

known as defensins are present at very high concentrations within the phagosome and insert themselves into the microbial membranes to form destabilising ion channels.

- Further damage is inflicted on the bacterial membrane by the neutral proteinase cathepsin G and via direct transfer to the microbial surface of a protein, which increases bacterial permeability.
- The enzyme lysozyme cleaves the peptidoglycan, a major component of bacterial cell walls.
- Lactoferrin, by complexing with iron, denies the growing bacteria an essential nutrient. It is said that nitric oxide behaves in the same way, but there are probably more complex mechanisms at work.
- The pH of the vacuole eventually drops and a variety of proteolytic and other hydrolytic enzymes digest the killed organisms.

Chronic granulomatous disease (CGD)

Although phagocytosis is rather ancient in evolutionary terms, the disease chronic granulomatous disease in which an inherited gene defect results in the inability of phagocytic cells to efficiently kill microorganisms vividly illustrates its continued importance to our survival.

Antibody and complement help phagocytosis

The phagocytosis of microorganisms by cells of the innate immune response can be greatly aided if the organism becomes coated or opsonised with antibody, produced by B-cells of adaptive immune response. The molecules coating the microorganism are known as opsonins and the process by which they coat is called opsonisation.

Many bacteria have a capsule on their surface. This capsule can protect the bacteria from phagocytosis. In the presence of only the innate immune system, such bacteria can survive relatively well. However, if antibodies were to coat the bacteria, then opsonisation would lead to the rapid clearance of the bacteria as the antibody would bind to bacterial surface antigens via its variable region and the Fc receptor on phagocytic cells via its constant region, thereby acting as a bridge.

The binding of a single antibody molecule to a phagocytic cell leads to only a low affinity interaction. However, bacteria usually possess multiple copies of surface antigens and therefore, can bind many antibody molecules of identical specificity. The bacterium coated with the many antibody molecules will form a high avidity multiple bond with the phagocytic cell. This high avidity multiple bond with cross-linking of the receptors leads to the production of a signal that is transduced into the phagocytic cell and leads to its activation and rapid phagocytosis of the opsonized bacteria.

In addition, when antibody binds to antigen, complement is activated and is able to opsonize microorganisms, further facilitating phagocytosis.

In addition, complement acts as a chemotactic factor to recruit the phagocytes to the site of infection and also causes mast cells to release histamines,