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Gene Section
Review

CCND1 (B-cell leukemia/lymphoma 1)

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Abstract

Review on CCND1, with data on DNA, on the protein encoded, and where the gene is implicated.

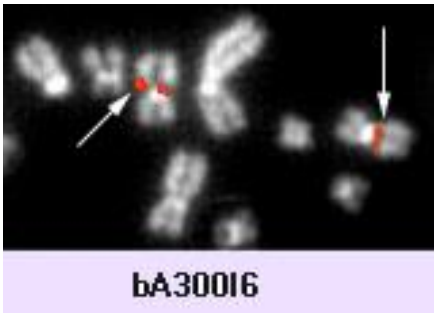
Keywords
CCND1; Cell cycle

Identity

HGNC (Hugo): CCND1

Location: 11q13.3

Other names: BCL1, U21B31, D11S287E



BCL1 (11q13) - Courtesy Mariano Rocchi.

DNA/RNA

Description

Located in the long (q) arm of chromosome 11 in the 13th band, the length of the CCND 1 gene is about 13.38 Kb (precisely 13,388 bases), contains 5 exons and is arranged in a telomere to centromere orientation.

Transcription

According to Ensembl, the full length, functional transcript of CCND1 (Transcript ID ENST00000227507) is 4307 bp in length, encoding 5 coding exons.
From the total of 6 transcripts generated, only two are protein coding.

Pseudogene

None reported.

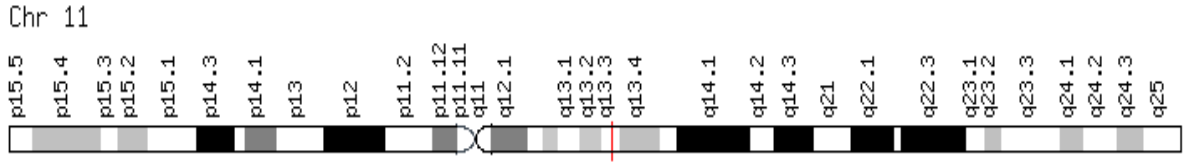
Protein

Description

The full length CCND1 protein has a length of 295 amino acids, having a molecular weight of 33729 Da. CCND1 is a member of the cyclin family, Cyclin D subfamily and contains 1 cyclin N-terminal domain.

Localisation

Nuclear, cytoplasmic and membrane. Accumulation of CCND1- CDK4 complexes occur in the nuclear membrane, which are then transported to the nucleus through interactions with KIP-CIP family member proteins (By similarity, a LaBaer et. al.,1997).



The figure shows the chromosomal location of CCND1 (Red line). Image courtesy genecards.org

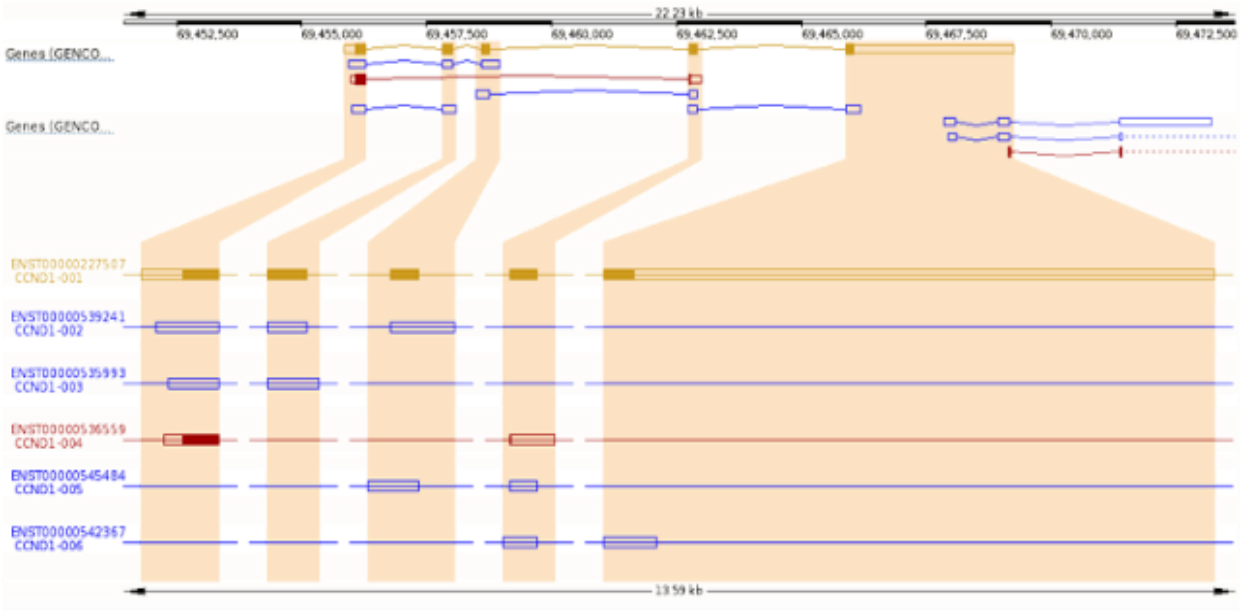
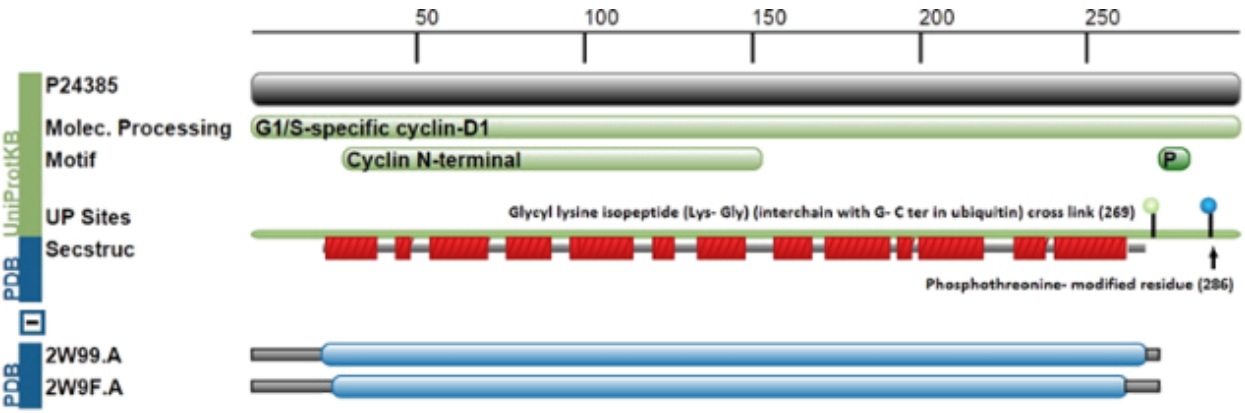
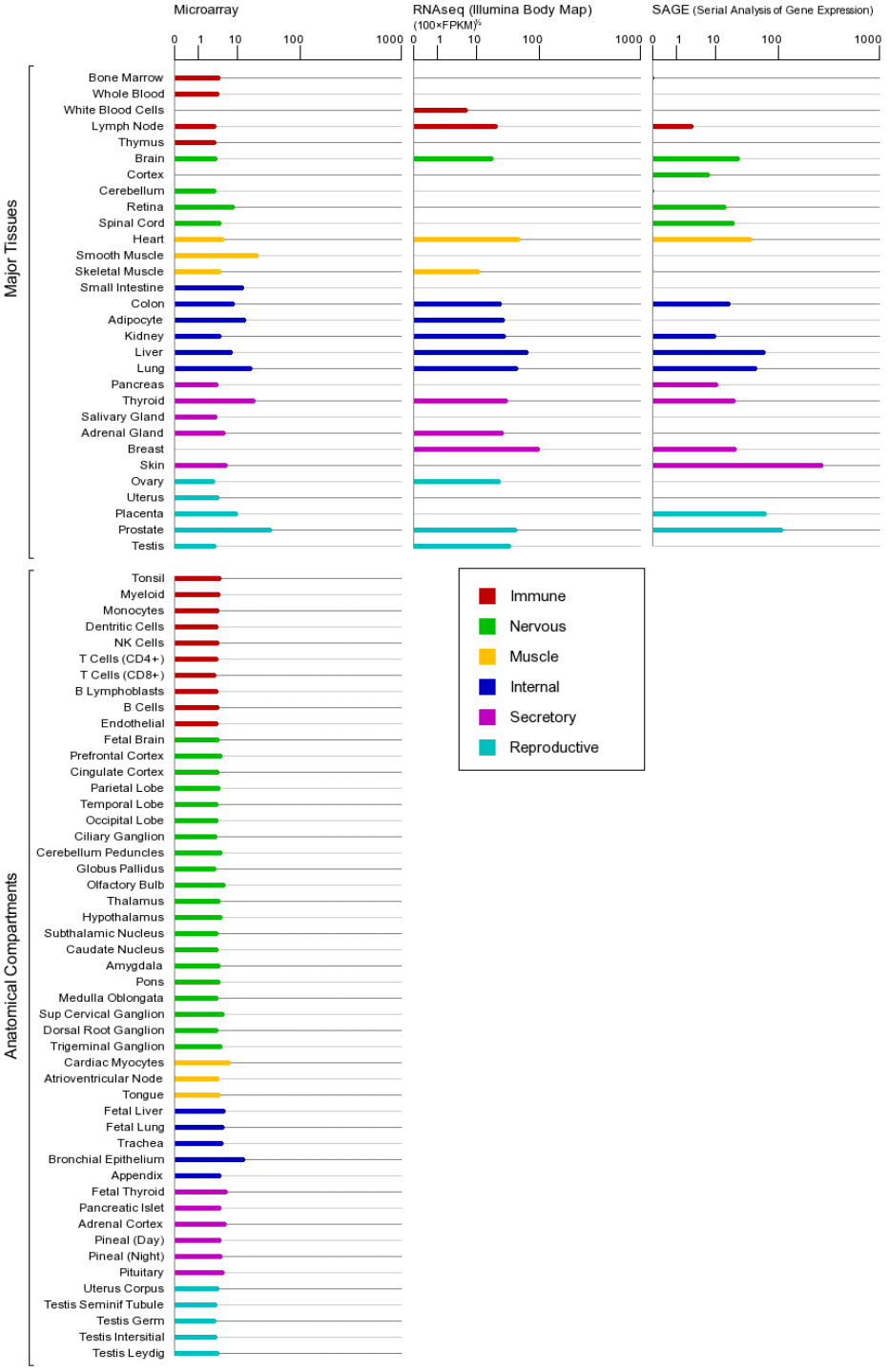


