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NMI Antibody (N-term) Blocking Peptide
Synthetic peptide
Catalog # BP9525a

Specification

NMI Antibody (N-term) Blocking Peptide - Product Information

Primary Accession Q13287

NMI Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 9111

Other Names
N-myc-interactor, Nmi, N-myc and STAT interactor, NMI

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

NMI Antibody (N-term) Blocking Peptide - Protein Information

Name NMI (HGNC:7854)

Function
Acts as a signaling pathway regulator involved in innate immune system response (PubMed:9989503, PubMed:26342464, PubMed:29038465, PubMed:29350881). In response to interleukin 2/IL2 and interferon IFN-gamma/IFNG, interacts with signal transducer and activator of transcription/STAT which activate the transcription of downstream genes involved in a multitude of signals for development and homeostasis (PubMed:9989503, PubMed:29377960). Enhances the recruitment of CBP/p300 coactivators to STAT1 and STAT5, resulting in increased STAT1- and STAT5-dependent transcription (PubMed:9989503). In response to interferon IFN-alpha, associates in a complex with signaling pathway regulator IFI35 to regulate immune response; the complex formation prevents proteasome-mediated degradation of IFI35 (PubMed:10779520, PubMed:10950963). In complex with IFI35, inhibits virus-triggered type I IFN-beta production when ubiquitinated by

ubiquitin-protein ligase TRIM21 (PubMed:26342464). In complex with IFI35, negatively regulates nuclear factor NF-kappa-B signaling by inhibiting the nuclear translocation, activation and transcription of NF-kappa-B subunit p65/RELA, resulting in the inhibition of endothelial cell proliferation, migration and re-endothelialization of injured arteries (PubMed:29350881). Negatively regulates virus-triggered type I interferon/IFN production by inducing proteosome-dependent degradation of IRF7, a transcriptional regulator of type I IFN, thereby interfering with cellular antiviral responses (By similarity). Beside its role as an intracellular signaling pathway regulator, also functions extracellularly as damage-associated molecular patterns (DAMPs) to promote inflammation, when actively released by macrophage to the extracellular space during cell injury or pathogen invasion (PubMed:29038465). Macrophage-secreted NMI activates NF-kappa-B signaling in adjacent macrophages through Toll-like receptor 4/TLR4 binding and activation, thereby inducing NF-kappa-B translocation from the cytoplasm into the nucleus which promotes the release of pro-inflammatory cytokines (PubMed:29038465).

Cellular Location
Cytoplasm. Nucleus. Secreted Note=Cytoplasmic NMI localizes in punctate granular structures (PubMed:9781816, PubMed:10950963). Nuclear localization increased following IFN-alpha treatment (PubMed:9781816, PubMed:10950963) Extracelullar following secretion by macrophage (PubMed:29038465)

Tissue Location
Expressed in adult spleen, liver, and kidney (PubMed:9781816). Expressed in fetal thymus, liver, placenta, spleen, lung, and kidney but not brain (PubMed:9781816). Expressed in macrophages (PubMed:29038465).

NMI Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

NMI Antibody (N-term) Blocking Peptide - Images

NMI Antibody (N-term) Blocking Peptide - Background

NMI is a protein that interacts with NMYC and CMYC (two members of the oncogene Myc family), and other transcription factors containing a Zip, HLH, or HLH-Zip motif. The NMI protein also interacts with all STATs except STAT2 and augments STAT-mediated transcription in response to cytokines IL2 and IFN-gamma.

NMI Antibody (N-term) Blocking Peptide - References

Davila, S., et al. Genes Immun. (2010) In press : Fillmore, R.A., et al. Int. J. Cancer 125(3):556-564(2009) Quaye, L., et al. Br. J. Cancer 100(6):993-1001(2009) Vega, A., et al. Gynecol. Oncol. 112(1):210-214(2009) Quaye, L., et al. Clin. Cancer Res. 14(18):5833-5839(2008) Bannasch, D., et al. Oncogene 18(48):6810-6817(1999)