Cell & Developmental Biology

ISSN: 2168-9296

OPEN ACCESS Freely available online

Mini Review

Mitochondrial Distress Signals are sent to the Heart by Fat Cells

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When fat cells become physiologically stressed and dysfunctional, they start spewing out chunks of mitochondria that act as warning signals to the heart, according to the authors of a report published today in Cell Metabolism (August 20). The mitochondrial signals in cardiac cells create a burst of reactive oxygen species (ROS), which appears to prime and protect the organ against further injury. Scott Summers, a diabetes and metabolism expert at the University of Utah who was not involved in the project, adds, "It's a remarkable observation." “I think we'll all be looking to see if this [mitochondrial shuttling] turns out to be a major regulatory route for organ behaviour change.” “It's also really remarkable that they saw this as producing a protective response in the heart,” he says [1].

Fat stores energy and vitamins, regulates hunger, metabolism, and immunity, and serves to insulate and protect the body's organs. Secreting hormones like leptin and adiponectin are responsible for some of fat's regulating functions. Extracellular vesicles, small lipid-bound particles released from cells, are another way fat communicates with organs. According to cell biologist Philipp Scherer of the University of Texas Southwestern Medical Center, roughly half of the extracellular vesicles seen in the blood come from adipose tissue. Extracellular vesicles, he and his colleagues reasoned, were the prime suspects behind an unexplained effect of fat on the heart they'd previously reported: when they stressed mouse adipose tissue by engineering fat cells to have overactive mitochondrial ferritin (a mitochondrial matrix enzyme) and feeding the animals a high-fat diet oxidative stress levels in the heart, where mitocarriage occurs, were elevated [2,3].

When fat cells become physiologically stressed and dysfunctional, they start spewing out chunks of mitochondria that act as warning signals to the heart, according to the authors of a report published today in Cell Metabolism (August 20). The mitochondrial signals in cardiac cells create a burst of reactive oxygen species (ROS), which appears to prime and protect the organ against further injury. Fat tissue normally stores extra calories as lipids, but metabolic stress can lead this tissue to become dysfunctional over time. As a result, lipids are more likely to build up in organs like the heart, liver, and kidneys, resulting in lipotoxicity, a condition that can cause a range of health concerns. Surprisingly, lipotoxicity does not appear to be linked to obesity; in fact, some claim that obesity can help protect against the damage, at least temporarily [4].

The team also looked at plasma samples from human volunteers. They discovered that a higher proportion of extracellular vesicles in plasma from overweight, metabolically unwell persons contained mitochondrial DNA than vesicles from plasma from lean people, implying that mitochondrial fragments may be used as markers in people as well.Dr. Crewe, Dr. Scherer, and their colleagues used genetic technologies to speed up the loss of mitochondrial mass and function in mice in their work. When these animals consume a high-fat diet, they get obese. Researchers discovered that as mouse fat cells got obese, they began releasing extracellular vesicles containing dying mitochondrial pieces. Some of these mitochondrial fragments make their way to the heart through the bloodstream, generating oxidative stress, which causes cells to release damaging free radicals [5].

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Received: September 7, 2021; **Accepted:** September 21, 2021; **Published:** September 28, 2021

Citation: Dola S (2021). Mitochondrial Distress Signals are sent to the Heart by Fat Cells. Cell Dev Biol.10:238.