

Role of myoferlin in mitochondrial dynamics and metabolic fitness of pancreas cancer



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Introduction

Pancreatic cancer is the 7th most common cause of cancer mortality in the world. It is predicted to become the second leading cause of cancer-related death in 2030. Myoferlin is a 230 kDa protein overexpressed in pancreatic cancer. Recently, our team showed a fragmentation of the mitochondrial network in PDAC cells when myoferlin was depleted using siRNA. Understanding the mechanism underlying this mitochondrial disruption would be of great interest as mitochondria are major actors in cancer development, progression and resistance.

Results

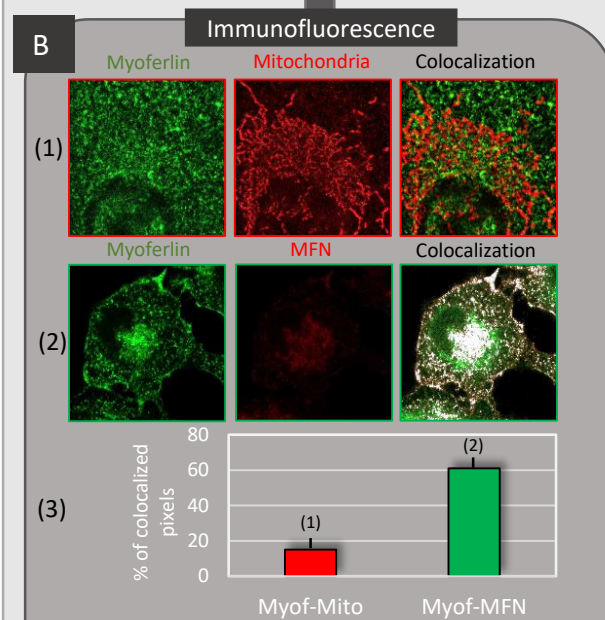
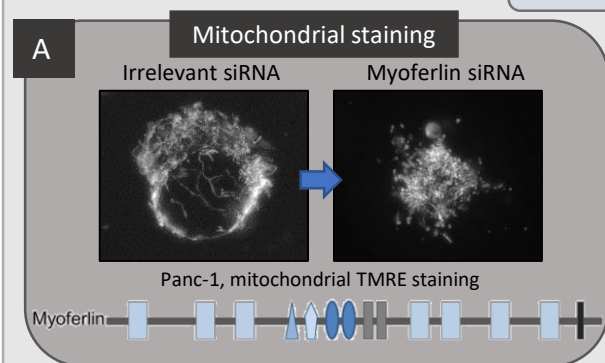


Figure B: Immunofluorescence in PANC-1 cells. (1) Myoferlin-mitochondria and (2) myoferlin-MFN colocalization map. (3) Percentage of pixel colocalizing between myoferlin-mitochondria and myoferlin-MFN using Manders coefficients

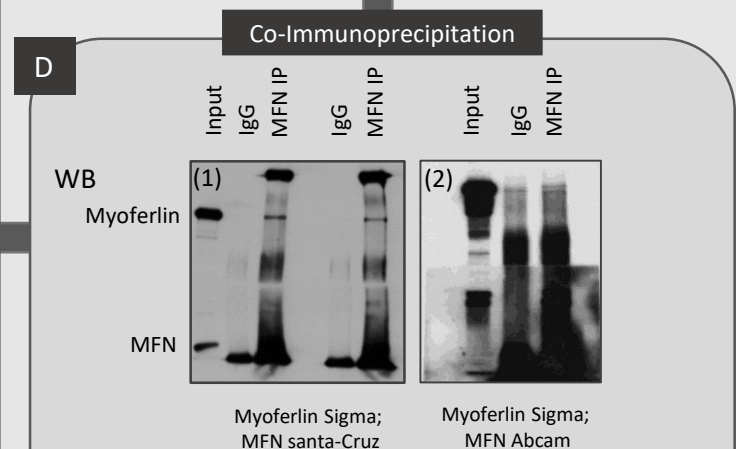
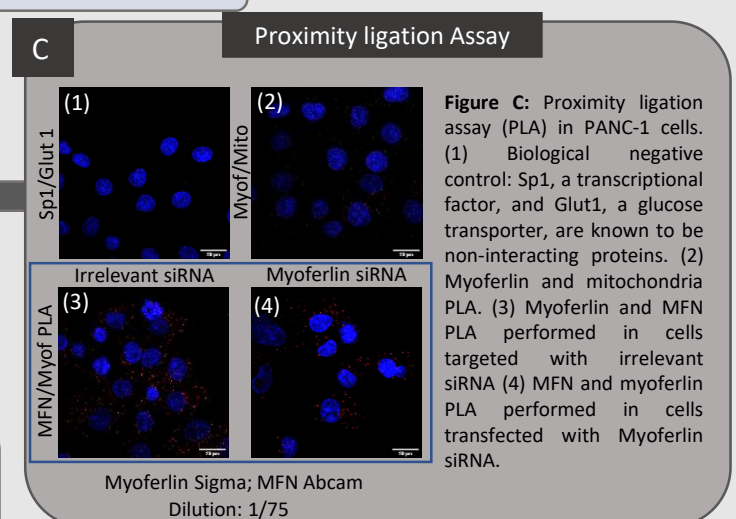


Figure D : Immunoprecipitation of MFN in PANC-1 cells using (1) Santa-Cruz antibody and (2) Abcam antibody. Detection of co-immunoprecipitated myoferlin was performed in both experiments.

Conclusion

Recently, our team showed a mitochondrial fragmentation using siRNA targeting Myoferlin. However, mechanisms underlying this process was still unclear. Our data strongly suggest that myoferlin interacts with MFN. Indeed, if myoferlin is a part of the mitochondrial fusion machinery, its silencing together with an unopposed fission would lead to mitochondrial fragmentation.



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