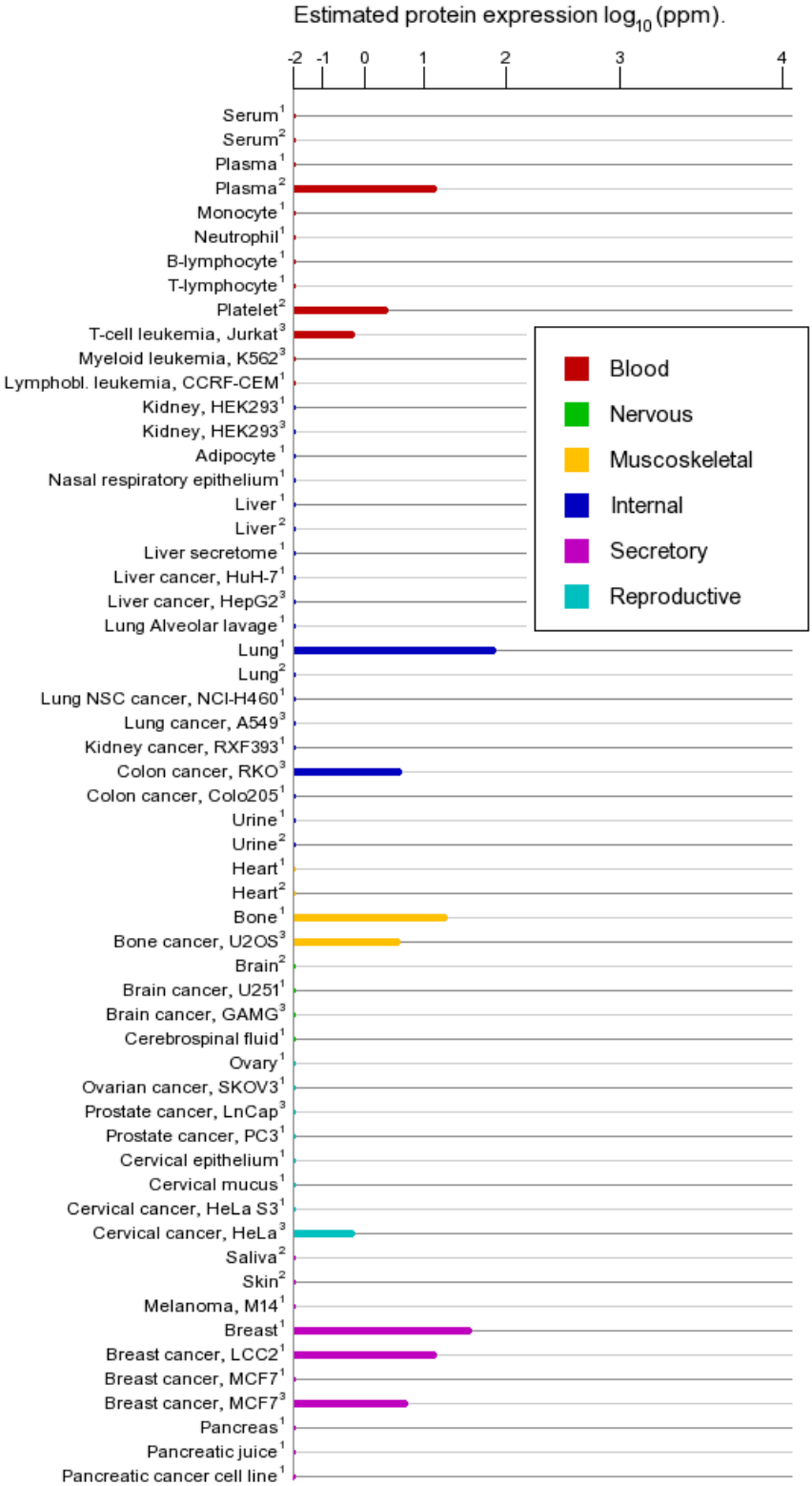
Diagram shows the different transcripts of CCND1 (BROWN, BLUE AND MAROON BOXES). Beginning of boxes represents transcription start sites. Filled areas represent translated regions. The brown box representing transcript CCND- 001 forms the full length, active protein. Image adapted from Ensembl.org



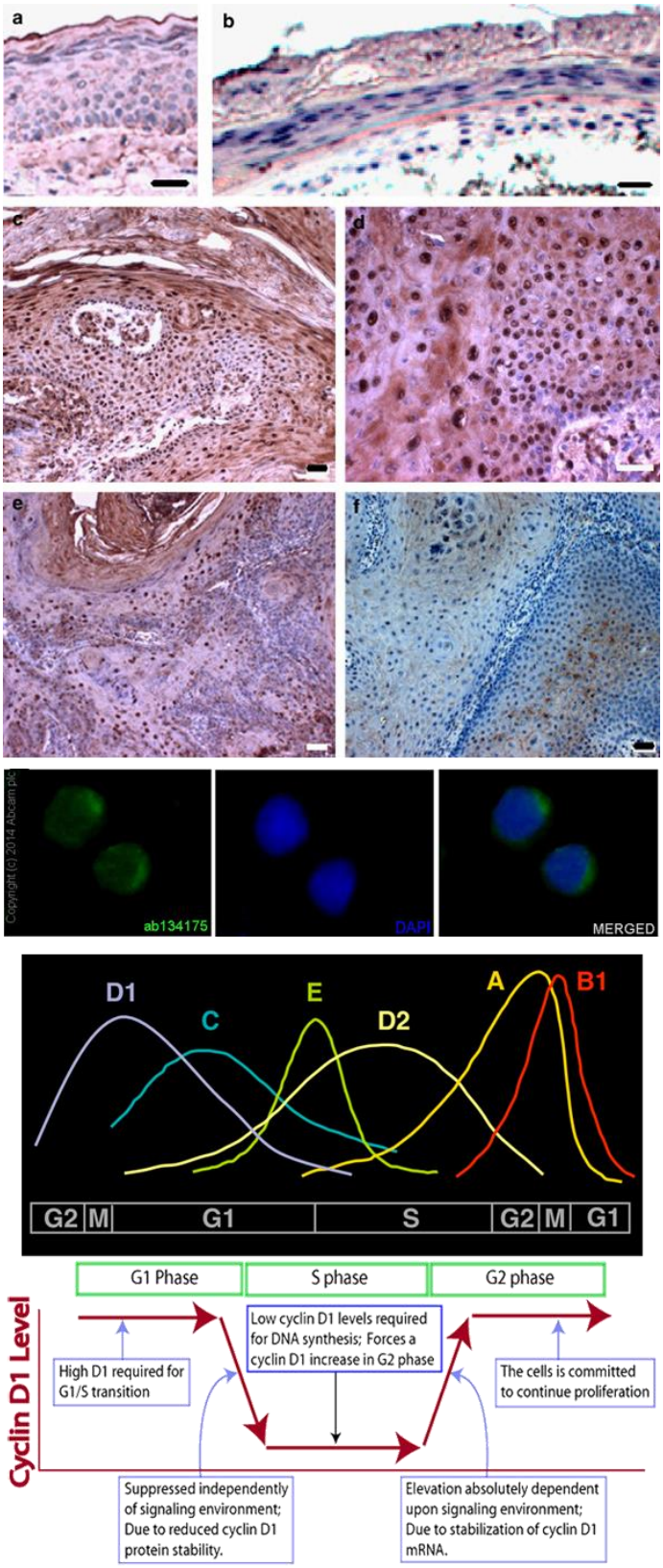
Schematic diagram of full length CCND1, showing different domains. Adapted from PDB P24385.
Data origin/ Colour codes: Data in Green originates from UniProtKB.; Data in blue originates from PDB. Secstruc- Secondary structure projected from representative PDB entries onto the UniProt sequence. a. Red box- Helix. b. Grey tube- Coil. Data in red indicates combined ranges of Homology Models from SBKB and the Protein Model Portal.



