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Editorial

MicroRNAs (miRNAs) are a family of short non-coding RNA molecules which contain about 18-24 nucleotides. miRNAs play significant regulatory roles in biological processes (cell proliferation, differentiation, survival and motility). miRNAs are essential regulators of coding genes at the post-transcriptional level. The biogenesis of miRNAs is composed of complex processes, and human genome contains approximately two thousand miRNAs (http:// www.mirbase.org) [1,2]. Expression patterns of miRNAs have been found to have links with pathogenesis of many diseases including neurodegenerative diseases, diabetes, cardiovascular diseases, and particularly cancer. miRNAs have been identified as a critical biomarker for treatment, diagnosis, and progression of cancer. Experimental and computational data indicates that miRNAs are up-regulated or down-regulated in cancer cells, and therefore, they have oncogenic (oncomirs) or tumor-suppressive roles in apoptosis, angiogenesis, proliferation, metastasis, and differentiation in cancer cells [1,3,4]. Therefore, inhibition of oncomirs and restoration of tumor suppressive miRNAs are considered two main strategies for

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MicroRNAs in Oncology

development of miRNA-based cancer therapeutics. The relationship between miRNAs expression and tumorogenesis was first determined in 2002. miR-15a and miR-16-1 are down-regulated as tumor suppressor genes in B cell chronic lymphocytic leukemia cells [5]. The connection between miRNAs expression and heat shock proteins and pseudogenes are extensively studied in human lung and breast cancer by our research group. Relationships between heat shock protein isoforms and pseudogenes with miRNAs were determined on NCI-60 lung and breast cancer cell lines by using CellMiner analysis tool (http://discover.nci.nih.gov/cellminer/) [4].

miRNAs participate in regulation of each step of the oncogenic signaling pathways. Therefore, miRNA-based therapeutics emerged as a potential candidate in target-specific and personalized cancer treatment.

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