Review Article

MicroRNAs: New Roles in Cancer Treatment and Diagnosis

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Abstract

Small no protein-coding RNAs called microRNAs control the expression of a wide range of genes by base-pairing on the untranslated region (UTR) of their target mRNAs, which either causes the target mRNA to degrade or inhibits its translation. In human breast cancer, aberrant miRNA expression has been associated with tumour formation, metastasis, diagnosis, prognosis, and therapeutic response. Certain miRNAs have been thought to have potential clinical uses as a diagnostic and therapeutic tool for breast cancer. Here, we outline and discuss the several lines of evidence that point to the critical connection between miRNAs and breast cancer, as well as its treatment options.

Keywords: Diagnosis; MicroRNAs; Cancer treatment; erapeutic tool; Breast cancer; Diagnostic/prognostic biomarkers

Introduction

Signi cant advancements in cancer biology have been made possible by the identi cation of a class of tiny non-protein-coding RNAs known as microRNAs (miRNAs). MiRNAs are 19–25 nucleotide regulatory, non–protein–coding RNA molecules that control the expression of many di erent genes by base-pairing on the 3UTRs of the target mRNA, which causes mRNA degradation or inhibits translation. MiRNA expression patterns are carefully regulated and have signi cant e ects on ontogenesis. In the last ten years, more and more human genes have been discovered to be regulated by miRNAs. Several studies have shown a correlation between miRNA expression and di erent malignancies, with miRNAs being both tumour suppressors and oncogenes, MicroRNAs [1-3] (Figure 1).

Subsets of miRNAs that are down-regulated or accumulate suggest a tumour suppressor or oncogenic function, respectively. Examples

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death under glucose shortage," demonstrates how forced oxidative phosphorylation and mitochondrial complex I inhibitors work together to induce cancer cell death. Surprisingly, cancer cells that have been immortalised or grown under high glucose circumstances do not respond to combined treatments that impact glycolysis and cause mitochondrial dysfunction. ese ndings imply that an alternate therapeutic strategy to raise the sensitivity of cancer cells to death may involve forcing the switch from glycolysis to oxidative phosphorylation along with the use of mitochondrial inhibitors [15].

Discussion

Many families of miRNAs have also been found to be markedly down regulated in the majority of malignancies, according to research. A large body of recent data strongly implies that the let-7 family can considerably reduce proliferation, inhibit invasion, and diminish cell growth in cancer cells, as well as make them more sensitive to therapy. According to Chen et al., overexpression of let-7a in vitro or in vivo dramatically desensitized acute myeloid leukaemia (AML) to the therapy with cytarabine (commonly known as Ara-C) [16], and let-7a levels in patients with AML corresponded signi cantly with a better prognosis. eir team discovered that CXCR4 controls let-7a in acute myeloid leukaemia. In SK-BR-3 cells, it was discovered that lin28 control of let-7a also a ected chemoresistance [17]. High levels of lin28 were linked with metastasis and/or relapse, and their downregulation reduced paclitaxel resistance. demonstrated that pancreatic cancer cell lines' processing of let-7a was controlled by lin28 and SET, Also, they discovered a correlation between resistance to gemcitabine and an increase in RRM2, a putative target of let-7a that is involved in the conversion of rib nucleotides to deoxyribonucleotides, as well as a build-up of unprocessed pre-let-7. In light of this, let-7 mimics and lin28 may both be signi cant therapeutic targets [18].

e miR-200 family is a new class of metastasis suppressors and therapeutic sensitizers. Although the miR-200 family is best known for its function in EMT suppression, it is now being shown to also play a factor in modulating the response of cancer cells to conventional treatment regimens. MiR-141 and miR-429 have received signi cantly less attention in recent miR-200 family research, which has largely concentrated on miR-200a, -200b, and -200c [19]. Di uorinated curcumin (CDF), a curcumin analogue, was shown to be able to upregulated miR-200a, -200b, and -200c in pancreatic cancer lines.

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