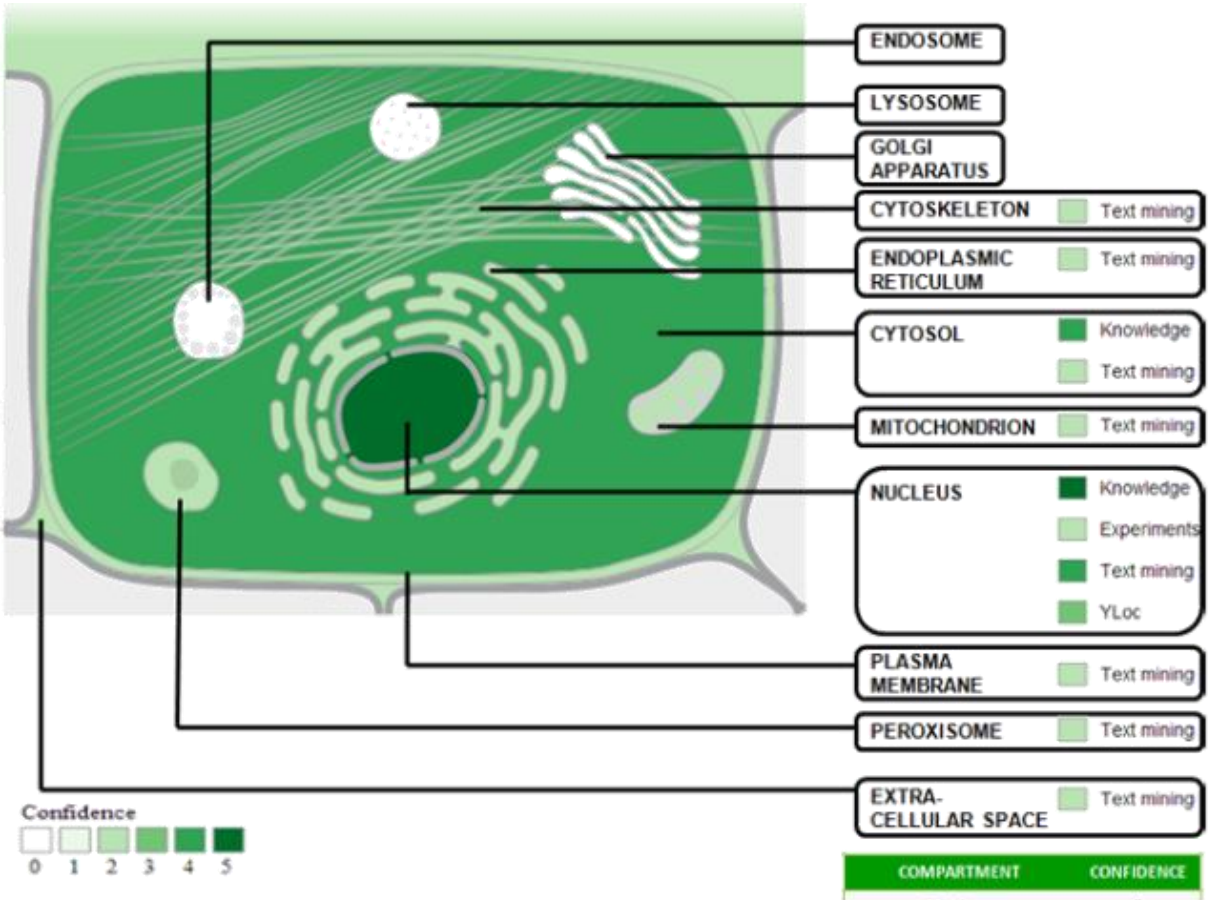
The RNA expression data of CCND1 based on data from BioGPS, Illumina Human BodyMap, and SAGE, with SAGE tags from CGAP, Figure shows RNA expression data (presence/absence) for RNA genes is according to H-InvDB, NONCODE, miRBase, and RNAdb. The expression images based on data from BioGPS, Illumina Human BodyMap, and SAGE, with SAGE tags from CGAP. BioGPS: 76 normal tissues were used and compartments hybridized against HG-U133A, with Affeymetrix MAS5 algorithm used in array processing. Illumina body map: Transcripts were mapped to genes from 16 normal human tissues by sequencing. Cufflinks program was used to calculate Fragments per Kilobase of exon per Million fragments mapped (FPKM) and rescaled by multiplying FPKM by 100 and calculating the root. CGAP: SAGE Normal. For Serial Analysis of Gene Expression (SAGE) of 19 normal human tissues, Hs frequencies and Hs libraries in CGAP datasets are mined for information regarding the number of SAGE tags per tissue. Unigene clustering was applied to Tags, followed by a particular gene by mining Hs best gene, Hs best tag and Hs GeneData. The number of appearances of the corresponding tag divided by the total number of tags in libraries derived from that tissue was used in calculating the level of expression of a particular gene, which were then rescaled by making the genomic mean of all tissues equal. Intermediate between log and linear scales are normalized intensities drawn on root scale, with values not comparable between datasets (i.e. Microarray, RNAseq and SAGE). Figure courtesy: genecards.org.



Presentation of protein expression images for 35 tissues, fluids and cells. Data sources: MOPED - Eugene Kolker, Bioinformatics & High-throughput Analysis Lab, Seattle Children's Research Institute; PaxDb - Christian von Mering, Bioinformatics Group, Institute of Molecular Life Sciences, University of Zurich; MAXQB - Matthias Mann, Department of Proteomics and Signal Transduction, Max-Planck Institute of Biochemistry, Germany. The data was normalized as follows: For each sample, ppm protein values were calculated, if not provided so by data sources. For each sample from MAXQB, iBAQ expression values were divided by sum of values of each sample, and multiplied by 1,000,000. For all samples, data was gene centrically aggregated by summing expression values of all isoforms for each gene. For better visualization of graphs, expression values are drawn on a root scale, which is an intermediate between log and linear scales as used for our mRNA expression graphs (PMID 12519968).



Expression in tissues: Top: Cyclin D1 overexpression in keratoacanthomas (KAs) and squamous cell carcinomas (SCCs). CCND1 (brown), counterstain hemalaun (blue). (a) Normal skin and (b) actinic keratosis, a precursor lesion of SCCs. (c-f) Representative KAs (c) Higher magnification of a different tumor (d); medium expression (e); and low expression (f) of cyclin D1. Bar=50 micro-m. Image courtesy Burnworth et. al., 2006. Middle: The figure shows the localization of CCND1 in Ramos cells. Image courtesy Abcam. Bottom: Expression during cell cycle: Image shows the levels expression of CCND1 during different phases of the cell cycle (left panel) and the function associated in each phase (right). Image courtesy kinexux.ca (left) and Yang et. al., 2006 (right).



COMPARTMENT	CONFIDENCE
Nucleus	5
Cytosol	5
Cytoskeleton	2
Endoplasmic reticulum	2
Extracellular	2
Mitochondria	2
Peroxisome	2
Plasma membrane	2

LEGEND DIAGRAM:
The subcellular localizations of CCND1 in different cellular compartments are shown.
The information is derived from database annotations, automatic text mining of the biomedical literature, and sequence-based predictions.
The confidence or probability of localization of CCND1 in any compartment is represented by a range of colours from white (low or no probability of protein being present) to the darkest green (protein will always be present).
Confidence score for presence of CCND1 in different compartments is given in the table.
Picture adapted from genecards.org.

Function

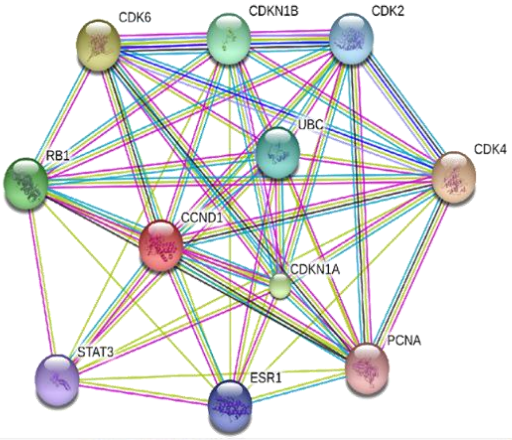
CCND1 binds and activates the G1 cyclin dependent kinases, Cdk4 and Cdk6. The complex then phosphorylates and inhibits members of the retinoblastoma (RB) family of protein including RB1, thereby regulating the G1/S transition in the cell cycle (Kato et al., 1993).

CCND1 has a kinase-independent function of sequestering CDK inhibitors such as p27 Kip1 and p21Cip1and promoting efficient activation of Cyclin E/CDK2-containing complexes (Polyak et al., 1994; Sherr and Roberts, 1999).

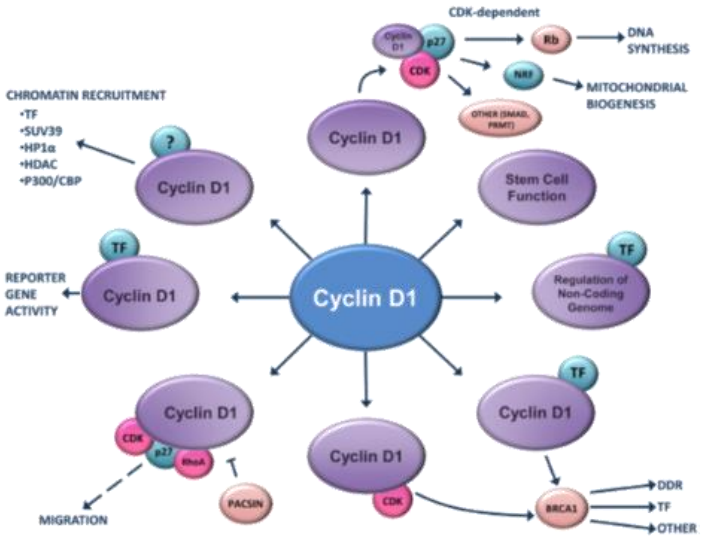
CCND1 phosphorylates Smad3 and inhibits its transcriptional activity and antiproliferative function (Matsuura et. al., 2004).

Homology

The CCND1 gene is conserved in chimpanzee, Rhesus monkey, dog, cow, mouse, rat, chicken, zebrafish, and frog (According to Homologene, NCBI).



	PREDICTED FUNCTIONAL PARAMETERS								SCORE
	CDK 4: Cyclin dependent kinase 4; Probably involved in the control of the cell cycle (303 aa).								0.999
	CDK 6: Cyclin dependent kinase 6; Probably involved in the control of the cell cycle. Interacts with D -type G1 cyclins (326 aa).								0.999
	CDK N1A: Cyclin dependent kinase inhibitor 1A (p21, Cip 1); May be the important intermediate by which p53 mediates its role as in inhibitor of cellular proliferation in response to DNA damage. Binds to and inhibits cyclin-dependent kinase activity, preventing phosphorylation of critical cyclin- dependent substrates and blocking cell cycle progression (164 aa).								0.999
	RB1: Retinoblastoma 1; Key regulator of entry into cell division that acts as a tumor suppressor. Acts as a transcription repressor of E2F1 target genes. The underphosphorylated, active form of RB1 interacts with E2F1 and represses its transcription activity, leading to cell cycle arrest. Directly involved in heterochromatin formation by maintaining overall chromatin structure and, in particular, that of constitutive heterochromatinby stabilizing histone methylation.Recruits and targets histone methyltransferases SUV39H1, SUV420H1 and SUV420H2, leading to epigenetic transcription (928 aa).								0.999
	CDK N1B: Cyclin dependent kinase inhibitor 1B (p27, Kip 1); Important regulator of cell cycle progression. Involved in G1 arrest. Potent inhibitor of cyclin E and cyclin A-CDK2 complexes. Positive regulator of cyclin D-dependent kinases such as CDK4. Regulated by phosphorylation and degradation events (198 aa).								0.999
	UCB: Ubiquitin C (685 aa).								0.900
	CDK2: Cyclin dependent kinase 2; Involved in the control of the cell cycle. Interacts with cyclins A, B1, B3, D or E. Activity of CDK2 is maximal during S phase and G2 (298 aa).								0.999
	ESR1: Estrogen receptor 1; Nuclear hormone receptor. The steroid hormones and their receptors are involved in the regulation of eukaryotic gene expression and affect cellular proliferation and differentiation in target tissues (595 aa).								0.999
	STAT3: Signal transduction and activation of transcription factor 3 (acute phase response factor); Transcription factor that binds to the interleukine -6 (IL-6)- responsive elements identified in the promoters of various acute-phase protein genes. Activated by IL31 through IL31RA (770 aa).								0.999
	PCNA: Proliferating cell nuclear antigen; This protein is an auxiliary protein of DNA polymerase delta and is involved in the control of eukaryotic DNA replication by increasing the polymerase's processibility during elongation of the leading strand (by similarity) (261 aa).								0.999

