Mitochondrial dysfunction may play role in heart failure

While all cells in the body rely on metabolic pathways that take place within the mitochondria, the cells of some organs, such as the brain and heart, have an essential demand for constant energy. Maintaining the proper mitochondrial homeostasis is critical for overall tissue health, as defective mitochondria have been linked to a number of disease states.

Now, researchers at Case Western Reserve University have discovered that a protein called Kruppel-like Factor 4 (KLF4) is pivotal in catalyzing energy production within mitochondria. Specifically, they saw that the absence of KLF4 led to reduced energy production—a phenotype that is particularly problematic for cardiac cells.

“Some cells are incredibly dependent on mitochondria, particularly the heart and brain,” explained lead author Xudong Liao, Ph.D., an assistant professor of medicine at Case Western Reserve University School of Medicine. “The brain is working all the time, too, even while we are sleeping, so it is particularly sensitive to mitochondrial function. Cancer also hijacks mitochondrial machinery to drive its spread. Therefore, the identification of KLF4 as a major regulator of mitochondrial health may have implications beyond those we detailed in this article.”

KLFs are a family of zinc finger DNA-binding proteins that act as transcription factors to regulate gene expression in a number of tissues throughout the body. In the current study, the investigators found that KLF4 was involved in mitochondrial biogenesis, metabolic function, dynamics, and autophagic clearance. Moreover, the researchers found that cardiac specific KLF4 deficiency had a powerful effect on heart failure in adult mice.

The GLP aggregated and excerpted this blog/article to reflect the diversity of news, opinion and analysis. Read full, original post: Link between Mitochondrial Protein and Heart Failure Uncovered