



<http://www.uniprot.org/citations/9703991> target="\_blank">9703991</a>). Acts as a protease that cleaves the N-terminal of most dual specificity mitogen-activated protein kinase kinases (MAPKKs or MAP2Ks) (except for MAP2K5): cleavage invariably occurs within the N-terminal proline-rich region preceding the kinase domain, thus disrupting a sequence involved in directing specific protein-protein interactions necessary for the assembly of signaling complexes (PubMed:<a href="http://www.uniprot.org/citations/10475971" target="\_blank">10475971</a>, PubMed:<a href="http://www.uniprot.org/citations/11104681" target="\_blank">11104681</a>, PubMed:<a href="http://www.uniprot.org/citations/14718925" target="\_blank">14718925</a>, PubMed:<a href="http://www.uniprot.org/citations/9563949" target="\_blank">9563949</a>, PubMed:<a href="http://www.uniprot.org/citations/9703991" target="\_blank">9703991</a>). Also cleaves mouse Nlrp1b: host Nlrp1b cleavage promotes ubiquitination and degradation of the N-terminal part of Nlrp1b by the proteasome, thereby releasing the cleaved C-terminal part of Nlrp1b, which polymerizes and forms the Nlrp1b inflammasome followed by host cell pyroptosis (PubMed:<a href="http://www.uniprot.org/citations/10338520" target="\_blank">10338520</a>, PubMed:<a href="http://www.uniprot.org/citations/19651869" target="\_blank">19651869</a>, PubMed:<a href="http://www.uniprot.org/citations/30872531" target="\_blank">30872531</a>, PubMed:<a href="http://www.uniprot.org/citations/31268597" target="\_blank">31268597</a>). Able to cleave mouse Nlrp1b alleles 1 and 5, while it is not able to cleave Nlrp1b alleles 2, 3 and 4 (PubMed:<a href="http://www.uniprot.org/citations/16429160" target="\_blank">16429160</a>, PubMed:<a href="http://www.uniprot.org/citations/19651869" target="\_blank">19651869</a>). In contrast, does not cleave NLRP1 human ortholog (PubMed:<a href="http://www.uniprot.org/citations/19651869" target="\_blank">19651869</a>). LF is not toxic by itself and only acts as a lethal factor when associated with protective antigen (PA) to form the lethal toxin (LeTx): PA is required for LF translocation into the host cytosol (PubMed:<a href="http://www.uniprot.org/citations/10475971" target="\_blank">10475971</a>, PubMed:<a href="http://www.uniprot.org/citations/11104681" target="\_blank">11104681</a>, PubMed:<a href="http://www.uniprot.org/citations/9563949" target="\_blank">9563949</a>, PubMed:<a href="http://www.uniprot.org/citations/9703991" target="\_blank">9703991</a>).

### Cellular Location

Secreted. Host cytoplasm, host cytosol Note=Translocation into host cytosol is mediated via interaction with the cleaved form of protective antigen (PA-63): following secretion, LF binds via its N-terminal region to the upper rim of the ring-shaped homooligomer formed by PA-63 on the host cell membrane (PubMed:21037566, PubMed:32810181). In this PA-63 pre-pore state, the N-terminal segment of LF refolds into an alpha helix engaged in the alpha-clamp of the PA-63 pre-pore (PubMed:32047164, PubMed:32521227) Loaded complexes are then endocytosed, followed by a conformational change of oligomerized PA-63 from the pre-pore to pore state, which is triggered by the low pH in the endosome (PubMed:10085027, PubMed:12551953, PubMed:3711080, PubMed:8380282). LF is then unfolded to pass through the PA-63 pore and translocate into the host cytosol (PubMed:21037566, PubMed:32047164, PubMed:32521227)

### Anthrax Lethal Factor Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### Anthrax Lethal Factor Antibody - Images

### Anthrax Lethal Factor Antibody - Background

**Anthrax Lethal Factor Antibody:** Anthrax infection is initiated by the inhalation, ingestion, or cutaneous contact with *Bacillus anthracis* endospores. *B. anthracis* produces three polypeptides that comprise the anthrax toxin: protective antigen (PA), lethal factor (LF), and edema factor (EF). PA binds to two related proteins on the cell surface; these are termed tumor epithelial marker 8 (TEM8)/anthrax toxin receptor (ATR) and capillary morphogenesis protein 2 (CMG2), although it is still unclear which is physiologically relevant. Following PA binding to its receptor, PA is cleaved into two fragments by a furin-like protease. The bound fragment binds both LF and EF; the resulting complex is then endocytosed which allows the translocation of LF and EF into the cytoplasm. LF is the primary toxin of anthrax and functions as a highly specific protease that cleaves members of the mitogen-activated protein kinase kinase (MAPKK) family near their amino terminus, interfering with MAPK signaling and inducing apoptosis.

### **Anthrax Lethal Factor Antibody - References**

- Schwartz MN. Recognition and management of anthrax - an update. *New Engl. J. Med.* 2001; 345:1621-6.
- Moayeri M and Leppla SH. The roles of anthrax toxin in pathogenesis. *Curr. Opin. Microbiol.* 2004; 7:19-24.
- Bradley KA, Mogridge J, Mourez M, et al. Identification of the cellular receptor for anthrax toxin. *Nature* 2001; 414:225-9.
- Scobie HM, Rainey GJ, Bradley KA, et al. Human capillary morphogenesis protein 2 functions as an anthrax toxin receptor. *Proc. Natl. Acad. Sci. USA* 2003; 100:5170-4.