

# Researchers discover novel mechanism linking changes in mitochondria to cancer cell death

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Credit: University of Notre Dame

To stop the spread of cancer, cancer cells must die. Unfortunately, many types of cancer cells seem to use innate mechanisms that block cancer cell death, therefore allowing the cancer to metastasize. While seeking to further understand cancer cell death, researchers at the University of

Notre Dame discovered that the activation of a specific enzyme may help suppress the spread of tumors.

The findings, published in *Nature Cell Biology*, demonstrate that the enzyme RIPK1 decreases the number of mitochondria in a cell. This loss of mitochondria leads to oxidative stress that can potentially kill [cancer cells](#), though researchers speculate the cancer cells could find ways to shut down this effect.

During the study, the Notre Dame team discovered that when the enzyme RIPK1 is activated in a cell, it can cause the number of mitochondria—considered the "power plants" of the cell—to decrease as the cell consumes its own components for energy, a process called mitophagy. This was surprising, given RIPK1 was well-known to regulate necrosis, a completely distinct [cell death](#) mechanism.

"We really thought this was going to be a story about necrosis, but we weren't able to see evidence of it, and knew there had to be something we're missing," said Zachary Schafer, Coleman Foundation Associate Professor of Cancer Biology in the Department of Biological Sciences and senior author on the study. "Looking at mitochondrial numbers fundamentally changed our thinking, and got us focused on a different way that RIPK1 may cause cells to die."

Schafer focuses on metastasis, the process by which cancer cells leave the primary tumor and disseminate to other parts of the body. Cell death, a barrier to metastasis, can be induced when [epithelial cells](#), those lining the surfaces of body cavities, detach from the meshwork of scaffolding-like proteins called the extracellular matrix (ECM). As a tumor progresses, it can become resistant to this type of cell death.

"While our findings are in their infancy, we hope we can take advantage of the liability associated with RIPK1's function in order to design a

strategy aimed at eliminating ECM-detached cancer [cells](#), which would ultimately improve patient survival," said Mark Hawk, a graduate student at Notre Dame and lead author of the study. Hawk added that more studies are needed to delineate which cancer types are most susceptible to this approach.

**More information:** Mark A. Hawk et al. RIPK1-mediated induction of mitophagy compromises the viability of extracellular-matrix-detached cells, *Nature Cell Biology* (2018). [DOI: 10.1038/s41556-018-0034-2](https://doi.org/10.1038/s41556-018-0034-2)

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