The role of p58IPK in connecting ER stress to mitochondriopathies

Ravi Singhal

In eukaryotes, endoplasmic reticulum (ER) and mitochondria play a major role in maintain the cell stability and uniformity. ER and mitochondria decide the cell fate during the stress condition. During the progress of stress both, the organelles co-ordinate together structurally and functionally by number of physiological process like Ca^{2+} signaling, fatty acid metabolism and cell death.

The ER under the stress condition, signals a response known as unfolded protein response, which helps in rescuing the ER from the stress condition. ER under stress can lead to excess of Ca^{2+} release and increase in unfolded protein in the ER leading to cell death. Coping with ER stress takes place by the unfolded protein response by co-ordination of series of proteins, and one of the proteins which, I am interested in this study is $P58^{IPK}$. $P58^{IPK}$ is a cytosolic chaperone, which is recruited as a late response to rescue the ER from the stress and reduce the unfolded protein burden in the ER. The aim of the project is to characterize the role of $P58^{IPK}$ connecting ER stress to mitochondrial disorders, in the human patients suffering from $P58^{IPK}$ mutation. To achieve this, I have established a standard experiment procedure to analyze the link the link between ER and mitochondria in the mutated patients.

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Supervisor: Prof. Doron Rapaport