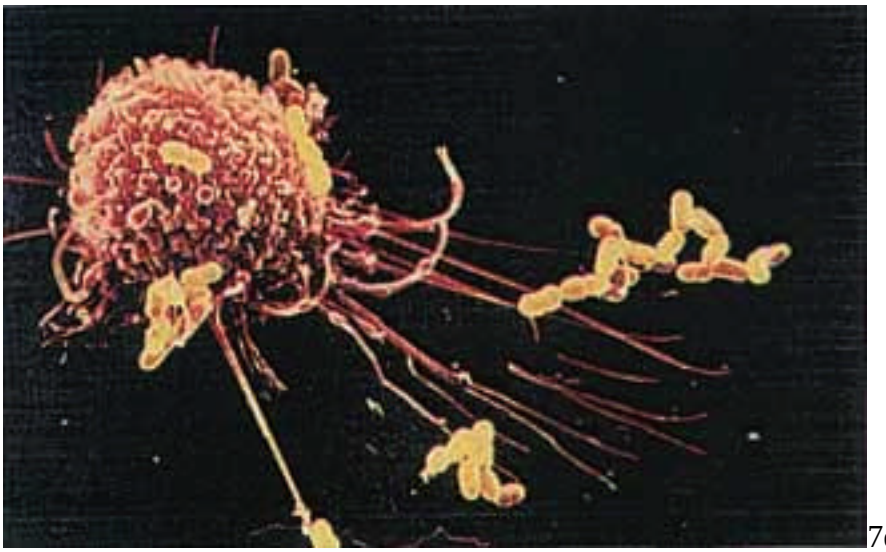
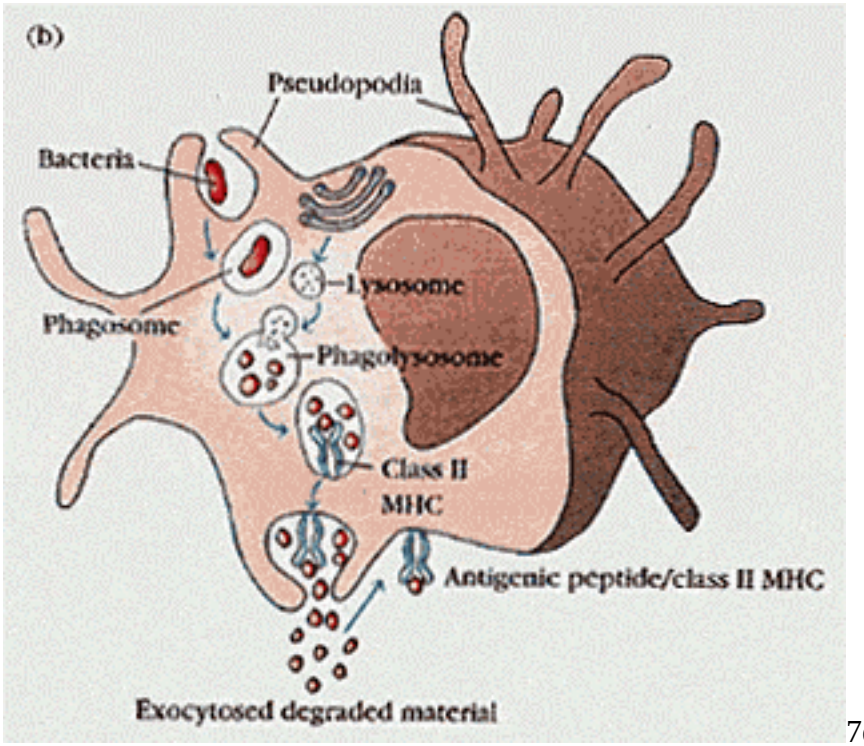



FIGURE 3-11

Drawings showing typical morphology of a monocyte and macrophage. Macrophages are five- to tenfold larger than monocytes and contain more organelles, especially lysosomes.


7e-1


(b)
Pseudopodia
Bacteria
Lysosome
Phagosome
Phagolysosome
Class II
MHC
Antigenic peptide/class II MHC
Exocytosed degraded material
7e-2

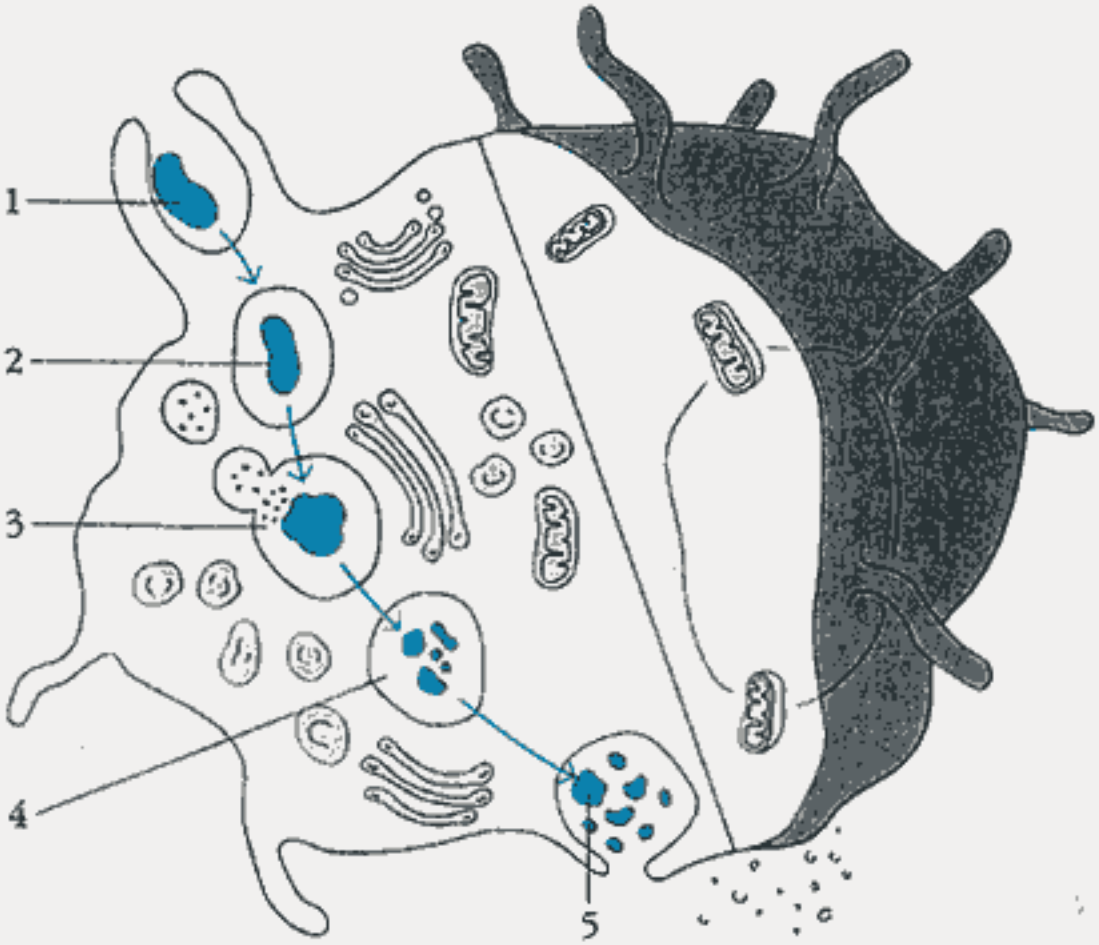
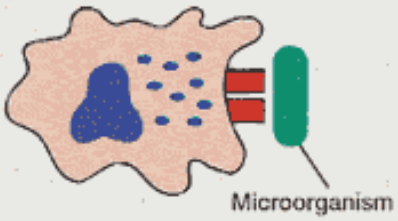
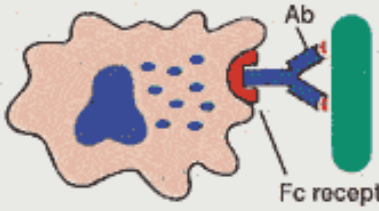
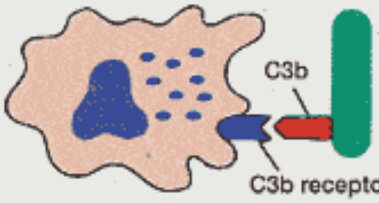
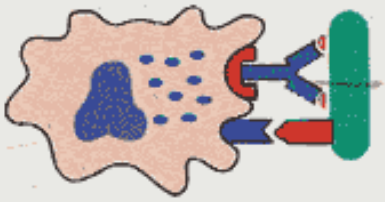



