

<http://www.uniprot.org/citations/9703991> target="_blank">9703991). 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:10475971, PubMed:11104681, PubMed:14718925, PubMed:9563949, PubMed:9703991). 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:10338520, PubMed:19651869, PubMed:30872531, PubMed:31268597). Able to cleave mouse Nlrp1b alleles 1 and 5, while it is not able to cleave Nlrp1b alleles 2, 3 and 4 (PubMed:16429160, PubMed:19651869). In contrast, does not cleave NLRP1 human ortholog (PubMed:19651869). 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:10475971, PubMed:11104681, PubMed:9563949, PubMed:9703991).

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

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