MitoInsights

Bringing mitochondrial science closer to the people it matters to most

Science moves fastest when everyone can take part. That's why MitoCanada is committed to empowering our community with knowledge. By translating cutting-edge mitochondrial research into clear, easy-to-understand summaries, we aim to nurture curiosity, strengthen understanding, and build confidence in the science driving hope and progress.

Lay Summary: Silencing Mitochondrial Genes Expression in Living Cells

Authors:
Cruz-Zaragoza, L.D., Andree, M., Richter-Dennerlein, R., Mårtensson, C.U., Deimling, L., Culley, S., Peter, B., Grzyb, K., Silvander, C., Lindberg, M., Zickermann, V., Arlt, H., Schliebs, W., Amunts, A., Spåhr, H., Spang, A., Rugarli, E.I., Wiedemann, N., Callegari, S., Larsson, N.-G., and *Dennerlein, S.*

What's this research about?

Every cell in our body contains mitochondria, tiny structures that act like power plants, producing the energy our bodies need to function.
Mitochondria have their own small set of genes, separate from the DNA in the cell's nucleus. Understanding what these mitochondrial genes do is crucial for discovering how energy production works and why it sometimes fails in mitochondrial disease.

Until now, scientists had no reliable way to "turn off" or silence specific mitochondrial genes inside living cells. This made it hard to study exactly how each gene affects mitochondrial function.

This study, led by Dr. Luis D. Cruz-Zaragoza and colleagues, reports a new method that allows researchers to selectively silence mitochondrial genes in living cells, giving them a powerful tool to study how those genes work.

Why is this important?

Understanding what each mitochondrial gene does is essential to uncovering how mitochondrial function normally, and what goes wrong when they don't.

Many mitochondrial diseases are caused by mutations in mitochondrial DNA, but because researchers haven't been able to directly study how each gene contributes to energy production, it has been difficult to pinpoint exactly how these mutations lead to disease.

This new tool fills an important gap. It doesn't treat or repair mitochondrial DNA, but it gives scientists a much clearer way to study the function of each gene and how its loss affects the cell. With this knowledge, researchers can build a more complete picture of how mitochondrial dysfunction develops, paving the way for more targeted investigations in the future.

How did they study this?

To create this new method, the researchers designed a small molecular tool that can enter mitochondria and attach itself to the messenger molecules that carry the instructions for making mitochondrial proteins. When the tool binds to a chosen target, it reduces the activity of that specific gene, essentially silencing its ability to produce its protein.

The team tested this method in living human cells grown in the lab. They confirmed that the tool successfully reached the mitochondria and reduced the levels of the target protein, without significantly affecting other genes. By measuring the cells' ability to produce energy, they could also see how turning off one gene altered mitochondrial function.

What did they find?

The researchers found that their new approach could precisely and temporarily silence individual mitochondrial genes inside living cells. When they silenced specific genes, the cells showed clear changes in their energy production and mitochondrial performance.

These results confirmed that the tool worked as intended and that its effects were specific, rather than causing broad or unintended changes elsewhere in the cell.

This discovery shows that mitochondrial gene activity can now be studied in far greater detail than before, offering a more direct way to link specific genes to their roles in energy metabolism and cell health.

What does this mean for mitochondrial disease research?

While this study does not present a therapy or treatment, it provides a vital new research tool that will help scientists understand the underlying biology of mitochondrial diseases.

In short, this work equips the scientific community with a new way to explore the basic building blocks of mitochondrial biology, a necessary step before effective therapies can be developed.

In simple terms

This study introduces a way for scientists to switch off one mitochondrial gene at a time in living cells and watch what happens next. It's like being able to press pause on one tiny part of the mitochondria's instruction manual to see how important that page is to the overall power plant. By doing this, researchers can learn more precisely how mitochondria make energy and what goes wrong when certain genes don't work properly.

Why this matters to the mito community

For people living with mitochondrial disease, this type of research represents progress at the foundation level, the kind of work that helps scientists truly understand how mitochondria function and fail. By improving the tools used to study these essential genes, the scientific community can generate stronger evidence, create more accurate disease models, and design better studies in the years ahead.

While this discovery is not a treatment, it strengthens the path toward one. Every improvement in understanding how mitochondria work brings researchers, patients, and families closer together in the shared effort to one day find solutions for mitochondrial disease.

Acknowledgment

This research was led by Dr. Luis D. Cruz-Zaragoza **and** Dr. Stefan Dennerlein, with contributions from an international team of scientists including Dr. Nils-Göran Larsson**,** Dr. Simona Callegari**,** Dr. Elena Rugarli, and collaborators across Sweden, Germany, and Italy. Together, their work represents a major collaborative effort to expand the scientific tools available for studying mitochondrial gene function in living cells.