

# Gene Section

## Short Communication

### MIR23A (microRNA 23a)

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#### Identity

**Other names:** MIRN23A, hsa-mir-23a, miRNA23A

**HGNC (Hugo):** MIR23A

**Location:** 19p13.13

#### DNA/RNA

##### Description

miRNA23A is a non-coding RNA (ncRNAs). This gene is located at chromosome 19 at location p13.13. It ranges from 13947401 to 13947473 on reverse strand.

##### Transcription

This gene has one transcript and one coding exon. And its transcript length is 73 bps. This transcript does not have a protein product.

In human, the miR-23a has two mature miRNAs: hsa-mir-23a-5p and hsa-mir-23a-3p. The hsa-mir-23a-5p, also described as hsa-mir-23a\*, locates from 13947444 to 13947465 (22 bps) while hsa-mir-23a-3p, shortly named as hsa-mir-23a, ranges from 13947409 to 13947429 (21 bps).

Sequences:

hsa-mir-23a-5p: ggguuuccuggggauagggaauu

hsa-mir-23a-3p: aucacaugccagggaauucc

The miR-23a forms cluster with miR-27a and miR-24-2, namely miR-23a~27a~24-2 cluster and encode primary miRNAs transcript (pri-miRNAs). The promoter of this cluster has lack of several promoter elements: TATA box, initiator element, downstream core promoter element, TFIIB recognition element, downstream core element and multiple start site downstream elements (Smale and Kadonaga, 2003).

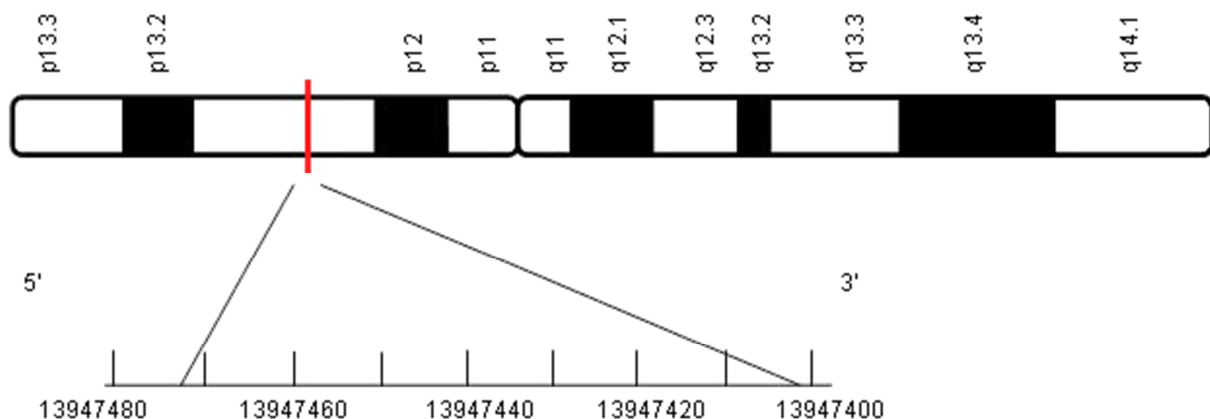


Figure 1: The microRNA 23a gene location (from 13947401 bps to 13947473 bps) was redrawn from Chhabra et al., 2010.

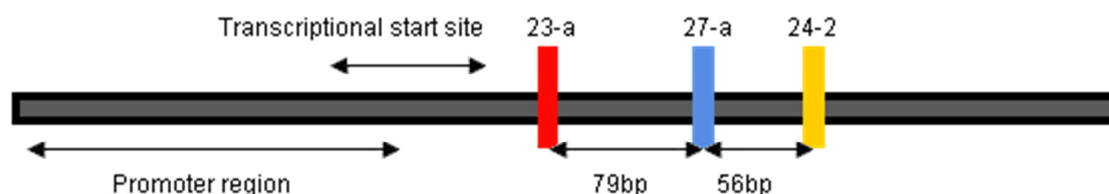


Figure 2: The gene structure of miR-23a-27a-24-2 at chromosome 9q22 was redrawn from Chhabra et al., 2010.

