Contributor(s)

Written 05-2008 Laura Lentini, Aldo Di Leonardo

Department of Cellular and Developmental Biology A, Monroy, University of Palermo, viale delle Scienze, Palermo, Italy

AURKA (aurora kinase A)

Identity

Other names: Aurora A
ARK1
AURA
Aurora2
BTAK
MGC34538
STK15
STK6
STK7

Hugo: AURKA

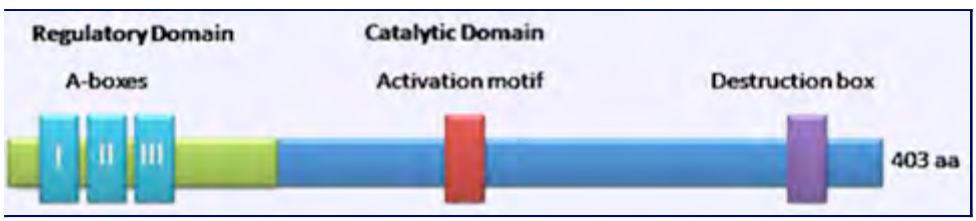
Location: 20q13.31

DNA/RNA

Description: The gene encompasses 22,8 kb of DNA; 9 Exons.

Transcription: 2253 bp mRNA.

Protein



Description: 403 amino acids, 46kDa protein. At the amino terminal domain,

	three putative conserved Aurora boxes (A-boxI, A-boxII and A-boxIII) can be identified. The functional significance of these boxes are not yet clearly known and there is suggestive evidence in the literature that these may be involved in subcellular localization or substrate recognition for the proteins. One of the serine residues in the A-boxII of Aurora-A kinase has recently been shown to be involved in the degradation of the protein. One activation motif and a destruction box in C-terminal. AURKA is regulated by phosphorylation in a cell cycle dependent manner. This phosphorylation occurs on a conserved residue, threonine 288, within the activation loop of the catalytic domain of the kinase and results in a significant increase in the enzymatic activity. AURKA protein is able to physically associate with multiple important cellular proteins such as p53, BRCA1, and TACC1. The interactions of AURKA with those critical molecules have been shown to disrupt/alter their physiological functions and may play roles in tumorigenesis.
Expression	Widely expressed, AURKA mRNA and protein expression levels are low during G1 and S phase and peak during the G2/M phase of the cell cycle. Kinase activity of the protein is also cell cycle regulated and the highest activity coincides with the most elevated expression level of the protein during mitosis.
Localisation	AURKA localizes next to the centrosome late in the G1 phase and early in the S phase. As the cell cycle progresses, concentration of AURKA increases and the kinase associates with the mitotic poles and the adjacent spindle microtubules. AURKA remains associated with the spindles through telophase.
Function	Serine/threonine kinase identified as key regulator of the mitotic cell division process. Known to be involved in the regulation of centrosome function, bipolar spindle assembly and chromosome segregation processes. AURKA is critical for proper formation of mitotic spindle. It is required for the recruitment of several different proteins important to the spindle formation. Among these target proteins are TACC, a microtubule-associated protein that stabilizes centrosomal microtubules and Kinesin 5, a motor protein involved in the formation of the bipolar mitotic spindle. Two upstream regulators of AURKA, Ajuba and targeting protein for Xklp2 (TPX2) are known. TPX2 is a MT-binding protein involved in spindle pole formation and is the best characterized RanGTP-dependent spindle activator. TPX2 is required for

AURKA binding to spindle MTs and its binding to AURKA holds the latter in an active conformation. Ajuba and AURKA interact in mitotic cells and become phosphorylated. In vitro analyses revealed that Ajuba induces the autophosphorylation and consequent activation of AURKA. Depletion of Ajuba prevented activation of AURKA at centrosomes in late G2 phase and inhibited mitotic entry.
AURKA induces p53 degradation by phosphorylation of Ser-315. It was demonstrated that DNA binding and transactivation activity of p53 was abrogated by AURKA. AURKA phosphorylates p53 at Ser-215 in vitro and in vivo. The inhibition of p53 DNA binding and transactivation activity by AURKA depends on phosphorylation of Ser-215 but not Ser-315. Further, AURKA phosphorylation of Ser-215 of p53 is associated with AURKA -regulated cell cycle progression, cell survival, and transformation. The p53 is a physiological substrate of AURKA and that AURKA exerts its function through phosphorylation of Ser-215 of p53.