FIGURE 1-4

Phagocytosis of bacteria. Schematic diagram of the steps in phagocytosis: (1) attachment of a bacterium (blue) to long membrane evaginations, called pseudopodia; (2) ingestion of bacterium forming a phagosome, which moves toward a lysosome; (3) fusion of the lysosome and phagosome, releasing lysosomal enzymes into the phagosome; (4) digestion of ingested material; and (5) release of digestion products from the cell.

| Phagocytic cell | Degree of binding | Opsonin |
|---|---|---|
|  (a) Attachment by nonspecific receptors | ± | – |
|  (b) | + | Antibody |
|  (c) | + + | Complement C3b |
|  (d) | + + + + | Antibody and complement C3b |

7g

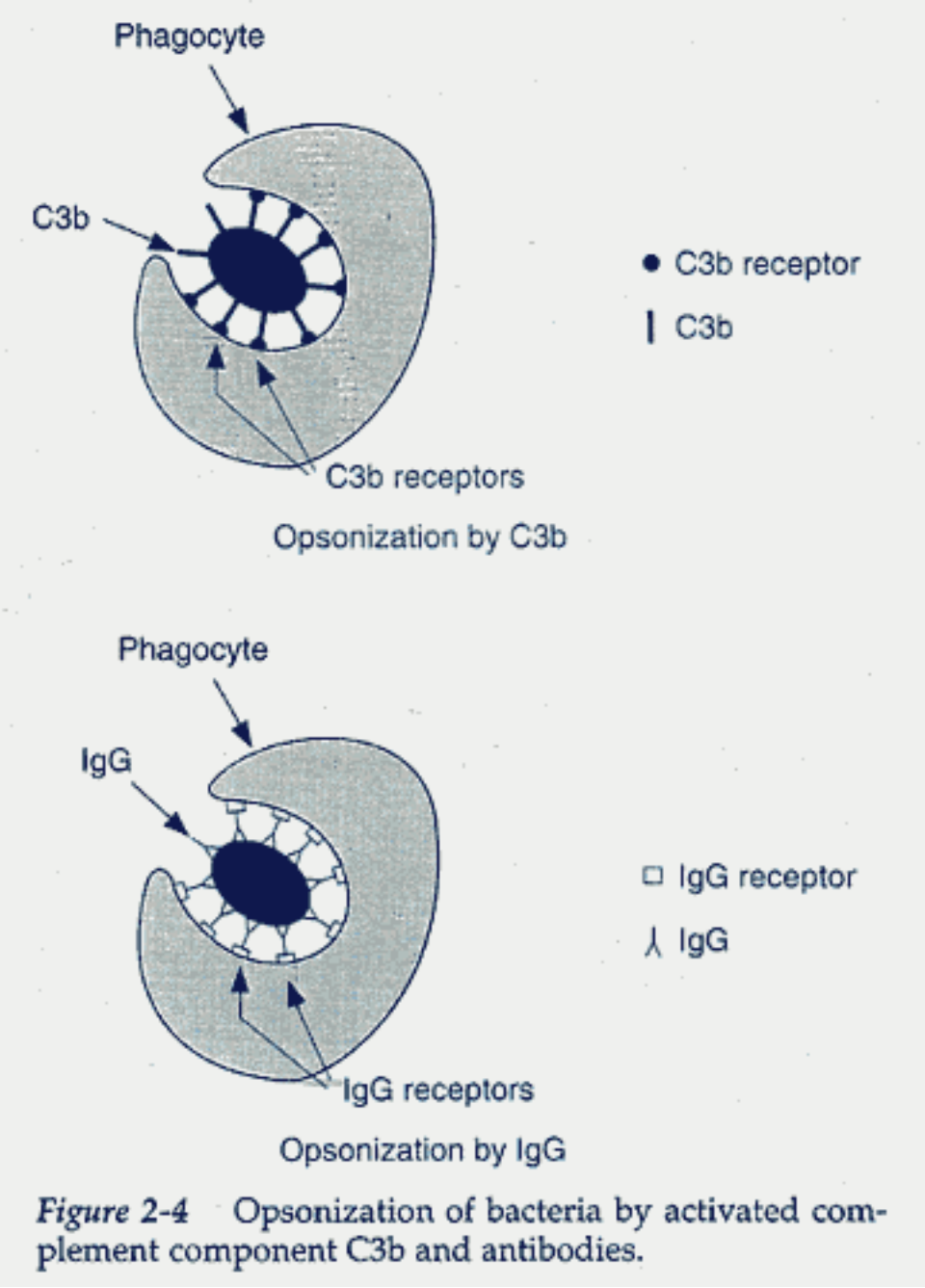



*Figure* 2-4 Opsonization of bacteria by activated complement component C3b and antibodies.