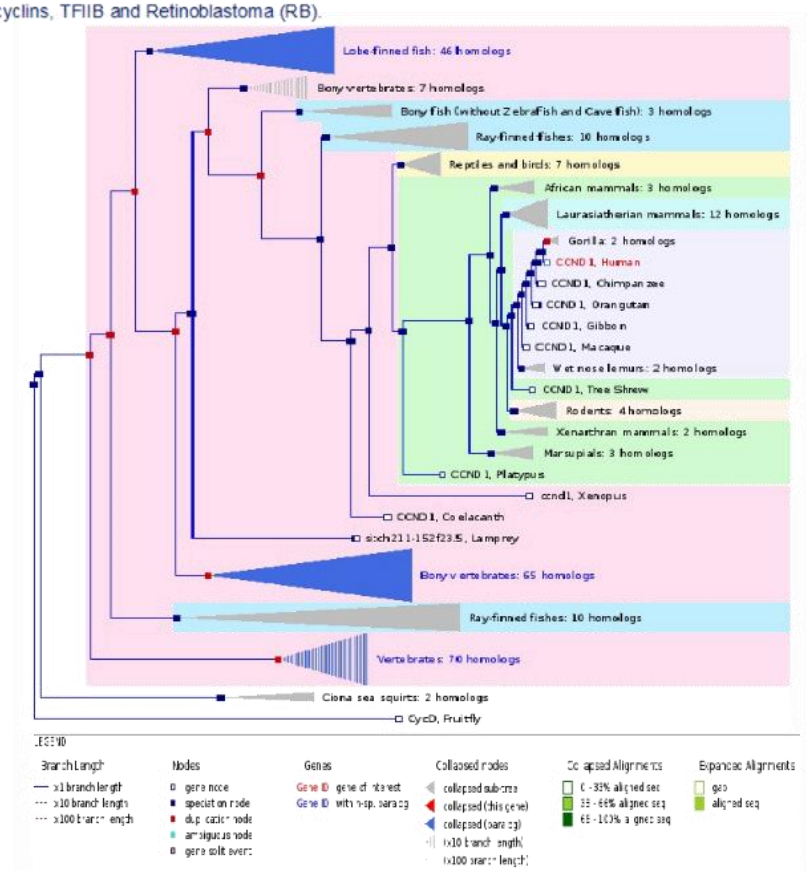


Top: String model depicting probable binding partners on CCND1. Image adapted from string-db.org; Bottom: The figure shows the different proteins with which CCND1 interact and the different functions that result from such interactions. Picture courtesy: Pestell, 2013.

ORGANISM		NCBI REF. NO.	CTDSPL HOMOLOGY DOMAIN
CCND1, *H.sapiens*	Humans	NP_444284.1	295 aa
CCND1, *M.mulatta*	Rhesus monkey	XP_001101029.1	295 aa
CCND1, *C.lupus*	Grey wolf	NP_001005757.1	295 aa
CCND1, *B.taurus*	Cow	NP_001039738.1	295 aa
Ccnd1, *M.musculus*	Mouse	NP_031657.1	295 aa
Ccnd1, *R.norvegicus*	Rat	NP_741989.3	295 aa
CCND1, *G.gallus*	Chicken	NP_990712.1	292 aa
ccnd1, *X.tropicalis*	Frog	NP_001005452.1	291 aa
ccnd1, *D.rerio*	Zebrafish	NP_571100.1	291 aa
CCND1, *P.troglodytes*	Chimpanzee	XP_003952008.1	564 aa

Cyclin, C-terminal domain.

Cyclin box fold. Protein binding domain functions in cell-cycle and transcription control. Present in cyclins, TFIIB and Retinoblastoma (RB).



Gene tree of CCND1 Human has been encircled in red. Adapted from ensembl.org.

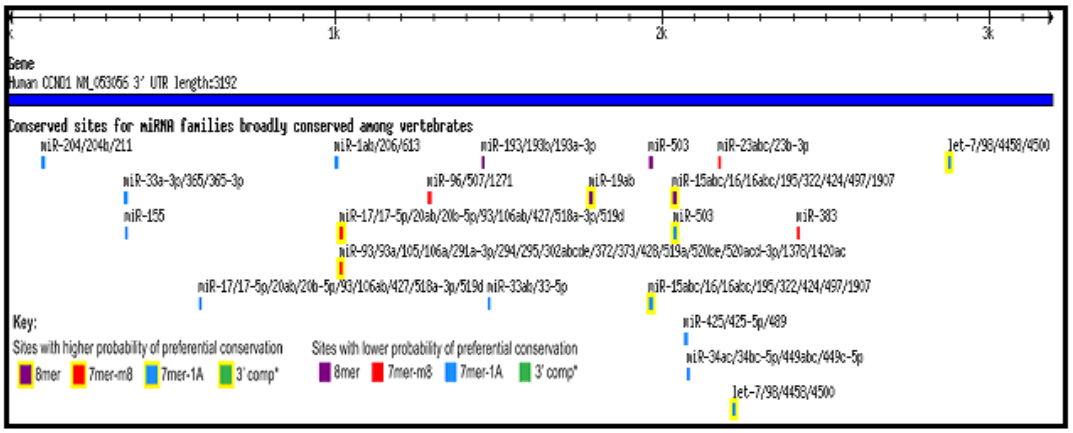


Figure shows the predicted miRNA binding sites in the 3' UTR of CCND1. Image courtesy TargetScan 6.2.

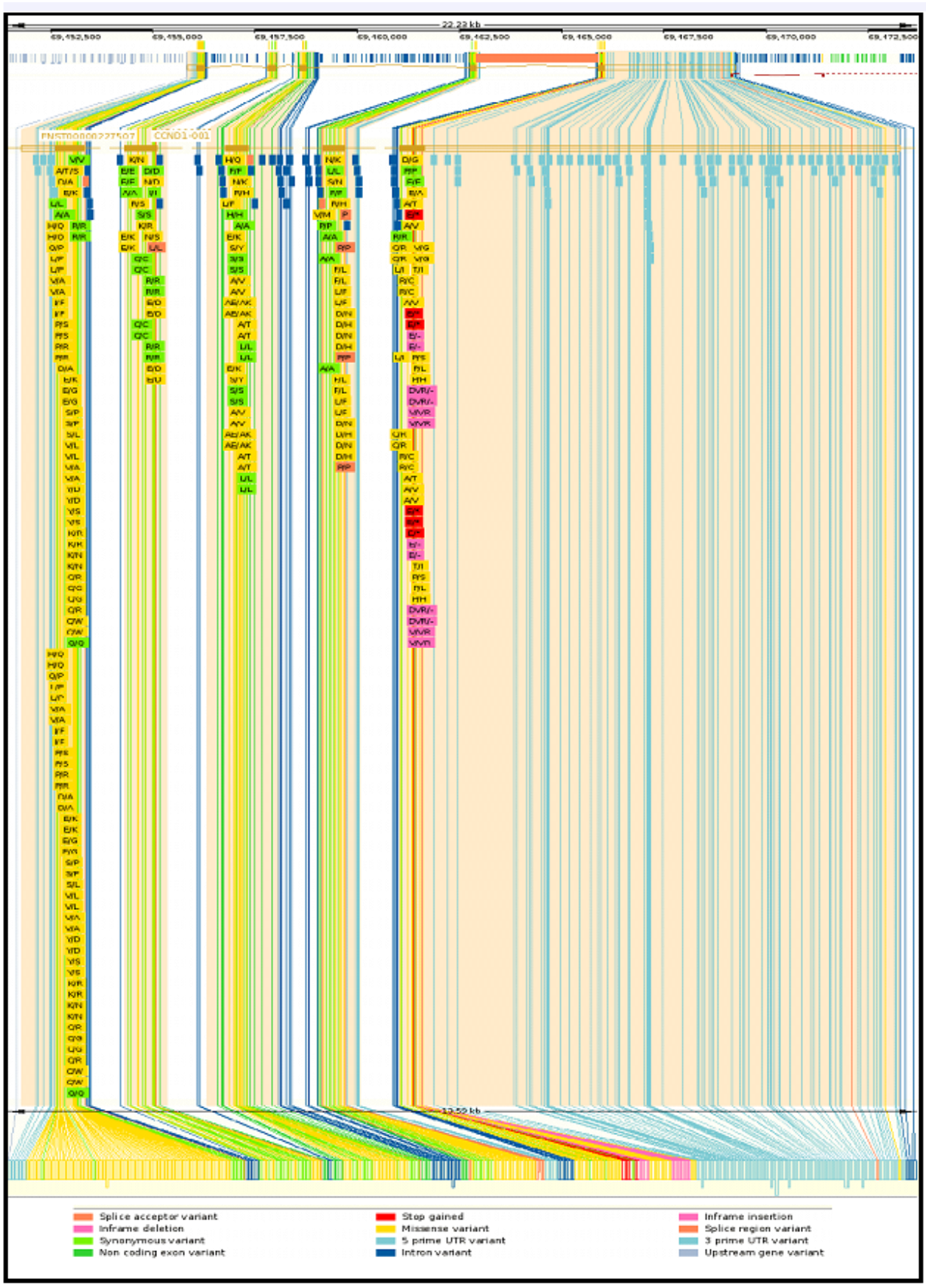


Figure represents the types and percentages of various of mutations observed in CCND1. Image adapted from COSMIC gene analysis.

Color	Mutation Type	Mutant samples	Percentage
	Substitution nonsense	2	3.51
	Substitution missense	43	75.44
	Substitution synonymous	9	15.79
	Insertion inframe	1	1.75
	Insertion frameshift	0	0.00
	Deletion inframe	3	5.26
	Deletion frameshift	0	0.00
	Complex	0	0.00
	Other	0	0.00
	Total	57	100

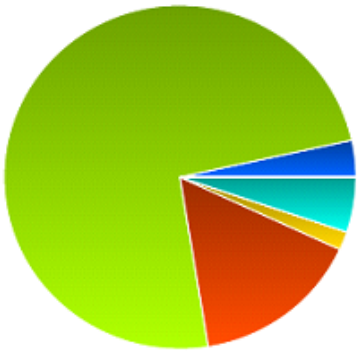


Figure represents the types and percentages of various of mutations observed in CCND1. Image adapted from COSMIC gene analysis.

