Membrane protein Oca3 is essential to keep structural integrity of mitochondria and endoplasmic reticulum

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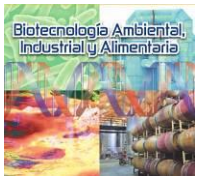

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ABSTRACT

Mitochondrial function is tightly conserved through evolution since it becomes essential for the fitness of any eukaryotic cell. Defective function of this organelle represents the cellular basis of some severe diseases in humans. Thus, the characterization of genes involved in the correct mitochondrial structure and function is critical to understand and treating these diseases. In our laboratory, using the fission yeast as a model, we are characterising the function of *oca3* gene, the ortholog of EMC2 gene in human. This gene is predicted to be a member of the ER membrane protein complex involved in the mitochondrion-endoplasmic reticulum membrane tethering[1]. We find the protein in the non-aqueous phase in cell extracts and Oca3-mCherry tagging actually decorates most cell membranes. Oca3 over-expression cause lethality[2] and the gene deletion becomes cold-sensitive. In both situations aberrant mitochondria aggregations are observed and endoplasmic reticulum seems disorganised. Interestingly, addition of Tween20 restores the viability of *oca3* deletion at low temperature. This result suggests that Oca3 may have a role in membrane fluidity homeostasis. In addition to this, we will analyse different gene interaction between some of the EMC complex members to clarify both, the importance of Oca3 for the complex and the importance of the complex itself for the cell.

REFERENCES

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