

# APEX1 Antibody

Purified Mouse Monoclonal Antibody (Mab)

Catalog # AW5625

## Product Information

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<b>Application</b>	IF, IHC, WB
<b>Primary Accession</b>	<a href="#">P27695</a>
<b>Reactivity</b>	Human
<b>Host</b>	Mouse
<b>Clonality</b>	Monoclonal
<b>Calculated MW</b>	35555
<b>Isotype</b>	IgG1,K
<b>Antigen Source</b>	HUMAN

## Additional Information

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<b>Gene ID</b>	328
<b>Antigen Region</b>	1-318
<b>Other Names</b>	DNA-(apurinic or apyrimidinic site) lyase, 31--, APEX nuclease, APEN, Apurinic-apyrimidinic endonuclease 1, AP endonuclease 1, APE-1, REF-1, Redox factor-1, DNA-(apurinic or apyrimidinic site) lyase, mitochondrial, APEX1, APE, APE1, APEX, APX, HAP1, REF1
<b>Dilution</b>	IF~~1:25 IHC~~1:100~500 WB~~0.25
<b>Target/Specificity</b>	This APEX1 antibody is generated from a mouse immunized with a recombinant protein of human APEX1.
<b>Storage</b>	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
<b>Precautions</b>	APEX1 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

## Protein Information

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<b>Name</b>	APEX1
<b>Synonyms</b>	APE, APE1, APEX, APX, HAP1, REF1
<b>Function</b>	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 (PubMed: <a href="#">11118054</a> , PubMed: <a href="#">11452037</a> , PubMed: <a href="#">15831793</a> , PubMed: <a href="#">18439621</a> , PubMed: <a href="#">18579163</a> ,

PubMed:[21762700](#), PubMed:[24079850](#), PubMed:[8355688](#), PubMed:[9108029](#), PubMed:[9560228](#)). Functions as an apurinic/apyrimidinic (AP) endodeoxyribonuclease in the 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 (PubMed:[15380100](#), PubMed:[16617147](#), PubMed:[18439621](#), PubMed:[19123919](#), PubMed:[19188445](#), PubMed:[19934257](#), PubMed:[20699270](#), PubMed:[21762700](#), PubMed:[24079850](#), PubMed:[8932375](#), PubMed:[8995436](#), PubMed:[9804799](#)). Operates at switch sites of immunoglobulin (Ig) constant regions where it mediates Ig isotype class switch recombination. Processes AP sites induced by successive action of AICDA and UNG. Generates staggered nicks in opposite DNA strands resulting in the formation of double-strand DNA breaks that are finally resolved via non-homologous end joining repair pathway (By similarity). Has 3'-5' exodeoxyribonuclease activity on mismatched deoxyribonucleotides at the 3' termini of nicked or gapped DNA molecules during short-patch BER (PubMed:[11832948](#), PubMed:[1719477](#)). Possesses DNA 3' phosphodiesterase activity capable of removing lesions (such as phosphoglycolate and 8-oxoguanine) blocking the 3' side of DNA strand breaks (PubMed:[15831793](#), PubMed:[7516064](#)). Also acts 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 (PubMed:[19188445](#), PubMed:[19401441](#), PubMed:[21762700](#)). 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 (PubMed:[9207062](#)). 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 (PubMed:[10023679](#), PubMed:[11118054](#), PubMed:[11452037](#), PubMed:[18579163](#), PubMed:[8355688](#), PubMed:[9108029](#)). 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 (PubMed:[11809897](#), PubMed:[14633989](#), PubMed:[8621488](#)). May also play a role in the epigenetic regulation of gene expression by participating in DNA demethylation (PubMed:[21496894](#)). Stimulates the YBX1-mediated MDR1 promoter activity, when acetylated at Lys-6 and Lys-7, leading to drug resistance (PubMed:[18809583](#)). Plays a role in protection from granzyme-mediated cellular repair leading to cell death (PubMed:[18179823](#)). Binds DNA and RNA. Associates, together with YBX1, on the MDR1 promoter. Together with NPM1, associates with rRNA (PubMed:[19188445](#), PubMed:[19401441](#), PubMed:[20699270](#)).

## 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.

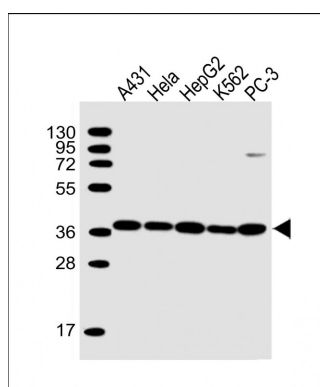
## Background

Multifunctional protein that plays a central role in the cellular response to oxidative stress. The two major activities of APEX1 in DNA repair and redox regulation of transcriptional factors. Functions as a apurinic/apyrimidinic (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. Does also incise 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 a 3'-5' exoribonuclease activity on mismatched deoxyribonucleotides at the 3' termini of nicked or gapped DNA molecules during short-patch BER. Possesses a 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 the protection from granzymes-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.

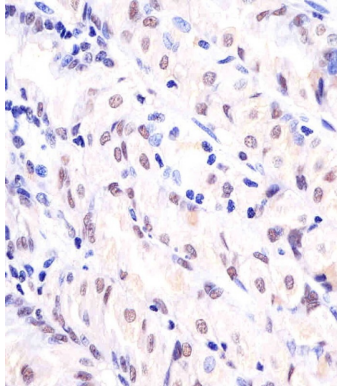
## References

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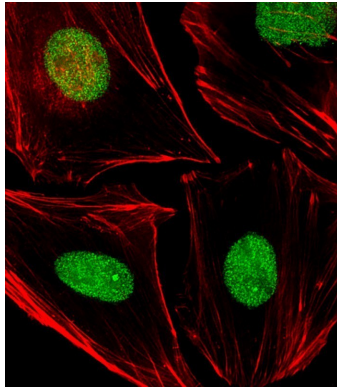
## Images



All lanes : Anti-APEX1 Antibody at 1:2000 dilution Lane 1: A431 whole cell lysate Lane 2: HeLa whole cell lysate Lane 3: HepG2 whole cell lysate Lane 4: K562 whole cell lysate Lane 5: PC-3 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-mouse IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 36 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



AW5623 staining APEX1 in human stomach sections by Immunohistochemistry (IHC-P - paraformaldehyde-fixed, paraffin-embedded sections). Tissue was fixed with formaldehyde and blocked with 3% BSA for 0.5 hour at room temperature; antigen retrieval was by heat mediation with a citrate buffer (pH6). Samples were incubated with primary antibody (1/25) for 1 hour at 37°C. A undiluted biotinylated goat polyvalent antibody was used as the secondary antibody.



Immunofluorescent analysis of 4% paraformaldehyde-fixed, 0.1% Triton X-100 permeabilized HeLa (human cervical epithelial adenocarcinoma cell line) cells labeling Pdx1 with AW5623 at 1/25 dilution, followed by Dylight® 488-conjugated goat anti-mouse IgG (NA166821) secondary antibody at 1/200 dilution (green). Immunofluorescence image showing nucleus staining on HeLa cell line. Cytoplasmic actin is detected with Dylight® 554 Phalloidin (PD18466410) at 1/100 dilution (red).

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.