

Gene name: **CCR7** Previous HGNC Symbols for CCR7 Gene: CMKBR7, EBI1

External Ids for CCR7 Gene: HGNC: [1608](#) NCBI Gene: [1236](#) Ensembl: [ENSG00000126353](#)

OMIM®: [600242](#) UniProtKB/Swiss-Prot: [P32248](#)

NCBI Gene Summary: The protein encoded by this gene is a member of the G protein-coupled receptor family. This receptor was identified as a gene induced by the Epstein-Barr virus (EBV), and is thought to be a mediator of EBV effects on B lymphocytes. This receptor is expressed in various lymphoid tissues and activates B and T lymphocytes. It has been shown to **control the migration of memory T cells to inflamed tissues, as well as stimulate dendritic cell maturation**. The chemokine (C-C motif) ligand 19 (CCL19/ECL) has been reported to be a specific ligand of this receptor. Signals mediated by this receptor regulate T cell homeostasis in lymph nodes, and may also function in the activation and polarization of T cells, and in chronic inflammation pathogenesis. Alternative splicing of this gene results in multiple transcript variants.

GeneCards Summary: CCR7 (C-C Motif Chemokine Receptor 7) is a Protein Coding gene. Diseases associated with CCR7 include [Chronic Graft Versus Host Disease](#) and [Tetanus](#). Among its related pathways are [Class A/1 \(Rhodopsin-like receptors\)](#) and [GPCR downstream signalling](#). Gene Ontology (GO) annotations related to this gene include *G protein-coupled receptor activity* and *chemokine (C-C motif) ligand 19 binding*. An important paralog of this gene is [CCR9](#).

UniProtKB/Swiss-Prot Summary: Receptor for the MIP-3-beta chemokine. Probable mediator of EBV effects on B-lymphocytes or of normal lymphocyte functions. ([CCR7_HUMAN,P32248](#))

Cellular localization: mainly in mitochondria and plasma membranes.

Full Name: *C-C Motif Chemokine Receptor 7*

Protein Type: G protein-coupled receptor (GPCR)



Biological Function of CCR7

- CCR7 encodes a chemokine receptor that plays a central role in:
 - Immune cell trafficking
 - Lymph node homing
 - T cell and dendritic cell migration
- Ligands for CCR7:
 - CCL19 (MIP-3β)
 - CCL21 (SLC)
 - Both are expressed in lymphoid tissues, especially the T cell zones of lymph nodes and high endothelial venules (HEVs).

Main Actions of CCR7:

- Guides immune cells to secondary lymphoid organs:
 - Directs naive T cells, central memory T cells, and dendritic cells (DCs) to lymph nodes.
- Facilitates antigen presentation:
 - Enables DCs to migrate from infected tissues to lymph nodes where they prime T cells.
- Helps organize the architecture of lymphoid tissues:
 - Maintains the structure and function of T-cell zones in lymph nodes and spleen.
- Supports immune surveillance and the initiation of adaptive immune responses.

Role of CCR7 in Sepsis

- In sepsis, CCR7 is often dysregulated, which can alter immune cell localization and function:

Early Sepsis:

- CCR7 expression on DCs and T cells is critical for effective immune priming.
- Enables pathogen-loaded DCs to reach lymph nodes and activate T cells.

In Prolonged or Severe Sepsis:

- Downregulation of CCR7 can impair:
 - DC migration → reduced antigen presentation.
 - T cell homing → weakened adaptive immune response.
 - Lymphoid tissue function → contributes to immune paralysis.
- Conversely, overexpression of CCR7 in some contexts can enhance inflammation and immune cell recruitment, potentially exacerbating systemic inflammation and tissue damage.

Clinical Relevance of CCR7 in Sepsis

Diagnostic Role:

- Changes in CCR7 expression can serve as a marker of immune dysfunction in septic patients.
- Low CCR7 levels on DCs and T cells may indicate loss of immune coordination and dysfunctional antigen presentation.

Prognostic Role:

- Impaired CCR7 expression correlates with:
 - Immunosuppression
 - Higher risk of secondary infections
 - Poor clinical outcomes

- Strategies to restore CCR7 signaling may enhance immune responsiveness.
- Targeting CCR7 could help regulate immune cell trafficking to limit harmful inflammation or enhance pathogen clearance.

