# FADD Blocking Peptide – >85% Purity

## PRODUCT OVERVIEW

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| **Product Name** | FADD, Blocking Peptide |
| **Full Product Name** | FADD Blocking Peptide |
| **Product Gene Name** | FADD blocking peptide |
| **Product Synonym Names** | MORT1; FAS-associated death domain protein; FAS-associating death domain-containing protein; Growth-inhibiting gene 3 protein; Mediator of receptor induced toxicity; Protein FADD |
| **Host** | Synthetic |
| **Species Reactivity** | Human, Monkey |
| **Form/Format** | Lyophilized Powder |
| **Purity/Purification** | >85% |
| **Preparation and Storage** | Shipped at 4 degree C. Store at -20 degree C for one year. |
| **Antibody/Peptide Pairs** | FADD peptide (MBS8243565) is used for blocking the activity of FADD antibody (MBS8241841) |
| **Molecular Weight** | 23,279 Da |
| **OMIM** | 602457 |
| **Quality Control** | The quality of the peptide was evaluated by reversed-phase HPLC and by mass spectrometry. |
| **Applications Tested/Suitable for FADD blocking peptide** | Blocking (BL) |
| **Application Notes for FADD blocking peptide** | Blocking Peptide to the diluted primary antibody in a molar ratio of 10:1 (peptide to antibody) and incubate the mixture at 4 degree C for overnight or at room temperature for 2 hours. |
| **Related Product Information for FADD blocking peptide** | MBS8243565 is a blocking peptide targeting FADD. Reactive in Human, Monkey. Host: Synthetic. The peptide is used to block Anti-FADD Antibody reactivity. |
| **Other Notes** | Small volumes of FADD blocking peptide vial(s) may occasionally become entrapped in the seal of the product vial during shipment and storage. If necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the container`s cap. Certain products may require to ship with dry ice and additional dry ice fee may apply. |

## ADDITIONAL DETAILS

NCBI/Uniprot data below describe general gene information for FADD. It may not necessarily be applicable to this product.

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| **NCBI GI #** | 2498355 |
| **NCBI GeneID** | 8772 |
| **NCBI Accession #** | Q13158.1 |
| **UniProt Primary Accession #** | Q13158 |
| **UniProt Secondary Accession #** | Q14866; Q6IBR4 |
| **UniProt Related Accession #** | Q13158 |
| **NCBI Official Full Name** | FAS-associated death domain protein |
| **NCBI Official Synonym Full Names** | Fas associated via death domain |

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| **NCBI Official Symbol** | FADD |
| **NCBI Official Synonym Symbols** | GIG3; MORT1 |
| **UniProt Protein Name** | FAS-associated death domain protein |
| **UniProt Synonym Protein Names** | FAS-associating death domain-containing protein; Growth-inhibiting gene 3 protein; Mediator of receptor induced toxicity; Protein FADD |
| **Protein Family** | Fas-associated death domain protein |
| **UniProt Gene Name** | FADD |
| **UniProt Synonym Gene Names** | MORT1 |
| **UniProt Entry Name** | FADD_HUMAN |
| **NCBI Protein Information** | FAS-associated death domain protein |
| **NCBI Summary for FADD** | The protein encoded by this gene is an adaptor molecule that interacts with various cell surface receptors and mediates cell apoptotic signals. Through its C-terminal death domain, this protein can be recruited by TNFRSF6/Fas-receptor, tumor necrosis factor receptor, TNFRSF25, and TNFSF10/TRAIL-receptor, and thus it participates in the death signaling initiated by these receptors. Interaction of this protein with the receptors unmasks the N-terminal effector domain of this protein, which allows it to recruit caspase-8, and thereby activate the cysteine protease cascade. Knockout studies in mice also suggest the importance of this protein in early T cell development. [provided by RefSeq, Jul 2008] |
| **UniProt Comments for FADD** | FADD: an adaptor molecule that mediates apoptosis. Recruited through its C-terminal death domain by Fas-receptor, tumor necrosis factor receptor, TNFRSF25, and TRAIL-receptor, participating in the death signaling initiated by these receptors. Interaction with the receptors unmasks the N-terminal effector domain of this protein, which recruits caspase-8, and thereby activates the cysteine protease cascade. Knockout studies in mice also suggest the importance of this protein in early T cell development.<br><br>Protein type: Adaptor/scaffold; Apoptosis<br><br>Chromosomal Location of Human Ortholog: 11q13.3<br><br>Cellular Component: CD95 death-inducing signaling complex; cytoplasm; cytosol; lipid raft; neuron projection; plasma membrane<br><br>Molecular Function: death receptor binding; identical protein binding; protease binding; protein binding; protein complex binding; tumor necrosis factor receptor binding; tumor necrosis factor receptor superfamily binding<br><br>Biological Process: apoptosis; caspase activation; cell surface receptor linked signal transduction; defense response to virus; induction of apoptosis via death domain receptors; innate immune response; lymph node development; MyD88-independent toll-like receptor signaling pathway; negative regulation of caspase activity; positive regulation of activated T cell proliferation; positive regulation of adaptive immune response; positive regulation of apoptosis; positive regulation of I-kappaB kinase/NF-kappaB cascade; positive regulation of interferon-gamma production; positive regulation of interleukin-8 production; positive regulation of macrophage differentiation; positive regulation of proteolysis; positive regulation of T cell mediated cytotoxicity; positive regulation of transcription from RNA polymerase II promoter; positive regulation of tumor necrosis factor production; programmed cell death; protein heterooligomerization; spleen development; T cell differentiation in the thymus; T cell homeostasis; thymus development; toll-like receptor 3 signaling pathway; toll-like receptor 4 signaling pathway; toll-like receptor signaling pathway; tumor necrosis factor-mediated signaling pathway; viral reproduction<br><br>Disease: Infections, Recurrent, With Encephalopathy, Hepatic Dysfunction, And Cardiovascular Malformations |
| **Research Articles on FADD** | **1.** Data (including data obtained in transgenic mice) suggest FADD is key in genesis of neural tube defects in pups of diabetic mice; unfolded protein response/endoplasmic reticulum stress was prevented by over-expression of human dominant negative FADD. |
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