Mitochondrial Contribution to the Oocyte and Embryonic Development

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Abstract

The energy supply through oxidative phosphorylation (OXPHOS) is mainly considered an essential function of mitochondrion. However, there are alternative roles of mitochondria relevant to female reproduction, such as the synthesis of biomolecules, epigenetic regulation, and the storage of the mitochondrial (mt) genome. Indeed, the complex I of OXPHOS is silenced in the oocyte, and the early embryo relies on glycolysis. We hypothesized that mitochondrial insufficiency does not jeopardize fertilization and early embryonic development. Therefore, we developed a mouse model of a deficient mitochondrial population in the oocyte: transcriptional factor A, mitochondrial (*Tfam*) conditional knock-out (*Tfam*^{loxPloxP}/*Zp3-Cre*) females as donors of *Tfam*^{null} oocytes. We observed that TFAM insufficiency significantly decreases mitochondrial population in *Tfam*^{null} oocytes. Surprisingly, a decreased amount of mtDNA does not affect either the fertilization rate of *Tfam*^{null} oocytes or the success of early development of *Tfam*$^{+/-}$ embryos. We observed a negligible amount of TFAM expressed from parental allele after embryonic genome activation and, accordingly, blastocysts were developed in spite of an imperceptible amount of TFAM. Taken together, we suggest the indispensability of mitochondrial population in the oocyte and early embryonic development due to OXPHOS silence due to the late blastocyst. We expect the Warburg effect to cover the energy demand of the oocyte and early preimplantation embryo; on the other hand, the genetic aspect of mitochondrial insufficiency should be considered whereas impaired biogenesis can lead to the accumulation of mtDNA mutations.

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