7h

## Effect of antibody and complement on rate of clearance of virulent bacteria from the blood

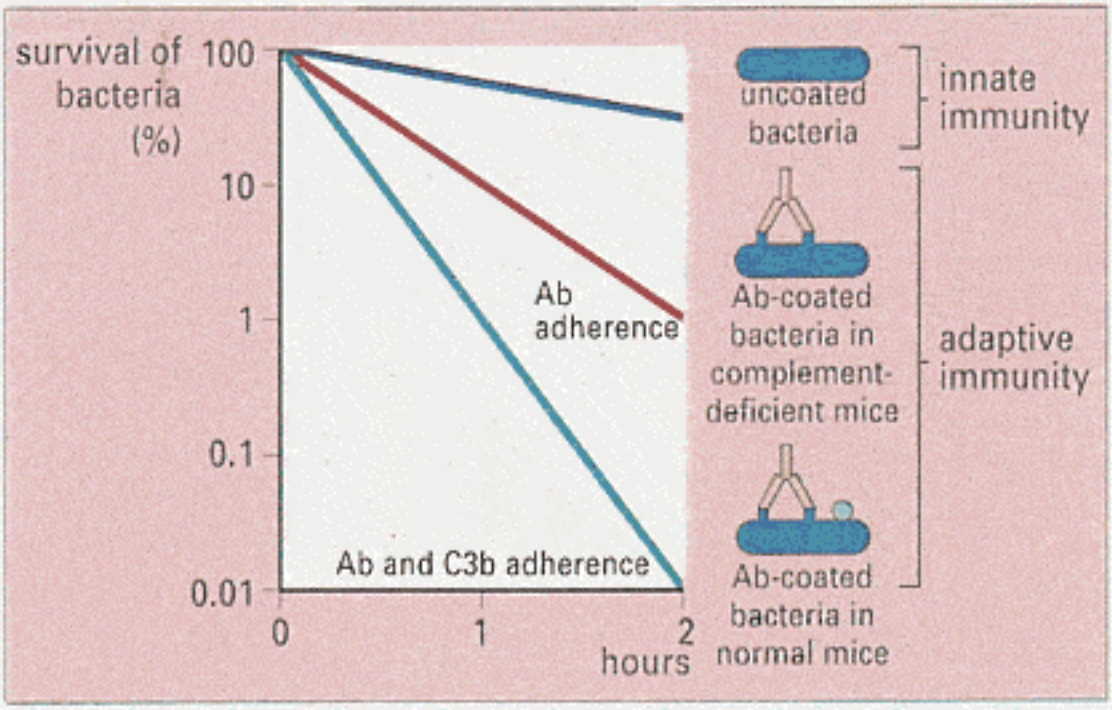



**Fig. 17.6** Uncoated bacteria are phagocytosed rather slowly (unless the alternative pathway is activated by the strain of bacterium); on coating with antibody, adherence to phagocytes is increased many-fold. The adherence is somewhat less effective in animals temporarily depleted of complement.