Tissue	Point Mutations			Copy Number Variation		
	% Mutated	(N)	Tested	Variant %	(N)	Tested
Breast	0.09	(1)	1174	33.9	(289), 8.6 (73)	852
Central nervous system			1159	2.4	(15), 11.6 (74)	638
Large intestine	0.83	(6)	724	5.9	(32), 15.2 (82)	540
Lung	0.67	(8)	1191	27.9	(133), 12.0 (57)	476
Ovary	0.18	(1)	562	31.4	(145), 12.6 (58)	462
Kidney			796	6.0	(26), 3.9 (17)	435
Endometrium	7.01	(19)	271	6.9	(17), 5.3 (13)	246
Pancreas			700	33.9	(80), 9.3 (22)	236
Haematopoietic and lymphoid	1.43	(16)	1117	3.6	(7)	192
Liver	0.12	(1)	827	2.6	(2)	78
NS	0.94	(2)	213	23.3	(7)	30
Urinary tract	1.9	(2)	105			-
Oesophagus	0.58	(1)	173			-
Prostate			482			-
Autonomic ganglia			362			-
Skin			326			-
Upper aerodigestive tract			169			-
Bone			75			-
Meninges			55			-
Salivary gland			49			-
Stomach			47			-
Small intestine			42			-
Eye			34			-
Adrenal gland			23			-
Thyroid			17			-
Parathyroid			16			-
Soft tissue			16			-
Cervix			14			-
Biliary tract			11			-
Pleura			1			-

Mutations and copy number variations of CCND1 in different organs. Red bar: Loss. Grey bar: Gain. Adapted from COSMIC gene analysis.

Mutations

Somatic

CD95 somatic mutations have been reported in several cancers.

Epigenetics

CCND1 and miRNAs:
miR365
in **Gastric cancer cell line BGC-823** Binds to 3' UTR of CCND1 in gastric cancer. miR-365 markedly decreased the expression (mRNA and protein) of CCND1. Conversely, miR365 knockdown repressed cell growth, which can be overcome by CCND1 over-expression. Similar inverse co-relation was obtained between miR-365 and CCND1 expression in patient samples. (Long-Guo et. al., 2013).
in **Vascular smooth muscle cell (VSMC)** miR-365 suppresses CCND1 significantly in mRNA and protein levels in primary rat VSMC. CCND1 is a direct target of miR-365 in vascular smooth muscle cells, as shown by significant inhibition of the luciferase activity of wild type CCND1 3' UTR, but not the mutant cyclinD1 3' UTR with the mutant biding site of miR-365 (Zhang et. al., 2014). CCND1 is a potential target of mir-365 through direct binding. (Kim et. al., 2014).
in **Colon cancer** miRNA directly binds to the 3'UTR of CCND1, proved by luciferase reporter assay. Transfection of miR365 significantly decreased CCND1 expression in HT29 and LoVo cells. Pearson's co-relation between miR-365 levels and CCND1 expression by qRT-PCR and western blot showed that they were inversely correlated (Nie et. al., 2012).
miR-338-3p in Hepatocyte cell line LO2 miR-338-3p binds at two regions in the 3' UTR of CCND1(mainly at the site spanning nucleotides 2397-2403). Overexpression of miR-338-3p downregulates endogenous CyclinD1 protein, while inhibition upregulates CyclinD1 protein, without any change in CCND1 mRNA levels. miR-338-3p post-transcriptionally regulates CCND1 (Fu et. al., 2012).
miR-19a in Human umbilical vein endothelial cells (HUVECs) miR-19a binding site (nucleotides 1,778-1,785 in human CCND1) identified by sequence alignment, which is highly conserved among different species. Binding of miR-19a to 3' UTR of CCND1 verified by luciferase assay. CCND1 protein expression markedly reduced upon over-expression of miR-19a, although no change in RNA expression. miR-19a post-transcriptionally regulates CCND1 expression (Qin et. al., 2010).
miR-490-3p in A549 Lung cancer cell line miR-490-3p binds to 3' UTR of CCND1. Over-expression decreased the expression of CCND1, both at the RNA and protein levels (Gu et. al., 2014).
miR-302 in Endometrial cell line Ishikawa Directly targets CCND1 and significantly inhibited protein expression (Yan et. al., 2014).
miR-449-a in Gastric cancer cell line SGC7901 miR-449a inhibited SGC7901 cells proliferation and enhanced cisplatin chemosensitivity by downregulating expression of CCND1, respectively, via directly targeting the 3'-untranslated regions of CCND1 mRNA (Hu et. al., 2014).
miR-16 in Bladder cancer cell line TCHu-1 Binding of miR16 to 3' UTR of CCND1 and its reduced expression was validated by luciferase assay, while the reverse result was obtained by mutation of the conserved miR-16 binding motif. Overexpression of miR-16 in TCHu-1 cells led to reduced CCND1 protein expression, whereas its inhibition led to an increased expression of CCND1 (Jiang et. al., 2013).
miR-9 in Gastric cancer Databases indicated potential binding site of miR-9 with high complementarity at CCND1 39-UTR (bases 2974-2995), which was validated by luciferase reporter assay. Significant inverse correlation between miR-9 expression and CCND1 transcript levels in gastric cancer tissues and cell lines. Overexpression of miR9 in gastric cancer cell lines SGC-7901 and AGS resulted in reduced RNA and protein expression of CCND1, whereas knockdown of miR-9 produced the opposite result, proving that miR-9 considerably inhibited the expression of CCND1 through post-transcriptional repression. Results validated by in-vitro experiments (Zheng et. al., 2013).
miR-195 in **Glioma** Analysis using publicly available algorithms (TargetScan, Pictar, miRANDA) indicates that CCND1 is a predicted target of miR-195, which was validated by overexpression of miR- 195, which reduced, but inhibition of miR-195 increased, the luciferase activity of CCND1-39UTR in a consistent and dose-dependent manner. Upregulation of miR-195 decreased, but inhibition of miR-195 increased, the expression levels of CCND1 in LN18 and T98G glioma cells. The findings were also validated in a model system in mice (Hui et. al., 2013).
miR-155 in **Human extravillous trophoblast derived HTR-8/SVneo cells** Bioinformatics analysis showed that, at the 3' untranslated region (UTR) of CCND1, six bases are complementary to the seed region of miR-155. Luciferase assays and CCND1 3'UTR transfection assays validated that CCND1 3'UTR was the target of miR-155 in HTR-8/SVneo cells. Overexpression of miR-155 in HTR-8/SVneo cells reduced the level of CCND1 protein (Dai et. al., 2012).
miR-143 in Mesenchymal stem cells from the bone marrow of male Fischer 344 rats Ectopic expression of miR-143 also increased CCND1 in the native MSC as compared with scramble transfected cells .On the contrary, pre-treatment of AAMSC

with miR-143 specific antagomir significantly abolished CCND1 expression (Lai et. al., 2012).

miR-21

in **Mouse liver regeneration** Cyclin D expression and G1 phase transition of hepatocytes after 2/3 PH depend on induced miR-21 expression. Knockdown of miR-21 impaired progression of hepatocytes into S phase of the cell cycle, mainly through a decrease in levels of cyclinD1 protein, but not Ccnd1 mRNA, whereas increased miR-21 expression facilitated CCND1 translation in the early phase of liver regeneration (Ng et. al., 2012).

in **Renal cancer** miR-21 controlled the expression of CCND1 through NFkB-dependent transcription and mediated renal cancer cell proliferation by CCND1 (Bera et. al., 2013).

miR-520-b in **Hepatoma cell lines** miR520-b directly targets the 3 'UTR of CCND1; proved by dual luciferase reporter system. Down-regulation of protein levels of CCND1 occurred on over-expression of miR520-b in HepG2 and H7402 cells, while the over-expression occurred on inhibition in miR520-b. Tumors in mice over-expressing miR520-b also showed lower CCND1 expression (Zhang et. al., 2012).

miR-193b in **Melanoma** TargetScan showed that miR193b binds to the 3'UTR of CCND1, which was proved by luciferase reporter assay. miR-193b over-expression led to nearly 50% reduction in CCND1 mRNA and protein levels in Malme-3M cells than in control (Chen et. al., 2010).

miR-17/20 in **Breast cancer** Levels of the miR-17-5p/miR-20a miRNA cluster were inversely correlated to CCND1 abundance in human breast tumors and cell lines. miR 17/20 negatively regulates the expression of CCND1 by binding to a conserved 3'UTR region (nucleotides 2,109-2,117) of the gene (Yu et. al., 2008).

miR-20 and miR106-a in **Spermatogonial stem cells (SCC)** They promote renewal at the post-transcriptional level via targeting CCND1. Knockdown of CCND1 results in renewal of SCCs (He et. al., 2013).

miR-503 in **Endometrioid endometrial cancer (EEC)** Binds to 5' UTR of CCND1 and its expression is inversely co-related with CCND1 in EEC tissues and cell lines (Xu et. al., 2013).

miR-449b in **SW116 colon cancer stem cell** Transfecting pre-miR-449b and inhibiting miR-449b altered protein expression levels of CCND1 (Fang, 2013).

miR-15a and miR16-1 in **Osteosarcoma** They bind to 3'-UTR of CCND1 and suppress transcription of CCND1 (Cai et. al., 2012).

miR-138 in **Nasopharyngeal carcinoma** CCND1 is a novel direct target of miR138. mRNA levels of CCND1 were inversely correlated with miR-138 expression (Liu et. al., 2012).

miR-34a in **A549 cell line** Ectopic expression of miR-34a reduces both mRNA and protein levels of CCND1 by targeting the 3'-untranslated mRNA region of CCND1 (Sun et. al., 2008).

miR-29a in **Breast cancer cell lines** Over-expression of miR29a down-regulation of CCND1 expression in MDA-MB-453 cells, whereas in MCF-10A cells with Mir-29a knockdown, CCND1 was up-regulated (Wu et. al., 2013).

miR-7 in **Colorectal cancer cell lines** Over-expression of miR-7 significantly decreased CCND1 expression (Xu et. al., 2014).

miR-545 in **Lung cancer** miR-545 caused cell cycle arrest at the G0/G1 phase and induced cell apoptosis in lung cancer cells by targeting CCND1. The effects of CCND1 down-regulated by miR-545 were similar to those caused by siRNAs of CCND1 and over-expression of CCND1 could abolish the miR-545-induced inhibition of cell proliferation (Du et. al., 2014).

miR-125b in **Melanoma** Cells over-expressing miR-125b exhibited reduced expression of CCND1 (Nyholm et. al., 2014).

miR-147 in Colon and lung cancer cells. Transfection of miR147 led to down-regulation of CCND1 (Lee et. al., 2014).

