

## DESCRIPTION

<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human LITAF in Western blots.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Antigen Affinity-purified
<b>Immunogen</b>	<i>E. coli</i> -derived recombinant human LITAF Met1-Ala111 Accession # Q99732
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

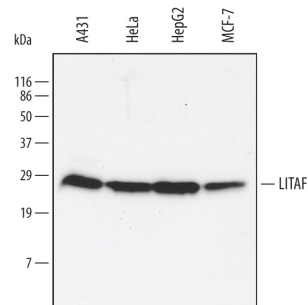
## APPLICATIONS

**Please Note:** Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Western Blot</b>	1 µg/mL	See Below
<b>Immunocytochemistry</b>	5-15 µg/mL	See Below
<b>Immunohistochemistry</b>	1-15 µg/mL	See Below

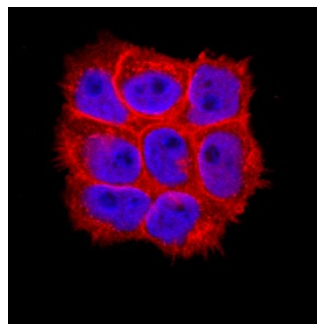
## DATA

### Western Blot



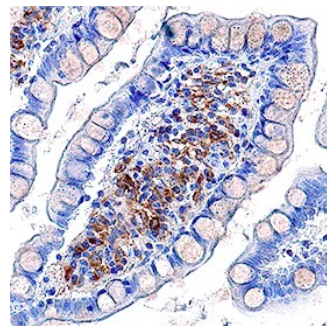
**Detection of Human LITAF by Western Blot.** Western blot shows lysates of A431 human epithelial carcinoma cell line, HeLa human cervical epithelial carcinoma cell line, HepG2 human hepatocellular carcinoma cell line, and MCF-7 human breast cancer cell line. PVDF membrane was probed with 1 µg/mL of Goat Anti-Human LITAF Antigen Affinity-purified Polyclonal Antibody (Catalog # AF4695) followed by HRP-conjugated Anti-Goat IgG Secondary Antibody (Catalog # HAF109). A specific band was detected for LITAF at approximately 28 kDa (as indicated). This experiment was conducted under reducing conditions and using [Immunoblot Buffer Group 1](#).

### Immunocytochemistry



**LITAF in A431 Human Cell Line.** LITAF was detected in immersion fixed A431 human epithelial carcinoma cell line using Goat Anti-Human LITAF Antigen Affinity-purified Polyclonal Antibody (Catalog # AF4695) at 10 µg/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Goat IgG Secondary Antibody (red; Catalog # NL001) and counterstained with DAPI (blue). Specific staining was localized to cell surfaces. View our protocol for [Fluorescent ICC Staining of Cells on Coverslips](#).

### Immunohistochemistry



**LITAF in Human Small Intestine.** LITAF was detected in immersion fixed paraffin-embedded sections of human small intestine using Goat Anti-Human LITAF Antigen Affinity-purified Polyclonal Antibody (Catalog # AF4695) at 1.7 µg/mL overnight at 4 °C. Tissue was stained using the Anti-Goat HRP-DAB Cell & Tissue Staining Kit (brown; Catalog # CTS008) and counterstained with hematoxylin (blue). Specific staining was localized to cytoplasm of lymphocytes in lamina propria in intestinal villi. View our protocol for [Chromogenic IHC Staining of Paraffin-embedded Tissue Sections](#).

## PREPARATION AND STORAGE

<b>Reconstitution</b>	Reconstitute at 0.2 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

#### BACKGROUND

LITAF (LPS-induced TNF- $\alpha$  factor), initially identified through its interaction with the TNF- $\alpha$  promoter, is a transcription factor that contributes to the regulation of several inflammatory cytokines in response to LPS or p53 stimulation. LITAF interacts directly with LPS-induced STAT6(B) in the cytoplasm, this complex then translocates into the nucleus, where it significantly up-regulates the transcription of other inflammatory mediators such as, GRO, IL-1 $\alpha$ , TNF- $\alpha$ , MCP-2 and IFN- $\gamma$ . Phosphorylation of LITAF by p38 $\alpha$  via the TLR pathway is also required for nuclear translocation. Mutations in LITAF have been associated with CMT1C (Charcot-Marie-Tooth neuropathy type 1C) an autosomal dominant demyelinating form of peripheral neuropathy.