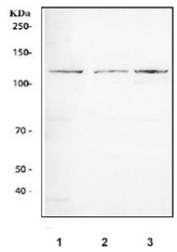
TRIF Antibody / TICAM1 (R32274)

Catalog No.	Formulation	Size
R32274	0.5mg/ml if reconstituted with 0.2ml sterile DI water	100 ug

Bulk quote request

Availability	1-3 business days
Species Reactivity	Mouse, Rat
Format	Antigen affinity purified
Host	Rabbit
Clonality	Polyclonal (rabbit origin)
Isotype	Rabbit IgG
Purity	Antigen affinity
Buffer	Lyophilized from 1X PBS with 2% Trehalose
UniProt	Q80UF7
Applications	Western blot: 0.5-1ug/ml
Limitations	This TRIF antibody is available for research use only.



Western blot testing of 1) rat testis, 2) mouse testis and 3) mouse thymus tissue lysate with TRIF antibody. A single TRIF band was detected between ~100-110 kDa, migrating above the predicted 76 kDa, consistent with the well-documented phosphorylation-dependent and proline-rich region-related upward mobility of endogenous TRIF reported in the literature.

Description

TRIF antibody detects TIR-domain-containing adaptor molecule 1 (TICAM1), commonly known as TRIF, a critical adaptor protein in the Toll-like receptor (TLR) signaling pathway. The UniProt recommended name is TIR domain-containing adapter molecule 1 (TICAM1). TRIF functions as a central mediator of innate immune responses by linking pattern-recognition receptor activation to type I interferon production, inflammatory gene expression, and antiviral defense. As the primary adaptor for TLR3 and the alternative adaptor for TLR4, TRIF integrates extracellular and intracellular pathogen-sensing cues to shape cytokine responses across immune and non-immune cell types.

Functionally, TRIF antibody identifies a 712-amino-acid cytoplasmic protein containing a Toll/interleukin-1 receptor (TIR) domain essential for interactions with upstream receptors and downstream effectors. Following TLR3 activation by double-stranded RNA or TLR4 activation through endosomal signaling, TRIF recruits proteins such as TRAF3, TBK1, IRF3, RIPK1, and NF-kappaB-activating adaptors. This cascade results in the induction of type I interferons, chemokines, costimulatory molecules, and proinflammatory cytokines. TRIF uniquely enables the antiviral interferon response downstream of TLR3, while its TLR4-associated signaling complements MyD88-dependent pathways to provide a balanced inflammatory output. TRIF also regulates programmed cell death pathways, including necroptosis, through interactions with RIPK1 and RIPK3, linking innate sensing to cell-fate decisions.

The TICAM1 gene is located on chromosome 19p13.3 and is expressed broadly in immune tissues such as spleen, lymph nodes, dendritic cells, macrophages, and microglia, as well as in non-immune cells that rely on TLR3-mediated antiviral responses, including epithelial cells and fibroblasts. Within dendritic cells, TRIF plays a central role in promoting maturation, antigen processing, and cross-priming of CD8-positive T cells. In epithelial barriers, TRIF-mediated TLR3 signaling supports antiviral immunity by inducing interferon-stimulated genes, restricting viral replication, and activating paracrine defense programs.

TRIF's cellular localization is predominantly cytoplasmic, where it interacts with activated TLRs following ligand-induced receptor endocytosis. Its activity is regulated through proteolytic processing, ubiquitination, and phosphorylation by kinases associated with antiviral signal transduction. Depending on cellular context, TRIF may function not only in host defense but also in maintaining epithelial barrier integrity and modulating inflammatory thresholds during chronic stimulation.

Pathologically, dysregulation of TRIF signaling contributes to a spectrum of immune and inflammatory diseases. Gain-of-function or sustained TRIF activation amplifies interferon signaling and proinflammatory gene expression, contributing to chronic inflammation, autoimmune activation, and tissue injury. Enhanced TRIF pathway activity has been observed in disorders such as viral encephalitis, inflammatory bowel disease, and certain autoinflammatory syndromes. Conversely, loss-of-function mutations or impaired TRIF signaling weaken antiviral immunity, leading to increased susceptibility to RNA virus infections and defective dendritic cell maturation. In cancer biology, TRIF influences tumor-associated inflammation, antitumor immune priming, and responses to nucleic acid-based therapeutics, underscoring its relevance in immunotherapy research. TRIF also participates in cell death pathways; excessive TRIF-RIPK signaling may contribute to tissue injury in sepsis, shock, and neurodegenerative conditions through induction of necroptosis.

Research using TRIF antibody supports investigations in innate immunity, pathogen recognition, interferon biology, and programmed cell death. TRIF antibody is validated for use in relevant research applications to detect TICAM1 expression and examine its involvement in TLR signaling, antiviral responses, and immune regulation. NSJ Bioreagents provides TRIF antibody reagents optimized for immunology, virology, inflammation, and cell signaling studies.

Application Notes

Optimal dilution of the TRIF antibody should be determined by the researcher.

Immunogen

Amino acids QDTEARVSLESLKMNTVAQLVAHQWADMETTE of mouse TRIF were used as the immunogen for the TRIF antibody.

Storage

After reconstitution, the TRIF antibody can be stored for up to one month at 4oC. For long-term, aliquot and store at -20oC. Avoid repeated freezing and thawing.

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