



[8524823](http://www.uniprot.org/citations/8524823) target="\_blank">8524823</a>). Acts as a more potent activator of the IFN-beta (IFNB) gene than the IFN-alpha (IFNA) gene and plays a critical role in both the early and late phases of the IFNA/B gene induction (PubMed:<a href="http://www.uniprot.org/citations/16846591" target="\_blank">16846591</a>, PubMed:<a href="http://www.uniprot.org/citations/16979567" target="\_blank">16979567</a>, PubMed:<a href="http://www.uniprot.org/citations/20049431" target="\_blank">20049431</a>, PubMed:<a href="http://www.uniprot.org/citations/36603579" target="\_blank">36603579</a>). Found in an inactive form in the cytoplasm of uninfected cells and following viral infection, double-stranded RNA (dsRNA), or toll-like receptor (TLR) signaling, is phosphorylated by IKBKE and TBK1 kinases (PubMed:<a href="http://www.uniprot.org/citations/22394562" target="\_blank">22394562</a>, PubMed:<a href="http://www.uniprot.org/citations/25636800" target="\_blank">25636800</a>, PubMed:<a href="http://www.uniprot.org/citations/27302953" target="\_blank">27302953</a>, PubMed:<a href="http://www.uniprot.org/citations/36603579" target="\_blank">36603579</a>). This induces a conformational change, leading to its dimerization and nuclear localization and association with CREB binding protein (CREBBP) to form dsRNA-activated factor 1 (DRAF1), a complex which activates the transcription of the type I IFN and ISG genes (PubMed:<a href="http://www.uniprot.org/citations/16154084" target="\_blank">16154084</a>, PubMed:<a href="http://www.uniprot.org/citations/27302953" target="\_blank">27302953</a>, PubMed:<a href="http://www.uniprot.org/citations/33440148" target="\_blank">33440148</a>, PubMed:<a href="http://www.uniprot.org/citations/36603579" target="\_blank">36603579</a>). Can activate distinct gene expression programs in macrophages and can induce significant apoptosis in primary macrophages (PubMed:<a href="http://www.uniprot.org/citations/16846591" target="\_blank">16846591</a>). In response to Sendai virus infection, is recruited by TOMM70:HSP90AA1 to mitochondrion and forms an apoptosis complex TOMM70:HSP90AA1:IRF3:BAX inducing apoptosis (PubMed:<a href="http://www.uniprot.org/citations/25609812" target="\_blank">25609812</a>). Key transcription factor regulating the IFN response during SARS-CoV-2 infection (PubMed:<a href="http://www.uniprot.org/citations/33440148" target="\_blank">33440148</a>).

#### Cellular Location

Cytoplasm. Nucleus Mitochondrion. Note=Shuttles between cytoplasmic and nuclear compartments, with export being the prevailing effect (PubMed:10805757, PubMed:35922005). When activated, IRF3 interaction with CREBBP prevents its export to the cytoplasm (PubMed:10805757). Recruited to mitochondria via TOMM70:HSP90AA1 upon Sendai virus infection (PubMed:25609812).

#### Tissue Location

Expressed constitutively in a variety of tissues.

#### IRF3 Rabbit mAb - 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](#)

#### IRF3 Rabbit mAb - Images



