SANTA CRUZ BIOTECHNOLOGY, INC.

cyclin T2a/b (2128C1a): sc-81243

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The Power to Question

BACKGROUND

Cyclin T1 was identified as a partner for Cdk9, an RNA polymerase II (RNAPII) transcription elongation factor. Cyclin T1 interacts with the transactivation domain of the HIV-1 Tat protein. The interaction of Tat with cyclin T1 enhances the affinity of Tat for the viral TAR RNA stem-loop structure, suggesting that Tat can recruit cyclin T1/Cdk9 to RNAPII through cooperative binding to TAR. The human positive transcription elongation factor b (P-TEFb) consists of a cyclin dependent kinase, Cdk9, paired with a cyclin T. Cdk9 may be paired with either cyclin T1 or cyclin T2, in a mutually exclusive manner. Two forms of cyclin T2, T2a and T2b, are due to alternative splicing. The binding of Tat to TAR was shown to be facilitated by human cyclin T1, but not by cyclins T2a or T2b. Cyclin T2 binds to Cdk9 but not to Tat, and cyclin T2 can inhibit cyclin T1-mediated Tat activity.

REFERENCES

1. Herrmann, C.H., et al. 1995. Lentivirus Tat proteins specifically associate with a cellular protein kinase, TAK, that hyperphosphorylates the carboxyl-terminal domain of the large subunit of RNA polymerase II: candidate for a Tat cofactor. J. Virol. 69: 1612-1620.
2. Yang, X., et al. 1997. TAK, an HIV Tat-associated kinase, is a member of the cyclin-dependent family of protein kinases and is induced by activation of peripheral blood lymphocytes and differentiation of promonocytic cell lines. Proc. Natl. Acad. Sci. USA 94: 12331-12336.
3. Wei, P., et al. 1998. A novel CDK9-associated C-type cyclin interacts directly with HIV-1 Tat and mediates its high-affinity, loop-specific binding to TAR RNA. Cell 92: 451-462.
4. Peng, J., et al. 1998. Identification of multiple cyclin subunits of human P-TEFb. Genes Dev. 12: 755-762.
5. Wimmer, J., et al. 1999. Interactions between Tat and TAR and human immunodeficiency virus replication are facilitated by human cyclin T1 but not cyclins T2a or T2b. Virology 255: 182-189.

CHROMOSOMAL LOCATION

Genetic locus: CCNT2 (human) mapping to 2q21.3.

SOURCE

cyclin T2a/b (2128C1a) is a mouse monoclonal antibody raised against a recombinant protein corresponding to an internal region of cyclin T2a/b of human origin.

PRODUCT

Each vial contains 100 µg IgG_1 in 1.0 ml of PBS with < 0.1% sodium azide and 1.0% stabilizer protein.

STORAGE

For immediate and continuous use, store at 4° C for up to one month. For sporadic use, freeze in working aliquots in order to avoid repeated freeze/thaw cycles. If turbidity is evident upon prolonged storage, clarify solution by centrifugation.

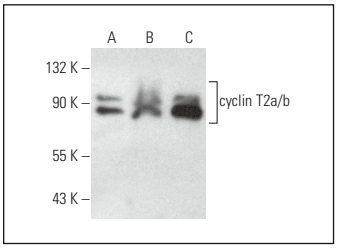
APPLICATIONS

cyclin T2a/b (2128C1a) is recommended for detection of cyclin T2a and cyclin T2b of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)].

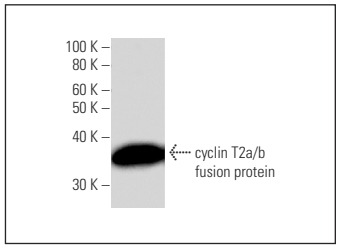
Suitable for use as control antibody for cyclin T2a/b siRNA (h): sc-37601, cyclin T2a/b shRNA Plasmid (h): sc-37601-SH and cyclin T2a/b shRNA (h) Lentiviral Particles: sc-37601-V.

Positive Controls: HeLa whole cell lysate: sc-2200, IMR-32 cell lysate: sc-2409 or Hep G2 cell lysate: sc-2227.

DATA



cyclin T2a/b (2128C1a): sc-81243. Western blot analysis of cyclin T2a/b expression in HeLa (**A**), IMR-32 (**B**) and Hep G2 (**C**) whole cell lysates.



cyclin T2a/b (2128C1a): sc-81243 Western Blot analysis of human recombinant cyclin T2a/b fusion protein.

SELECT PRODUCT CITATIONS

1. Blazek, D., et al. 2011. The cyclin K/Cdk12 complex maintains genomic stability via regulation of expression of DNA damage response genes. Genes Dev. 25: 2158-2172.
2. Liu, W., et al. 2013. Brd4 and JMJD6-associated anti-pause enhancers in regulation of transcriptional pause release. Cell 155: 1581-1595.
3. Li, C.F., et al. 2014. AMACR amplification in myxofibrosarcomas: a mechanism of overexpression that promotes cell proliferation with therapeutic relevance. Clin. Cancer Res. 20: 6141-6152.
4. Dahan, S., et al. 2021. VEGFA's distal enhancer regulates its alternative splicing in CML. NAR Cancer 3: zcab029.
5. Cheng, S.S., et al. 2022. Inhibition of the CDK9-cyclin T1 protein-protein interaction as a new approach against triple-negative breast cancer. Acta Pharm. Sin. B 12: 1390-1405.
6. Hafer, T.L., et al. 2023. A CRISPR screen of HIV dependency factors reveals that CCNT1 is non-essential in T cells but required for HIV-1 reactivation from latency. Viruses 15: 1863.
7. Hafer, T.L., et al. 2023. A CRISPR screen of HIV dependency factors reveals CCNT1 is non-essential in T cells but required for HIV-1 reactivation from latency. bioRxiv. E-published.

RESEARCH USE

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