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Gene Section
Short Communication

MIR429 (microRNA 429)

Yaguang Xi, Hong Chang

Mitchell Cancer Institute, University of South Alabama, USA. xi@health.southalabama.edu; hchang@health.southalabama.edu

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Abstract

Review on MIR429, with data on RNA, and where it is implicated.

Keywords: MIR429

Identity

HGNC (Hugo): MIR429

Location: 1p36.33

Note

MicroRNA 429 belongs to microRNA 200 family. MicroRNA 200 family consists of five microRNAs, microRNA 200b, microRNA 200a, microRNA 429, microRNA 200c and microRNA 141. MicroRNA 200b, microRNA 200a and microRNA 429 were transcribed as a single ploycistronic transcript from chromosome 1. And microRNA 200 family were demonstrated important roles in EMT (epithelial-mesenchymal transition) progress.

DNA/RNA

Description

microRNA 429 located in chromosome 1 and microRNA 429 was transcribed with microRNA 200b and microRNA 200a as a ploycistronic transcript. The putative transcription start site locates about 6129 bp upstream of the precursor of microRNA 429.

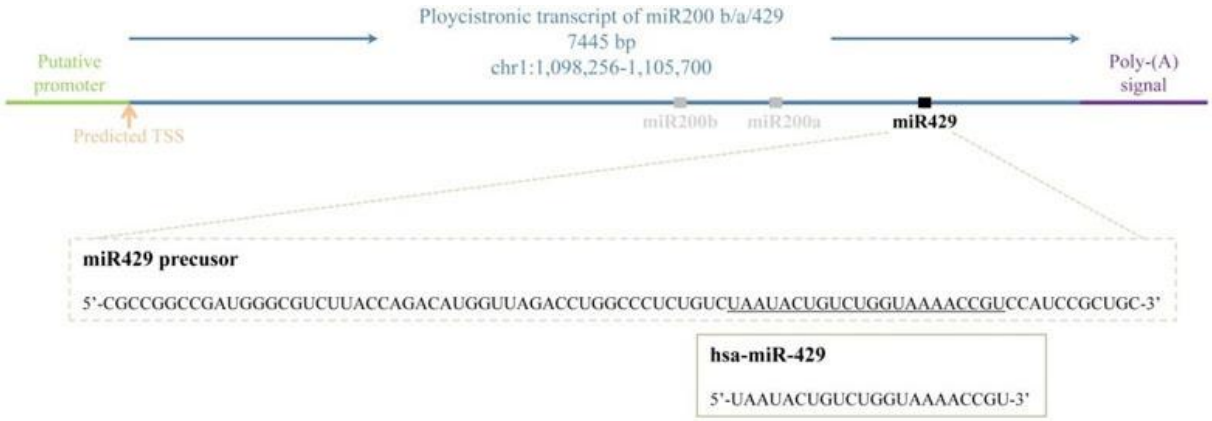
Transcription

MiR-429 precursor: 5'-CGCCGGCCGAUGGGCGUCUUACCAGACAUGGUUAGACCUGGCCCUCUGUCUAAUACUGUCUGGUAAAACCGUCCAUCCGCUGC-3'

Mature miR-429: 5'-UAAUACUGUCUGGUAAAACCGU-3'

Pseudogene

No pseudogene was found.



Protein

Note
microRNAs are not translated into proteins.

Implicated in

Tumor cell proliferation

Abnormal expression of miR-200a was reported in various tumors, including human breast cancer, gastric carcinoma, endometrial adenocarcinoma, gliomas, and colorectal carcinoma. MiR-429 could inhibit cell growth and induce apoptosis by directly targeting ONECUT2 in colorectal carcinoma and targeting BCL2 in esophageal carcinoma (Sun, Shen et al. 2014).
Also, MiR-429 could induce apoptosis and suppress invasion by targeting BCL2 and SP1 in esophageal carcinoma (Wang, Li et al. 2013). However, miR-429 was also showed its anti-apoptosis function in colorectal cancer by targeting SOX2 (Li, Du et al. 2013).

Tumor cells invasion and cancer metastasis

Members of miR-200 family showed vital roles in tumor cells invasion and cancer metastasis. As to miR-429, studies demonstrated it could repress tumor cell invasion and cancer metastasis by targeting ONECUT2 and SP1 in different tumors (Sun, Shen et al. 2014; Wang, Li et al. 2013).

Repression of epithelial-mesenchymal transition (EMT)

EMT is an important feature of tumor cells.
Tumor cells underwent EMT showed more invasion and metastatic properties.
Studies demonstrated members of miR-200 family could directly target ZEB1 and ZEB2 and could be regulated as a feedback loop during EMT.
Ectopic over-expression of miR-429 induces mesenchymal-to-epithelial transition (MET) in metastasizing ovarian cancer cells (Chen, Wang et al. 2011).
In colorectal carcinoma cells, miR-429 could regulate EMT-related markers such as ZEB2, Vimentin, SNAI2 (SLUG) and SNAI1 (SNAIL) by targeting ONECUT2 (Sun, Shen et al. 2014).

Chemoresistance

Cancer stem cells showed highly resistance to chemotherapy which make relapse of tumor.
It was found that over-expression of miR-429 could increase drug sensitivity in metastasizing ovarian (Wang, Mezencev et al. 2014).
However, it was reported that miR-429 was highly expressed in hepatocellular carcinoma tissues and enrichment of miR-429 in liver tumour-initiating cells with EPCAM expression would contributed to hepatocyte self-renewal, malignant proliferation, chemoresistance and tumorigenicity (Li, Tang et al. 2014).

References

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