SANTA CRUZ BIOTECHNOLOGY, INC.

Pdcd-4 (k4C1): sc-130545

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The transformation suppressor gene Pdcd-4 (programmed cell death gene 4) inhibits the tumor-promoter mediated transformation of mouse keratinocytes and is a potential tumor suppressor gene in the development of human lung cancer. Biochemical analysis suggests that the Pdcd-4 protein is involved in protein translation as well as in nuclear events. Pdcd-4 directly interacts with the RNA helicase eIF4A and inhibits protein synthesis by interfering with the assembly of the cap-dependent translation initiation complex. Pdcd-4 also suppresses the transactivation of AP-1 responsive promoters by c-Jun, suggesting that the transformation-suppressor activity of Pdcd-4 might be due, at least in part, to the inhibition of c-Jun activity. In addition to affecting c-Jun phosphorylation, Pdcd-4 blocks the recruitment of the co-activator p300 by c-Jun.

REFERENCES

1. Bohm, M., et al. 2003. The transformation suppressor protein Pdcd-4 shuttles between nucleus and cytoplasm and binds RNA. Oncogene 22: 4905-4910.
2. Lankat-Buttgereit, B., et al. 2003. Programmed cell death protein 4 (Pdcd-4): a novel target for antineoplastic therapy? Biol. Cell 95: 515-519.
3. Bitomsky, N., et al. 2004. Transformation suppressor protein Pdcd-4 interferes with JNK-mediated phosphorylation of c-Jun and recruitment of the co-activator p300 by c-Jun. Oncogene 23: 7484-7493.

CHROMOSOMAL LOCATION

Genetic locus: PDCD4 (human) mapping to 10q25.2.

SOURCE

Pdcd-4 (k4C1) is a mouse monoclonal antibody raised against full-length recombinant Pdcd-4 of human origin.

PRODUCT

Each vial contains 50 µg IgG_1 in 0.5 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

Pdcd-4 (k4C1) is recommended for detection of Pdcd-4 of 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)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for Pdcd-4 siRNA (h): sc-106389, Pdcd-4 shRNA Plasmid (h): sc-106389-SH and Pdcd-4 shRNA (h) Lentiviral Particles: sc-106389-V.

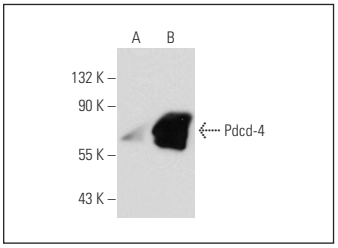
Molecular Weight of Pdcd-4: 54 kDa.

Positive Controls: Pdcd-4 (h): 293T Lysate: sc-176183, Hep G2 cell lysate: sc-2227 or SK-BR-3 nuclear extract: sc-2134.

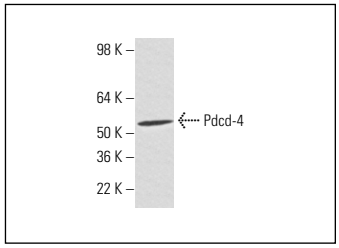
STORAGE

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

DATA



Pdcd-4 (k4C1): sc-130545. Western blot analysis of Pdcd-4 expression in non-transfected: sc-117752 (**A**) and human Pdcd-4 transfected: sc-176183 (**B**) 293T whole cell lysates.



Pdcd-4 (k4C1): sc-130545. Western blot analysis of Pdcd-4 expression in Hep G2 whole cell lysate.

SELECT PRODUCT CITATIONS

1. Cheng, Y., et al. 2009. MicroRNA-21 protects against the H_2O_2-induced injury on cardiac myocytes via its target gene Pdcd-4. J. Mol. Cell. Cardiol. 47: 5-14.
2. Chen, X. and Qin, Z. 2011. Post-transcriptional regulation by microrna-21 and let-7a microRNA in paediatric cholesteatoma. J. Int. Med. Res. 39: 2110-2118.
3. Kolpa, H.J., et al. 2013. miR-21 represses Pdcd-4 during cardiac valvulogenesis. Development 140: 2172-2180.
4. Teng, Y., et al. 2013. Endocrine disruptors fludioxonil and fenhexamid stimulate miR-21 expression in breast cancer cells. Toxicol. Sci. 131: 71-83.
5. Teng, Y., et al. 2014. Dehydroepiandrosterone-induces miR-21 transcription in Hep G2 cells through estrogen receptor β and androgen receptor. Mol. Cell. Endocrinol. 392: 23-36.
6. Liang, H., et al. 2016. miR-93 functions as an oncomiR for the downregulation of Pdcd-4 in gastric carcinoma. Sci. Rep. 6: 23772.
7. Liu, Y., et al. 2016. miR-181b functions as an oncomiR in colorectal cancer by targeting Pdcd-4. Protein Cell 7: 722-734.
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10. Ji, C., et al. 2017. miR-93 enhances hepatocellular carcinoma invasion and metastasis by EMT via targeting PDCD4. Biotechnol. Lett. 39: 1621-1629.
11. Vandewalle, V., et al. 2021. miR-15a-5p and miR-21-5p contribute to chemoresistance in cytogenetically normal acute myeloid leukaemia by targeting PDCD4, ARL2 and BTG2. J. Cell. Mol. Med. 25: 575-585.

RESEARCH USE

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