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*Editorial*

# Involvement of microrna-183 in Cancer Progression

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## EDITORIAL NOTE

Many types of cancers have aberrant expression of MicroRNA-183 (miR-183). It has a role in the beginning and progression of cancers. The expression of miR-183 is controlled by a variety of mechanisms. The mechanism of action of miR-183 in cancer is complex, and inconsistent findings are frequently drawn. It was elevated in 18 cancer types and downregulated in six cancer types. There are also seven other forms of cancer, with both upregulated and downregulated reports available. Evidence suggests that miR-183 can act as an oncogene or an anti-oncogene, as well as control the expression of other oncogenes or anti-oncogenes in various cancer types. The regulation of miR-183 is discussed in this review, as well as the expression of miR-183 in various malignancies. We also tallied the miR-183 target genes and their functional roles. Furthermore, we concentrated on miR-183's effects in cell migration, cell invasion, and microangiogenesis, which are all critical in cancer processes. It explains why miR-183 appears to play diverse functions in different malignancies. Furthermore, miR-183 and its downstream effectors have the potential to be useful cancer prognostic and therapeutic markers.

The expression of miR-183 varies dramatically amongst cancer types. To understand the varied up-and-down regulation events of miR-183 in different diseased tissues, it is required to outline the upstream regulatory factors of miR-183. Although the upstream regulation of the miR-183 cluster has been established, only a portion of the factors directly affect miR-183 expression. According to the research, there are eight regulatory variables that can increase miR-183 expression levels. By binding to the upstream promoter region of miR-183, they can boost its transcriptional expression. The most researched aspect of miR-183 regulation is the Wnt/beta-catenin (CTNNB1) signalling pathway. CTNNB1 activation upregulates miR-183 expression, while CTNNB1 knockdown results in miR-183 downregulation, suggesting that miR-183 is a key downstream target gene in the regulation of the Wnt signalling pathway. In human gastric cancer cells, further research have found that activation of GSK3 inhibits miR-183 production via the Catenin/TCF/LEF-1 pathway, whereas -Catenin/TCF/LEF-1 binds to the miR-183 promoter and so promotes miR-183 transcription. They discovered that TFAP2C prevented AKAP12-mediated cyclin D1 inhibition via increasing the overexpression of miR-183 in non-small cell lung cancer, indicating that TFAP2C is one of the upstream regulators of miR-183. ZEB1 and HSF2 regulate miR-183 transcription in breast cancer, and HSF2 can upregulate miR-183 expression. The regulatory effect of ZEB1 on miR-183, on the other hand, is more complicated. MiR-183 transcriptional expression can be downregulated by ZEB1, although ZEB1 can also be targeted by miR-183.

While in cancer, the pattern of miR-183 changes differs substantially between cancer types. In different cancer types, distinct upstream regulators play a vital role in miR-183 expression. We can deduce that miR-183 is increased in the majority of cancers. Meanwhile, 8 upstream regulators can upregulate miR-183 expression, whereas 5 upstream regulators can downregulate miR-183 expression. Because there are more upregulating effect transcription factors than downregulating effect transcription factors, we believe miR-183 is elevated in most cancer types. This shows that miR-183 has a variety of functions in various cancers, and its molecular mechanisms are complicated and varied, requiring additional investigation. Furthermore, aberrant miR-183 expression in cancer progression can be employed as a biological molecule for cancer detection and treatment; it is extremely important for cancer diagnosis and drug development.

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