

Anti-Cyclin D1 CCND1 Rabbit Monoclonal Antibody

Catalog Number: M00149-1

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Overview

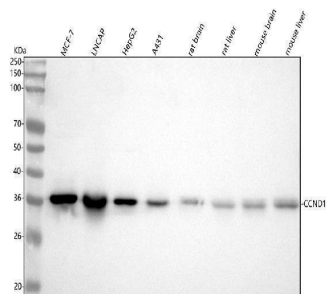
Product Name	Anti-Cyclin D1 CCND1 Rabbit Monoclonal Antibody
Reactive Species	Human, Mouse, Rat
Description	Boster Bio Anti-Cyclin D1 CCND1 Rabbit Monoclonal Antibody catalog # M00149-1. Tested in WB, IHC, ICC/IF, IP applications. This antibody reacts with Human, Mouse, Rat.
Application	IP, IF, IHC, ICC, WB
Clonality	Monoclonal DAD-3
Formulation	Rabbit IgG in stabilizing components, phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol. *This antibody is supplied in a stabilized formulation. Compatibility with conjugation reactions depends on the chemistry of the conjugation method used. For conjugation methods that are not compatible with the stabilizing components present in this formulation, a carrier-free antibody format is required.
Storage Instructions	Store at -20°C for one year. For short term storage and frequent use, store at 4°C for up to one month. Avoid repeated freeze-thaw cycles.
Host	Rabbit
Uniprot ID	P24385

Technical Details

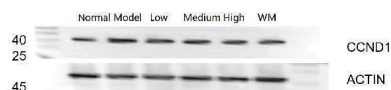
Immunogen	A synthesized peptide derived from human Cyclin D1
Isotype	Rabbit IgG
Form	Liquid
Concentration	0.5mg/ml
Purification	Affinity-chromatography
Suggested Dilutions	WB 1:500-2000

	IHC 1:50-200 ICC/IF 1:50-200 IP 1:20
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Anti-Cyclin D1 CCND1 Rabbit Monoclonal Antibody (M00149-1) Images

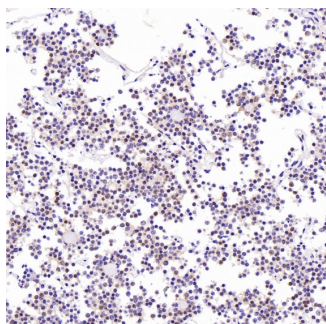
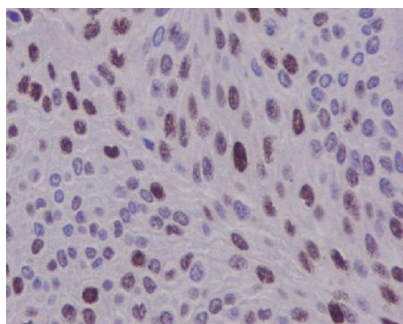


Western blot analysis of Cyclin D1 using anti-Cyclin D1 antibody (M00149-1). Electrophoresis was performed on a 5-20% SDS-PAGE gel at 70V (Stacking gel) / 90V (Resolving gel) for 2-3 hours. The sample well of each lane was loaded with 30 ug of sample under reducing conditions. Lane 1: human MCF-7 whole cell lysates, Lane 2: human LNCAP whole cell lysates, Lane 3: human HepG2 whole cell lysates, Lane 4: human A431 whole cell lysates, Lane 5: rat brain tissue lysates, Lane 6: rat liver tissue lysates, Lane 7: mouse brain tissue lysates, Lane 8: mouse liver tissue lysates. After electrophoresis, proteins were transferred to a nitrocellulose membrane at 150 mA for 50-90 minutes. Blocked the membrane with 5% non-fat milk/TBS for 1.5 hour at RT. The membrane was incubated with rabbit anti-Cyclin D1 antigen affinity purified monoclonal antibody (Catalog # M00149-1) at 1:1000 overnight at 4°C, then washed with TBS-0.1%Tween 3 times with 5 minutes each and probed with a goat anti-rabbit IgG-HRP secondary antibody at a dilution of 1:1000 for 1.5 hour at RT. The signal is developed using an Enhanced Chemiluminescent detection (ECL) kit (Catalog # EK1002) with Tanon 5200 system. A specific band was detected for Cyclin D1 at approximately 34 kDa. The expected band size for Cyclin D1 is at 34 kDa.

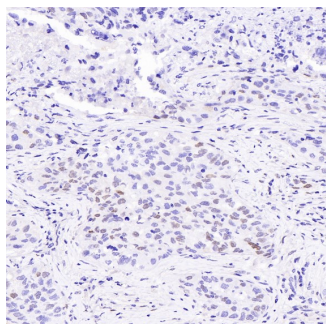


Western blot analysis of Cyclin D1 using anti-Cyclin D1 antibody (M00149-1). Electrophoresis was performed on a 5-20% SDS-PAGE gel at 70V (Stacking gel) / 90V (Resolving gel) for 2-3 hours. The sample well of each lane was loaded with 30 ug of sample under reducing conditions. Lane 1: Normal group-rat colon tissue lysates, Lane 2: Model group-rat colon tissue lysates, Lane 3: Triditional Chinese medicine treatment (low dose)-rat colon tissue lysates, Lane 4: Triditional Chinese medicine treatment (medium dose)-rat colon tissue lysates, Lane 5: Triditional Chinese medicine treatment (high dose)-rat colon tissue lysates, Lane 6: Western medicine-rat colon tissue lysates. After electrophoresis, proteins were transferred to a nitrocellulose membrane at 150 mA for 50-90 minutes. Blocked the membrane with 5% non-fat milk/TBS for 1.5 hour at RT. The membrane was incubated with rabbit anti-Cyclin D1 antigen affinity purified monoclonal antibody (Catalog # M00149-1) at 1:1000 overnight at 4°C, then washed with TBS-0.1%Tween 3 times with 5 minutes each and probed with a goat anti-rabbit IgG-HRP secondary antibody for 1 hour at RT. The signal is developed using an Enhanced Chemiluminescent detection (ECL) kit (Catalog # EK1002) with ChemiDoc MP system. A specific band was detected for Cyclin D1 at approximately 36 kDa. The expected band size for Cyclin D1 is at 34 kDa.

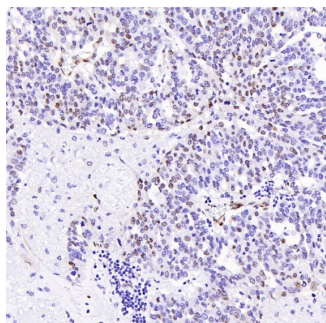
Immunohistochemical analysis of paraffin-embedded human bladder, using Cyclin D1 Antibody.



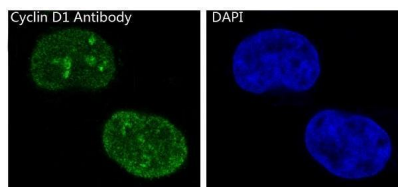
Immunohistochemical analysis of paraffin-embedded Human pituitary tumor, using the Antibody at 1:100 dilution.



Immunohistochemical analysis of paraffin-embedded Human squamous cell carcinoma, using the Antibody at 1:100 dilution.

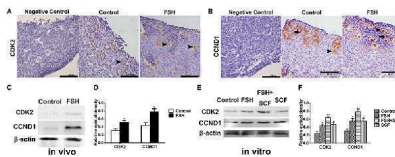
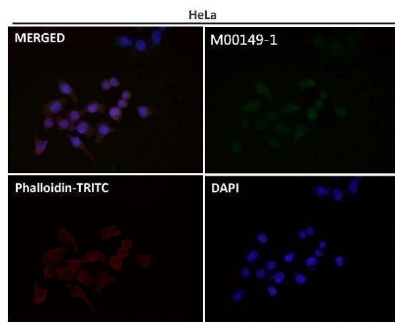


Immunohistochemical analysis of paraffin-embedded Human ovarian cancer, using the Antibody at 1:500 dilution.

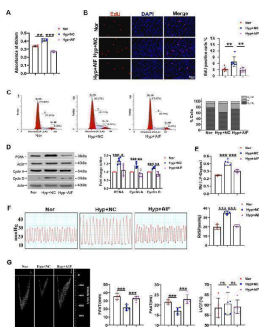


Immunofluorescent analysis of MCF-7 cells, using Cyclin D1 Antibody.

Immunofluorescent analysis using the Antibody at 1:50 dilution.

