miR-126

miR-126 is a small non-coding RNA molecule belonging to the family of microRNAs (MicroRNAs).

miR-126 is known for its importance in vascular biology and angiogenesis. It is highly enriched in endothelial cells, the cells that line the interior of blood vessels and has been found to be a critical regulator of vascular integrity and function. Some key features and functions of miR-126 include:

Angiogenesis: miR-126 promotes angiogenesis, the process by which new blood vessels are formed from existing ones. It does so by regulating the expression of genes involved in endothelial cell migration, proliferation, and tube formation, which are essential steps in the angiogenic process.

Vascular Integrity: miR-126 contributes to maintaining vascular integrity by regulating the expression of genes involved in endothelial barrier function. It helps prevent excessive vascular leakage and inflammation, thereby preserving the health of blood vessels.

Cardiovascular Health: miR-126 has been implicated in various cardiovascular diseases, including atherosclerosis and ischemic heart disease. Dysregulation of miR-126 expression can affect endothelial cell function and contribute to the development and progression of these diseases.

Cancer: miR-126 also plays a role in cancer biology. In some cancers, miR-126 acts as a tumor suppressor by inhibiting tumor cell proliferation, migration, and invasion. It can also influence the tumor microenvironment and affect cancer progression.

The dysregulation of miR-126 expression has been associated with several diseases and disorders, highlighting its significance in various biological processes. Researchers continue to study miR-126 and its target genes to better understand its role in health and disease, with the hope of potentially utilizing it as a therapeutic target or diagnostic biomarker in certain conditions.

A study explored the role of MicroRNA (miR)-126 in ICH. Adult male Wistar rats were randomly assigned to the ICH model and sham groups. ICH was induced by intracerebral injection of collagenase. The mRNA expression levels of miR-126 in the two groups were determined. The miR-126 lentivirus expression vector pWPXL-miR-126 or negative control vector was then constructed and delivered via intraparenchymal injection. Following transduction, behavioral testing (rotarod and limb placement tests), relative hemorrhagic lesion size, apoptotic cells, and protein levels of vascular endothelial growth factor (VEGF)-A and caspase-3 were determined. The relative expression levels of miR-126 were significantly decreased in the ICH group compared to the sham group (P=0.026). Overexpression of miR-126 significantly improved the relative duration of stay on the rotarod at day 2 (P=0.029) and 3 (P=0.033), and statistically reduced the deficit score (P=0.036), the relative size of the hemorrhagic lesion (P=0.019) and the number of apoptotic cortical neurons (P=0.024) compared with the sham group. Additionally, the protein levels of VEGF-A were significantly elevated, however, levels of caspase-3 were downregulated by overexpression of miR-126 compared with the negative control group. MiR-126, therefore, exhibits a protective role in ICH. Overexpression of miR-126 protects against ICH, and may be involved in the process of angiogenesis and exhibit an anti-apoptotic effect [1)].

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Kong F, Zhou J, Zhou W, Guo Y, Li G, Yang L. Protective role of microRNA-126 in intracerebral

hemorrhage. Mol Med Rep. 2017 Jan 19. doi: 10.3892/mmr.2017.6134. [Epub ahead of print] PubMed PMID: 28112373.