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# COT (MAP3K8/MEKK8) Antibody (C-term) Blocking peptide

**Synthetic peptide**
**Catalog # BP7913a**

## Specification

### COT (MAP3K8/MEKK8) Antibody (C-term) Blocking peptide - Product Information

Primary Accession P41279

### COT (MAP3K8/MEKK8) Antibody (C-term) Blocking peptide - Additional Information

**Gene ID** 1326

**Other Names**
Mitogen-activated protein kinase kinase kinase 8, Cancer Osaka thyroid oncogene, Proto-oncogene c-Cot, Serine/threonine-protein kinase cot, Tumor progression locus 2, TPL-2, MAP3K8, COT, ESTF

**Target/Specificity**
The synthetic peptide sequence used to generate the antibody <a href=/product/products/AP7913a>AP7913a</a> was selected from the C-term region of human MEKK8 . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**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.

### COT (MAP3K8/MEKK8) Antibody (C-term) Blocking peptide - Protein Information

**Name** MAP3K8

**Synonyms** COT, ESTF

**Function**
Required for lipopolysaccharide (LPS)-induced, TLR4-mediated activation of the MAPK/ERK pathway in macrophages, thus being critical for production of the pro-inflammatory cytokine TNF-alpha (TNF) during immune responses. Involved in the regulation of T-helper cell differentiation and IFNG expression in T-cells. Involved in mediating host resistance to bacterial infection through negative regulation of type I interferon (IFN) production. In vitro, activates MAPK/ERK pathway in response to IL1 in an IRAK1-independent manner, leading to up-regulation of IL8 and CCL4. Transduces CD40 and TNFRSF1A signals that activate ERK in B-cells and macrophages, and thus may play a role in the regulation of immunoglobulin production. May also play a role in the transduction of TNF signals that activate JNK and NF-kappa-B in some cell types.

In adipocytes, activates MAPK/ERK pathway in an IKBKB- dependent manner in response to IL1B and TNF, but not insulin, leading to induction of lipolysis. Plays a role in the cell cycle. Isoform 1 shows some transforming activity, although it is much weaker than that of the activated oncogenic variant.

**Cellular Location**
Cytoplasm

**Tissue Location**
Expressed in several normal tissues and human tumor-derived cell lines

## COT (MAP3K8/MEKK8) Antibody (C-term) Blocking peptide - Protocols

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

- Blocking Peptides

## COT (MAP3K8/MEKK8) Antibody (C-term) Blocking peptide - Images

## COT (MAP3K8/MEKK8) Antibody (C-term) Blocking peptide - Background

Mitogen-activated protein kinase (MAPK) signaling cascades include MAPK or extracellular signal-regulated kinase (ERK), MAPK kinase (MKK or MEK), and MAPK kinase kinase (MAPKKK or MEKK). MAPKK kinase/MEKK phosphorylates and activates its downstream protein kinase, MAPK kinase/MEK, which in turn activates MAPK. The kinases of these signaling cascades are highly conserved, and homologs exist in yeast, Drosophila, and mammalian cells. MEKK8 is able to activate NF-kappa-B 1 by stimulating proteasome-mediated proteolysis of NF-kappa-B 1/p105. The protein appears to play an important role in the cell cycle. This cytoplasmic protein is expressed in several normal tissues and human tumor-derived cell lines. The 58 kDa form is activated specifically during the S and G2/M phases of the cell cycle. The longer form undergoes phosphorylation on Ser residues mainly, and the shorter form on both Ser and Thr residues.

## COT (MAP3K8/MEKK8) Antibody (C-term) Blocking peptide - References

Sanchez-Gongora, E., et al., J. Biol. Chem. 275(40):31379-31386 (2000).Aoki, M., et al., J. Biol. Chem. 268(30):22723-22732 (1993).Chan, A.M., et al., Oncogene 8(5):1329-1333 (1993).Miyoshi, J., et al., Mol. Cell. Biol. 11(8):4088-4096 (1991).Aoki, M., et al., Oncogene 6(9):1515-1519 (1991).