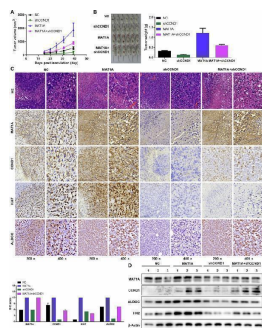


Combined effects of FSH and SCF on the expressions of cell cycling proteins CCND1 and CDK2. (A-D) 4-day-old chickens were treated by FSH in vivo . Brown staining represents the immunohistochemical antigen. (E,F) 4-day-old chicken ovaries were treated by FSH or SCF for 2 days in vitro . (A) Arrowheads represent oocytes stained with CDK2 antibody. (B) Arrowheads and arrows represent oocytes and somatic cells stained with CCND1 antibody. (C,D) Western blot and gray analysis indicate that FSH promoted the CDK2 and CCND1 protein expression in vivo . (E,F) Western blot and gray analysis indicate that SCF enhanced the effect of FSH on promoting the CDK2 and CCND1 protein expression in vitro . T -tests were used to determine statistically significant differences. The values are the mean $\pm$ SEM of six experiments. Asterisks indicate significant differences (* $P < 0.05$, ** $P < 0.01$). Scale bars: 50 μ m. Index in PubMed under a CC BY license. PMID: 30837955

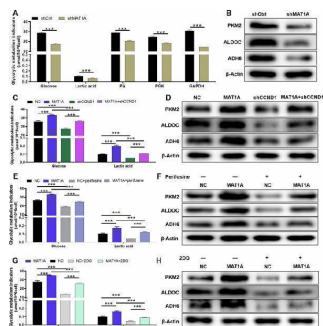


AIF blocks hypoxia-induced progression of PH in vitro and in vivo. A Hypoxia increased the viability of PSMCs after growth arrest for 24 h, and this effect was decreased by AIF ($n = 4$). B Pretreatment with an AIF overexpression plasmid blocked the effects of hypoxia on EdU incorporation in cells ($n = 6$). Scale bars: 50 μ m. C Cell cycle analysis by flow cytometry indicated that hypoxia stimulated cell progression into G2/M + S phase, and this effect was inhibited by AIF overexpression ($n = 3$). D Effects of AIF on the expression of PCNA, Cyclin A and Cyclin D under hypoxia ($n = 4-5$). E Represents weight ratio of the right ventricular (RV)/left ventricular (LV) + Septum ($n = 6$); F Represents the right ventricular systolic pressure (RVSP) from mice ($n = 5$); G pulmonary artery velocity time integral (PAVTI), pulmonary artery acceleration time (PAAT) and left ventricular ejection fraction (LVEF) of the hypoxic mouse model infected with AAV5-NC and AAV5-AIF ($n = 6$). All data are presented as the means $\pm$ standard deviation. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Nor normoxia, Hyp hypoxia, NC negative control Index in PubMed under a CC BY license. PMID: 35090552

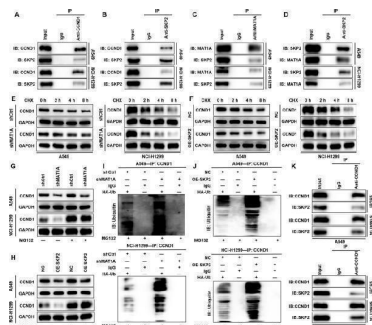
The MAT1A/CCND1 signaling axis promotes glycolysis and tumorigenesis in NSCLC. The xenograft tumor model in nude mice established by A549 cells that interfered with MAT1A and CCND1 expression. After 39 days of monitoring, A tumor volume and B tumor weight was measured. C HE, IHC staining and D WB analysis was performed to determine the



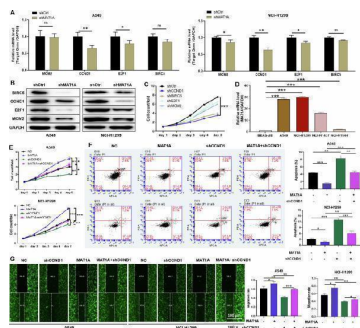
expression of MAT1A, CCND1, proliferation marker Ki67 and glycolytic enzymes (ALDOC, HK2) after tumor tissue sections or protein extraction. Index in PubMed under a CC BY license. PMID: 39438468



MAT1A enhances the glycolysis of NSCLC cells through CCND1. The metabolic profile of glycolysis in MAT1A knockdown A549 cells was analyzed, including (A) glucose utilization, lactic acid production, PA, PGK, GAPDH activity and (B) expression of pivotal glycolytic enzymes (PKM2, ALDOC, ADH6). C , D Measurement of glucose utilization, lactic acid production and (D) expression of pivotal glycolytic enzymes (PKM2, ALDOC, ADH6) in A549 cells (NC, as negative control; MAT1A-overexpressing; CCND1 knockdown; MAT1A-overexpressing and CCND1 knockdown). E , F MAT1A-overexpressed A549 cells treated with AKT inhibitor (perifosine) and measurement of (E) glucose utilization, lactic acid production and (F) expression of pivotal glycolytic enzymes (PKM2, ALDOC, ADH6). G , H MAT1A-overexpressed A549 cells treated with glycolysis inhibitor 2-deoxy-d-glucose (2-DG) and measurement of (G) glucose utilization, lactic acid production and (H) expression of pivotal glycolytic enzymes (PKM2, ALDOC, ADH6). Results were shown as mean $\pm$ SD. * $p < 0.05$, ** $p < 0.05$, *** $p < 0.001$. Index in PubMed under a CC BY license. PMID: 39438468



