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Gene Section
Review

MIR373 (microRNA 373)

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Abstract

Review on MIR373, with data on DNA/RNA and where the gene is implicated.

Identity

Other names: MIRN373, hsa-mir-373, miRNA373

HGNC (Hugo): MIR373

Location: 19q13.42

Local order: Based on UCSC genome browser, genes flanking MIR373 oriented from centromere to telomere on 19q13.42 are:

- DPRX: divergent-paired related homeobox, 19q13.42,
- C19MC: microRNA cluster, 19q13.42,
- MIR371A: microRNA-371a, 19q13.42,
- MIR371B: microRNA-371b, 19q13.42,
- MIR372: microRNA-372, 19q13.42,
- **MIR373: microRNA 373, 19q13.42**,
- NLRP12: NLR family, pyrin domain containing 12, 19q13.42,
- MYADM: myeloid-associated differentiation marker, 19q13.42.

DNA/RNA

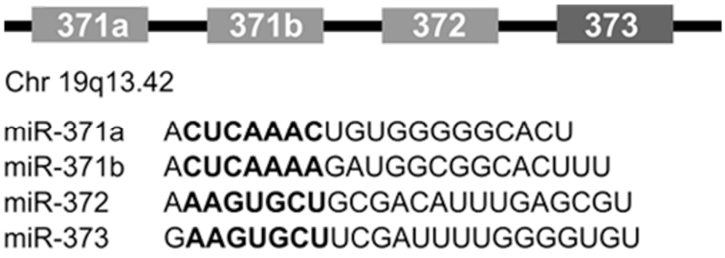
Description

miR-373 belongs to a cluster of four microRNAs located on chromosome 19q13.

The cluster is located approximately 20 kb downstream of the largest known cluster of miRNAs in the human genome, C19MC. miR-373 belongs to a family of miRNAs; (miR-302abcd/372/373/520be/520acd-3p) which are predicted to target the same genes due to the similarity in seed sequence (2-7 nucleotide, miRBASE v12.0).

Transcription

hsa-mir-373 (precursor miRNA)
Accession: MIMAT0000725
Length: 68 bp
Sequence:
5'-
GGGAUACUCAAAAUGGGGGCGCUUUCCUU
UUGUCUGUACUGGGAAGUGCUUCGAUUUU
GGGGUGUCCC-3'



The miR-371-373 cluster maps in the chromosomal region 19q13.42. However, miR-371ab and miR-372/373 differ in their seed sequences and as such display separate functions based on their target genes.

hsa-miR-373 (mature miRNA) Accession: MIMAT0000726

Length: 23 bp
Sequence:
5'-GAAGUGCUUCGAUUUUGGGGUGU-3'
The miR-373 cluster was first described to be specifically expressed in embryonic stem cells and transcribed as a polycistronic transcript (Suh et al., 2004).
The neighbouring C19MC cluster is thought to have originated from the miR-373 cluster (Zhang et al., 2008). miR-373 has been described to be epigenetically regulated by DNA methylation in colon cancer (Tanaka et al., 2011) and in a rare cancer type, hilar cholangiocarcinoma (Chen et al., 2012).
Chromosomal alterations have been also linked to aberrant expression of miR-371-373 cluster. Indeed, 19q13.4 translocation in thyroid adenomas has been shown to activate both, C19MC and miR-371-373 clusters which are in close proximity to the chromosomal breakpoint cluster (Rippe et al., 2010). miR-371-373 cluster has also been reported to be transcriptionally regulated by WNT-signaling through β-catenin/LEF1 transactivation of the miR-371-373 promoter in both, breast (MCF-7) and colon (HCT-116) cancer cells (Zhou et al., 2012). Furthermore, miRNA microarray analysis revealed miR-373 to be upregulated under hypoxic conditions in a HIF-1α-dependent manner, in both HeLa and MCF-7 cancer cell lines that were exposed to hypoxia (0,01% O_2) (Crosby et al., 2009).

Pseudogene

No reported pseudogenes.

Protein

Note
microRNAs are not translated into amino acids.

Mutations

Note
Gene mutations have not been described.

