SANTA CRUZ BIOTECHNOLOGY, INC.

HPV16 E6 (N-17): sc-1584

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The Power to Question

BACKGROUND

Human papilloma viruses (HPVs) can be classified as either high risk or low risk according to their association with cancer. HPV16 and HPV18 are the most common of the high risk group while HPV6 and HPV11 are among the low risk types. Approximately 90% of cervical cancers contain HPV DNA of the high risk types. Mutational analysis have shown that the E6 and E7 genes of the high risk HPVs are necessary and sufficient for HPV transforming function. The specific interactions of the E6 and E7 proteins with p53 and pRB, respectively, correlate with HPV high and low risk classifications. The high risk HPV E7 proteins bind to pRB with a higher affinity than do the low risk HPV proteins, and only the high risk HPV E6 proteins form detectable complexes with p53 *in vitro*.

REFERENCES

1. Reich, N.C., et al. 1983. Two distinct mechanisms regulate the levels of a cellular tumor antigen, p53. Mol. Cell. Biol. 3: 2143-2150.
2. Munger, K., et al. 1989. Complex formation of human papillomavirus E7 proteins with the retinoblastoma tumor suppressor gene product. EMBO J. 8: 4099-4105.
3. Hawley-Nelson, P., et al. 1989. HPV16 E6 and E7 proteins cooperate to immortalize human foreskin keratinocytes. EMBO J. 13: 3905-3910.
4. Munger, K., et al. 1989. The E6 and E7 genes of the human papillomavirus type 16 together are necessary and sufficient for transformation of primary human keratinocytes. J. Virol. 63: 4417-4421.
5. Riou, G., et al. 1990. Association between poor prognosis in early-stage invasive cervical carcinomas and non-detection of HPV DNA. Lancet 335: 1171-1174.

SOURCE

HPV16 E6 (N-17) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of HPV16 E6.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-1584 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

HPV16 E6 (N-17) is recommended for detection of HPV16 E6 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).

Molecular Weight of HPV16 E6: 17 kDa.

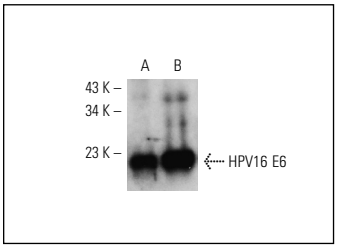
STORAGE

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.

DATA



Western blot analysis of HPV16 E6 expression in RKO E6 (**A**) and Ca Ski (**B**) whole cell lysates immunoprecipitated with HPV16 E6/18 E6 (C1P5): sc-460 and detected with HPV16 E6 (N-17): sc-1584.

SELECT PRODUCT CITATIONS

1. Lee, S.J., et al. 2001. Both E6 and E7 oncoproteins of human papillomavirus 16 inhibit IL-18-induced IFN-γ production in human peripheral blood mononuclear and NK cells. J. Immunol. 167: 497-504.
2. Cho, Y.S., et al. 2001. Down modulation of IL-18 expression by human papillomavirus type 16 E6 oncogene via binding to IL-18. FEBS Lett. 501: 139-145.
3. Fernandez, A.F., et al. 2009. The dynamic DNA methylomes of double-stranded DNA viruses associated with human cancer. Genome Res. 19: 438-451.
4. Chuang, C.Y., et al. 2012. Differential impact of IL-10 expression on survival and relapse between HPV16-positive and -negative oral squamous cell carcinomas. PLoS ONE 7: e47541.
5. Chen, T.H., et al. 2012. Human papilloma virus 16 E6 oncoprotein associated with p53 inactivation in colorectal cancer. World J. Gastroenterol. 18: 4051-4058.
6. Sung, W.W., et al. 2013. IL-10 promotes tumor aggressiveness via upregulation of CIP2A transcription in lung adenocarcinoma. Clin. Cancer Res. 19: 4092-4103.
7. Tung, M.C., et al. 2013. Association of epidermal growth factor receptor mutations with human papillomavirus 16/18 E6 oncoprotein expression in non-small cell lung cancer. Cancer 119: 3367-3376.
8. Zeng, W., et al. 2014. αNAC inhibition of the FADD-JNK axis plays anti-apoptotic role in multiple cancer cells. Cell Death Dis. 5: e1282.