MiR-320b-targeted TRIAP1 prevents mitochondria fragmentation and apoptosis by controlling cytochrome *c* release in nasopharyngeal carcinoma

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Mitochondria play critical roles in apoptosis. Imbalance of mitochondria fusion/fission leads to mitochondrial fragmentation and initiates intrinsic apoptosis, but the regulation of mitochondrial fragmentation in nasopharyngeal carcinoma (NPC) remains undefined. Here we show that TRIAP1 is aberrantly overexpressed and associated with poor survival in NPC patients. TRIAP1 overexpression promotes cell growth and suppresses NPC cell death *in vitro* and *in vivo*, whereas TRIAP1 knockdown inhibits cell tumorigenesis and enhances apoptosis through induction of mitochondrial fragmentation, membrane potential alteration and release of cytochrome *c* from mitochondria to cytosol. In intersection with our previous miRNA data and available bioinformatics algorithms, we identified miR-320b as a negative regulator of TRIAP1 expression. Further studies show that miR-320b suppresses NPC cell proliferation and enhances mitochondrial fragmentation and apoptosis by targeting TRIAP1. Moreover, loss of miRA-320b expression is conversely correlated with TRIAP1 overexpression and poor prognosis in NPC patients. This study reveals the regulation of miR-320b/TRIAP1 in mitochondrial fragmentation and apoptosis, and suggests potential therapeutic targets for NPC treatment.