Implicated in

Breast cancer

Oncogenesis
In a miRNA genetic screen to identify regulators of cancer cell migration, Huang and colleagues (Huang et al., 2008) used a miRNA library consisting of 450 retro-viral miRNA vectors to transduce the non-invasive and non-metastatic estrogen-dependent breast cancer cell line MCF-7, which was then subjected to trans-well migration (Huang et al., 2008). Subsequent analysis of the migrated cancer cell population by miR-array (Voorhoeve et al., 2006) revealed that several members of the miR-373 family were enriched in the migrating cells compared with their abundance in the total cell population (Huang et al., 2008). The authors further demonstrated a pro-metastatic role of miR-373 and miR-520c in vivo, using xenograft mouse models for pulmonary metastasis, which mechanistically could be explained by direct targeting of the hyaluronan receptor CD44 (Huang et al., 2008). In agreement with these findings, qRT-PCR analysis of 11 paired primary breast tumors and lymph-node metastases (LN) revealed that miR-373 expression was upregulated in metastases compared to primary tumors (Huang et al., 2008). Similarly, the average miR-373 expression was upregulated in 34 primary breast tumors of patients who were positive for LN compared to 38 LN negative primary tumors (Huang et al., 2008). In another study, using a SILAC-based quantitative proteomics approach, Yan and colleagues identified 30 genes involved in cancer invasion and metastasis to be downregulated at the protein level by miR-373 in MCF-7 cells, while the tumor suppressors TXNIP and RABEP1 were validated as direct targets (Yan et al., 2011).
However, the role of the miR-373 family in cancer development and progression may be cancer type and/or context-dependent. Keklikoglou and colleagues analyzed a panel of 76 primary breast tumors with known LN and estrogen receptor (ER) status for miR-373 and miR-520c expression by qRT-PCR. Interestingly, miR-520c was found to be downregulated in tumors of patients with LN metastasis compared to tumors of patients without, specifically in ER negative tumors (Keklikoglou et al., 2012). However, this effect was restricted in ER negative tumors as no significant difference in miR-520c expression was observed between the LN positive and LN negative groups in ER positive patients (Keklikoglou et al., 2012). It is noteworthy, that miR-373 expression analysis was not feasible due to the low detection levels in both ER positive and ER negative patients (Keklikoglou et al., 2012). In the same study, several members of miR-373 family were shown to downregulate the secretion of pro-inflammatory and pro-angiogenic cytokines by mammary epithelial and cancer cells, while cancer cell invasion and intravasation of the highly invasive and ER negative breast cancer cell line, MDA-MB-231, was inhibited both in vitro and in vivo, by targeting RELA and TGFBR2, simultaneously (Keklikoglou et al., 2012).
Diagnosis: analysis of circulating miR-373 in the plasma of breast cancer patients revealed that miR-373 was upregulated in 35 patients with metastatic breast cancer to the lymph-nodes compared to 25 patients with non-metastatic disease (Chen et al., 2012). Furthermore, increased amounts of circulating miR-373 in the serum of breast cancer patients have been reported when compared to

healthy donors, however, no difference in circulating miR-373 amounts was observed between patients with metastatic and non-metastatic disease (Eichelser et al., 2013).

Prostate cancer

Oncogenesis

miR-373 has been reported to be downregulated in 5 matched prostate tumor specimens compared to benign tissue (Yang et al., 2009). However, overexpression of miR-373 or miR-520c markedly reduced CD44 total protein levels, while it stimulated prostate cancer cell invasion in vitro (Yang et al., 2009). On the contrary, Walter and colleagues analyzed 37 prostate cancer specimens that were manually microdissected to obtain cancer cells, adjacent stroma cells and normal epithelium, which were then analyzed by qRT-PCR using RT2-PCR arrays for miRNA expression (Walter et al., 2013). The profiling revealed that miR-372, a member of the miR-373 family, was upregulated in both tumor and stroma compartments compared to normal epithelium (Walter et al., 2013). In addition, the expression of miR-373 was found to be significantly upregulated in high grade tumors (Gleason score ≥8) compared to low grade tumors (Gleason score <6) (Walter et al., 2013).

