Novel crosstalk mechanism between mitochondrial translation and cytoplasmic translation

Under the support by the National Natural Science Foundation of China, the research group led by Prof Qin Yan (秦燕) at the Key Laboratory of RNA Biology, Institute of Biophysics, Chinese Academy of Sciences, Beijing, China reported recently on the crosstalk mechanism between mitochondrial translation and cytoplasmic translation, which was published online on 11 April 2016 in *Nature Structural & Molecular Biology*.

Protein translation is mainly done on the ribosome, and mitochondrial translation elongation factor 4 (mtEF4) is a quality control factor of protein translation. In this study, by using a systemic mtEF4 gene knockout mouse model, researchers found out that mtEF4 knockout damages the oxidative phosphorylation function in germ cells of the male mice, thus resulting in male sterility. Further study found that the rate of mitochondrial protein translation increased after mtEF4 was knocked out; however, the price was the lower protein "qualified rate" and the shorter protein half-life. In order to keep step with the "quickened" mitochondrial translation, somatic cells activate the mTOR signaling pathway to accelerate cytoplasmic translation to balance the mitochondrial translation. In this way, somatic cells have successfully resolved the negative impact of the high-speed mitochondrial translation. In contrast, the mTOR signaling pathway could not be activated in germ line cells, the mitochondrial complex assembly of germ cells failed, and the sperm maturation process was stagnated at the round sperm stage, which ultimately resulted in the male sterility. This study reveals a new information exchange way within a cell (see figure below): the mTOR signaling pathway balances the dynamics between mitochondrial translation and cytoplasmic translation. When the mitochondrial translation rate increases, the mTOR signaling pathway is activated, thus causing the increase of cytoplasmic protein translation rate to counteract the pressure from the increased mitochondrial translation, which is a new evolutionary adaptation mechanism. Meanwhile, this study reveals a new way of male infertility, also of great value for the clinical treatment of male infertility.

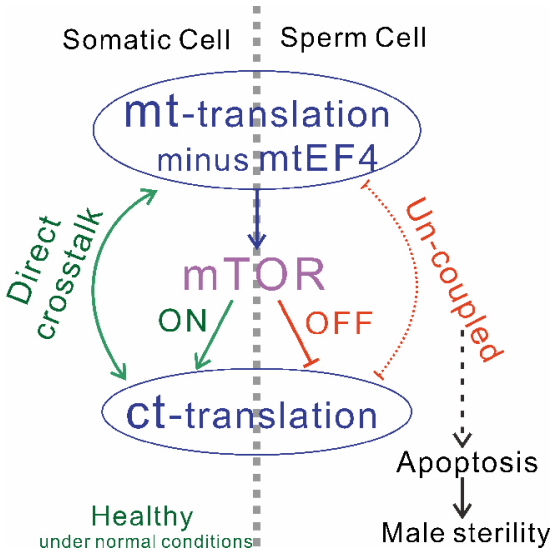

Figure The crosstalk mechanism between mitochondrial translation and cytoplasmic translation.