Evidence has suggested that retinal noncoding RNA3 (RNCR3) is a GBM-associated noncoding RNA and is under-expressed in glioblastoma. However, the function and mechanism of RNCR3 on GBM cell growth and apoptosis are still uncertain.

In a study, Zhang et al., found that the level of RNCR3 is decreased in U87, U251, U373, and A172 GBM cell lines when compared with the normal human astrocytes. Elevating Long non-coding RNA RNCR3 expression markedly inhibits U87 and U251 cell survival and proliferation. Further studies indicated that RNCR3 overexpression promotes U87 and U251 cell apoptosis and activity caspase-3/7. Moreover, they found that RNCR3 overexpression promotes Krüppel-like factor 16 (KLF16) expression through inhibiting the level of miR-185-5p. They demonstrated that KLF16 is a direct target of miR-185-5p. An increased miR-185-5p level by a miR-185-5p mimic or decreased KLF16 by KLF16 small interfering RNA both reversed the function of RNCR3 overexpression on GBM cell growth and apoptosis. In summary, this study focuses on investigating the key molecular mechanisms of RNCR3 involved in GBM cell growth and apoptosis. Our data indicated that RNCR3 overexpression inhibits cell growth and induces its apoptosis through the miR-185-5p/KLF16 axis [1].

1)
Zhang L, Cao Y, Wei M, Jiang X, Jia D. Long non-coding RNA-RNCR3 overexpression deleteriously affects the growth of glioblastoma cells through miR-185-5p/Krüppel-like factor 16 axis. J Cell Biochem. 2018 Jun 28. doi: 10.1002/jcb.27167. [Epub ahead of print] PubMed PMID: 29953649.

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