ABNORMAL MITOCHONDRIAL FUSION EFFECT ON SCNT EMBRYOS DEVELOPMENT

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Somatic cell nuclear transfer (SCNT) is a powerful technique, but still very inefficient despite 20 years passed by since the cloned mammal was born. We have recently shown that the major cause of abnormalities observed in cloned fe tuses are mitochondrial dysfunction s in placenta collected from cloned sheep. Investigations on mitochondria in SCNT are limited to the mtDNA hetero/homoplasmy in cloned offspring, whereas no data is available for an eventual role of mitochondria dysfunction on the developmental failure of cloned animals. Here we wanted to know whether mitochondrial abnormalities are observed already in cloned embryos since mitochondrial replication does not occur before the hatched blastocysts stage . SCNT and natural mated (NM) mouse early embryos were produced and analysed for mitochondrial structure and functionality. The results shown a lower expression of major mitochondrial fusion proteins as well as mRNA (mitofusin 1, mitofusin 2, Opa1) in SCNT embryos comparing to NM once. Moreover, time – lapse analysis shown minimal mitochondrial fusions in SCNT blastocysts. Additionally, decreased density of mature mitochondria, very high degree of cytoplasmic vacuolisation, numerous cytoplasmic vesicle and autophagosomes were observed in SCNT embryos by transmission electron microscope (TEM) analysis. The obtained results clearly shown that mitochondrial abnormalities are already observed in SCNT blastocysts stage embryos . I t is important to point out that activity of mitochondria are strictly controlled by nuclear signals; thus our results may suggest that incomplete nuclear reprogramming can also affect genes that control mitochondrial function, with the ensuing mitochondrial dysfunction concurring to the developmental restraints of cloned embryos/fetuses.