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Anti-p48-DDB2 (C-terminal specific) (RABBIT) Antibody

p48 DDB2 Antibody
Catalog # ASR3723

Specification

Anti-p48-DDB2 (C-terminal specific) (RABBIT) Antibody - Product Information

Host	**Rabbit**
Conjugate	**Unconjugated**
Target Species	**Human**
Reactivity	**Human**
Clonality	**Polyclonal**
Application	**WB, IHC, E, IP, I, LCI**
Application Note	**This antibody reacts with human DDB2 tested by western blot and immunoprecipitation. The antibody immunoprecipitates in vitro translated protein and protein from cell lysates (using 293T, and others). Coimmunoprecipitation of related proteins has not been tested. A 47.8 kDa band corresponding to human DDB2 is detected. Most cell lines expressing DDB2 can be used as a positive control. Researchers should determine optimal titers for other applications.**
Physical State	**Liquid (sterile filtered)**
Immunogen	**This antibody was prepared from whole rabbit serum produced by repeated immunizations with a synthetic peptide corresponding to amino acids 419-427 of Human DDB2 (C-terminal) coupled to KLH.**
Preservative	**0.01% (w/v) Sodium Azide**

Anti-p48-DDB2 (C-terminal specific) (RABBIT) Antibody - Additional Information

Gene ID 1643

Other Names
1643

Purity
This product is monospecific antiserum processed by delipidation and defibrination followed by sterile filtration. This product reacts with human DDB2. Cross reactivity with DDB2 from other sources is not known.

Storage Condition
Store vial at -20° C prior to opening. Aliquot contents and freeze at -20° C or below for extended storage. Avoid cycles of freezing and thawing. Centrifuge product if not completely clear after standing at room temperature. This product is stable for several weeks at 4° C as an undiluted liquid. Dilute only prior to immediate use.

Precautions Note
This product is for research use only and is not intended for therapeutic or diagnostic applications.

Anti-p48-DDB2 (C-terminal specific) (RABBIT) Antibody - Protein Information

Name DDB2

Function
Protein, which is both involved in DNA repair and protein ubiquitination, as part of the UV-DDB complex and DCX (DDB1-CUL4-X-box) complexes, respectively (PubMed:10882109, PubMed:11278856, PubMed:11705987, PubMed:12732143, PubMed:15882621, PubMed:16473935, PubMed:18593899, PubMed:32789493, PubMed:9892649). Core component of the UV-DDB complex (UV-damaged DNA-binding protein complex), a complex that recognizes UV-induced DNA damage and recruit proteins of the nucleotide excision repair pathway (the NER pathway) to initiate DNA repair (PubMed:10882109, PubMed:11278856, PubMed:11705987, PubMed:12944386, PubMed:14751237, PubMed:16260596, PubMed:32789493). The UV-DDB complex preferentially binds to cyclobutane pyrimidine dimers (CPD), 6-4 photoproducts (6-4 PP), apurinic sites and short mismatches (PubMed:10882109, PubMed:11278856, PubMed:11705987, PubMed:12944386, PubMed:16260596). Also functions as the substrate recognition module for the DCX (DDB2-CUL4-X-box) E3 ubiquitin-protein ligase complex DDB2-CUL4-ROC1 (also known as CUL4-DDB-ROC1 and CUL4- DDB-RBX1) (PubMed:12732143, PubMed:15882621, PubMed:16473935, PubMed:18593899, PubMed:26572825). The DDB2-CUL4-ROC1 complex may ubiquitinate histone H2A, histone H3 and histone H4 at sites of UV- induced DNA damage (PubMed:16473935, PubMed:16678110). The ubiquitination of histones may facilitate their removal from the nucleosome and promote subsequent DNA repair (PubMed:16473935, PubMed:16678110). The DDB2-CUL4-ROC1 complex also ubiquitinates XPC, which may enhance DNA-binding by XPC and promote NER (PubMed:15882621). The DDB2-CUL4-ROC1 complex also ubiquitinates KAT7/HBO1 in response to DNA damage, leading to its degradation: recognizes KAT7/HBO1 following phosphorylation by ATR (PubMed:<a href="http://www.uniprot.org/citations/26572825"

target="_blank">26572825).

Cellular Location
Nucleus. Chromosome. Note=Accumulates at sites of DNA damage following UV irradiation.

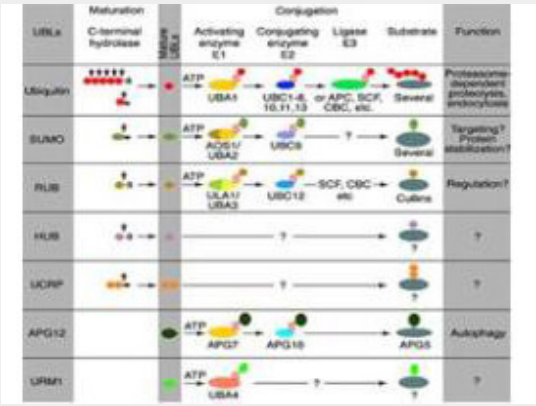
Tissue Location
Ubiquitously expressed; with highest levels in corneal endothelium and lowest levels in brain. Isoform D1 is highly expressed in brain and heart. Isoform D2, isoform D3 and isoform D4 are weakly expressed.

Anti-p48-DDB2 (C-terminal specific) (RABBIT) Antibody - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Anti-p48-DDB2 (C-terminal specific) (RABBIT) Antibody - Images



Most modifiers mature by proteolytic processing from inactive precursors (a; amino acid). Arrowheads point to the cleavage sites. Ubiquitin is expressed either as polyubiquitin or as a fusion with ribosomal proteins. Conjugation requires activating (E1) and conjugating (E2) enzymes that form thiolesters (S) with the modifiers. Modification of cullins by RUB involves SCF(SKP1/cullin-1/F-box protein) /CBC(cullin-2/elongin B/elonginC) -like E3 enzymes that are also involved in ubiquitination. In contrast to ubiquitin, the UBLs do not seem to form multi-UBL chains. UCRP(ISG15) resembles two ubiquitin moieties linked head-to-tail. Whether HUB1 functions as a modifier is currently unclear. APG12 and URM1 are distinct from the other modifiers because they are unrelated in sequence to ubiquitin. Data contributed by S.Jentsch.

Anti-p48-DDB2 (C-terminal specific) (RABBIT) Antibody - Background

DDB2 is also known as Damage-specific DNA binding protein 2, DDB p48 subunit, DDBb, UV-damaged DNA-binding protein 2 and UV-DDB 2. The DDB2 gene encodes the small subunit (p48) of DNA damage-binding protein, which is a heterodimer, composed of a large (p127 DDB1) and a small subunit. The DDB2 subunit appears to be required for DNA binding. This nuclear

protein functions in nucleotide-excision repair resulting from UV-damaged DNA by binding to pyrimidine dimers. Its defective activity causes the repair defect in the patients with xeroderma pigmentosum complementation group E (XPE). XP-E is a rare human autosomal recessive disease characterized by solar sensitivity, high predisposition for developing cancers on areas exposed to sunlight and, in some cases, neurological abnormalities. However, it remains for mutation analysis to demonstrate whether the defect in XPE patients is in this gene or the gene encoding the large subunit.