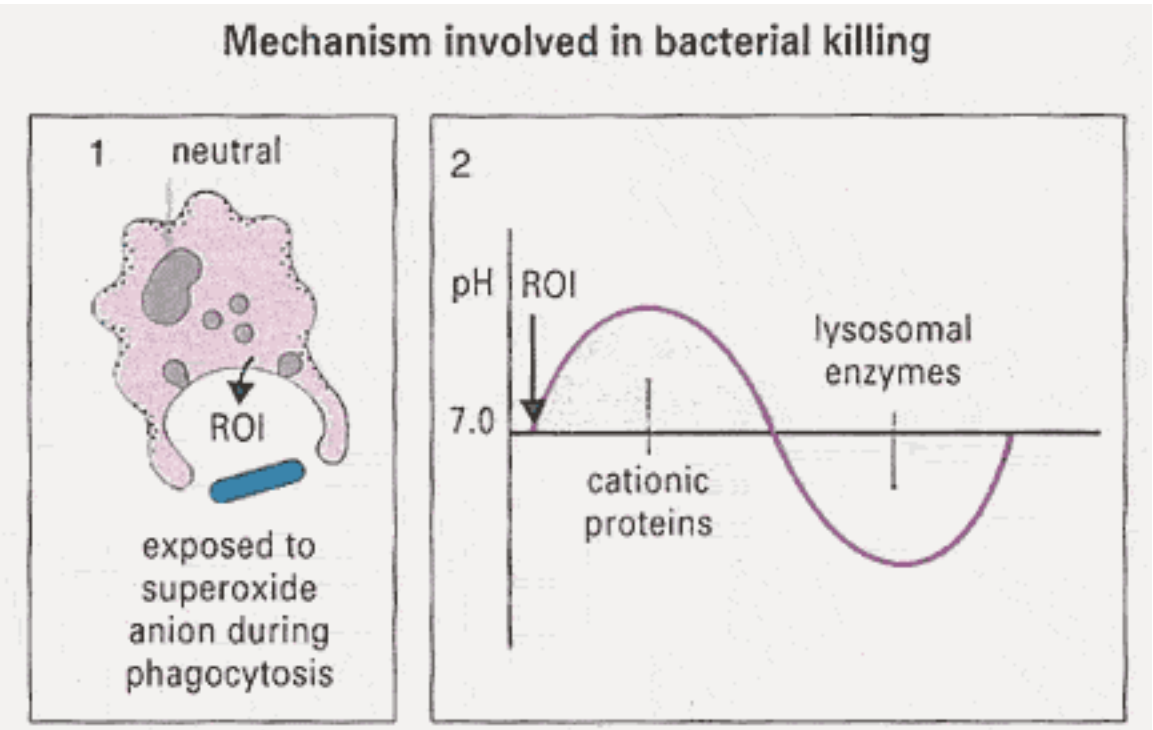



**Fig. 17.11** During phagocytosis there is immediate exposure to reactive oxygen intermediates (ROIs) (**1**). This leads to a transient increase in pH, when cationic proteins may be effective (**2**). Subsequently the pH falls, as $H^+$ ions are pumped into the phagolysosome, and lysosomal enzymes with low pH optima become effective. Lactoferrin acts by chelating free iron, and can do so at alkaline or acidic pH.

