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Protein balance in the reproductive system can prevent disease

Scientists from the University of Cologne found that the balance status of proteins (protein homeostasis) of germline cells influences protein aggregation in other tissues by long-distance signaling | Publication in 'Science Advances'

A recent study shows that a healthy reproductive system can prevent disease-related protein accumulation in distant tissues, such as neurons, and alteration of mitochondria - the power plants of cells. An imbalance of proteins, for example an aggregation of damaged proteins in brain cells, can lead to diseases like Alzheimer's, Huntington's disease or Amyotrophic Lateral Sclerosis (ALS). Since these diseases are associated with aging and the reproductive system is one of the first tissues to decline during aging, Dr David Vilchez and his group investigated whether the protein homeostasis (proteostasis) of germline cells (the cells that form the egg, sperm and the fertilized egg) influences other tissues and organs. Using the model organism *Caenorhabditis elegans*, the scientists show that when the germline accumulates damaged protein aggregates, it releases specific signals (Wnt singaling) which in turn induce changes in mitochondria, the powerhouses of the cells, leading to protein aggregation in other tissues such as muscle or neurons. The article 'Systemic regulation of mitochondria by germline proteostasis prevents protein aggregation in the soma of C. elegans' has now been published in *Science Advances*.

'We were very excited when we saw that just by inducing accumulation of protein aggregates in the germline, we could change the mitochondrial network of the entire organism inducing protein aggregation in neurons,' said Guiseppe Calculli, lead author of the study.

In the future, the group plans to examine whether germ-line specific proteins also aggregate during the aging process and whether this process contributes to the age-associated aggregation of proteins characteristic of pathologies like Huntington's disease or ALS.

‘Our findings open a new door to understanding why protein aggregates accumulate in the neurons of patients with Huntington’s disease and ALS. Since these aggregates can contribute to the neurodegeneration characteristic of these diseases, which remain incurable, a better understanding of the process discovered here might lead to novel therapeutic approaches,’ explained Vilchez, research group leader at the CECAD Cluster of Excellence in Aging Research and the Center for Molecular Medicine Cologne (CMMC) and leader of this study.

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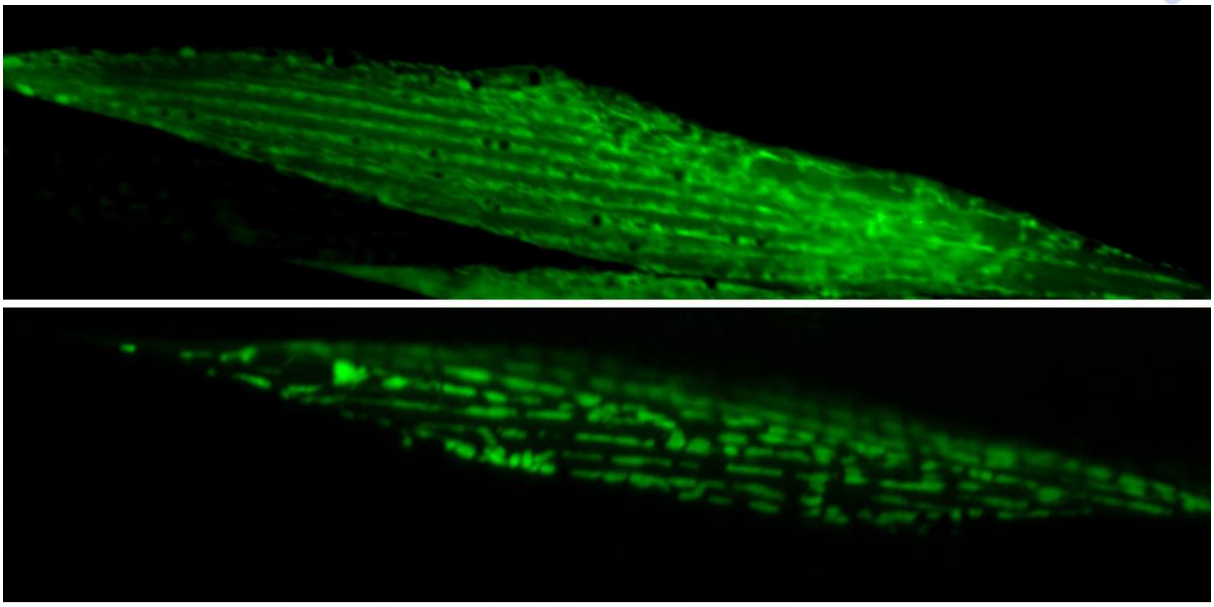
Picture:



On top: mitochondria in healthy muscle of *C. elegans.* Bottom: fragmented mitochondria in the muscle after induction of aggregation of proteins in the germline. Picture credit: Guiseppe Calculli