SANTA CRUZ BIOTECHNOLOGY, INC.

DDB2 (S-16): sc-16295

BACKGROUND

Damaged DNA binding protein (DDB) is a heterodimer composed of two subunits, p127 and p48, which are designated DDB1 and DDB2, respectively. The DDB heterodimer is involved in repairing DNA damaged by ultraviolet light. Specifically, DDB, also designated UV-damaged DNA binding protein (UV-DDB), xeroderma pigmentosum group E binding factor (XPE-BF) and hepatitis B virus X-associated protein 1 (XAP-1), binds to damaged cyclobutane pyrimidine dimers (CPDs). Mutations in the DDB2 gene are implicated as causes of xeroderma pigmentosum group E, an autosomal recessive disease in which patients are defective in nucleotide excision DNA repair. XPE is characterized by hypersensitivity of the skin to sunlight with a high frequency of skin cancer as well as neurologic abnormalities. The hepatitis B virus (HBV) X protein interacts with DDB1, which may mediate HBx transactivation.

REFERENCES

1. Dualan, R., et al. 1995. Chromosomal localization and cDNA cloning of the genes (DDB1 and DDB2) for the p127 and p48 subunits of a human damage-specific DNA binding protein. Genomics 29: 62-69.
2. Nichols, A.F., et al. 1996. Mutations specific to the xeroderma pigmentosum group E DDB-phenotype. J. Biol. Chem. 271: 24317-2420.
3. Stohr, H., et al. 1998. Refined mapping of the gene encoding the p127 kDa UV-damaged DNA-binding protein (DDB1) within 11q12-q13.1 and its exclusion in Best's vitelliform macular dystrophy. Eur. J. Hum. Genet. 6: 400-405.
4. Lin, G.Y., et al. 1998. The V protein of the paramyxovirus SV5 interacts with damage-specific DNA binding protein. Virology 249: 189-200.

CHROMOSOMAL LOCATION

Genetic locus: DDB2 (human) mapping to 11p11.2; Ddb2 (mouse) mapping to 2 E1.

SOURCE

DDB2 (S-16) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the C-terminus of DDB2 of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-16295 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

APPLICATIONS

DDB2 (S-16) is recommended for detection of DDB2 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

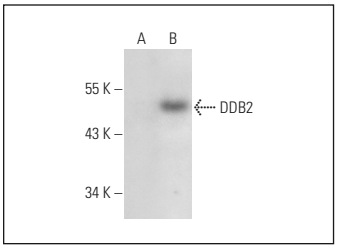
DDB2 (S-16) is also recommended for detection of DDB2 in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for DDB2 siRNA (h): sc-37799, DDB2 siRNA (m): sc-37800, DDB2 shRNA Plasmid (h): sc-37799-SH, DDB2 shRNA Plasmid (m): sc-37800-SH, DDB2 shRNA (h) Lentiviral Particles: sc-37799-V and DDB2 shRNA (m) Lentiviral Particles: sc-37800-V.

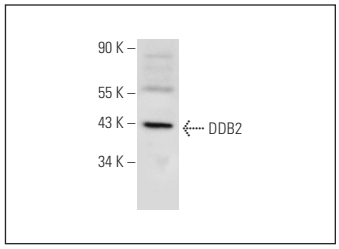
Molecular Weight of DDB2: 48 kDa.

Positive Controls: HeLa nuclear extract: sc-2120, DDB2 (h): 293T Lysate: sc-371088 or HeLa whole cell lysate: sc-2200.

DATA



DDB2 (S-16): sc-16295. Western blot analysis of DDB2 expression in non-transfected: sc-117752 (**A**) and human DDB2 transfected: sc-371088 (**B**) 293T whole cell lysates.



DDB2 (S-16): sc-16295. Western blot analysis of DDB2 expression in HeLa nuclear extract.

SELECT PRODUCT CITATIONS

1. Lo, P.K., et al. 2004. The prosurvival activity of p53 protects cells from UV-induced apoptosis by inhibiting c-Jun NH_2-terminal kinase activity and mitochondrial death signaling. Cancer Res. 64: 8736-8745.
2. Kapetanaki, M.G., et al. 2006. The DDB1-CUL-4ADDB2 ubiquitin ligase is deficient in xeroderma pigmentosum group E and targets Histone H2A at UV-damaged DNA sites. Proc. Natl. Acad. Sci. USA 103: 2588-2593.
3. Latimer, J.J., et al. 2010. Nucleotide excision repair deficiency is intrinsic in sporadic stage I breast cancer. Proc. Natl. Acad. Sci. USA 107: 21725-21730.