Homology — Aurora related kinases, AURKA and AURKB are present in Drosophila,C.elegans and X. laevis. Among mammals, three members of the family, AURKA, -B and -C have so far been identified. The three members of the mammalian kinases of varying peptide lengths share similar catalytic domains located in the carboxyl terminus but their amino terminal extensions are of variable length and display little or no similarity. By examination of the AURKA cDNA sequence the threonine at residue 288 in the catalytic domain was found to be highly conserved in all Aurora family members as well as in various other serine/threonine kinases.

Mutations

Germinal — No germline mutations have been reported.

Somatic — Recent studies have demonstrated the overexpression and amplification of AURKA in many malignant human cancers and cell lines including breast, ovarian, colon, prostate, and neuroblastoma cancer cell lines. AURKA overexpression induces supernumerary centrosomes aneuploidy and cells transformation. It has been also reported that ectopic overexpression of AURKA in NIH3T3 and immortalized Rat1 induces cells transformation that generates tumor when implanted in nude mice. Elevated AURKA expression amplification

overrides the checkpoint mechanism that monitors mitotic spindle assembly, inducing resistance to the chemotherapeutic agent paclitaxel. Cells overexpressing Aurora-A inappropriately enter anaphase despite defective spindle formation, and the persistence of MAD2 at the kinetochores, marking continued activation of the spindle assembly checkpoint. These findings suggest that enhanced AURKA expression causes resistence to apoptosis induced by mitotic inhibitors in human cancer cells.

Implicated in

Entity Breast cancer

Disease Breast cancer is the most common cause of cancer in women and the second most common cause of cancer death in women. Some of the patients are hereditary, with a large proportion characterized by mutation in BRCA1 and/or BRCA2 genes.

Oncogenesis The protein level of AURKA is increased at the G2 to M phase transition in normal cells, where AURKA is specifically localised at centrosomes and mitotic spindles. By contrast, analysis of tumour cells of the breast for AURKA expression patterns shows that overexpression of AURKA was present in 94% of the cases, regardless of their cell-cycle phases, and is diffusely detected in the cytoplasm. Amplification of AURKA has been detected at higher frequency in tumors from BRCA1 and BRCA2 mutation carriers than in sporadic breast tumors, suggesting that overexpression of AURKA and inactivation of BRCA1 and BRCA2 cooperate during tumor development and progression. The F31I polymorphism in AURKA has been associated with breast cancer risk in the homozygous state in prior studies. Studies have demonstrated that AURKA overexpression contributes to genetic instability and tumourigenesis by disrupting the proper assembly of the mitotic checkpoint complex and occurs in a high proportion of breast cancers.

Entity Pancreatic cancer

Disease Pancreatic ductal adenocarcinoma is one of the most fatal malignancies. Intensive investigation of molecular pathogenesis might lead to identifying useful molecules for diagnosis and treatment of the disease.

Oncogenesis Pancreatic ductal adenocarcinoma harbors complicated aberrations of alleles including losses of 1p, 6q, 9p, 12q, 17p, 18q, and 21q, and gains of 8q and 20q. Pancreatic cancer is usually initiated by mutation of KRAS and aberrant expression of SHH. Overexpression of AURKA mapping on 20q13.2 may significantly enhance overt tumorigenesity. AURKA was a direct downstream target of MAPK1, suggesting that the overexpression of AURKA without gene amplification may be induced by constitutive activation of MAPK1 in cancer cells. The constitutive activation of MAPK1 is frequently observed in pancreatic cancer.

