

**APEX1**  
**Purified Mouse Monoclonal Antibody**  
**Catalog # AO2532a****Specification**

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**APEX1 - Product Information**

Application	<b>E, WB, IHC</b>
Primary Accession	<a href="#">P27695</a>
Reactivity	<b>Human, Rat, Monkey</b>
Host	<b>Mouse</b>
Clonality	<b>Monoclonal</b>
Isotype	<b>Mouse IgG1</b>
Calculated MW	<b>35.6kDa KDa</b>

**Immunogen**

Purified recombinant fragment of human APEX1 (AA: 219-318) expressed in E. Coli.

**Formulation**

Purified antibody in PBS with 0.05% sodium azide

**APEX1 - Additional Information**

**Gene ID** 328

**Other Names**

APE; APX; APE1; APEN; APEX; HAP1; REF1

**Dilution**

E~~ 1/10000  
WB~~ 1/500 - 1/2000  
IHC~~ 1/200 - 1/1000

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

APEX1 is for research use only and not for use in diagnostic or therapeutic procedures.

**APEX1 - Protein Information**

**Name** APEX1

**Synonyms** APE, APE1, APEX, APX, HAP1, REF1

**Function**

Multifunctional protein that plays a central role in the cellular response to oxidative stress. The two major activities of APEX1 are DNA repair and redox regulation of transcriptional factors. Functions

as an apurinic/aprimidinic (AP) endodeoxyribonuclease in the DNA base excision repair (BER) pathway of DNA lesions induced by oxidative and alkylating agents. Initiates repair of AP sites in DNA by catalyzing hydrolytic incision of the phosphodiester backbone immediately adjacent to the damage, generating a single-strand break with 5'-deoxyribose phosphate and 3'-hydroxyl ends. Also incises at AP sites in the DNA strand of DNA/RNA hybrids, single-stranded DNA regions of R-loop structures, and single-stranded RNA molecules. Has 3'-5' exoribonuclease activity on mismatched deoxyribonucleotides at the 3' termini of nicked or gapped DNA molecules during short-patch BER. Possesses DNA 3' phosphodiesterase activity capable of removing lesions (such as phosphoglycolate) blocking the 3' side of DNA strand breaks. May also play a role in the epigenetic regulation of gene expression by participating in DNA demethylation. Acts as a loading factor for POLB onto non-incised AP sites in DNA and stimulates the 5'-terminal deoxyribose 5'-phosphate (dRp) excision activity of POLB. Plays a role in protection from granzyme-mediated cellular repair leading to cell death. Also involved in the DNA cleavage step of class switch recombination (CSR). On the other hand, APEX1 also exerts reversible nuclear redox activity to regulate DNA binding affinity and transcriptional activity of transcriptional factors by controlling the redox status of their DNA-binding domain, such as the FOS/JUN AP-1 complex after exposure to IR. Involved in calcium-dependent down- regulation of parathyroid hormone (PTH) expression by binding to negative calcium response elements (nCaREs). Together with HNRNPL or the dimer XRCC5/XRCC6, associates with nCaRE, acting as an activator of transcriptional repression. Stimulates the YBX1-mediated MDR1 promoter activity, when acetylated at Lys-6 and Lys-7, leading to drug resistance. Acts also as an endoribonuclease involved in the control of single-stranded RNA metabolism. Plays a role in regulating MYC mRNA turnover by preferentially cleaving in between UA and CA dinucleotides of the MYC coding region determinant (CRD). In association with NMD1, plays a role in the rRNA quality control process during cell cycle progression. Associates, together with YBX1, on the MDR1 promoter. Together with NPM1, associates with rRNA. Binds DNA and RNA.

