SANTA CRUZ BIOTECHNOLOGY, INC.

Mat1 (FL-309): sc-6234

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Progression through the cell cycle requires activation of a series of enzymes designated cyclin dependent kinases (Cdks). The monomeric catalytic subunit, Cdk2, a critical enzyme for initiation of cell cycle progression, is completely inactive. Partial activation is achieved by the binding of regulatory cyclins such as cyclin D1, while full activation requires phosphorylation at Thr-160. The enzyme responsible for phosphorylation of Thr-160 in Cdk2 and also Thr-161 in Cdc2 p34, designated Cdk-activating kinase (CAK), has been partially purified and shown to be comprised of a catalytic subunit, a regulatory subunit and a subunit of unknown function. The regulatory subunit is a novel cyclin (cyclin H) and is required for activation of Cdk7. This previously undescribed protein, now termed Mat1, has been cloned as a protein that associates with the cyclin H-Cdk7 nuclear complex at all stages of the cell cycle. Cyclin H-Cdk7-Mat1 complexes display kinase activity towards Cdk activation domains, and the carboxy terminus of RNA polymerase II. Mat1 appears to constitute the first example of an assembly factor, essential for the formation of an active Cdk-cyclin complex.

CHROMOSOMAL LOCATION

Genetic locus: MNAT1 (human) mapping to 14q23.1; Mnat1 (mouse) mapping to 12 C3.

SOURCE

Mat1 (FL-309) is a rabbit polyclonal antibody raised against amino acids 1-309 representing full length Mat1 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.

APPLICATIONS

Mat1 (FL-309) is recommended for detection of Mat1 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).

Mat1 (FL-309) is also recommended for detection of Mat1 in additional species, including equine, canine, bovine, porcine and avian.

Suitable for use as control antibody for Mat1 siRNA (h): sc-35861, Mat1 siRNA (m): sc-35862, Mat1 shRNA Plasmid (h): sc-35861-SH, Mat1 shRNA Plasmid (m): sc-35862-SH, Mat1 shRNA (h) Lentiviral Particles: sc-35861-V and Mat1 shRNA (m) Lentiviral Particles: sc-35862-V.

Molecular Weight of Mat1: 36 kDa.

Positive Controls: A-431 nuclear extract: sc-2122, A-673 nuclear extract: sc-2128 or NIH/3T3 nuclear extract: sc-2138.

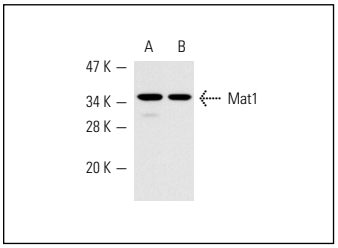
STORAGE

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



Mat1 (FL-309): sc-6234. Western blot analysis of Mat1 expression in A-431 (**A**) and A-673 (**B**) nuclear extracts.

SELECT PRODUCT CITATIONS

1. Kilbey, A., et al. 1999. Loss of cell cycle control by deregulation of cyclin-dependent kinase 2 kinase activity in Evi-1 transformed fibroblasts. Cell Growth Differ. 10: 601-610.
2. Wang, J.G., et al. 2006. Retinoic acid induces leukemia cell G_1 arrest and transition into differentiation by inhibiting cyclin-dependent kinase-activating kinase binding and phosphorylation of PML/RARα. FASEB J. 20: 2142-2144.
3. Luo, P., et al. 2007. Intrinsic retinoic acid receptor α-cyclin-dependent kinase-activating kinase signaling involves coordination of the restricted proliferation and granulocytic differentiation of human hematopoietic stem cells. Stem Cells 25: 2628-2637.
4. Helenius, K., et al. 2009. Mat1 inhibits peroxisome proliferator-activated receptor γ-mediated adipocyte differentiation. Mol. Cell. Biol. 29: 315-323.
5. Watanabe, G., et al. 2009. αB-crystallin: a novel p53-target gene required for p53-dependent apoptosis. Cancer Sci. 100: 2368-2375.
6. Ola, A., et al. 2010. The mixed-lineage kinase 1-3 signalling pathway regulates stress response in cardiac myocytes via GATA-4 and AP-1 transcription factors. Br. J. Pharmacol. 159: 717-725.
7. Arab, H.H., et al. 2010. Dissociation of CAK from core TFIIH reveals a functional link between XP-G/CS and the TFIIH disassembly state. PLoS ONE 5: e11007.
8. Zhu, Q., et al. 2012. Lack of CAK complex accumulation at DNA damage sites in XP-B and XP-B/CS fibroblasts reveals differential regulation of CAK anchoring to core TFIIH by XPB and XPD helicases during nucleotide excision repair. DNA Repair 11: 942-950.