Classical pathway of complement activation

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The classical complement activation pathway is evolutionarily younger than the alternative.

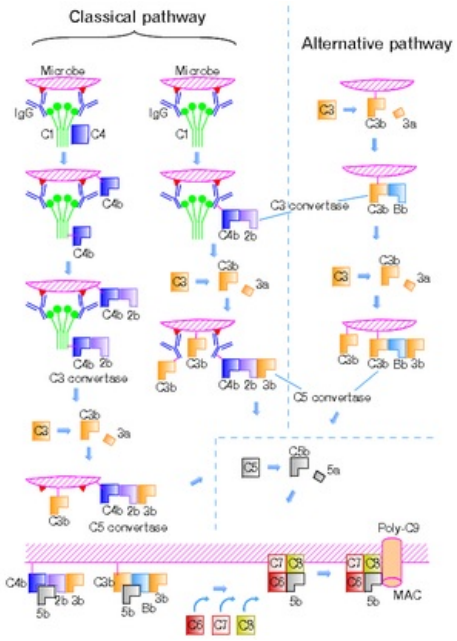
Initiated on surfaces where **antibodies** are bound (mainly IgG, IgM). Binding to a surface (e.g. bacteria) changes the conformation of the antibody molecule → reveals the binding site for **C1**. It starts by binding **C1q** to the **Fc** fragment of the immunoglobulin. **C1** (= C1q + x C1r + 2xC1s) also changes shape and becomes proteolytically active upon binding to the antibody → begins to cleave **C4** and **C2**. **C4b** and **C2a** bind to the surface of the challenged microorganism → forms ***classic C3-convertase*** → cleaves a lot of C3 into C3a and C3b. Then another enzyme is formed - ***classic C5-convertase*** (**C4bC3bC2a**) → cleaves C5 into **C5a** and **C5b**.

Pentraxins can also start the classical pathway: CRP, serum amyloid P (acute phase reactants).

Terminal (lytic) phase of the complement cascade

C5b forms a complex with other components - **C6, C7, C8**. This complex burrows into the surface of the lipid membrane of the attacked cell and attaches to a ring of **13-18 C9** molecules. A **pore** is formed in the membrane - cytoplasmic components leak out, osmotic balance is disturbed, cells can lyse.

Most microorganisms are **resistant** (protection by the cell wall).



Classical and alternative pathways of complement activation

Links

Related articles

- Complement system
- Alternative pathway for complement activation
- Mannose-binding lectin deficiency
- Lectin pathway of complement activation

External links

- HOŘEJŠÍ, Václav – BARTŮŇKOVÁ, Jiřina. *Základy imunologie.* 3. edition. Praha : Triton, 2008. 280 pp. ISBN 80-7254-686-4.