**MicroRNA-1827 Represses MDM2 to Positively Regulate Tumor Suppressor P53 and Suppress Tumorigenesis**

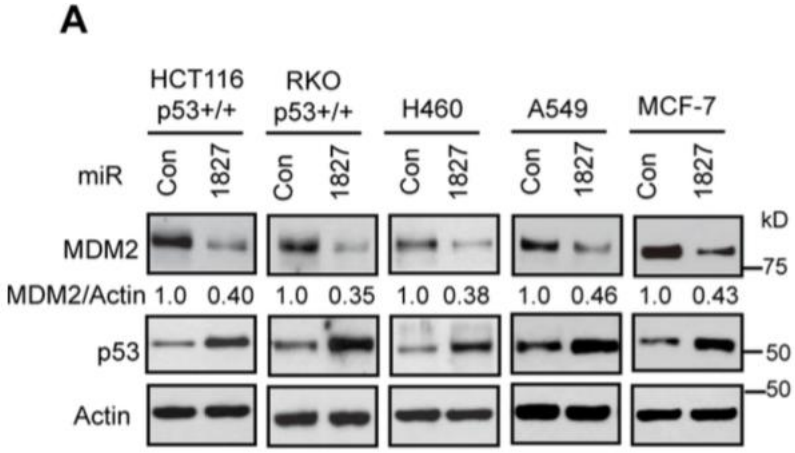
Zheng, Cen, et. al Oncotarget 7.8 (2016): 1-14. Web.

**Summary**

The p53 gene is a highly specialized protein designated with preventing tumor growth in cells. P53 protein loss of function or mutation can lead to cellular stress, apoptosis and cell cycle arrest. The ubiquitin ligase MDM2 a key damaging regulator of p53 plays a critical role in overall p53 function by degradation due binding directly to the p53 protein, creating a negative feedback loop. The regulation of p53 gene expression is highly controlled by microRNAs, a certain miRNA in this case has been chosen to protect p53 from inhibiting factors such as MDM2.

In question is miRNA-1827, the study proposed by Zheng et. al aimed specifically to target MDM2 by binding the untranslated region of the 3' end. Conversely, in the presence of miRNA-1827, MDM2 is negatively regulated which results in MDM2 suppression and overall increase in transcriptional activity and p53 protein levels. In the experiment colorectal cancer activity was tested to show mi-1827 is indeed able to regulate p53 through decreasing MDM2 activity, this proved that Zheng et. al discovered a "new" mechanism of tumor suppression not previously known.

To test the effectiveness of miRNA-1827 regulation the experimental procedure consisted mainly of two types of cells, wild type HCT116 p53 +/+,-/- and RKO p53 +/+,-/-.



The levels of MDMA as well as Actin mRNAs in the cell were measured by Taqman real-time PCR assays by biotinylation (attaching biotin to a protein). Figure A serves to reiterate the fact that MDM2 mRNA was enriched significantly in the miRNA-1827 pull-down assay for both the HCT116 p53+/+ and RKO+/+ cells. The band strength suggests miRNA-1827 does directly bind to MDM2, proving miRNA-1827 enhances the overall transcriptional activity of p53.