Implicated in

t(11;14)(q13;q32)/B-cell malignancies CCND1/ IgH

Disease

The t(11;14) is mainly found in mantle cell lymphoma; also in: B-prolymphocytic leukaemia, plasma cell leukaemia, splenic lymphoma with villous lymphocytes; rarely in: chronic lymphocytic leukaemia, multiple myeloma

Prognosis

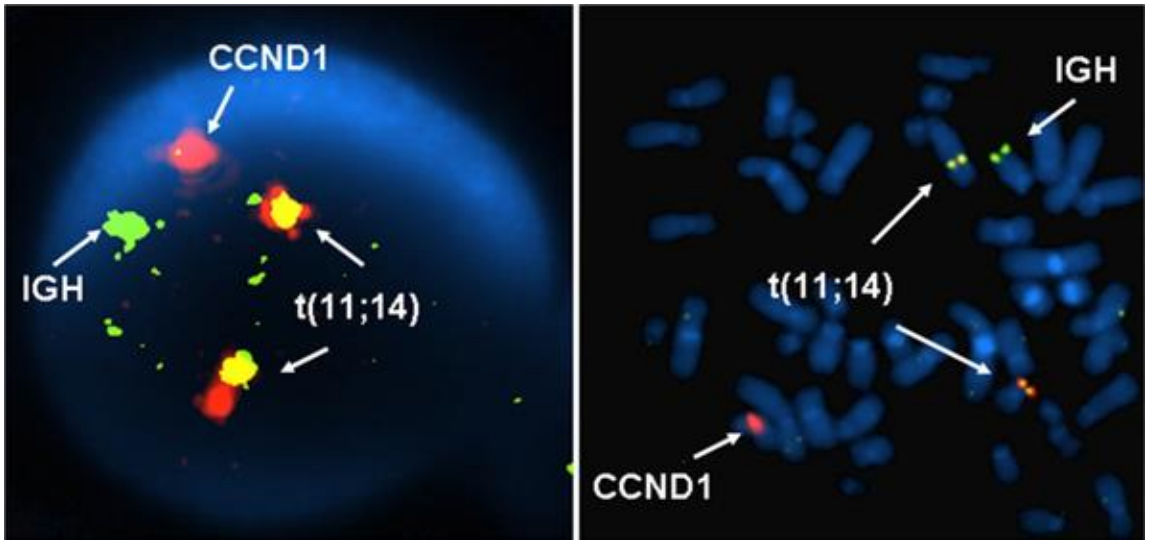
according to the disease.

Cytogenetics

Complex karyotypes.

Hybrid/Mutated gene

5' CCND1 translocated on chromosome 14 near JH (junctions genes of IgH) and C in 3'.



Fluorescence in situ hybridization (FISH) for identification of t(11;14)(q13;q32) chromosomal translocation in metaphase nuclei. Orange probe represents CCND1 (chromosome 11q13), green represents IGH (chromosome 14q32). Fusion signals representing translocations are encircled in white. Image courtesy Ghielmini et. al., 2009.

Abnormal protein

no fusion protein, but promoter exchange; the immunoglobulin gene enhancer stimulates the expression of CCND1.

Oncogenesis

Overexpression of CCND1 accelerates the cell transit through the G1 phase (Williams et. al., 1993, Williams et. al., 1994, Rimokh et. al., 1994, Wlodarska et. al., 1994, de Boer et. al., 1997, Stilgenbauer et. al., 1998, Donnellan et. al., 1998, Li et. al., 1999, Wlodarska et. al., 2004, Sander et. al., 2008).

t(11;19)(q13;p13) CCND1/ FSTL3

Found in a case of chronic lymphocytic leukaemia (Hayette et al., 1998).

Acute Lymphoblastic Leukemia (ALL)

Routinely used ALL drugs: Routinely used drugs failed to bind to CCND1 in in vitro docking studies (Jayaraman et. al., 2014).

Adrenocortical tumors (AC)

CCND1 was over-expression in 31.0% (13/42) in AC tumors compared to 17.5 % (4/23) in normal adrenal samples. Similarly, mRNA of CCND1 was significantly over-expressed in AC compared to normal samples (Mitsui et. al., 2014).

B cell neoplasia

Strong CCND1 mRNA over-expression was detected in mantle cell lymphomas (23 of 23), hairy cell leukemias (5 of 19), and multiple myelomas (7 of 23) with particularly high levels in 2 of the latter cases. Intermediate CCND1 transcripts were detected in multiple myeloma (5 / 23), hairy cell leukemia (7 / 19) Low of no CCND1 was detected in B -cell chronic lymphocytic leukemias (10 / 10), follicular lymphomas (9 / 9), mucosa associated lymphoid tissue lymphomas (5 / 5) and reactive lymphoid tissues (Specht et. al., 2002).

Biliary Intraepithelial Neoplasia (BilIN) / Pancreatic Intraepithelial Neoplasia (PanIN)

Immunohistochemical expression of CCND1 was absent or focal in nonneoplastic epithelium of the bile ducts and the pancreatic ducts, and were occasionally observed in BilIN-1 and PanIN-1 and more frequently in BilIN-2/3 and PanIN-2/3. No significant difference was obtained between expression of BilIN and PanIN in semi-quantitative analysis (Sato et. al., 2014).

Bladder cancer tumors

Increased CCND1 levels were not correlated with OS with a pooled HR estimate, but were significantly correlated with progression-free survival (Ren et. al., 2014). Over-expression of Pin X1 in T24 cells leads to greater than 2 fold increase in mRNA expression of CCND1 than in control cell, with similar results obtained by Western blotting. A significant correlation between the immune-histochemical expression of PinX1 and CCND1 was also observed in the UCB tissues (Liu et. al., 2013). Ursane triterpenoid isopropyl 3-hydroxyurs-12-en-28-oat (UA17) (Natural compound): Protein level of CCND1 was down-regulated in a dose-dependent manner when treated with UA17or Cisplatin in NTUB1 cells. Enhanced decrease of level of CCND1 when treated with a combination of Cisplatin + UA17 (Lin et. al., 2014) Metformin: Treatment with metformin leads to reduction in expression of CCND1 in a dose-dependent manner. Metformin treatment also markedly reduced the expression of CCND1 in Human Bladder Tumor Xenografts in

Nude Mice compared to control (Zhang et. al., 2013).

Breast cancer

CCND1 induction of Dicer coordinates microRNA biogenesis by its transcriptional targeting (Yu et. al., 2013). CCND1 overexpression is associated with longer DSS, but not recurrence-free survival, in patients with breast cancer (Chung et. al., 2014). There was a statistically significant reverse relationship of CCND1 with tumor grade and both ER and PR hormone receptors (Mohammadizadeh et. al., 2013). CCND1 was one of the most frequently altered genes in breast cancer (Wheler et. al., 2014). Activation of Notch-1 signaling up-regulated expression of CCND1 through NF-kB (Li et. al., 2014). Acylglycerol kinase (AGK) over-expression led to concurrent increase in levels of CCND1 (Wang et. al., 2014). Over-expression led to blockade of CCND1 expression via BCAS2 and beta-catenin (Sengupta et. al., 2014). Enhanced expression of Vav1 led to the elevation of CCND1 and the progression of cell cycle (Du et. al., 2014). CCND1 was frequently more positive in ER alpha positive and Bmi1 positive breast tumors than ER alpha negative and Bmi1 negative groups (Wang et. al., 2014). Progesterone induced the assembly of a transcriptional complex among AP-1, Stat3, PR, and ErbB-2 at the CCND1 promoter, which functions as an enhanceosome to drive breast cancer growth (Flaqué et. al., 2013). Prolactin-induced protein (PIP) silenced cells showed marked decrease in CCND1 expression (Naderi et. al., 2014). Calcitriol (Natural compound): In calcitriol-treated cells, the presence of antiestrogen ICI-182 down-regulated CCND1 gene expression (Martinez et. al., 2014). Euginol (Natural compound): Treatment of euginol decreased CCND1 level 3 fold in MDA-MB-231 cells and 20 fold in MCF7 cells compared to control (Sharif et. al., 2013). Fangchinoline (Fan) (Natural compound): Fan decreased the expression of CCND1 both in the RNA and protein level (Wang et. al., 2014). Gallotannin (Natural compound): Nanostring and qPCR data showed that CCND1 was exclusively downregulated on treatment with gallotannin in triple negative breast cancer (Zhao et. al., 2014). 8-bromo-7-methoxychrysin (BrMC) (Natural compound): BrMC caused a dose-dependent reduction of CCND1 in HER2/neu over-expressing breast cancer cells (Cao et. al., 2014). Panepoxydone (Natural compound): CCND1 was down-regulated by dose-dependent treatment of Panepoxydone (Arora et. al., 2014). Thymus caramanicus extract (TCE) (Natural compound): TCE led to reduction in expression of CCND1, either alone or in combination with Vincristine (Mahani et. al., 2014). Tea polyphenols (Natural compound): Tea polyphenols did not significantly alter the expression of CCND1 in breast cancer cell lines (Chen et. al., 2014). Fenofibrate: Fenofibrate decreased the expression of CCND1 in a time and dose dependent manner in Triple negative breast cancer cells (Li et. al., 2014). Obatoclax analog SC-2001: SC-2001 down-regulated CCND1 in TNBC cell lines in a dose- dependent manner (Liu et. al., 2014).

Chronic Myeloid Leukemia tumors (CML)

Resveratrol (Res) (Natural compound): Res reduced expression of CCND1 in K562 cells (Siu et. al., 2014). Quercetin (Natural compound): CML KBM7 Cells demonstrated reduction in expression on CCND1 on treatment with quercetin ((Li et. al., 2014).

Colorectal cancer

There was significant association between post-menopausal hormone therapy (HRT) and CCND1 negative-tumors, as well as significantly increased risk in CCND1 positive tumours (Brändstedt et. al., 2014). High height and weight was associated with risk of CCND1 positive CRC in women. Increased hip circumference, high BMI, high WHR and high waist circumference was associated with CCND1 positive tumours in men (Brändstedt et. al., 2013). CCND1 over-expression was significantly associated with both poor OS, DFS, relatively older patients (over 60 years), T3,4 tumor invasion, N positive and distant metastasis (Li et. al., 2104). Galectin-3 knockdown decreased the mRNA expression level of CCND1, whereas epirubicin significantly up-regulated their expression. Combined treatment effectively reduced the mRNA expression of CCND1 (Lee et. al., 2013). HMGCR expression was significantly associated with expression of CCND1 (Bengtsson et. al., 2014). CoCl2: Treatment of COCl2 leads to dose-dependent decrease in expression of CCND1 and cell cycle arrest (Lopez-Sanchez et. al., 2014). SW620-S and TGF-b1: Fibroblasts induced by Colorectal cancer cells, treated with SW620-S and TGF-b-1 separately showed high expression of CCND1 (Rao et. al., 2014).

