Technical Data Sheet Purified Mouse Anti-Human LITAF

Product Information

611614
LPS Induced TNFa Factor
50 µg
250 µg/ml
30/LITAF
Human LITAF aa. 1-212
Mouse IgG1
QC Testing: Human
24 kDa
Aqueous buffered solution containing BSA, glycerol, and $\leq 0.09\%$ sodium azide.

Description

TNF α (Tumor Necrosis Factor α) is a pleiotropic cytokine that conveys either beneficial (prevention of cancers and infection, activation of inflammatory responses) or detrimental (mediation of septic shock, inflammation, and apoptosis) effects to the host. It is produced and secreted primarily by monocytes and macrophages in response to stimulation with lipopolysaccharide (LPS), a principle endotoxic component. LPS-induced TNF α factor (LITAF) is a nuclear factor that was purified as a result of its ability to bind to a DNA fragment from the TNF α promoter region. LITAF expression is dependent on LPS induction and PMA differentiation in THP-1 cells and is observed at high levels in spleen, lymph node, and peripheral blood leukocytes. Antisense inhibition of LITAF causes a reduction in TNF α expression in THP-1 cells. In addition, p53 induces the expression of a transcript called PIG7, which has 98% homology with LITAF. Thus, LITAF is thought to link p53 pathways with the induction of TNF α gene expression in response to LPS stimulation and to function, possibly as a transcription factor, to regulate the expression of this highly potent cytokine. This antibody was generated to the LITAF aa 1-212 sequence (Genbank accession # U77396). Recent reports indicate that the nucleotide coding sequence for LITAF may differ and instead, encode for a protein designated as SIMPLE. Under the sequence for SIMPLE, the LITAF aa 1-212 sequence used to generate this antibody corresponds to the aa 1-126 region of SIMPLE.

This antibody is routinely tested by western blot analysis. Other applications were tested at BD Biosciences Pharmingen during antibody development only or reported in the literature.





Immunofluorescence staining of A431 cells (Human

epithelial carcinoma; ATCC CRL-1555).

Western blot analysis of LITAF on a A431 cell lysate (Human epithelial carcinoma; ATCC CRL-1555). Lane 1: 1:5000, lane 2: 1:10,000, lane 3: 1:20,000 dilution of the mouse anti- human LITAF antibody.

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Preparation and Storage

The monoclonal antibody was purified from tissue culture supernatant or ascites by affinity chromatography. Store undiluted at -20° C.

Application Notes

Application

Western blot	Routinely Tested
Immunofluorescence	Tested During Development

Recommended Assay Procedure:

Western blot: Please refer to http://www.bdbiosciences.com/pharmingen/protocols/Western_Blotting.shtml

Suggested Companion Products

Catalog Number	Name	Size	Clone
611447	A431 Cell Lysate	500 μg	(none)
554002	HRP Goat Anti-Mouse Igs	1.0 ml	(none)
554001	FITC Goat Anti-Mouse Igs	0.5 mg	Polyclonal

Product Notices

1. Since applications vary, each investigator should titrate the reagent to obtain optimal results.

Please refer to www.bdbiosciences.com/pharmingen/protocols for technical protocols. 2.

- 3. Caution: Sodium azide yields highly toxic hydrazoic acid under acidic conditions. Dilute azide compounds in running water before discarding to avoid accumulation of potentially explosive deposits in plumbing.
- Source of all serum proteins is from USDA inspected abattoirs located in the United States. 4.

References

Moriwaki Y, Begum NA, Kobayashi M, Matsumoto M, Toyoshima K, Seya T. Mycobacterium bovis Bacillus Calmette-Guerin and its cell wall complex induce a novel lysosomal membrane protein, SIMPLE, that bridges the missing link between lipopolysaccharide and p53-inducible gene, LITAF(PIG7), and estrogen-inducible gene, EET-1. J Biol Chem. 2001; 276(25):23065-23076.(Immunogen)

Myokai F, Takashiba S, Lebo R, Amar S. A novel lipopolysaccharide-induced transcription factor regulating tumor necrosis factor alpha gene expression: molecular cloning, sequencing, characterization, and chromosomal assignment. Proc Natl Acad Sci U S A. 1999; 96(8):4518-4523.(Biology) Saifi GM, Szigeti K, Wiszniewski W, et al. SIMPLE mutations in Charcot-Marie-Tooth disease and the potential role of its protein product in protein degradation. Hum Mutat. 2005; 25(4):372-383.(Immunogen)

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