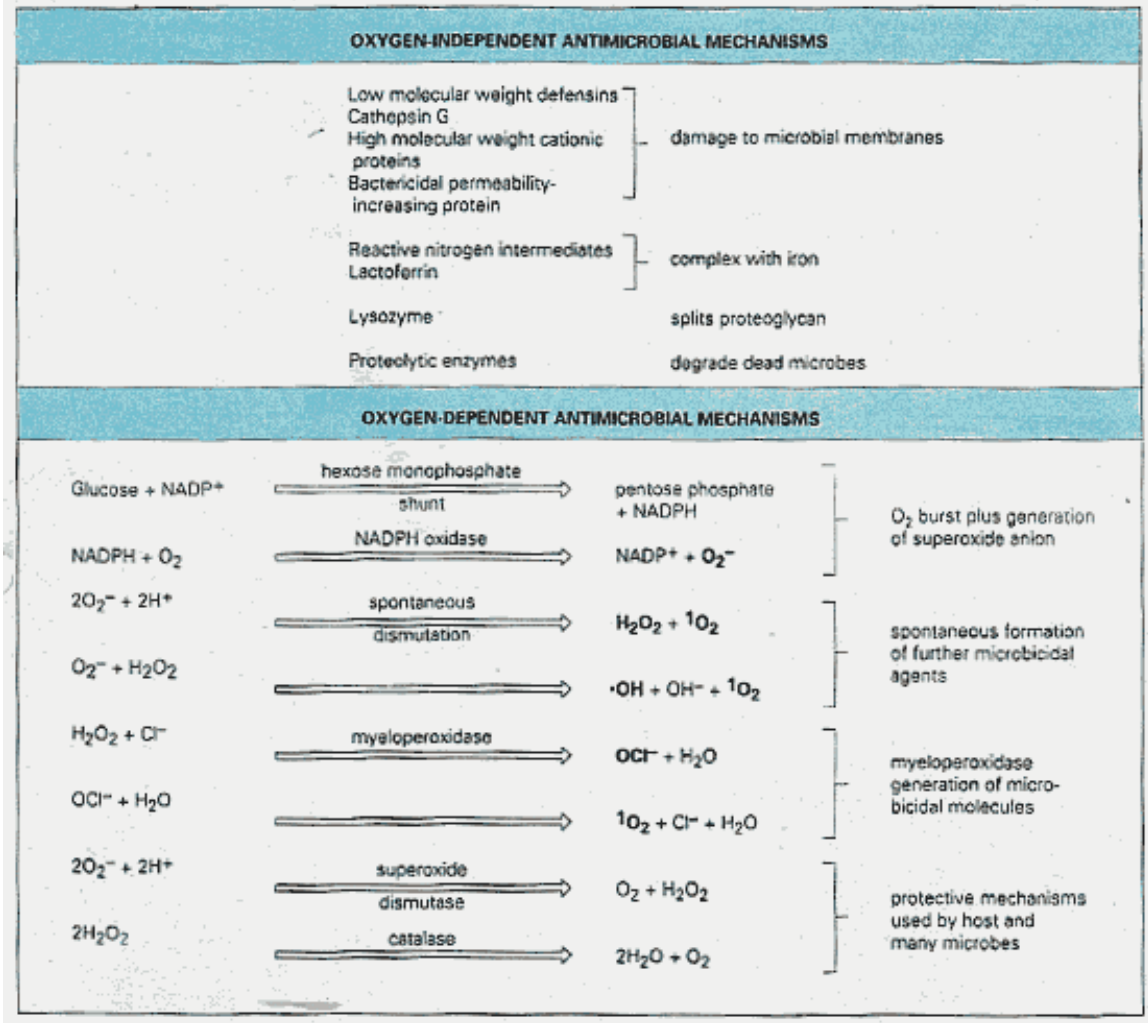



**Fig. 4.13** Antimicrobial mechanisms in phagocytic vacuoles. Microbicidal species in bold letters. $O_2^-$, superoxide anion; $^1O_2$, singlet (activated) oxygen; ·OH, hydroxyl free radical. Reactive nitrogen intermediates such as nitric oxide (NO) are derived from asparagine. NADPH, reduced nicotinamide adenine dinucleotide phosphate; $NAPD^+$, oxidized NADPH; $H_2O_2$, hydrogen peroxide.

7k

**Table 13-1. Major immunodeficiency states**

| |
|---|
| B cell and immunoglobulin deficiency, reflected by an increase in extracellular bacterial infections |
| Transient neonatal hypogammaglobulinemia |
| Bruton's X-linked agammaglobulinemia |
| Common variable hypogammaglobulinemia |
| Immunoglobulin A deficiency |
| T cell deficiencies, reflected by an increase in many viral, fungal, and intracellular bacterial infections |
| Nezelof syndrome |
| DiGeorge syndrome |
| Wiskott-Aldrich syndrome |
| Ataxia-telangiectasia |
| Purine enzyme deficiency (adenosine deaminase and purine nucleotide phosphorylase) |
| AIDS |
| Phagocytic deficiency, reflected by an increase in bacterial diseases |
| Chronic granulomatous disease |
| Chédiak-Higashi syndrome |
| Myeloperoxidase deficiency |
| Leukocyte adhesion deficiency |
| Lazy leukocyte syndromes |
| Glucose-6-phosphate dehydrogenase deficiency |
| Complement and complement regulatory deficiencies, reflected by an increase in bacterial infections |
| Hereditary angioneurotic edema |
| Paroxysmal nocturnal hemoglobinuria |
| Late-acting component deficiency |