## Mutations

### Note

N/A

## Implicated in

### Hepatocellular carcinoma

#### Note

In the study by Huang et al. (2008), up-regulation of the cluster hindered TGF- $\beta$  induced apoptotic cell death and supported cell growth in hepatocellular carcinoma. Activation of miR-23a by STAT-interleukin 6 negatively regulated PGC-1 $\alpha$  and glucose-6-phosphatase catalytic subunit (G6PC), leading to decreased glucose production that favours hepatocellular carcinoma (Wang et al., 2012). Coptidis Rhizoma (CR, huanglian in Chinese) and its active compound, berberine has been shown to elicit anti-cancer properties in different cell lines and animal models (Wang et al., 2010; Feng et al., 2011).

Zhu et al. (2011) has reported up-regulation of miR-23a after treatment with CR in hepatocellular carcinoma (HCC) cell lines, suggesting miR-23a can be one of the targets and biomarker alter for CR treatment in HCC cell lines and implying the potential application of CR on treatment of HCC.

### Acute promyelocytic leukemia

#### Note

Saumet et al. (2009) has observed PML-RARA oncogene dependent characteristic of miR-23a cluster and significant repression of miR-23a by PML-RARA. The association of retinoic acid receptor alpha (RARA) gene with promyelocytic leukemia (PML) protein in chromosomal translocation process favoured the protein expression in acute promyelocytic leukemia (APL).

### Other cancers

#### Note

Increased expression of miR-23a has been reported to promote cancer growth in bladder cancer, gastric adenocarcinoma and colorectal cancer (Mi et al., 2007; Gottardo et al., 2007; Zhu et al., 2010; Jahid et al., 2012).

## Angiogenesis

### Note

Suppression of angiogenesis in vitro and postnatal retinal vascular development in vivo was reported in response to the reduced expression of miR-23. Loss of miR-23 gene has hindered laser-induced choroidal neovascularization in mouse model. These functions were postulated to be caused by the inhibition of Sprouty2 and Sema6A, which negatively regulate MAPK and VEGFR2 factors (Zhou et al., 2011). Poliseno et al. (2006) has stated the presence of receptors of angiogenic factors in human umbilical vein endothelial cells (HUVECs) as target of miR-23a.

## Neural differentiation

### Note

A study by Kawasaki and Taira (2003) has demonstrated the regulation of Hes-1 gene by miR-23, thereby supporting the neuronal differentiation of NT-2 cells at post-transcriptional level.

The reduced expression of miR-23 is associated with accumulation of Hes1 gene, a basic helix loop helix differentiation suppressor, resulting in blockage of retinoic acid induced neuronal differentiation.

## Muscular atrophy / Cardiac hypertrophy

### Note

The miR-23a inhibited translation of muscle-specific ubiquitin ligases, MAFbx/atrogen-1 and muscle RING-finger 1 (MuRF1), thus promoting protection against skeletal muscle atrophy (Wada et al., 2011).

In the report by Lin et al. (2008), nuclear factor of activated T cells (NFATc3) is proposed to regulate cardiac hypertrophy by transcriptionally activate miR-23a. Muscle RING-finger 1 (MuRF1) is the target of miR-23a where hypertrophic signal is conveyed by miR-23a through suppressing the translation of MURF1.

## Development of primary hematopoietic cells

### Note

Inhibition of B lymphopoiesis both in vitro and in vivo by miR-23a cluster expressing hematopoietic progenitor was reported by Kong et al. (2010). The cluster was recognised as the downstream target of

transcription factor PU.1. PU.1 has four conserved binding sites for promoter of miR-23a cluster. It showed that the cluster promoted myelopoiesis, while blocked the development of B lymphoid cells.

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