Entity Ovarian cancer

Disease Ovarian cancer is the most lethal gynecologic malignancy in developed countries. The exact cause is usually unknown. The risk of developing ovarian cancer appears to be affected by several factors.

Oncogenesis Recent studies have shown that DNA gains of the chromosomal region 20q13 and overexpression of the centrosomal kinase AURKA, the gene of which is found within the 20q13 region, are hallmarks of ovarian cancer. Amplification of AURKA has been reported in ovarian tumors, suggesting a role in ovarian cancer pathology. AURKA is polymorphic with two single nucleotide substitutions (449t/a and 527g/a) in evolutionarily conserved regions causing amino acid changes (F31I and V57I). Two other nucleotide substitutions (287c/g and 1891g/c) of unknown significance are in 5' and 3' untranslated regions (UTR), respectively. AURKA overexpression represented a survival factor for tumor cells and a negative prognostic molecular marker.

Entity Human esophageal squamous cell carcinoma

Disease Human esophageal squamous cell carcinoma (ESCC) is one of the most frequent malignancies worldwide and occurs at a very high frequency in the People's Republic of China, South Africa, France, and Italy. A number of epidemiological investigations have shown that esophageal carcinogenesis and the malignant development of esophageal cancers are complex and associated with multiple etiologic factors, including genetic backgrounds, environmental stimuli, nutritional conditions, and cultural habits.

Oncogenesis Despite some epidemiological observations, the biological mechanism(s) that is involved in ESCC occurrence and progression remains to be elucidated. It has been shown that point mutations of the tumor suppressor gene p53 are detected in 40% of human esophageal cancers. The Rb gene is also frequently mutated in ESCC. Amplification of the cellular protooncogenes Myc, EGFR, HST1, INT2, and cyclin D1 are often found in this malignant disease. A recent demonstration indicates that AURKA polymorphisms are associated with advanced disease status of ESCC, and there are strong evidence that AURKA is overexpressed in human ESCC and may play a role in carcinogenesis and malignancy development of ESCC.

Entity Human Bladder Cancer

Disease Bladder tumors are among the most common human cancers, with approximately 55 000 new cases detected each year in the United States. Bladder cancers, which represent a group of tumors with diverse morphologic and clinical behavior, exhibit one of the strongest relationships seen in any cancer between clinical aggressiveness and degree of aneuploidy.

Oncogenesis Bladder cancers arise from at least two distinct, albeit sometimes overlapping, pathways that lead to the development of papillary and solid or nonpapillary tumors. Most superficially growing, low-grade papillary lesions are diploid or near-diploid. Although they often recur, they are unlikely to invade the bladder wall and metastasize. By contrast, virtually all nonpapillary tumors are highly aneuploid and have a strong propensity to invade the stroma and metastasize. Superficial bladder tumors that are aneuploid are also likely to progress to invasive clinically aggressive carcinomas, which may metastasize. AURKA amplification is frequently overexpressed in bladder tumors tested by FISH and the strong association of the gene amplification and overexpression levels with the degree of aneuploidy suggests that AURKA may play an important role in bladder carcinogenesis by contributing to the development of aneuploid cell populations with aggressive phenotypes.

Entity Human colon cancer

Disease — It is the third most common form of cancer and the second leading cause of cancer-related death in the Western world. Colorectal cancer causes 655,000 deaths worldwide per year, including about 16,000 in the UK, where it is the second most common site (after lung) to cause cancer death. The most common colon cancer cell type is adenocarcinoma which accounts for 95% of cases. Other, rarer types include lymphoma and squamous cell carcinoma. Adenocarcinoma is a malignant epithelial tumor, originating from glandular epithelium of the colorectal mucosa. It invades the wall, infiltrating the muscularis mucosae, the submucosa and thence the muscularis propria.