MAT1A inhibited the ubiquitination of CCND1 by blocking the binding of SKP2 to CCND1. In co-immunoprecipitation experiments, the total lysates of A549 and NCI-H1299 cells were immunized with (A) anti-CCND1 or (B) anti-SKP2 and WB, respectively. In the co-immunoprecipitation experiments, total lysates of A549 and NCI-H1299 cells were immunized with (C) anti-MAT1A or (D) anti-SKP2 and WB. E MAT1A knockdown and control A549 and NCI-H1299 cells were treated with protein synthesis inhibitor (CHX, cycloheximide from microbial, 10 μ mol/L) and then subjected to cycloheximide chase assays. F SKP2 overexpressed and control A549 and NCI-H1299 cells were treated with CHX (10 μ mol/L) to detect the relative protein levels of CCND1 at indicated time points. CCND1 protein level was determined in (G) MAT1A-depleted or (H) SKP2-overexpressed A549 and NCI-H1299 cells treated with or without proteasome inhibitor (MG132, 10 μ mol/L). The ubiquitin of CCND1 immunoprecipitated in (I) MAT1A-depleted or (J) SKP2-overexpressed A549 and NCI-H1299 cells was detected. K Total lysates of A549 and NCI-H1299 cells with or without MAT1A knockdown were immunized with anti-CCND1 and WB. Index in PubMed under a CC BY license. PMID: 39438468



MAT1A facilitated NSCLC cell proliferation and migration through CCND1. A Relative mRNA levels of top 4 DEGs were detected by qPCR analysis in A549 and NCI-H1299 cell after MAT1A depletion. B Relative protein levels of CCND1, BIRC5, E2F1 and MCM2 in A549 and NCI-H1299 cell after MAT1A depletion were determined by WB assays. C Cell viabilities of A549 cell after depletion of CCND1, BIRC5, E2F1 and MCM2 were assessed by Celigo cell count assays at indicated times. D CCND1 expression levels in human NSCLC cell lines (A549, NCI-H1299 and NCI-H1944) and normal human lung epithelial cell line (BEAS-2B) were determined by qPCR. A549 and NCI-H1299 cells were grouped as NC (empty vector, as negative control), MAT1A (MAT1A-overexpressing), shCCND1 (CCND1 knockdown) and MAT1A+shCCND1 (MAT1A-overexpressing and CCND1-knockdown) groups, performed (E) Celigo cell count assays, (F) flow cytometry, (G) wound-healing assays. Results were shown as mean $\pm$ SD. * $p < 0.05$, ** $p < 0.05$, *** $p < 0.001$. Index in PubMed under a CC BY license. PMID: 39438468

27 Publications Citing This Product

1. PubMed ID: 31467112, Chen Q, Deng X, Hu X, Guan S, He M, Wang Y, Wei B, Zhang J, Zhao H, Yao W, Jin F, Liu Y, Chen J, Olapade OI, Wu H, Wei M. Breast Cancer Risk-Associated SNPs in the mTOR Promoter Form De Novo KLF5- and ZEB1-Binding Sites that Influence the Cellular Response to Paclitaxel. *Mol Cancer Res*. 2019 Nov;17(11):2244-2256. doi:10.1158/1541-7786.MCR-18-1072. Epub 2019 Aug 29. PMID: 31467112.
2. PubMed ID: 30610869, Xiang W, Jiang T, Hao X, Wang R, Yao X, Sun K, Guo F, Xu T. Primary cilia and autophagy interaction is involved in mechanical stress mediated cartilage development via ERK/mTOR axis. *Life Sci*. 2019 Feb 1;218:308-313. doi:10.1016/j.lfs.2019.01.001. Epub 2019 Jan 3. PMID: 30610869.
3. PubMed ID: 31102746, Zhang H, Su Y, Wang J, Gao Y, Yang F, Li G, Shi Q. Ginsenoside Rb1 promotes the growth of mink hair follicle via PI3K/AKT/GSK-3 β signaling pathway. *Life Sci*. 2019 Jul 15;229:210-218. doi:10.1016/j.lfs.2019.05.033. Epub 2019 May 16. PMID: 31102746.

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