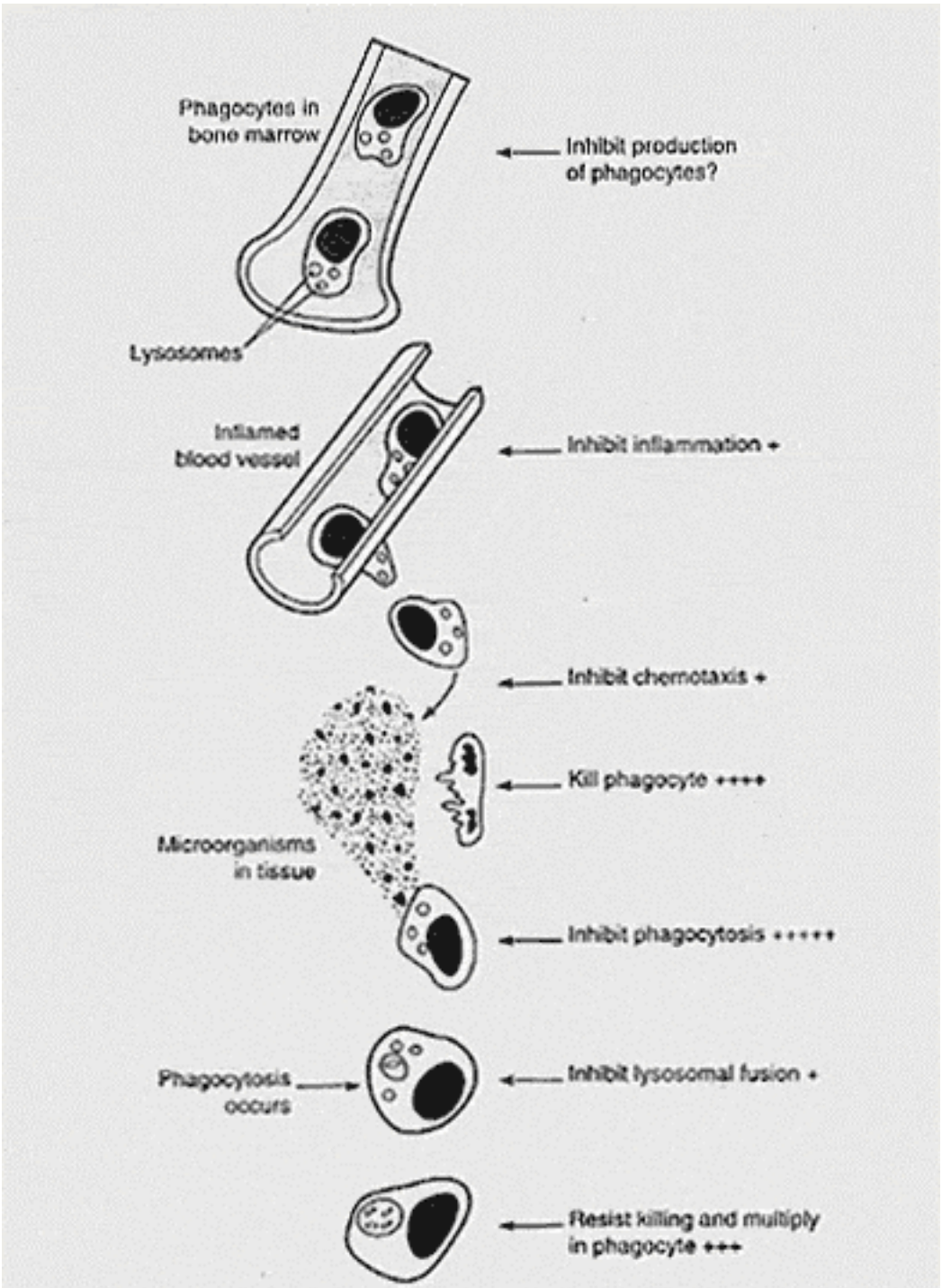



**Fig. 4.5** Antiphagocytic strategies available to microorganisms. Extent to which strategies are actually used by microorganisms are indicated by plusses.

7m

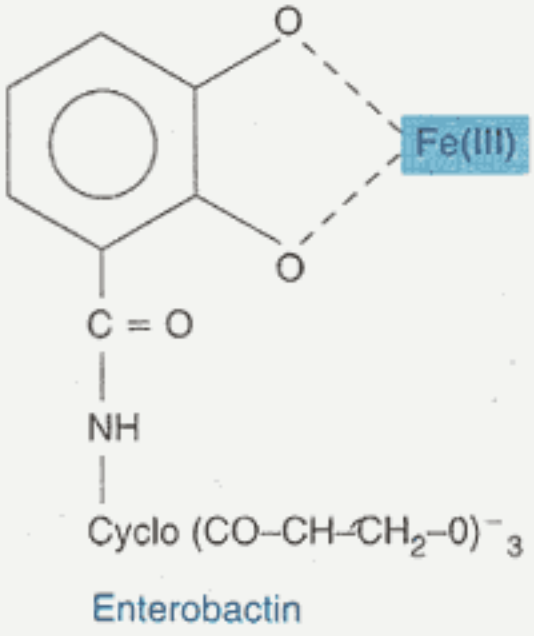



*Figure 3-5* Structure of one type of bacterial siderophore (enterobactin). Siderophores differ considerably in structure but are similar in that they are basically just iron chelators with a very high affinity for iron.

7n