Oncogenesis — Colorectal cancer is a disease originating from the epithelial cells lining the gastrointestinal tract. Hereditary or somatic mutations in specific DNA sequences, among which are included DNA replication or DNA repair genes and also the APC, K-Ras, NOD2 and p53 genes, lead to unrestricted cell division. Furthermore, genetic instability is expressed in colon cancer by an increased rate of a number of different genetic alterations. These different manifestations of genetic instability are classified into two major categories. The first one involves subtle changes in DNA sequences typically represented by microsatellite instability (MIN). The second one is characterized by gains and losses of whole or parts of chromosomes, named chromosomal instability (CIN), and it is considered a driving force for tumourigenesis. MIN occurs in approximately 15% of colon cancers and results from inactivation of the mismatch repair (MMR) system by either MMR gene mutations or hypermethylation of the MLH1 promoter. The mechanisms inducing CIN in cancer and more specifically in colon cancer are only partly understood. At least two possible causes, not mutually exclusive, could be responsible for CIN: mutations in genes encoding mitotic regulators, such as spindle checkpoint proteins, and defects in genes controlling centrosome homeostasis. The presence of mutations of the mitotic checkpoint regulators BUB1 and BUBR1 and amplification of AURKA in a subset of human colon cancers have suggested that CIN results primarily from deregulation of DNA replication and mitotic-spindle checkpoints.

Entity — Human Multiple myeloma

Disease — Multiple myeloma (also known as MM, myeloma, plasma cell myeloma, or as Kahler's disease after Otto Kahler) is a type of cancer of plasma cells which are immune system cells in bone marrow that produce antibodies. Myeloma is regarded as incurable, but remissions may be induced with steroids, chemotherapy, thalidomide and stem cell transplants. Myeloma is part of the broad group of diseases called hematological malignancies. Multiple myeloma (MM) is a malignancy characterized by genetic instability, suggesting a disruption of checkpoints that arrest cells at G2M when injury to the mitotic machinery occurs.

Oncogenesis — The expression of RHAMM and other centrosome-associated genes are known to correlate with the extent of centrosome amplification in multiple myeloma, and with poor prognosis. RHAMM has a significant interaction with TPX2, a protein which regulates the localization and action of AURKA at the spindle poles. AURKA is expressed ubiquitously in myeloma, to varying degrees. Aurora kinase inhibitor VE-465 also induces apoptosis and death in myeloma cell lines and primary myeloma plasma cells. The combination of VE-465 and dexamethasone improves cell killing compared with the use of either agent alone, even in cells resistant to the single agents.

Entity — Human hepatocellular carcinoma

Disease — Hepatocellular carcinoma (HCC, also called hepatoma) is a primary malignancy (cancer) of the liver. Most cases of HCC are secondary to either a viral hepatitide infection (hepatitis B or C) or cirrhosis (alcoholism being the most common cause of hepatic cirrhosis). In countries where hepatitis is not endemic, most malignant cancers in the liver are not primary HCC but metastasis (spread) of cancer from elsewhere in the body, e.g. the colon. Treatment options of HCC and prognosis are dependent on many factors but especially on tumor size and staging.

Oncogenesis — AURKA is overexpressed frequently in HCC, and correlated with high grade and high stage, indicating that overexpression of AURKA plays a role in the development and progression of HCC. Furthermore in HCC is frequently associated the homozygous deletion of p15E2 (MTS2/INK4b/CDKN2B) and p16E2 (MTS1/INK4a/CDKN2A) with overexpression of AURKA gene, this association may play a role in the oncogenesis and malignant progression of HCC.

