Gene name: **C3AR1**

External Ids for C3AR1 Gene : HGNC: 131 NCBI Gene: 719 Ensembl: ENSG00000171860 OMIM®: 605246 UniProtKB/Swiss-Prot: Q16581

NCBI Gene Summary: C3a is an anaphylatoxin released during activation of the **complement system**. The protein encoded by this gene is an orphan G protein-coupled receptor for C3a. Binding of C3a by the encoded receptor activates chemotaxis, granule enzyme release, superoxide anion production, and bacterial opsonization.

GeneCards Summary: C3AR1 (Complement C3a Receptor 1) is a Protein Coding gene. Diseases associated with C3AR1 include Atypical Hemolytic-Uremic Syndrome and Prostate Cancer. Among its related pathways are Class A/1 (Rhodopsin-like receptors) and Complement cascade. Gene Ontology (GO) annotations related to this gene include *G protein-coupled receptor activity* and *complement component C3a receptor activity*. An important paralog of this gene is C5AR1.

UniProtKB/Swiss-Prot Summary: Receptor for the chemotactic and inflammatory peptide anaphylatoxin C3a. This receptor stimulates chemotaxis, granule enzyme release and superoxide anion production. (C3AR_HUMAN,Q16581)

Cellular localization: Cell membrane; Multi-pass membrane protein is the most important.

Full name: Complement component 3a receptor 1 (C3AR1).

Receptor type: G protein-coupled receptor (GPCR).

Ligand: C3a, an anaphylatoxin generated during activation of the **complement system.**

Expression: Highly expressed on myeloid cells — particularly neutrophils, monocytes/macrophages, dendritic cells, and some endothelial and epithelial cells.

🧬 Biological Function of C3AR1

- Binds **C3a**, a cleavage product of complement protein C3
- Activates **pro-inflammatory signaling cascades**, leading to:
    - **Chemotaxis** of neutrophils and macrophages
    - **Release of reactive oxygen species (ROS)**
    - **Cytokine and chemokine secretion**
    - **Increased vascular permeability**
- Plays a key role in **innate immunity,** particularly in **early response to infection.**

🦠 Role in Sepsis

Sepsis is a dysregulated immune response to infection, and C3AR1 is central to the hyperinflammatory phase:

Early-phase sepsis (hyperinflammatory):

- C3AR1 is upregulated in sepsis
- Drives excessive neutrophil activation and cytokine storms
- Associated with endothelial damage, vascular leakage, and multi-organ dysfunction

Later-phase (immunosuppression):

- Some studies show compensatory downregulation or desensitization of C3AR1 as a mechanism to prevent tissue damage.

📈 Evidence in Sepsis Studies

Diagnostic Value

- C3AR1 gene expression is significantly upregulated in whole blood transcriptome studies of sepsis patients compared to healthy controls.
- Detected early in sepsis onset, including in neonates and adults.

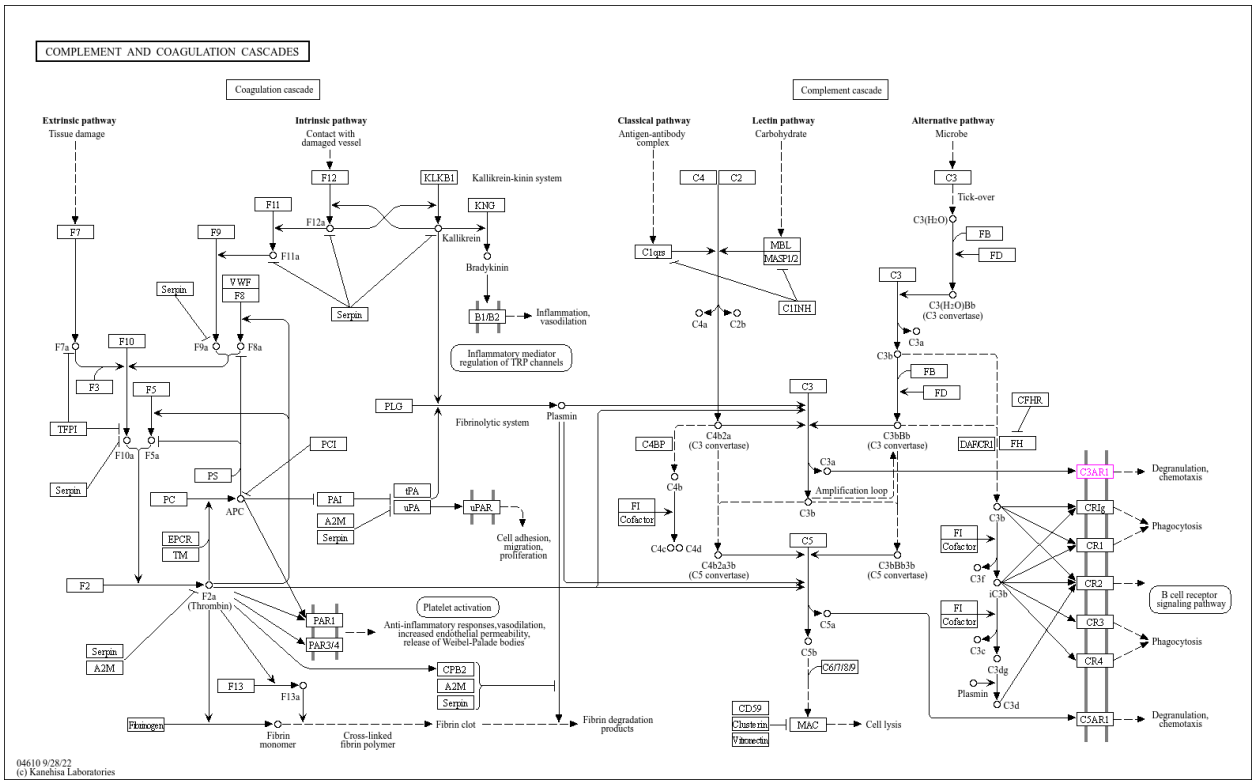
Prognostic Value

- High C3AR1 expression is associated with:
    - Increased mortality risk.
    - Organ failure severity (SOFA scores).
- May help predict poor outcomes, especially in patients with Gram-negative bacteremia or systemic inflammation.

📚 Supporting Literature

DOI: 10.1097/MD.0000000000037519
DOI: 10.1038/s41598-024-59400-0
DOI: 10.3389/fimmu.2019.00543
DOI: 10.1111/jth.12956

KEGG :



Enrichr-KG results: