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Editorial

Aberrant miRNA expression in brain tumors: a subject attracting an increasing amount of attention

Micro RNAs (miRNAs) are short strands of ribonucleotides that function to attenuate gene expression via base-pairing with complementary messenger RNA (mRNA) sequences; this usually results in the repression of corresponding protein expression, either by promoting mRNA degradation or by interfering with mRNA translation. The human genome encodes over 1000 miRNAs, the expression of which target mRNAs transcribed from at least 60% of all genes. Therefore, the expression of most of our genes is influenced by the cellular expression of miRNAs. The existence of miRNAs was first reported in 1993 (1), several years after the initial use of recombinant plasmids to express exogenous anti-sense RNAs for suppressing cellular gene expression (2), which is based largely on the same principle as cellular miRNAs. Thus, the anti-sense approach used by scientists for suppressing gene expression had always been part of the cell's repertoire for regulating its own gene expression. Describing the molecular mechanisms by which exogenous and endogenous RNAs suppress gene expression, referred to as RNA interference (RNAi) and initially described in 1998 (3), proved worthy of the 2006 Nobel Prize in Medicine (4).

Since developing an awareness of miRNA expression as a normal process for regulating gene expression, the field of cancer research has directed and continues to direct efforts toward understanding how aberrant miRNA expression contributes to tumor development. This research activity includes the discipline of neuro-oncology, illustrated by, among other metrics, the publication of 17 miRNA articles in *Neuro-Oncology* since 2010. Included among these is an article by Xia et al (5) in the current issue, which focuses on miR-218, an miRNA whose expression is frequently suppressed in glioma (6). In this report, the authors investigated the effects of transferring an miR-218 expression plasmid into glioma cells and showed that ectopic/exogenous miR-218 expression significantly increases apoptosis by suppressing the pro-tumorigenic activity of NF-κb through an effect on an upstream regulator of NF-κb activity, ECOP. The authors suggest that their results indicate a novel approach to glioma therapy. Indeed, results such as these have stirred substantial interest and enthusiasm for nucleic acid-based therapies for treating cancer that involve the process of RNA interference. We are, however, at the earliest of stages in exploring the use of nucleic acid therapies for treating human tumors, and the delivery of interfering RNAs, or expression constructs that produce interfering RNAs, to target cells in vivo has proved a significant challenge. It is too early to know whether the use of nucleic acids for treating brain tumors will prove a valuable addition to the clinical neuro-oncology armamentarium, but results such as those by Xia et al (5) suggest that this is a story worth following.

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