Entity	Human Upper gastrointestinal adenocarcinomas
Disease	Upper gastrointestinal adenocarcinomas are the second most common cause of cancer-related death in the world. and are characterized by complex molecular changes. Several epidemiological studies have indicated that the incidence of proximal adenocarcinomas of the gastroesophageal junction and lower esophagus is rising faster than ever before in the Western world. The incidence of adenocarcinoma of the cardia, gastroesophageal junction, and lower esophagus has been rapidly rising, 5-fold to 6-fold in the past few decades, especially in patients younger than 50 years of age.
Oncogenesis	Overexpression of AURKA is frequent in upper gastrointestinal adenocarcinomas and in recently works it was identified the AURKA/AKT axis as an important mechanism that provides cancer cells with potent antiapoptotic properties through regulating p53-dependent apoptosis. Frequent overexpression of AURKA at the mRNA and protein levels in upper gastrointestinal adenocarcinomas, and interestingly, this overexpression was more prevalent in gastroesophageal junction adenocarcinomas andlower esophageal, Barrett-related adenocarcinomas (BAs) than in antrum and body gastric adenocarcinomas. There are not an association between AURKA overexpression and histopathological paramters such as tumor grade, TNM classification, and lymph-node metastasis.

External links

	Nomenclature
Hugo	AURKA
GDB	AURKA
Entrez_Gene	AURKA 6790 aurora kinase A
	Cards
Atlas	**AURKAID730ch20q13.txt**
GeneCards	AURKA
Ensembl	AURKA [Search_View] ENSG00000087586 [Gene_View]
Genatlas	AURKA
GeneLynx	AURKA

eGenome	AURKA
euGene	6790
	Genomic and cartography
GoldenPath	AURKA - 20q13.31 chr20:54377852-54400758 - 20q13.2-q13.3 [Description] (hg18-Mar_2006)
Ensembl	AURKA - 20q13.2-q13.3 [CytoView]
NCBI	Mapview
OMIM	Disease map [OMIM]
HomoloGene	AURKA
	Gene and transcription
Genbank	AF008551 [ENTREZ]
Genbank	AF011468 [ENTREZ]
Genbank	AL711075 [ENTREZ]
Genbank	AM392948 [ENTREZ]
Genbank	BC001280 [ENTREZ]
RefSeq	NM_003600 [SRS] NM_003600 [ENTREZ]
RefSeq	NM_198433 [SRS] NM_198433 [ENTREZ]
RefSeq	NM_198434 [SRS] NM_198434 [ENTREZ]
RefSeq	NM_198435 [SRS] NM_198435 [ENTREZ]
RefSeq	NM_198436 [SRS] NM_198436 [ENTREZ]
RefSeq	NM_198437 [SRS] NM_198437 [ENTREZ]
RefSeq	AC_000063 [SRS] AC_000063 [ENTREZ]
RefSeq	NC_000020 [SRS] NC_000020 [ENTREZ]
RefSeq	NT_011362 [SRS] NT_011362 [ENTREZ]
RefSeq	NW_927339 [SRS] NW_927339 [ENTREZ]
AceView	AURKA AceView - NCBI
Unigene	Hs.250822 [SRS] Hs.250822 [NCBI] HS250822 [spliceNest]
Fast-db	13689 (alternative variants)
	Protein : pattern, domain, 3D structure
SwissProt	O14965 [SRS] O14965 [EXPASY] O14965 [INTERPRO]
Prosite	PS00107 PROTEIN_KINASE_ATP [SRS] PS00107 PROTEIN_KINASE_ATP [Expasy]
Prosite	PS50011 PROTEIN_KINASE_DOM [SRS] PS50011 PROTEIN_KINASE_DOM [Expasy]
Prosite	PS00108 PROTEIN_KINASE_ST [SRS] PS00108 PROTEIN_KINASE_ST [Expasy]

