

Phospho-ATF2-pS480 Blocking Peptide
Synthetic peptide
Catalog # BP3390a**Specification**

Phospho-ATF2-pS480 Blocking Peptide - Product InformationPrimary Accession [P15336](#)**Phospho-ATF2-pS480 Blocking Peptide - Additional Information****Gene ID** 1386**Other Names**

Cyclic AMP-dependent transcription factor ATF-2, cAMP-dependent transcription factor ATF-2, Activating transcription factor 2, Cyclic AMP-responsive element-binding protein 2, CREB-2, cAMP-responsive element-binding protein 2, HB16, Histone acetyltransferase ATF2, cAMP response element-binding protein CRE-BP1, ATF2, CREB2, CREBP1

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.

Phospho-ATF2-pS480 Blocking Peptide - Protein Information**Name** ATF2**Synonyms** CREB2, CREBP1**Function**

Transcriptional activator which regulates the transcription of various genes, including those involved in anti-apoptosis, cell growth, and DNA damage response. Dependent on its binding partner, binds to CRE (cAMP response element) consensus sequences (5'-TGACGTCA- 3') or to AP-1 (activator protein 1) consensus sequences (5'-TGACTCA- 3'). In the nucleus, contributes to global transcription and the DNA damage response, in addition to specific transcriptional activities that are related to cell development, proliferation and death. In the cytoplasm, interacts with and perturbs HK1- and VDAC1-containing complexes at the mitochondrial outer membrane, thereby impairing mitochondrial membrane potential, inducing mitochondrial leakage and promoting cell death. The phosphorylated form (mediated by ATM) plays a role in the DNA damage response and is involved in the ionizing radiation (IR)-induced S phase checkpoint control and in the recruitment of the MRN complex into the IR-induced foci (IRIF). Exhibits histone acetyltransferase (HAT) activity which specifically acetylates histones H2B and H4 in vitro (PubMed:10821277). In concert

with CUL3 and RBX1, promotes the degradation of KAT5 thereby attenuating its ability to acetylate and activate ATM. Can elicit oncogenic or tumor suppressor activities depending on the tissue or cell type.

Cellular Location

Nucleus. Cytoplasm. Mitochondrion outer membrane. Note=Shuttles between the cytoplasm and the nucleus and heterodimerization with JUN is essential for the nuclear localization. Localization to the cytoplasm is observed under conditions of cellular stress and in disease states. Localizes at the mitochondrial outer membrane in response to genotoxic stress. Phosphorylation at Thr-52 is required for its nuclear localization and negatively regulates its mitochondrial localization. Co-localizes with the MRN complex in the IR-induced foci (IRIF)

Tissue Location

Ubiquitously expressed, with more abundant expression in the brain

Phospho-ATF2-pS480 Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

Phospho-ATF2-pS480 Blocking Peptide - Images**Phospho-ATF2-pS480 Blocking Peptide - Background**

ATF2 is a transcription factor that is a member of the leucine zipper family of DNA binding proteins. This protein binds to the cAMP-responsive element (CRE), an octameric palindrome. The protein forms a homodimer or heterodimer with c-Jun and stimulates CRE-dependent transcription. The protein is also a histone acetyltransferase (HAT) that specifically acetylates histones H2B and H4 in vitro; thus it may represent a class of sequence-specific factors that activate transcription by direct effects on chromatin components.

Phospho-ATF2-pS480 Blocking Peptide - References

Kravets, A., et al., J. Biol. Chem. 279(19):19916-19923 (2004).
Hong, S., et al., J. Biol. Chem. 279(17):16996-17003 (2004).
Averous, J., et al., J. Biol. Chem. 279(7):5288-5297 (2004).
Berger, A.J., et al., Cancer Res. 63(23):8103-8107 (2003).
Wen-Sheng, W., Oncogene 22(7):955-963 (2003).