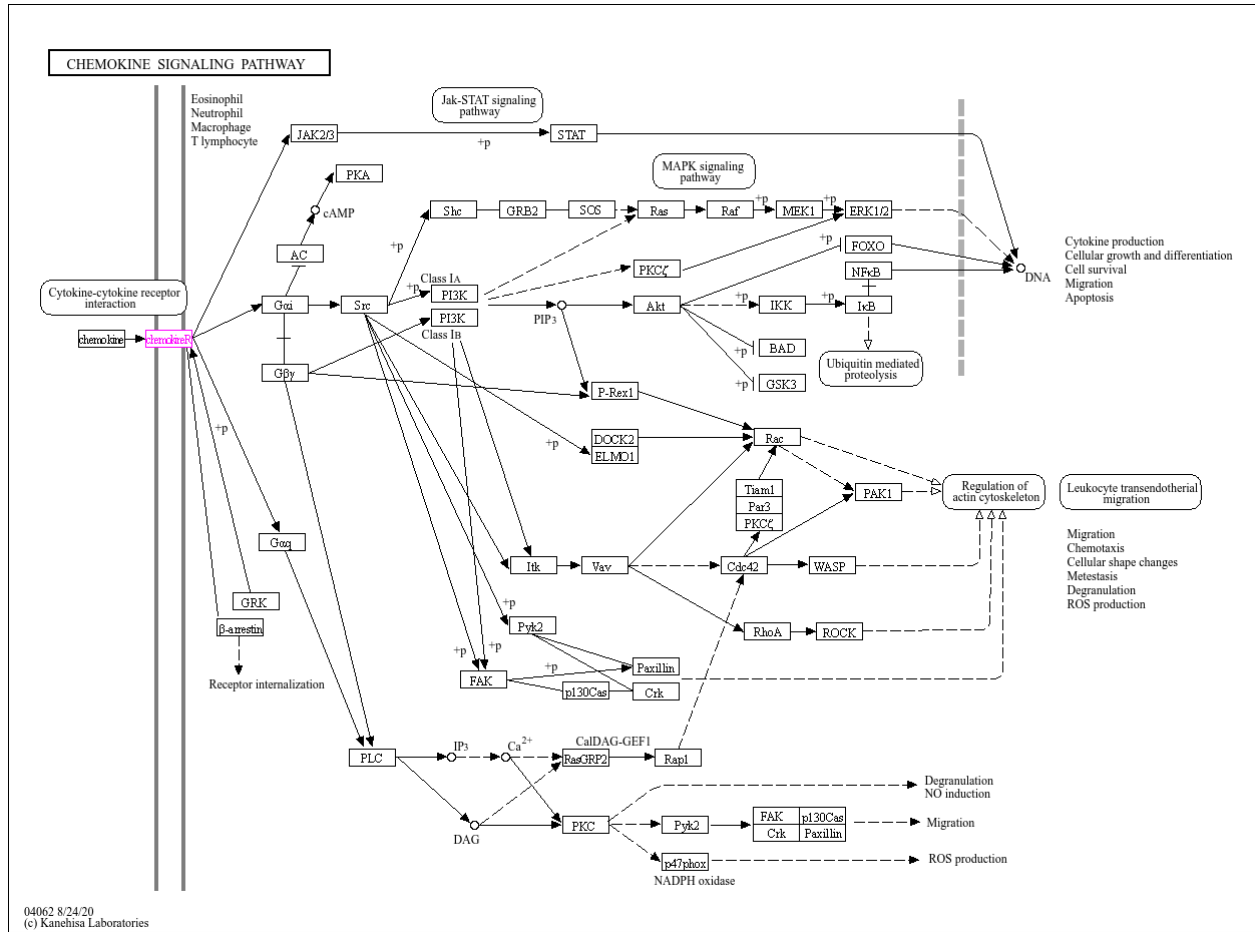


- ## Supporting Literature

Doi: 10.1155/2013/164246

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KEGG:



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