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Researchers discover mechanism in cells that leads to inflammatory diseases

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Cedars-Sinai researchers have unlocked the mystery of how an inflammatory molecule is produced in the body, a discovery they say could lead to advances in the treatment of rheumatoid arthritis, Type 2 diabetes and numerous other chronic diseases that affect tens of millions of people.

The study, funded by the National Institutes of Health, is published online by the peer-reviewed journal *Immunity* and will appear in the March print edition.

The researchers identified for the first time the mechanism that leads to the production of the molecule interleukin-1beta. It is a major contributor to inflammation, which lies at the root of many serious health conditions, including atherosclerotic heart disease and some types of strokes.

Current drug therapies seek to block this molecule's action after it is secreted by cells. However, the new research could lead to the development of treatments that would prevent the body from producing it, resulting in more effective medications and therapies for inflammatory diseases.

"If we understand how this molecule is made in the body, we may be able to block it before it is produced," said study author Dr. Moshe Arditi, executive vice chair of research in the Department of Pediatrics and director of the Division of Pediatric Infectious Diseases and

Immunology. "Until now, this was the missing piece of the puzzle."

Arditi, who also directs Cedars-Sinai's Infectious and Immunologic Diseases Research Center, found that damaged mitochondrial DNA activate specific proteins within dying cells, triggering the release of interleukin-1beta. Previous research has shown the molecule, when over-secreted by cells, can be a significant contributor to major inflammatory diseases.

Three of these diseases alone -- atherosclerosis, Type 2 diabetes and rheumatoid arthritis – affect an estimated 100 million Americans.

Arditi is planning further studies to build on the findings.

"The discovery by Arditi and colleagues has great potential to impact a wide range of inflammatory diseases, particularly in their early stages where an intervention could prevent more severe and debilitating ravages of such diseases," said Dr. Leon Fine, Cedars-Sinai's vice dean of research and chair of biomedical sciences. "This discovery, at last, may open the door to such therapy."

In addition to Cedars-Sinai, other research groups involved in the study include UCLA 's David Geffen School of Medicine, UC Merced's Health Sciences Research Institute and School of Natural Sciences; UC Riverside's Department of Chemistry and the University of Massachusetts Medical School's Department of Medicine.

Provided by Cedars-Sinai Medical Center