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Ultrastructural analysis reveals abnormal mitochondria in cloned blastocysts

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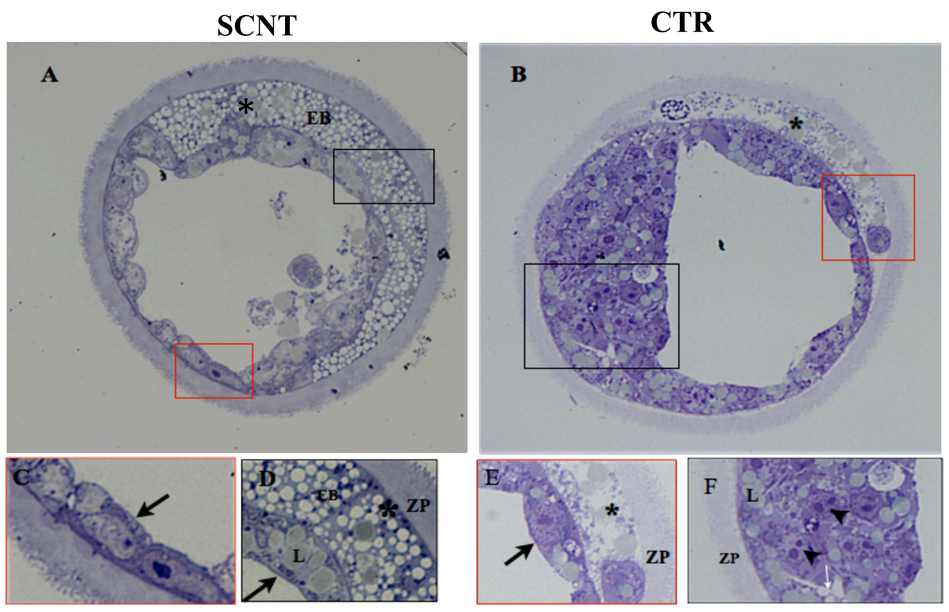
INTRODUCTION Somatic cell nuclear transfer (SCNT) is a powerful technique, but still very inefficient despite 20 years passed by since the first cloned mammal was born. We have recently shown that the major cause of abnormalities observed in cloned fetuses are mitochondrial dysfunctions in placenta collected from cloned sheep.

AIM Here we wanted to know whether mitochondrial abnormalities are observed already in cloned blastocysts. SCNT and *in vitro* processed (IVP) blastocysts were produced and analysed for mitochondrial structure and functionality.

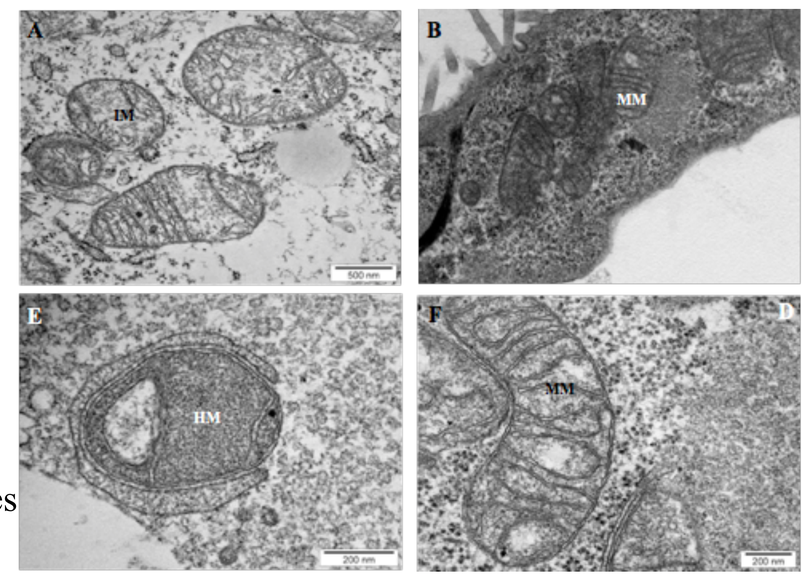
RESULTS

Drastic abnormalities in mitochondrial structure in SCNT blastocysts

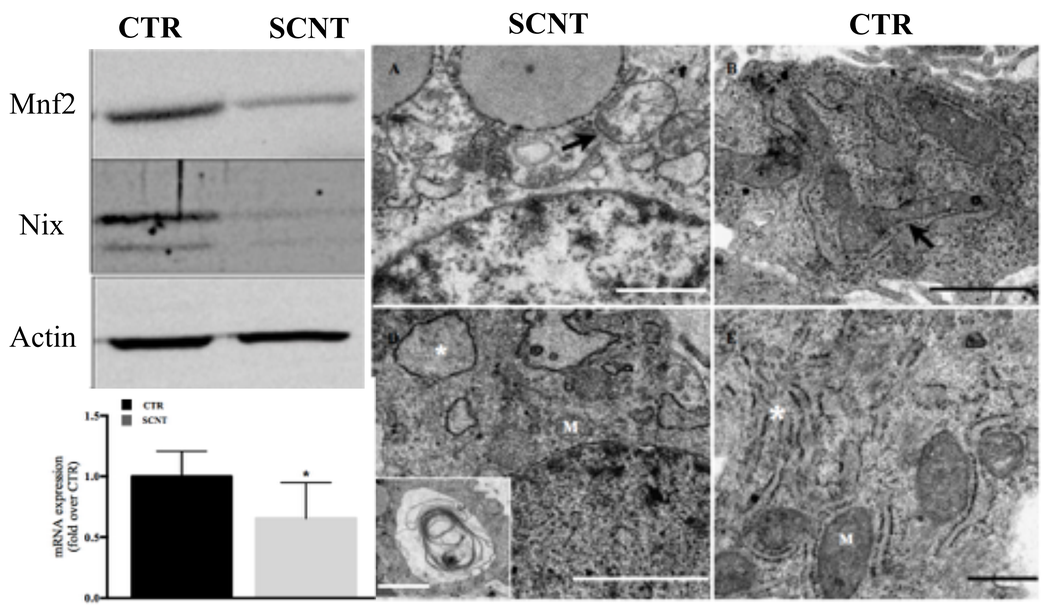


SCNT blastocysts

- intact zona pellucida (ZP);
- many lipid droplets (L);
- excluded blastomers (EB)
- accumulation of degenerated material within perivitelline space (*stars A, D*);
- immature mitochondria (IM) (C, E);
- lack of mitochondrial fusion;
- numerous incomplete autophagosomes (E);



Mitochondrial abnormalities in SCNT blastocysts affect on placenta development



SCNT placenta

- very low expression of mitochondrial proteins (Mfn2 and Nix) and mRNA;
- mitochondria radomly dispersed, swollen, fragmented and devoid of cristae (*black arrows*);
- swollen endoplasmic reticulum (*white stars*);
- lack of mitochondrial fusion process;

CONCLUSION Mitochondrial abnormalities are already observed in blastocysts stage embryos and affect on poor placenta development. Moreover, mitochondria are strictly controlled by nuclear signals, thus, incomplete nuclear reprogramming in cloned nucleus might be responsible for the impaired mitochondrial function in cloned embryos/fetuses.

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