SANTA CRUZ BIOTECHNOLOGY, INC.

E2F-5 (H-111): sc-1699

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The human retinoblastoma gene product appears to play an important role in the negative regulation of cell proliferation. Functional inactivation of Rb can be mediated either through mutation or as a consequence of interaction with DNA tumor virus encoded proteins. Of all the Rb associations described to date, the identification of a complex between Rb and the transcription factor E2F most directly implicates Rb in regulation of cell proliferation. E2F was originally identified through its role in transcriptional activation of the adenovirus E2 promoter. Sequences homologous to the E2F binding site have been found upstream of a number of genes that encode proteins with putative functions in the G_1 and S phases of the cell cycle. E2F-1 is a member of a broader family of transcription regulators including E2F-2, E2F-3, E2F-4, E2F-5 and E2F-6, each of which forms heterodimers with a second protein, DP-1, forming an "active" E2F transcriptional regulatory complex.

REFERENCES

1. Chellappan, S., et al. 1991. The E2F transcription factor is a cellular target for the Rb protein. Cell 65: 1053-1061.
2. Chittenden, T., et al. 1991. The T/E1A-binding domain of the retinoblastoma product can interact selectively with a sequence-specific DNA-binding protein. Cell 65: 1073-1082.
3. Helin, K., et al. 1993. Heterodimerization of the transcription factors E2F-1 and DP-1 leads to cooperative transactivation. Genes Dev. 7: 1850-1861.
4. Krek, W., et al. 1993. Binding to DNA and the retinoblastoma gene product promoted by complex formation of different E2F family members. Science 262: 1557-1560.
5. Ginsberg, D., et al. 1994. E2F-4, a new member of the E2F transcription factor family, interacts with p107. Genes Dev. 8: 2665-2679.

SOURCE

E2F-5 (H-111) is a rabbit polyclonal antibody raised against amino acids 89-200 of E2F-5 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.

Available as TransCruz reagent for Gel Supershift and ChIP applications, sc-1699 X, 200 µg/0.1 ml.

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.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.

APPLICATIONS

E2F-5 (H-111) is recommended for detection of E2F-5 and, to a lesser extent, E2F-1, E2F-2, E2F-3 and E2F-4 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); non cross-reactive with E2F-6.

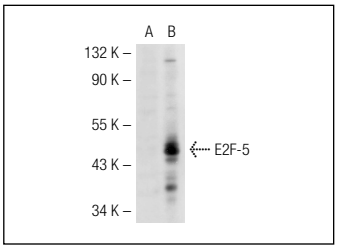
E2F-5 (H-111) is also recommended for detection of E2F-5 and, to a lesser extent, E2F-1, E2F-2, E2F-3 and E2F-4 in additional species, including equine, canine, bovine, porcine and avian.

E2F-5 (H-111) X TransCruz antibody is recommended for Gel Supershift and ChIP applications.

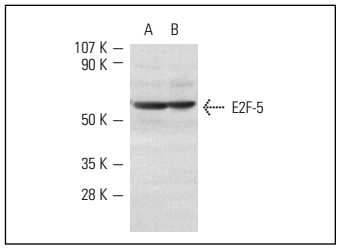
Molecular Weight of E2F-5: 59 kDa.

Positive Controls: E2F-5 (m): 293T Lysate: sc-119885, KNRK whole cell lysate: sc-2214 or K-562 whole cell lysate: sc-2203.

DATA



E2F-5 (H-111): sc-1699. Western blot analysis of E2F-5 expression in non-transfected: sc-117752 (**A**) and mouse E2F-5 transfected: sc-119885 (**B**) 293T whole cell lysates.



E2F-5 (H-111): sc-1699. Western blot analysis of E2F-5 expression in KNRK (**A**) and NIH/3T3 (**B**) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Campanero, M., et al. 1999. Distinct cellular factors regulate the c-Myb promoter through its E2F element. Mol. Cell. Biol. 19: 8442-8450.
2. Rathi, A.V., et al. 2007. Enterocyte proliferation and intestinal hyperplasia induced by simian virus 40 T antigen require a functional J domain. J. Virol. 81: 9481-9489.
3. Repici, M., et al. 2009. c-Jun N-terminal kinase binding domain-dependent phosphorylation of mitogen-activated protein kinase kinase 4 and mitogen-activated protein kinase kinase 7 and balancing cross-talk between c-Jun N-terminal kinase and extracellular signal-regulated kinase pathways in cortical neurons. Neuroscience 159: 94-103.