Interpro	IPR000719 Prot_kinase_core [SRS] IPR000719 Prot_kinase_core [EBI]
Interpro	IPR008271 Ser_thr_pkin_AS [SRS] IPR008271 Ser_thr_pkin_AS [EBI]
Interpro	IPR002290 Ser_thr_pkinase [SRS] IPR002290 Ser_thr_pkinase [EBI]
CluSTr	O14965
Pfam	PF00069 Pkinase [SRS] PF00069 Pkinase [Sanger] pfam00069 [NCBI-CDD]
Smart	SM00220 S_TKc [EMBL]
Prodom	PD000001 Prot_kinase[INRA-Toulouse]
Prodom	O14965 STK6_HUMAN [Domain structure] O14965 STK6_HUMAN [sequences sharing at least 1 domain]
Blocks	O14965
PDB	1MQ4 [SRS] 1MQ4 [PdbSum], 1MQ4 [IMB] 1MQ4 [RSDB]
PDB	1MUO [SRS] 1MUO [PdbSum], 1MUO [IMB] 1MUO [RSDB]
PDB	1OL5 [SRS] 1OL5 [PdbSum], 1OL5 [IMB] 1OL5 [RSDB]
PDB	1OL6 [SRS] 1OL6 [PdbSum], 1OL6 [IMB] 1OL6 [RSDB]
PDB	1OL7 [SRS] 1OL7 [PdbSum], 1OL7 [IMB] 1OL7 [RSDB]
PDB	2BMC [SRS] 2BMC [PdbSum], 2BMC [IMB] 2BMC [RSDB]
PDB	2C6D [SRS] 2C6D [PdbSum], 2C6D [IMB] 2C6D [RSDB]
PDB	2C6E [SRS] 2C6E [PdbSum], 2C6E [IMB] 2C6E [RSDB]
PDB	2DWB [SRS] 2DWB [PdbSum], 2DWB [IMB] 2DWB [RSDB]
PDB	2J4Z [SRS] 2J4Z [PdbSum], 2J4Z [IMB] 2J4Z [RSDB]
PDB	2J50 [SRS] 2J50 [PdbSum], 2J50 [IMB] 2J50 [RSDB]
PDB	2NP8 [SRS] 2NP8 [PdbSum], 2NP8 [IMB] 2NP8 [RSDB]
HPRD	04066
	Protein Interaction databases
DIP	O14965
IntAct	O14965
	Polymorphism : SNP, mutations, diseases
OMIM	114500;603072 [map]
GENECLINICS	114500;603072
SNP	AURKA [dbSNP-NCBI]
SNP	NM_003600 [SNP-NCI]
SNP	NM_198433 [SNP-NCI]

SNP	NM_198434 [SNP-NCI]
SNP	NM_198435 [SNP-NCI]
SNP	NM_198436 [SNP-NCI]
SNP	NM_198437 [SNP-NCI]
SNP	AURKA [GeneSNPs - Utah] AURKA] [HGBASE - SRS]
HAPMAP	AURKA [HAPMAP]
COSMIC	AURKA [Somatic mutation (COSMIC-CGP-Sanger)]
HGMD	AURKA
	General knowledge
Family Browser	AURKA [UCSC Family Browser]
SOURCE	NM_003600
SOURCE	NM_198433
SOURCE	NM_198434
SOURCE	NM_198435
SOURCE	NM_198436
SOURCE	NM_198437
SMD	Hs.250822
SAGE	Hs.250822
Enzyme	2.7.11.1 [Enzyme-SRS] 2.7.11.1 [Brenda-SRS] 2.7.11.1 [KEGG] 2.7.11.1 [WIT]
GO	nucleotide binding [Amigo] nucleotide binding
GO	protein kinase activity [Amigo] protein kinase activity
GO	protein serine/threonine kinase activity [Amigo] protein serine/threonine kinase activity
GO	protein binding [Amigo] protein binding
GO	protein binding [Amigo] protein binding
GO	ATP binding [Amigo] ATP binding
GO	nucleus [Amigo] nucleus
GO	centrosome [Amigo] centrosome
GO	spindle [Amigo] spindle
GO	protein amino acid phosphorylation [Amigo] protein amino acid phosphorylation
GO	protein amino acid phosphorylation [Amigo] protein amino acid phosphorylation

