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London: Scientists have identified a protein that plays a crucial role in correctly measuring stress levels, and also makes sure the pathways of cell repair or cell death in the body are effective.

Each cell in an organism has a sensor that measures the health of its "internal" environment, researchers said.

This "alarm" is found in the endoplasmic reticulum (ER), which is able to sense cellular stress and trigger either rescue responses or the death of the cell.

A team from the Institute for Research in Biomedicine (IRB), in Barcelona, found some of the molecular mechanisms that connect the protein Mitofusin 2 (Mfn2) to endoplasmic reticulum stress.

When the scientists removed Mfn2 from the cell under conditions of cell stress, the endoplasmic reticulum responded by over-activating the repair pathways.

By doing so, it contradictorily functioned worse, reducing the capacity of cells to overcome the stress insult and promoting to a lesser degree apoptotic cell death.

"When Mfn2 is removed, the cellular stress response pathways are completely disrupted," said Antonio Zorzano, coordinator of IRB's Molecular Medicine Programme.

The study investigated the relationship between mitochondria and the endoplasmic reticulum, and indicated that changes in mitochondria, caused by the loss of the Mfn2 protein, directly affect the endoplasmic reticulum function.

"We have shown that Mfn2 is important for cell viability and has implications for numerous diseases, such as neurodegeration, cancer, cardiovascular disease, in addition to diabetes," said postdoctoral researcher Juan Pablo Munoz, first author of the study.

"The fact that we can modulate cell damage response with Mfn2 opens a wide window of possible therapeutic avenues for further study," said Munoz.

Munoz explained that tumour cells don't activate cell death properly and proliferate uncontrolled.

"Cancer cells have already been noted to have low Mfn2 levels, and if we could increase such levels, we would be able to promote apoptosis," Munoz said.

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First Published: Saturday, September 21, 2013, 12:49