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Gene Section
Short Communication

MIR200B (microRNA 200b)

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Abstract

Review on MIR200B, with data on RNA, and where it is implicated.

Keywords
MIR200B

Identity

HGNC (Hugo): MIR200B

Location: 1p36.33

Note
MicroRNA 200b 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

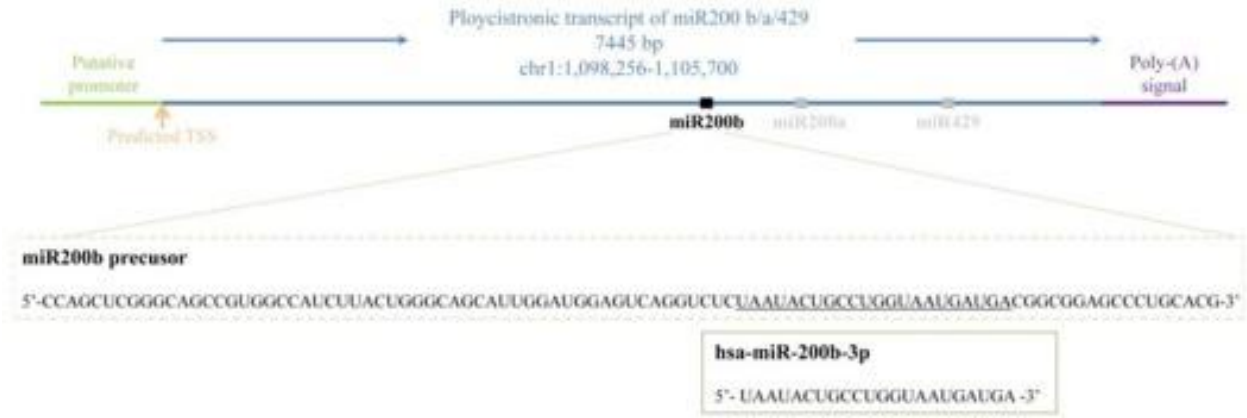
microRNA 200b located in chromosome 1 and microRNA 200b was transcribed with microRNA 200a and microRNA 429 as a ploycistronic transcript. The putative transcription start site locates about 4228 bp upstream of the precursor of microRNA 200b.

Transcription

MiR-200b precursor: 5'-CCAGCUCGGGCAGCCGUGGCCAUCUUACUGGGCAGCAUUGGAUGGAGUCAGGUCUCUAAUACUGCCUGGUAAUGAUGACGGCGGAGCCCUGCACG-3'
Mature miR-200b: 5'-UAAUACUGCCUGGUAAUGAUGA-3'

Pseudogene

No pseudogene was found.



Protein

microRNAs are not translated into proteins.

Implicated in

Tumor cell proliferation

Multiple roles of miR-200b in cell cycles regulation and tumor cell proliferation were reported in human cancers. MiR-200b could directly target RND3 which could induce expression of cyclin D1 thereby promoting S-phase entry (Xia, Li et al. 2010). MiR-200b could also regulate cell cycle by targeting GATA4 and its downstream protein cyclin D1 which could affect cell proliferation (Yao, Wei et al. 2013). In human TGFBR2-null colorectal cancers, miR-200b could stimulate tumor growth by targeting CDKN1B (p27/kip1). It was also found that miR-200b could promoter cell proliferation by targeting p27/kip1 (Fu, Liu et al. 2014).

Tumor cells invasion and cancer metastasis

Abnormal expressions of miR-200b were found in various cancers, including breast cancer, colon cancer, nasopharyngeal carcinoma, urothelial carcinoma and prostate cancer. MiR-200b could directly targeting SUZ12 and ROCK2 thus inhibit cholangiocarcinoma tumourigenesis and metastasis (Peng, Jiang et al. 2013). In gastric carcinoma, miR-200b was found to target ZEB2 and thereby repress tumor cell invasion and migration (Kurashige, Kamohara et al. 2012).

Repression of epithelial-mesenchymal transition (EMT)

EMT is an important pathological progression and studies revealed vital roles of miR-200b in EMT regulation. MiR-200b could inhibit TGFB1-induced epithelial-mesenchymal transition by targeting SMAD2 intestinal epithelial cells and SMAD2 could repress expression of vimentin, which indicated regulation role of miR-200b on vimentin (Chen, Xiao et al. 2013). Also, the role of miR-200b in EMT regulation was observed in EMT induced by transforming growth factor-beta1 in kidney proximal tubular cells by directly target the 3'-UTR of fibronectin (Tang, Chen et al. 2013).

Chemoresistance

Chemoresistance is the main cause of tumor relapse. Ectopic expression of miR-200b could reduce the chemoresistance of cells by targeting BIM1 in human tongue cancer cells. In human lung adenocarcinoma cells, miR-200b could target E2F3 to sensitize tumor cells to chemotherapy agents.

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