- Following the formation of the C3 convertase, a C5 covertase is formed by the addition of a newly active C3 to the convertase, which splits complement component C5 into C5a and C5b.
- C5b then forms a trimolecular complex with two further components C6 and C7. The complex inserts itself into the membrane and recruits the C8 molecule, which undergoes a conformational change before sitting inside the cell surface membrane at the side of the C5b67 complex.
- This In turn recruits the terminal component of the complement pathway, C9, which it turns inside out and converts to an amphipathic molecule capable of insertion into the lipid bilayer, where it can polymerize to form and annular membrane attack complex (MAC).
- The hydrophilic portions of the molecule now form a channel through the membrane allowing it to play a role in immunity.

Activities generated by the triggering of the complement cascade

The complement components involved in immune responses are C3a, C3b, C5a and the MAC.

- C3a and C5a and to a less of an extent, C4a, trigger the release of inflammatory mediators from tissue mast cells and blood basophils.
- C5a also acts as a chemotactic factor to attract phagocytic cells such as neutrophils to the site of infection.
- Complement component C3b (and C4b) can opsonise microorganisms and therefore bind to complement receptors on phagocytic cells, thereby enhancing phagocytosis.
- C3b can also form a component of immune complexes (antibodies bound to antigens), which are then bound to complement receptors on erythrocytes and transported to the liver and spleen for destruction of antigens by macrophages.
- The MAC inserts into bacterial membranes forming a channel through which small water-soluble solutes can flow, thereby upsetting the integrity of the cell and usually leading to lysis through osmotic influx of water and therefore leading to the death of the bacterium.
- It is known that complement component C3d is involved in activating B-cells. C3d is produced when C3b is cleaved into C3c and C3d.
- All of our cells are coated with complement regulatory proteins that prevent complement from damaging our cell.

Inflammation involves mediators released from tissue mast cells

C3a and C5a produced from the complement reaction can trigger degranulation of mast cells by binding to the surface receptors (encounter with PAMPs, damage to tissues and binding to IgE antibodies can also do this). This causes an increase in intracellular calcium and cAMP, which leads to the release of preformed granule contents to the exterior of the cell and activation of surface phospholipase A_2. This acts on arachidonic acid on the membrane to produce mediators, which belong to two different pathways.

- The lipoxygenase pathway