GO	cell cycle [Amigo] cell cycle
GO	spindle organization and biogenesis [Amigo] spindle organization and biogenesis
GO	mitosis [Amigo] mitosis
GO	kinase activity [Amigo] kinase activity
GO	transferase activity [Amigo] transferase activity
GO	regulation of protein stability [Amigo] regulation of protein stability
GO	phosphoinositide-mediated signaling [Amigo] phosphoinositide-mediated signaling
BIOCARTA	Role of Ran in mitotic spindle regulation [Genes]
PubGene	AURKA
TreeFam	AURKA
CTD	6790 [Comparative ToxicoGenomics Database]
	Other databases
	Probes
Probe	AURKA Related clones (RZPD - Berlin)
	PubMed
PubMed	121 Pubmed reference(s) in LocusLink

Bibliography

A homologue of Drosophila aurora kinase is oncogenic and amplified in human colorectal cancers.
Bischoff JR, Anderson L, Zhu Y, Mossie K, Ng L, Souza B, Schryver B, Flanagan P, Clairvoyant F, Ginther C, Chan CS, Novotny M, Slamon DJ, Plowman GD.
EMBO J. 1998 Jun 1;17(11):3052-65.
PMID 9606188

Identification of phosphorylated residues that affect the activity of the mitotic kinase Aurora-A.
Littlepage LE, Wu H, Andresson T, Deanehan JK, Amundadottir LT, Ruderman JV.
Proc Natl Acad Sci U S A. 2002 Nov 26;99(24):15440-5.
PMID 12422018

Amplification/Overexpression of a Mitotic Kinase Gene in Human Bladder Cancer.

Sen S, Zhou H, Zhang RD, Yoon DS, Vakar-Lopez F, Ito S, Jiang F, Johnston D, Grossman HB, Ruifrok AC, Katz RL, Brinkley W, Czerniak B.
J Natl Cancer Inst. 2002 Sep 4;94(17):1320-9.
PMID 12208897

Aurora-A and an interacting activator, the LIM protein Ajuba, are required for mitotic commitment in human cells.
Hirota T, Kunitoku N, Sasayama T, Marumoto T, Zhang D, Nitta M, Hatakeyama K, Saya H.
Cell. 2003 Sep 5;114(5):585-98.
PMID 13678582

The Aurora kinases: role in cell transformation and tumorigenesis.
Katayama H, Brinkley WR, Sen S.
Cancer Metastasis Rev. 2003 Dec; 22(4): 451-64. (Review).
PMID 12884918

STK15 polymorphisms and association with risk of invasive ovarian cancer.
Dicioccio RA, Song H, Waterfall C, Kimura MT, Nagase H, McGuire V, Hogdall E, Shah MN, Luben RN, Easton DF, Jacobs IJ, Ponder BA, Whittemore AS, Gayther SA, Pharoah PD, Kruger-Kjaer S.
Cancer Epidemiol Biomarkers Prev. 2004 Oct;13(10):1589-94.
PMID 15466974

Overexpression and amplification of Aurora-A in hepatocellular carcinoma.
Jeng YM, Peng SY, Lin CY, Hsu HC.
Clin Cancer Res. 2004 Mar 15;10(6):2065-71.
PMID 15041727

Aurora-A abrogation of p53 DNA binding and transactivation activity by phosphorylation of serine 215.
Liu Q, Kaneko S, Yang L, Feldman RI, Nicosia SV, Chen J, Cheng JQ.
J Biol Chem. 2004 Dec 10;279(50):52175-82. Epub 2004 Oct 6.
PMID 15469940

Overexpression of Aurora-A Contributes to Malignant Development of Human Esophageal Squamous Cell Carcinoma.
Tong T, Zhong Y, Kong J, Dong L, Song Y, Fu M, Liu Z, Wang M, Guo L, Lu S, Wu M, Zhan Q.
Clin Cancer Res. 2004 Nov 1;10(21):7304-10.
PMID 15534106

Two functional coding single nucleotide polymorphisms in STK15 (Aurora-A) coordinately increase esophageal cancer risk.
Kimura MT, Mori T, Conroy J, Nowak NJ, Satomi S, Tamai K, Nagase H.
Cancer Res. 2005 May 1;65(9):3548-54.
PMID 15867347

