

Oncogenesis and MicroRNA

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Description

MicroRNAs (miRNAs) were first found in 1993, when a non-coding short RNA gene called *lin-4* was discovered in a genetic screen looking for genes affecting developmental time in *Caenorhabditis elegans* larvae. Findings about *lin-4* and other newly discovered small non-coding RNAs with the ability to change gene expression post-transcriptionally in a variety of species, including mammalian species, have positioned miRNAs, which are endogenously synthesised, approximately 22 nucleotide-long, single-stranded, non-coding RNAs, as essential components of the noncoding genome [1-5].

Since it was initially shown the link between miRNAs and cancer by identifying miR-15-a and miR-16-1 as located on a chromosomal region that is frequently deleted in B-cell chronic lymphocytic leukaemia cases, miRNAs have become a very prominent issue in cancer research. Since then, significant progress has been made in understanding miRNAs and their role in the aetiology of various cancer types. They've been proven to have oncogenic or tumour suppressive properties. MiR-125b, miR-221/222, miR-21, miR-27a, and miR-106a, among others, have recently been implicated as important oncogenic miRNAs linked to cancer [2,3].

The most well-known tumour suppressor miRNAs in carcinogenesis include miR-145, miR-34c, and let-7c. MiRNAs have been linked to tumour development and metastasis, in addition to their oncogenic and tumour suppressor properties. Although the roles of miRNAs in these processes are yet unknown, particular miRNAs, including as let7c, miR-21, miR-34a, miR-145, and miR-205, have been implicated as important effectors in cell migration and metastatic pathways in various malignancies [4,5].

Furthermore, mature miRNAs are among the ideal candidate biomarkers for characterization of physio-pathological conditions, including cancer prognosis, due to several advantages such as their simplicity, lack of posttranscriptional modification, ease of detection of expressional changes, tissue and body fluid specific profile, high conservation among human and other model organisms, and high stability. For example, miR-221 has recently been shown to be related with prognosis in hepatocellular cancer. In prostate cancer patients, blood levels of miR-26a, miR-195, and let-7i were shown to be greater than in those with benign prostate hyperplasia. Deregulation of three miRNAs, miR-106a, and miR-24, in blood samples of early stage prostate cancer patients compared to healthy controls was discovered in another investigation. Furthermore, the fact that changed miRNA levels have been detected in a variety of tumour types, as well as their particular deregulation

pattern in different malignancies, suggests that miRNAs could be used as potential therapeutic targets and diagnostic markers.

Treatment resulted in inhibition of proliferation of human pancreatic ductal adenocarcinoma derived cell lines through induction of apoptosis in pancreatic ductal adenocarcinoma cases where high levels were reported. In addition, chemotherapeutics may be used in conjunction with treatment to minimise tumour size. MiRNA expression profiling is now critical for understanding the underlying mechanisms of cancer initiation, progression, invasion, and metastasis, as well as developing novel and promising therapeutic applications against cancer, following the discovery of more than 2500 mature miRNAs in *Homo sapiens*. Meanwhile, more research into the mechanisms of action of miRNAs is needed to learn more about how miRNAs operate in cancer pathogenesis and to solve potential hurdles like as side effects and delivery issues [1,2].

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Conflict of Interest

The author reported no potential conflict of interest.

References

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