

## PINK1 IS ESSENTIAL FOR MAINTENANCE OF MITOCHONDRIAL MORPHOLOGY IN PORCINE PREIMPLANTATION EMBRYOS

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### Introduction

PINK1 selectively locates to the outer membrane of impaired mitochondria and promotes their autophagy. PINK1 regulates mitochondrial dynamics through promoting mitochondrial fission in *Drosophila* and has a role Parkinson's disease. Mitochondrion is an important organelle in mammalian preimplantation embryos, but few studies focus on the mitochondrial dynamics during preimplantation embryo development.

### Materials and Methods

To investigate whether PINK1 are required for mitochondrial dynamics in porcine preimplantation embryos, gene knockdown were engaged in the present study. After injection of PINK1 dsRNA, the parthenotes was cultured in vitro to blastocyst stage. Immunofluorescence and qPCR were used to observe the changes in mitochondrial quality and oxidative stress.

### Results and Discussion

The results showed that the blastocyst formation was compromised significantly ( $42.9 \pm 1.9\%$  vs.  $36.2 \pm 1.4\%$ ,  $p < 0.01$ ) after PINK1 knockdown. Furthermore, knockdown of PINK1 induced mitochondrial elongation ( $1.93 \pm 0.06 \mu\text{m}$  vs.  $3.03 \pm 0.09 \mu\text{m}$ ,  $p < 0.001$ ) and mitochondrial copy number reduce ( $1.01 \pm 0.07$  vs.  $0.80 \pm 0.04$ ,  $p < 0.05$ ). The total ROS ( $10.79 \pm 0.97$  vs.  $20.46 \pm 1.86$ ,  $p < 0.001$ ) and mitochondrial derived ROS ( $6.69 \pm 0.71$  vs.  $13.06 \pm 3.07$ ,  $p < 0.05$ ) was observably increase, but the mitochondria were seriously depolarized. In addition, both of the autophagy ( $9.17 \pm 4.01$  vs.  $32.57 \pm 7.35$ ,  $p < 0.05$ ) and apoptosis ( $6.80 \pm 1.18\%$  vs.  $18.00 \pm 3.21\%$ ,  $p < 0.01$ ) were significantly increase after mitochondrial elongation. In conclusion, these data suggest that PINK1 promotes mitochondrial fission in porcine preimplantation embryos.