#### Cellular Location

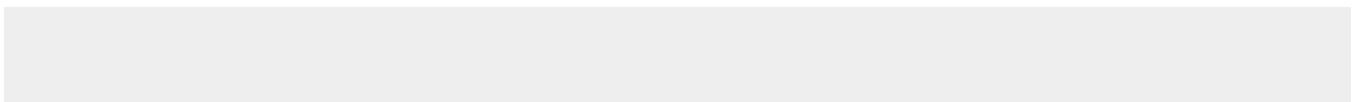
Nucleus {ECO:0000255|PROSITE-ProRule:PRU00764}. Nucleus, nucleolus. Nucleus speckle. Endoplasmic reticulum. Cytoplasm Note=Detected in the cytoplasm of B-cells stimulated to switch (By similarity). Colocalized with SIRT1 in the nucleus. Colocalized with YBX1 in nuclear speckles after genotoxic stress. Together with OGG1 is recruited to nuclear speckles in UVA-irradiated cells. Colocalized with nucleolin and NPM1 in the nucleolus. Its nucleolar localization is cell cycle dependent and requires active rRNA transcription. Colocalized with calreticulin in the endoplasmic reticulum. Translocation from the nucleus to the cytoplasm is stimulated in presence of nitric oxide (NO) and function in a CRM1-dependent manner, possibly as a consequence of demasking a nuclear export signal (amino acid position 64-80). S- nitrosylation at Cys-93 and Cys-310 regulates its nuclear-cytosolic shuttling. Ubiquitinated form is localized predominantly in the cytoplasm.

#### APEX1 - 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](#)

#### APEX1 - Images



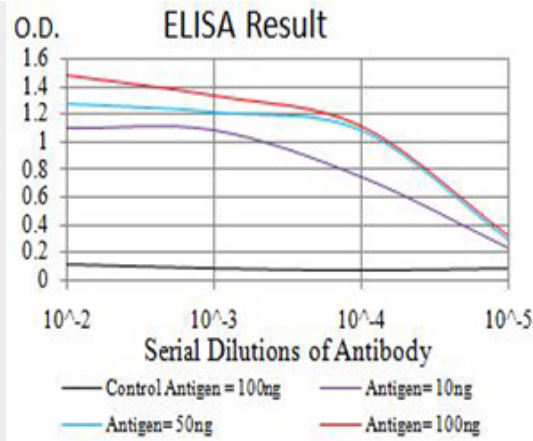


Figure 1: Black line: Control Antigen (100 ng); Purple line: Antigen (10ng); Blue line: Antigen (50 ng); Red line: Antigen (100 ng)

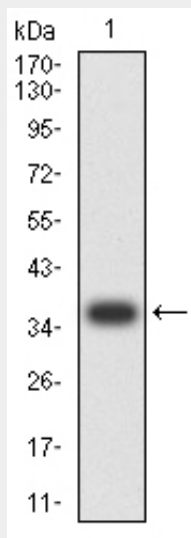


Figure 2: Western blot analysis using APEX1 mAb against human APEX1 (AA: 219-318) recombinant protein. (Expected MW is 37.4 kDa)

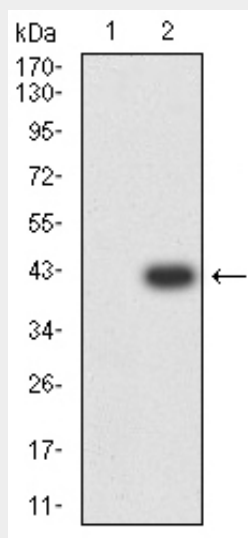


Figure 3: Western blot analysis using APEX1 mAb against HEK293 (1) and APEX1 (AA: 219-318)-hlgGfC transfected HEK293 (2) cell lysate.

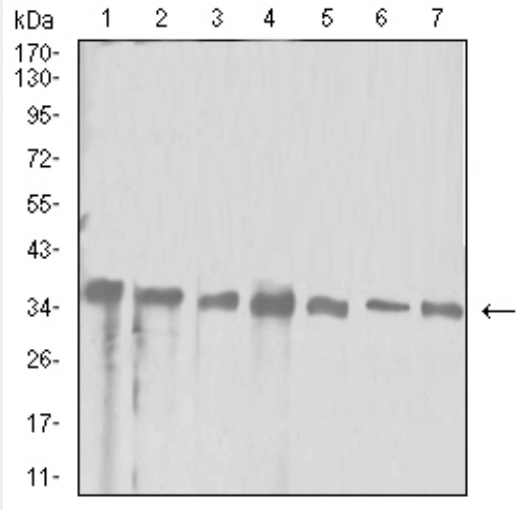


Figure 4:Western blot analysis using APEX1 mouse mAb against Hela (1), Jurkat (2), SW480 (3), A431 (4), HepG2 (5), NIH/3T3 (6), and PC-12 (7) cell lysate.

