Editorial

MiRNAs as Legitimate Targets for Cancer Therapy

Mounting evidence suggests that micorRNAs (miRNAs) play a critical role in the development and progression of human malignancies. There is a wealth of literature supporting that miRNAs are involved in governing multiple cellular processes including cell proliferation, apoptosis, invasion, and metastasis. Therefore, this special issue reviewed the current understanding of how miRNAs are regulated and involved in the tumorigenesis such as controlling cell growth and apoptosis, regulation of invasion, epithelial to mesenchymal transition (EMT), stem cell features, and drug resistance. Moreover, regulation of miRNAs by natural compounds will also be reviewed. Therefore, targeting miRNAs, specifically by natural compounds could open newer avenues for the prevention of tumor progression or treatment of human cancers.

It has been documented that miRNAs are involved in tumorigenesis in many human cancers. Pai *et al.* reviewed the advance in the role of miRNAs in the pathogenesis and aggressiveness of pancreatic cancer. Moreover, Chakraborty *et al.* summarized the function of miRNAs in insulin resistance and diabetes-associated pancreatic cancer. Dr. Syed *et al.* discussed the current knowledge of the role of miRNAs in the regulation of the human response upon exposure to UV radiation and UV-induced skin cancer. Dr. Rossi and colleagues discussed the role of miRNAs and miRNA-based therapies in multiple myeloma.

Recently, multiple studies have shown that miRNAs could regulate many cellular signaling pathways. Dr. Xie *et al.* summarized the miRNAs-FOXM1 signaling pathways in cancer initiation and progression. Targeting FOXM1 through regulation of miRNAs may have a potential role in cancer treatment. Dr. Mo and his colleagues discussed the recent findings on miRNAs interacting with Notch signaling pathway and highlighted the therapeutic potential of targeting Notch signaling pathway and its related miRNAs in human cancers. It is known that miRNAs are under epigenetic regulation. Thus, Dr. Azmi *et al.* described the mechanisms of miRNA nuclear transport mediated regulation and proposed a novel therapeutic strategy through blockade of this mechanism.

Altered expression of specific miRNAs has been found to be involved in drug-resistant cancer cells. Dr. Liu *et al.* summarized the recent advances in drug resistance related miRMAs in cancer, and also discussed the potential applications of miRNAs for cancer treatment to overcome drug resistance. Furthermore, Dr. Seca *et al.* reported that targeting miR-21 induced autophagy and chemosensitivity in leukemia cells. Accumulating evidence suggests that miRNAs are involved in human cancer stem cells (CSCs). Dr. Singh *et al.* summarized how miRNAs target multiple genes and signaling pathways to govern cancer stemness properties.

Notably, a line of evidence revealed that chemopreventive agents commonly known as natural compounds could target miRNAs in human cancer cells. Dr. Sarkar *et al.* described that several natural, nontoxic chemopreventive agents regulated the expression of miRNAs. Dr. Wang *et al.* summarized that miR-34 family could be up-regulated by natural compounds in human malignancies. Furthermore, Ma *et al.* observed that natural agent genistein exerts its anti-tumor activity in part through down-regulation of miR-223 in pancreatic cancer. Taken together, natural agents could be useful for cancer treatment especially when combined with conventional therapeutics.

Lastly, as the editor, I am grateful to the contributors for their promptness in preparing their articles. I am also impressed by their dedication and diligent work. I would like to acknowledge the referees for their professional and timely reviews. We also appreciate receiving help from the Publication Manager and the staff members of Bentham Science Publishers.

Zhiwei Wang, M.D. Ph.D.

(Guest Editor)
Department of Pathology
Beth Israel Deaconess Medical Center
Harvard Medical School
Boston, MA 02215

Tel: 617-735-2474

E-mail: zhiwei@gmail.com; zwang6@bidmc.harvard.edu