Diffuse large B-cell lymphoma

A case of diffuse large B-cell lymphoma was described, which developed within a rectal tubular adenoma with low-graded dysplasia. The mass showed positive staining of CCND1 (Genovese et. al., 2014).

Esophageal cancer

CCND1 G870A polymorphism had no significant association with esophageal squamous cell carcinoma (ESCC) or esophageal adenocarcinoma (EADC) in Caucasian or the Asian populations. However, the comparison of A vs. G in CCND1 G870A showed significant differential susceptibility

to esophageal cancer, suggesting that the CCND1 G870A polymorphism has no association with esophageal cancer risk in ethnicity and histology, respectively (He et. al., 2013). No significantly statistical differences between the two groups were observed in distribution of genotypes or alleles at CCND1 807 (Jang et. al., 2013).

Fibrosarcoma

KIOM-C (Natural compound): Treatment of HT1080 human fibrosarcoma cells led to down-regulated expression of CCND1 compared to control (Kim et. al., 2014).

Gastric cancer

Down regulation of CCND1 by ShCCND1 in NCI-N87 cells showed significant inhibition of cell proliferation, cell motility, clonogenicity, G1 arrest and apoptosis. Results were validated by in vivo studies in mice, suggesting the possibility of developing new gastric cancer therapies using lentivirus-mediated shRNA (Seo et. al., 2014). Odd-skipped related 1 (OSR1) suppressed the expression of CCND1 (Otani et. al., 2014). Knockdown of P115 led to reduction in expression of CCND1, whereas its over-expression led to up-regulation of CCND1 (Li et. al., 2013). Caudatin 3-O-D-cymaropyranosyl-(1 4)--D-oleandropyranosyl-(1 4)--D-cymaropyranosyl-(1 4) -D-cymaropyranoside (CGII) (Drug): CGII induced down-regulation of expression of CCND1 in a dose-dependent manner in Gastric Cancer SGC-7901 Cells (Wang et. al., 2013) Tetramethypyrazine (TMP) (Natural compound): Expression of CCND1 gradually decreased with increasing concentrations of TMP in Gastric cancer 7901 cells (Ji et. al., 2014). Resveratrol (Res) (Natural compound): Res reduced expression of CCND1 (Yang et. al., 2013) .

Glioma

Expression of Alpha enolase (ENO-1) inhibited the expression of CCND1 (Song et. al., 2014).

Hairy Cell Leukemia

CCND1 displayed nuclear staining at variable intensities but with high specificity and accuracy in HCL biopsies, thus representing it as a valuable tool in the differential diagnosis of HCL and its mimics (Toth-Liptak et. al., 2014).

Head and neck squamous cell carcinoma tumors

Amplification, over-expression and translocation of CCND1 has been reported (Akervall et. al., 1997, Akervall et. al., 2002, Utikal et. al., 2005, Sabbir et. al., 2006). However, expression of CCND1 did not change in post-therapy tumors compared to pre-therapy (Sarkar et. al., 2014).

Hepatocellular Carcinoma

CCND1 Ectopic expression of miR-184 led to down-regulation of the SOX7 protein, resulting in up-regulation of CCND1, cell proliferation and tumorigenesis (Wu et. al., 2014). SOX7-overexpression inhibited cell growth by down-regulating CCND1, which could be over-ridden by ectopic expression of CCND1 and induction of SOX7. Over-expression of SOX7 suppressed tumor formation with down-regulation of CCND1 in vivo (Wang et. al., 2014). Knockdown of TRIM24 led to decreased CCND1 expression (Liu et. al., 2014). KIF14 knockdown suppresses tumor cell growth through decrease in levels of cyclins including CCND1 (Xu et. al., 2014). Knockdown of expression of SHC SH2-domain binding protein 1 (SHCBP1) led to reduction in expression of CCND1 (Tao et. al., 2013). 7. 3, 3'-Di-O-methyl ellagic acid-4'-O-d-xylopyranoside (JNE2). JNE2 induced down-regulation of expression of CCND1 in HepG2 cells (Zhang et. al., 2014). Silybin (SIL) (Natural compound): Treatment of HepG2 cells with SIL led to down-regulation of expression of CCND1 (Zhang et. al., 2013). SL1122-37: SL1122-37 induced down-regulation of expression of CCND1 in PLC/ PRF/5 HCC cells (Qin et. al., 2013). IBN-65 (1-benzyl-2-phenyl-3-(4-isopropyl)-benzyl-imidazolium chloride) : IBN-65 decreased levels of CCND1 in Huh7 cells in Mouse model of HCC (Gopalan et. al., 2014). Sorafenib and YC-1 : Treatment with the sorafenib and YC-1 combination led to a significant reduction in CCND1 (Kong et. al., 2014).

Hepatoma

Over-expression of HA-FHIT inhibited the expression of CCND1 in the cells. In HepG2 cells which were transfected with a full-length CCND1 promoter-luciferase reporter, cotransfection with increasing quantities of FHIT plasmid DNA caused a concentration-dependent inhibition of the transcriptional activity of the CCND1 promoter (Ge et. al., 2014).

Lung Cancer

PAX6 down-regulation led to reduction in protein levels of CCND1 (Zhao et. al., 2014). Over-expression of Ubiquitin- conjugating enzyme E2C (UBE2C) increased expression of CCND1 in L-78 and SC-1680 cells, as well as in tumor transplants in nude mice (Tang et. al., 2014). Met- F-AEA in combination with URB597 induced down-regulation of CCND1 and subsequent G0/ G1 cell cycle arrest (Ravi et. al., 2014). Up-regulation of decorin led to significant decrease in expression of CCND1 (Liang et. al., 2013). The expression of CCND1 was significantly decreased upon knockdown of Claudin-2 in lung adenocarcinoma (Ikari et. al., 2014). Knockdown of JAM-A decreased protein levels of CCND1 (Zhang et. al., 2013). Tea polyphenols

(Natural compound): Epigallocatechin gallate, epicatechin gallate and theaflavin reduced the expression of CCND1 in benzo(a)pyrene-induced lung carcinogenesis in mice (Manna et al., 2009). Polydatin: PD suppressed expression of CCND1 in A549 and NCI-H1975 lung cancer cell lines (Zhang et. al., 2014).

Mantle Cell Lymphoma

Decrease in expression of CCND1 by RNSi induced partial inhibition and reduced expression of AKT and/or S6, which may in turn lead to decrease in NOXA mRNA levels (Dengler et. al., 2014). 85% were weakly positive and 15%, moderately positive with labelled streptavidin biotin, whereas 75% were weakly positive and 25% moderately positive for CCND1 with EnVision. All 20 mantle cell lymphoma cases were strongly CCN D1 positive with catalyzed signal amplification. No evidence of CCND1 immunostaining was obtained in any of the small lymphocytic lymphoma and follicular centre cell lymphoma instances with any of the three methods used (Barranco et. al., 2003). CCND1 showed exclusive nuclear staining and directly compared with the expression observed by immunoblot analysis with the same antibody, as well as with mRNA expression and with the occurrence of genomic rearrangements within the B CL-1 locus. 12/13 MCL showed over-expression by immunohistochemistry or immunoblot, with similar results for additional 13 MCLs, indicating its importance for routine diagnostic purposes (Boer et. al., 2014). CCND1 mRNA could be detected in 23 of 24 mantle-cell lymphomas by reverse transcription polymerase chain reaction (RT-PCR) whereas only 9 of 24 demonstrated a t(11;14) by PCR (Aguilera et. al., 1998). In 16 of 21 cases of MCL with overt disease, the ratio of CCND1 mRNA to 2-microglobulin mRNA was increased, but all 21 cases showed increased ratios of CCND1 mRNA to CD19 mRNA (Howe et. al., 2004)

Melanoma

Piperine (Natural compound): Piperine induced reduction in expression of CCND1 in a dose-dependent manner in SK MEL 28 and B16 F0 melanoma cells (Fofaria et. al., 2014).

Multiple myeloma

CCND1 expression was observed in 57% cases. CCND1 positive group had significantly lower hemoglobin level than CCND1 negative group, though both groups showed no statistical significance in regard to age, gender, Durie and Salmon stage, lytic bone lesions, light chain phenotype, creatinine, calcium, lactate dehydrogenase, leukocyte and platelet count and bone marrow histology (Padhi et. al., 2013).

Nasopharyngeal cancer

No significant association was found between CCND1 G870A polymorphism and nasopharyngeal carcinoma risk in total population meta-analysis. In the subgroup meta-analysis by ethnicity, a negative association was shown in Caucasian subgroup, and no significant association in any genetic models among Asians was observed (Li et. al., 2013). Indole-3-carbinol (I3C) : I3C induced G1 arrest by decreasing CCND1 expression (Chen et. al., 2013).

Neuroblastoma

CCND1 showed strong nuclear reactivity in a case study on Primary localized congenital sacrococcygeal neuroblastomas (SCNs) (Khandeparkar et. al., 2013). Over-expression of n-myc downstream regulated gene 2 (NDRG2) induced down-regulation of expression of CCND1 (Zhang et. al., 2014). A negative co-relation existed between WWOX and CCND1 expression (Nowakowska et. al., 2014)

Odontogenic tumors

Using immune-labelling of CCND1, no statistical difference was observed between primary and recurrent KOT (keratocystic odontogenic tumors), sporadic and NBCCS-KOT (nevoid basal cell carcinoma syndrome), and unicystic and solid AB (ameloblastomas) (Gurgel et. al., 2014).

Oral cancer

Expression of CCND1 in group 3 (leukoplakias exhibiting dysplasias) was significantly higher than in group 1 (normal buccal mucosa without any habits) and 2 (clinically normal mucosa from tobacco habits), expression in group 2 was significantly higher than in group and were statistically significant.