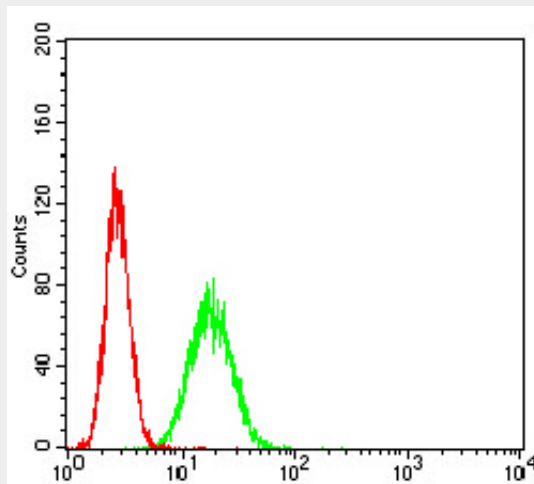


Figure 5:Flow cytometric analysis of HeLa cells using APEX1 mouse mAb (green) and negative control (red).

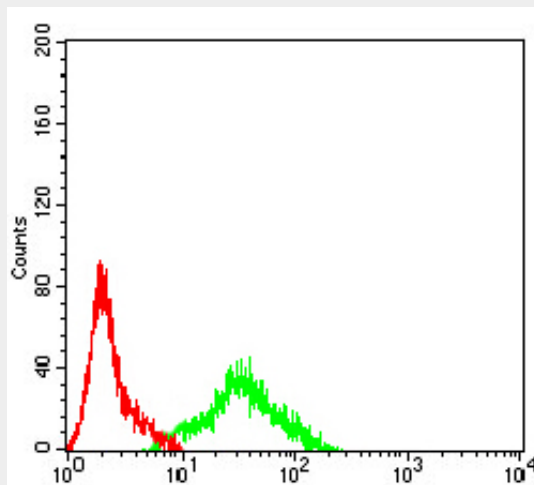


Figure 6:Flow cytometric analysis of SK-N-SH cells using APEX1 mouse mAb (green) and negative control (red).

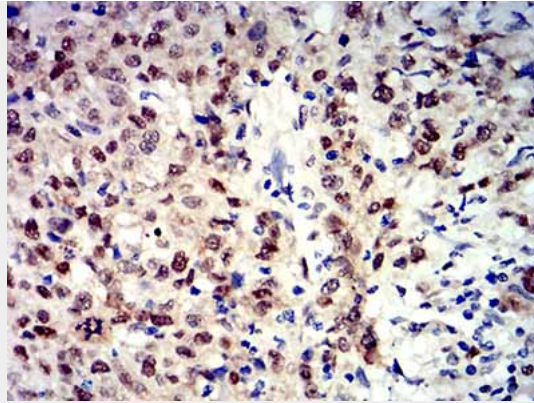


Figure 7: Immunohistochemical analysis of paraffin-embedded breast cancer tissues using APEX1 mouse mAb with DAB staining.

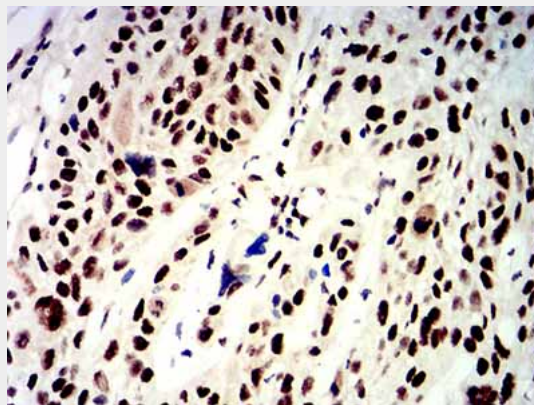


Figure 8: Immunohistochemical analysis of paraffin-embedded esophageal cancer tissues using APEX1 mouse mAb with DAB staining.

#### **APEX1 - References**

1. Mutat Res Genet Toxicol Environ Mutagen. 2015 Nov;793:19-29. 2. PLoS One. 2015 Dec 1;10(12):e0143289.