Childhood B-cell precursor acute lymphoblastic leukemia (pre-B-ALL)

Oncogenesis

miRNA expression profiling in blast cells revealed miR-373* to be downregulated in pre-B-ALL patients compared to their healthy counterparts (Ju et al., 2009).

Esophageal cancer

Oncogenesis

miR-373 has an oncogenic role in esophageal cancer cell lines and tumors by targeting the tumor suppressor LATS2 and as such, promoting cell growth (Lee et al., 2009). This tumorigenic role was further confirmed by qRT-PCR which revealed that miR-373 was upregulated in 11 out of 23 matched esophageal cancer tissues compared to their adjacent non-tumor counterparts (Lee et al., 2009).

Fibrosarcoma

Oncogenesis

miR-373 as well as miR-520c have been demonstrated to indirectly regulate MMP9 expression in human fibrosarcoma HT1080 cells by directly targeting mTOR and SIRT1, two negative regulators of RAS/MEK/ERK and NF-kB signaling, respectively (Liu and Wilson, 2012). Thus, miR-373 promotes cell migration and growth by inducing both, ERK- and NF-kB-dependent MMP9 expression (Liu and Wilson, 2012).

Gastric cancer

Oncogenesis

miR-373, as well as miR-520c-5p and miR-372 were reported to be upregulated in gastric cancer cells compared to cancer stem cells (CSCs) which had been previously sorted as CD44+ cells from the same gastric cancer cell line MKN-45 (Golestaneh et al., 2012).

Infection of gastric epithelial cells with Helicobacter pylori, a pathogen that is associated with the development of atrophic gastritis and gastric carcinoma, has been shown to cause miR-373 downregulation and as such cell cycle arrest of highly proliferating gastric epithelial cells through upregulation of LATS2, a miR-373 direct target (Belair et al., 2011).

Hepatocellular carcinoma (HCC)

Oncogenesis

miR-373 has been found to be upregulated in 26 HCC tissues compared with their matched normal adjacent counterparts, as analyzed by qRT-PCR (Wu et al., 2011). Moreover, miR-373 was shown to promote cell cycle progression from G1 to S phase and cell proliferation in HCC cell lines by targeting protein phosphatase 6 (PPP6C) (Wu et al., 2011). In agreement with this study, Toffanin and colleagues reported that miRNAs from the C19MC cluster were upregulated in a panel of 89 HCC patients, as determined by miRNA microarray profiling (Toffanin et al., 2011). In particular, miR-517a and miR-520c were associated with increased cell proliferation and tumor growth, using in vitro and in vivo approaches (Toffanin et al., 2011). Interestingly, miRNA microarray analysis revealed that miR-373 was upregulated in the HCC cells, HepG2, upon cell infection with the hepatitis B virus (HBV), a pathogen that is frequently associated with liver diseases, including inflammation, cirrhosis and cancer (Guo et al., 2011). In addition, miR-373 was shown to regulate the expression of the virus in the host cells by directly regulating NFIB expression (Guo et al., 2011). Nevertheless, it should be noted that other members of the miR-373 family that share identical seed sequence were found to be downregulated in HCC (Zhang et al., 2012a; Zhang et al., 2012b). For instance, the promoter of miR-520e has been shown to be hypermethylated in HCC tissues and cell lines, thus leading to abrogated miR-520e expression (Zhang et al., 2012a). In addition, miR-520b and miR-520e have been shown to inhibit tumor growth in HCC using xenograft mouse models, by targeting MEKK2, CCND1 and NIK, respectively (Zhang et al., 2012a; Zhang et al., 2012b).

Lung cancer

Oncogenesis

Overexpression of miR-373 in A549 lung cancer cells inhibited cell migration by increasing E-cadherin expression levels (Wu et al., 2012). Place and colleagues had previously reported that miR-373 may enhance E-cadherin expression at the transcriptional level by targeting its promoter sequence in PC-3 cells (Place et al., 2008).

Ovarian cancer

Oncogenesis

miR-373 as well as miR-372 were identified as inducers of cell proliferation in human epithelial ovary cancer cells, A2780, using a gain-of-function miRNA screen to identify miRNA regulators of ovarian cancer cell viability (Nakano et al., 2013).

