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Gene Section

MIR30A (microRNA 30a)

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Identity

Other names: MIRN30A

HGNC (Hugo): MIR30A

Location: 6q13

Local order: Based on Mapviewer, genes flanking MIR30A oriented from centromere to telomere on 6q13 are:

- B3GAT2, beta-1,3-glucuronyltransferase 2 (glucuronosyltransferase S)

- MIR30C2, microRNA 30c-2
- MIR30A, microRNA 30a
- RIMS1, regulating synaptic membrane exocytosis 1

- KCNQ5, potassium voltage-gated channel, KQT-like subfamily, member 5

DNA/RNA

Description

miR-30 microRNA precursor is a small non-coding RNA that regulates gene expression.

Animal microRNAs are transcribed as ~70 nucleotide stem-loop precursor and subsequently processed by the Dicer enzyme to give a mature ~22 nucleotide product. In this case the mature sequence comes from both the 3' (miR-30) and 5' (mir-97-6) arms of the precursor. The products are thought to have regulatory roles through complementarity to mRNA. A screen of 17 miRNAs that have been predicted to regulate a number of breast cancer associated genes found variations in the microRNAs miR-17 and miR-30c-1, these patients were noncarriers of BRCA1 or BRCA2 mutations, lending the possibility that familial breast cancer may be caused by variation in these miRNAs. Members of the miR-30 family have been found to be highly expressed in heart cells.

Transcription

miRNAs are transcribed by RNA polymerase II as part of capped and polyadenylated primary transcripts (primiRNAs) that can be either protein-coding or noncoding. The primary transcript is cleaved by the Drosha ribonuclease III enzyme to produce an approximately 70-nt stem-loop precursor miRNA (pre-miRNA), which is further cleaved by the cytoplasmic Dicer ribonuclease to generate the mature miRNA.

Pre-miR Length: 71 bases.

gcgactgtaa acateetega etggaagetg tgaageeaca gatgggettt eagteggatg tttgeagetg e

Pseudogene

No reported pseudogenes.



Protein

Note

miRNAs are not translated into amino acids.

Mutations

Note

Gene mutations have not been described.

Implicated in

Breast cancer

Disease

Overexpression of miR-30a suppressed the migration and invasiveness phenotypes of breast cancer cell lines. Moreover, reduced tumor expression of miR-30a in breast cancer patients was associated with an unfavorable outcome, including late tumor stage, lymph node metastasis, and worse progression (Cheng et al., 2012).

Prognosis

Higher expression levels of hsa-miR-30a-3p, hsa-miR-30c, and hsa-miR-182 were significantly associated with benefit of tamoxifen treatment and with longer PFS (Rodríguez-González et al., 2011).

Colon carcinoma

Disease

miR-30a-5p was shown to be down-regulated in human colorectal cancer compared with normal colon mucosa. Overexpression of miR-30-5p suppresses proliferation colon cancer cell lines by targeting denticleless protein homolog (DTL) (Baraniskin et al., 2012).

Non-small cell lung cancer

Disease

microRNA-30a expression was found inversely proportional to the invasive potential of various NSCLC cell lines, correlating positively with Ecadherin (epithelial marker) and negatively with Ncadherin (mesenchymal marker) expression. Lucerfise reporter assay indicates snail was a potential target of miR-30a (Kumarswamy et al., 2012).

Squamous cell carcinoma

Note

Diagnosis: a 5-microRNA classifier (hsa-miR-210, hsa-miR-182, hsa-miR-486-5p, hsa-miR-30a, and hsa-miR-140-3p) that could distinguish SCC from normal lung tissues (Tan et al., 2011).

Gastric cancer

Prognosis

Li et al identified seven-miRNA signature (miR-10b, miR-21, miR-223, miR-338, let-7a, miR-30a-5p, miR-126) for overall survival (p=0,0009) and relapse-free survival (p=0,0005) of gastric cancer patients (2010).

Thyroid carcinomas

Disease

miR-30a-5p was down-regulated in Thyroid carcinomas in comparison to normal thyroid tissue (Visone, 2007).

Chemotherapy resistance

Note

miR-30a in regulating beclin 1 expression and autophagy reveals a novel function for miRNA in a critical cellular event with significant impacts in cancer development, progression and treatment (Zhu et al., 2009).

miR-30a can sensitize tumor cells to cis-DDP via reducing beclin 1-mediated autophagy and that increasing miR-30a level in tumor cells represents a novel approach to enhance the efficacy of chemotherapy during cancer treatment (Zou et al., 2012).

Imatinib markedly inhibits expression of miR-30a in human CML cells. miR-30a is a potent inhibitor of autophagy by downregulating Beclin 1 and ATG5 expression. miR-30a mimic or knockdown of autophagy genes (ATGs) such as Beclin 1 and ATG5 by short hairpin RNA enhances imatinib-induced cytotoxicity and promotes mitochondria-dependent intrinsic apoptosis.

In contrast, knockdown of miR-30a by antagomir-30a increases the expression of Beclin 1 and ATG5, and inhibits imatinib-induced cytotoxicity (Yu et al., 2012)

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