A functional interplay between Aurora-A, Plk1 and TPX2 at spindle poles: Plk1 controls centrosomal localization of Aurora-A and TPX2 spindle association.
De Luca M, Lavia P, Guarguaglini G.
Cell Cycle. 2006 Feb;5(3):296-303. Epub 2006 Feb 7.
PMID 16418575

AURKA is one of the downstream targets of MAPK1/ERK2 in pancreatic cancer.
Furukawa T, Kanai N, Shiwaku HO, Soga N, Uehara A, Horii A.
Oncogene. 2006 Aug 10;25(35):4831-9.
PMID 16532023

Aurora-A and p16 polymorphisms contribute to an earlier age at diagnosis of pancreatic cancer in Caucasians.
Chen J, Li D, Wei C, Sen S, Killary AM, Amos CI, Evans DB, Abbruzzese JL, Frazier ML.
Clin Cancer Res. 2007 May 15;13(10):3100-4.
PMID 17505013

Consortium analysis of 7 candidate SNPs for ovarian cancer.
Couch FJ, Sinilnikova O, Vierkant RA, Pankratz VS, Fredericksen ZS, Stoppa-Lyonnet D, Coupier I, Hughes D, Hardouin A, Berthet P, Peock S, Cook M, Baynes C, Hodgson S, Morrison PJ, Porteous ME, Jakubowska A, Lubinski J, Gronwald J, Spurdle AB; kConFab, Schmutzler R, Versmold B, Engel C, Meindl A, Sutter C, Horst J, Schaefer D, Offit K, Kirchhoff T, Andrulis IL, Ilyushik E, Glendon G, Devilee P, Vreeswijk MP, Vasen HF, Borg A, Backenhorn K, Struewing JP, Greene MH, Neuhausen SL, Rebbeck TR, Nathanson K, Domchek S, Wagner T, Garber JE, Szabo C, Zikan M, Foretova L, Olson JE, Sellers TA, Lindor N, Nevanlinna H, Tommiska J, Aittomaki K, Hamann U, Rashid MU, Torres D, Simard J, Durocher F, Guenard F, Lynch HT, Isaacs C, Weitzel J, Olopade OI, Narod S, Daly MB, Godwin AK, Tomlinson G, Easton DF, Chenevix-Trench G, Antoniou AC; Consortium of Investigators of Modifiers of BRCA1/2.
Cancer Epidemiol Biomarkers Prev. 2007 Jul;16(7):1416-21.
PMID 17627006

Simultaneous Aurora-A/STK15 overexpression and centrosome amplification induce chromosomal instability in tumour cells with a MIN phenotype.
Lentini L, Amato A, Schillaci T, Di Leonardo A.
BMC Cancer. 2007 Nov 13;7:212.
PMID 17999753

Aurora A kinase RNAi and small molecule inhibition of Aurora kinases with VE-465 induce apoptotic death in multiple myeloma cells.
Evans R, Naber C, Steffler T, Checkland T, Keats J, Maxwell C, Perry T, Chau H, Belch A, Pilarski L, Reiman T.
Leuk Lymphoma. 2008 Mar;49(3):559-69.
PMID 18297535

REVIEW articles *automatic* search in PubMed
Last year publications *automatic* search in PubMed

Search in all EBI NCBI

Contributor(s)

Written 05-2008 Laura Lentini, Aldo Di Leonardo

Department of Cellular and Developmental Biology A, Monroy, University of Palermo, viale delle Scienze, Palermo, Italy

Citation

This paper should be referenced as such :

Lentini L, Di Leonardo A . AURKA (aurora kinase A). Atlas Genet Cytogenet Oncol Haematol. May 2008 .
URL : http://AtlasGeneticsOncology.org/Genes/AURKAID730ch20q13.html

 indexed on : Wed Jul 2 08:22:01 2008