Testicular germ cell tumors (TGCTs)

Oncogenesis

miR-373 was initially identified as an oncogene in testicular germ cell tumorigenesis. In particular, ectopic expression of miR-373 in primary fibroblasts (BJ/ET) which were subsequently transduced with oncogenic RAS (RAS^{V12}), inhibited RAS-induced senescence and enhanced cellular transformation along with the RAS oncogene, in the presence of wild-type p53 (Voorhoeve et al., 2006). Furthermore, miR-373 expression was detected in 4 out of 7 cell lines tested which were derived from nonseminomatous tumors, while miR-373-371 cluster was barely expressed in other somatic cancer cell lines tested originating from breast, lung and brain tumors, as determined by an RNase protection assay (RPA) (Voorhoeve et al., 2006). In a panel of primary seminomas, nonseminomas, and spermatocytic seminomas analyzed for miR-372 expression, this cluster was found to be upregulated in most seminomas (28/32) and nonseminomas (14/21), while no miR-372 expression was detected in neither spermatocytic-seminoma tumors nor in normal testis tissues. Of note, tumors with high miR-372-373 expression did not contain mutated p53 alleles (Voorhoeve et al., 2006). Last but not least, miR-372 and mir-373 were found to neutralize p53-mediated CDK inhibition, partially by directly targeting LATS2 tumor suppressor (Voorhoeve et al., 2006).

In another study, miR-373 and miR-372 expression was strongly upregulated in 11 seminoma tissues compared to 11 normal testicular tissues, as determined by miRNA microarray analysis (Bing et al., 2012).

Pancreatic cancer

Oncogenesis

The zinc importer ZIP4 has been recently shown to transcriptionally regulate miR-373 expression in pancreatic cancer through CREB activation and binding to the miR-373 promoter (Zhang et al., 2013).

In particular, pancreatic ductal adenocarcinoma cells (PDAC) MiaPaca-2 overexpressing ZIP4 were shown to also overexpress miR-373, as determined by miRNA microarray analysis. At the same time, dampening ZIP4 expression in AsPC-1 cells using a short hairpin RNA (shRNA) against ZIP4 resulted in downregulation of miR-373 expression compared to control cells (Zhang et al., 2013). Of note, ZIP4-dependent upregulation of miR-373 led to increased cell invasion, proliferation and accelerated tumor growth in vivo, using a xenograft mouse model of PDAC. The oncogenic function of miR-373 was shown to be mediated by direct targeting of CD44, LATS2 and TP53INP1 (Zhang et al., 2013).

Hilar cholangiocarcinoma

Prognosis

Analysis of 48 patients with hilar cholangiocarcinoma revealed that decreased expression of miR-373 was associated with poor cell differentiation, advanced clinical stage and shorter overall and disease-free survival (Chen et al., 2011a).

Oncogenesis

The promoter miR-373 has been found to be methylated in hilar cholangiocarcinomas and as such, its expression levels are decreased in malignant tissues (Chen et al., 2011a). Methyl-CpG-binding domain protein 2 (MBD2) is a direct target of miR-373 in hilar cholangiocarcinoma (Chen et al., 2011b).

Colon cancer

Oncogenesis

Using a microarray-based trasncriptomic approach, miR-373 was identified to be epigenetically regulated in the colon cancer cell line DLD-1 which was treated with 5'-aza'2'-deoxycytidine (DAC), as its expression levels were increased upon treatment compared to control cells (Tanaka et al., 2011). The epigenetic regulation of miR-373 was further confirmed by bisulfite sequencing analysis which revealed aberrant methylation of the miR-373 promoter in colon cancer cells. 40 colon cancer specimens were analyzed for miR-373 expression levels (Tanaka et al., 2011). In line with the aforementioned observations, miR-373 was downregulated in colon cancer specimens compared to their normal counterparts, while aberrant methylation of miR-373 promoter was identified in 87,5% of the colon cancer specimens using methylation-specific PCR (Tanaka et al., 2011). The oncogene RAB22A has been identified as a direct target of miR-373 in colon cancer (Tanaka et al., 2011).

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