CCND1 was mostly expressed in the lower third of epithelium. Highest expression was obtained in mild dysplasias, with expression consistently correlating with basilar hyperplasia among atypical morphological features (Ramakrishna et. al., 2013). Clinico-pathological correlation showed that CCND1 over-expression was related to increase in tumor size, tumor differentiation and higher clinical stages and lymph node metastasis and adversely affected overall survival (Zhao et. al., 2014). HPV-negative patients, heavy alcohol consumption was significantly associated with somatic copy-number alterations (SCNAs) in CCND1 (Urashima et. al., 2013).

The proportions of positive staining in well, moderately and poorly differentiated laryngeal SCC were 50, 66.7, 100%, respectively, for CCND1, and were statistically significant, with the expression being positively correlated with Ang-2 expression. Tumor grading and CCND1 were independent factors affecting laryngeal SCC patient survival by

the Cox regression model of risk factors proportion analysis, which may possess clinical significance in evaluating the prognosis and guiding the clinical treatment of SCC (Liu et. al., 2013). Knockdown of Nemo-like kinases (NLK) led to significant reduction in the levels of CCND1 (Dong et. al., 2013). 2,4-bis (p-hydroxyphenyl)-2-butenal : HPB 242 significantly decreased CCND1 expression in HN22 and HSC4 Oral squamous cell carcinoma cell lines (Chae et. al., 2014).

Osteosarcoma

Selective inhibition of Ether à go-go 1 (Eag1) led to significant decrease in expression of CCND1 (Wu et. al. 2014).

Ovarian serous carcinoma

Compared with NOT (Normal Ovarian Tissue), CCND1 expression in the OSA (ovarian serous cystadenomas) and OSC (Ovarian serous carcinoma) groups was significantly elevated. Expression of CCND1 was positively associated with lymphatic metastasis and the expression gradually increased in the NOT, OSA, OS-BT and OSC groups and was associated with tumor metastasis (Song et. al., 2014).

Pancreatic cancer

Silencing of Frizzled (Fz)2 by siRNA or shRNA induced significant reduction of expression on CCND1 (Tomizawa et. al., 2014). Down-regulation of miR-196a led to decrease in expression of CCND1 via Nuclear Factor Kappa-B-Inhibitor Alpha (Huang et. al., 2014). Diallyl trisulfide (DATS) (Natural compound): DATS reduced levels of CCND1 and DATS-induced apoptosis was correlated with down-regulation of CCND1 protein levels in Capan-2 cells (Ma et. al., 2014).
alpha-Mangostin (Natural compound): alpha--Mangostin led to decrease in expression of CCND1 (Xu et. al., 2014). Pristimerin (PM) : PM treatment produced decreased expression of CCND1 in MiaPaCa-2 and Panc-1 cells (Deeb et. al., 2014).

Plasmacytoma

A solitary plasmacytoma following complete remission from an intravascular large B-cell lymphoma, stained strongly for CCND1 while the initial tumor was negative for CCND1, proving different clonal origins of the tumors (Lee et. al., 2014).

Prostate cancer

CNCD1 staining was positive (expression in .5% of tumor cells) in 64 cases (75.4%) and negative (expression in 5% of tumor cells) in 21 cases (including 15 cases with no immunostaining) with normal prostate tissues being negative for CCND1. Patients with high grade Gleason score and perineural invasion showed significant association with CCND1 expression, but not with PSA levels or other parameters. Thus, high CCND1 expression could be a potential marker for tumor aggressiveness (Pereira et. al., 2014). Univariate analyses showed that lymph node positivity, surgical margin positivity, non-localized tumor, age at prostatectomy and CCND1 in malignant epithelium were significantly associated with time to BF (Biochemical failure) (Rizzardi et. al., 2014). Reevesioside A (Natural compound): Reevesioside A inhibited expression of CCND1 in Hormone-Refractory Prostate Cancers (Leu et. al., 2014). Scoparone (Natural compound): Scoparone suppressed the transcription of STAT3 target CCND1 in DU-145 cells (Kim et. al., 2013). Triptolide (Natural compound): Triptolide induced significant decrease of expression of CCND1 through EZH2 (Tamgue et. al., 2014). Pifithrin (PFT) : Combination therapy with suboptimal doses of PFT-m and HT decreased expression of CCND1 (Sekihara et. al., 2013).

Renal cancer

Microvessicles: CCND1 protein expression in tumor tissues was markedly up-regulated by MVs released from human Wharton's jelly mesenchymal stem cells (hWJ-MSCs) (Du et. al., 2014).

Sarcoma

Tea polyphenol epigallocatechin gallate (EGCG) did not alter expression of CCND1 in Sarcoma180 cells in vivo (Manna et. al., 2006)

T-cell Acute Lymphoblastic Leukemia

Resveratrol (Res) (Natural compound): Expression of CCND1 was attenuated in Res treated T-ALL CEM-C1-15 cells (Ge et. al., 2013).

Uterine cervical cancer

Bcl-1/Cyclin D1 alterations are associated with the development of uterine cervical carcinoma (Singh et. al., 2005).

Various cancers

Ursolic acid (UA) (Natural compound): UA in combination with other drugs led to down-regulation of expression of CCND1 (Doudican et. al., 2014). Salinomycin-: Salinomycin induced lowering of expression of CCND1 in Breast and prostate cancer cells (Lu et. al., 2014).

Non-cancerous tissues

Primary human cardiomyocytes: Thrombin time-dependently up-regulated CCND1 expression, with a significant response within 36-48 h (Chien et. al., 2014).
Human diploid fibroblast (HDFs): CCND1 gene was significantly up-regulated in irradiated (1 Gy) HDFs as compared to untreated control, while bothHDFs treated with Gelam honey and irradiated HDFs pre-treated with Gelam honey showed down-

regulation of cyclin D1 gene as compared to irradiated HDFs. HDFs treated with Gelam honey during radiation and post-irradiation however showed significant up-regulation of cyclin D1 gene as compared to untreated control (Ahmed et. al., 2014).

Human coronary artery smooth muscle cells (HCASMCs): FABP4 induced increase in expression of the downstream genes CCND1 (Girona et. al., 2013).

Vascular smooth muscle cells: STS (sodium tanshinone IIA silate) decreased the expression of cell cycle-associated protein, CCND1 (Wu et. al., 2014). PDGF-induced CCND1 mRNA and protein expression was inhibited by TGFb. PDGF-induced CCND1 expression requiring KLF5 was inhibited by TGFb via a Smad dependent mechanism, leading to G1 cell cycle arrest of VSMs (Garrido et. al., 2013).

Neuroectodermal stem cells: PGE2 (Prostaglandin E2) treatment significantly up-regulated CCND1 (Wong et. al., 2014).

Neurons: DYRK1A (dual specificity tyrosine-phosphorylation-regulated kinase 1A) reduced cellular CCND1 levels by phosphorylation on Thr286, which is known to induce proteasomal degradation (Soppa et. al., 2014).

Renal intestinal fibroblasts: Exposure of NRK-49F to resulted in reduced expression proliferation markers CCND1 in a dose and time dependent manner (Ponnusamy et. al., 2014).

Idiopathic pulmonary fibrosis (IPF): Cell cycle regulatory protein CCND1 was significantly enhanced in AEC (alveolar epithelial cell) within the remodelled fibrotic areas of IPF lungs but expression was negligible in myofibroblasts (Akram et. al., 2014).

Human Rheumatoid Arthritis Synovial Cells: The protein and mRNA levels of CCND1 decreased gradually with the increasing of thapsigargin concentration and treatment times (Wang et. al., 2014).

Other mammals

Mouse:

Adult mice cardiomyocytes: Silencing the CCND1 expression is necessary for the maintenance of the cell cycle exit, and suggests a mechanism that involves inhibition of M-phase entry (Tane et. al., 2004).

Mouse hair follicle (HF): Real-time PCR analysis revealed an inverse relationship between CCND1 expression pattern and that of Sfrp2 throughout the HF cycle (Kim et. al., 2014).

Mouse mammary gland: CCND1 was more frequently up-regulated in mammary tumors from transgenic mice (expressing myristoylated-Akt1 (myr-Akt1) under the control of the MMTV-LTR promoter) compared to tumors from wild-type mice. Increased expression of CCND1 was incompletely dependent on Akt1 expression. Low expression of CCND1 and increased expression of Twist and Slug was observed in mammary tumors that had metastasized to secondary sites (Wu et. al., 2014).

Atherosclerosis in mice: The expression levels of CCND1 in smooth muscle cells were restrained by CD59 and C-PC (C-phycocyanin) (Li et. al., 2013).

Mouse pancreatic cancer: Embelin-treated mice showed significant inhibition in tumor growth, which was associated with reduced expression of CCND1 (Huang et. al., 2014).

Human umbilical cord mesenchymal stem cells (hUCMSCs) in nude mice: In mice treated with hUCMSCs-LV-IL-21, Expression of cyclin-D1 was simultaneously low compared to control group, hUCMSCs group and hUCMSCs-LV-Vec group (Zhang et. al., 2014).

Partial hepatectomy: Following seventy percent partial hepatectomy (PH) in wild type (WT) mice IL-6 serum levels increased, resulting in increased CCND1 (Tachibana et. al., 2014).

Cow:

Dairy Cow Mammary Epithelial Cells: Treatment with leucine induced LeuRS, increasing CCND1 mRNA and protein expression (Wang et. al., 2014).

Rat:

Rat epithelial cells: CCND1 accumulation due to differential effects of of PKC was likely contribute to the opposing tumor suppressive and tumor promoting activities in the intestinal epithelium (Pyfz et. al., 2014).

Hyperbilirubinemic Gunn Rat: Decreased expression in CCND1 in the cerebellum of the hyperbilirubinemic Gunn rats led to significant increased cell cycle arrest in the late G0/G1 phase (Robert et. al., 2013).

Neonatal hypoxia-ischemia in rat: IGF-1R activation together with EGFR co-signaling decreased the percentage of cells in G1 and enhanced cell progression into S and G2 by increases in expression of CCND1 (Alagappan et. al., 2014).

Rat balloon injury model: CCND1 mRNA was significantly decreased by sodium ferulate in cells under serum stimulation (Zhang et. al., 2014).

Rat liver fibrosis: Sophocarpine inhibited the proliferation of HSCs by a decrease in the expression of CCND1 (Qian et. al., 2014).

Rat Airway Smooth Muscle Cells: Nicotine significantly increased expression of CCND1 (He et. al., 2014).

Chicken:

Chicken fetal myoblasts (CFMs): Increased CCND1 expression during acceleration of cell cycle at G1/ S phase in CMF was due to CARP (cardiac ankyrin repeat protein) over-expression (Ma. et. al., 2014).

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