

Gene name: **TREM1**

External Ids for TREM1 Gene: HGNC: [17760](#) NCBI Gene: [54210](#) Ensembl: [ENSG00000124731](#)
OMIM®: [605085](#) UniProtKB/Swiss-Prot: [Q9NP99](#)

NCBI Gene Summary: This gene encodes a receptor belonging to the Ig superfamily that is expressed on myeloid cells. This protein amplifies neutrophil and monocyte-mediated inflammatory responses triggered by bacterial and fungal infections by stimulating release of pro-inflammatory chemokines and cytokines, as well as increased surface expression of cell activation markers. Alternatively spliced transcript variants encoding different isoforms have been noted for this gene.

GeneCards Summary: TREM1 (Triggering Receptor Expressed On Myeloid Cells 1) is a Protein Coding gene. Diseases associated with TREM1 include [Maxillary Sinusitis](#) and [Pneumonia](#). Among its related pathways are [Innate Immune System](#) and [Toll-like receptor signaling pathway](#). Gene Ontology (GO) annotations related to this gene include *signaling receptor activity* and *scaffold protein binding*.

UniProtKB/Swiss-Prot Summary: [Isoform 1]: Cell surface receptor that plays important roles in innate and adaptive immunity by amplifying inflammatory responses (PubMed:[10799849](#), [21393102](#)). Upon activation by various ligands such as PGLYRP1, HMGB1 or HSP70, multimerizes and forms a complex with transmembrane adapter TYROBP/DAP12 (PubMed:[17568691](#), [25595774](#), [29568119](#)). In turn, initiates a SYK-mediated cascade of tyrosine phosphorylation, activating multiple downstream mediators such as BTK, MAPK1, MAPK3 or phospholipase C-gamma (PubMed:[14656437](#), [21659545](#)). This cascade promotes the neutrophil- and macrophage-mediated release of pro-inflammatory cytokines and/or chemokines, as well as their migration and thereby amplifies inflammatory responses that are triggered by bacterial and fungal infections (PubMed:[17098818](#), [17568691](#)). By also promoting the amplification of inflammatory signals that are initially triggered by Toll-like receptor (TLR) and NOD-like receptor engagement, plays a major role in the pathophysiology of acute and chronic inflammatory diseases of different etiologies including septic shock and atherosclerosis (PubMed:[11323674](#), [21393102](#)). ([TREM1_HUMAN,Q9NP99](#)) [Isoform 2]: Acts as a decoy receptor, counterbalancing TREM1 pro-inflammatory activity through the neutralization of its ligand. ([TREM1_HUMAN,Q9NP99](#)).

Gene Name: TREM1 (Triggering Receptor Expressed on Myeloid Cells 1).
TREM1 is an immunoreceptor primarily expressed on myeloid cells, such as: Neutrophils, Monocytes/macrophages. It acts as an **amplifier of the innate immune response**, especially during bacterial or fungal infections.



Molecular Function

- TREM1 is part of the immunoglobulin superfamily.
- It does not have a signaling domain itself, but signals via DAP12 (TYROBP), a protein with an ITAM (immunoreceptor tyrosine-based activation motif) that triggers intracellular activation.
- When activated, it induces secretion of pro-inflammatory cytokines like: TNF- α , IL-6, IL-8, MCP-1.

TREM1 in Sepsis

TREM1 is strongly upregulated during sepsis and other systemic inflammatory responses, particularly in:


- Sepsis-induced acute lung injury, Bacteremia and Septic shock. It works synergistically with Toll-like receptors (TLRs) – especially TLR4, amplifying their response to pathogen-associated molecular patterns (PAMPs).


 Inhibiting TREM1 signaling (such as, with TREM1 decoy peptides like LP17) in mouse models:

- Reduces cytokine storm
- Improves survival
- Prevents tissue damage

Diagnostic/Prognostic Value

- sTREM1 (soluble form of TREM1) can be measured in plasma or serum, and:
 - Is elevated in sepsis.
 - Correlates with severity (SOFA score, mortality risk).
 - May help differentiate bacterial infection from non-infectious inflammation.

 Clinical trials are exploring sTREM1 as a biomarker for: Early sepsis detection, Monitoring disease progression, Assessing response to treatment

 **Supporting Literature** Doi: 10.1016/j.cellimm.2011.10.006

Doi: 10.3389/fimmu.2022.907387

Doi:10.1002/eji.200636387

Enrichr-KG:

