

# Fat cells may influence how the body reacts to heart failure, study shows

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University of Alberta researchers have found that limiting the amount of fat the body releases into the bloodstream from fat cells during heart failure could help improve outcomes for patients.

In a recent study published in the *American Journal of Physiology*, Jason Dyck, professor of pediatrics in the Faculty of Medicine & Dentistry and director of the U of A's Cardiovascular Research Centre, found that mice with heart failure that were treated with a drug blocking the release of fat into the bloodstream from fat [cells](#) saw less inflammation in the heart and throughout the [body](#), and had better outcomes than a control group.

"Many people believe that, by definition, heart failure is only a condition of the heart. But it's much broader and multiple organs are affected by it," said Dyck, who holds the Canada Research Chair in Molecular Medicine and is a member of the Alberta Diabetes Institute and the Women and Children's Health Research Institute. "What we've shown in mice is that if you can target fat cells with a drug and limit their ability to release stored fat during heart failure, you can protect the heart and improve [cardiac function](#).

"I think it really opens the door for other avenues of investigation and therapies for treating heart failure," Dyck noted.

During times of stress, such as heart failure, the body releases stress hormones, such as epinephrine and norepinephrine, to help the heart compensate. But because the heart can't function any better—and is in fact damaged further by being forced to pump faster—the body releases more stress hormones and the process cascades, with heart function continuing to decline. This is why a common treatment for heart failure is beta-blocker drugs, which are designed to block the effects of stress hormones on the heart.

The release of stress hormones also triggers the release of fat from its storage deposits in fat cells into the bloodstream to provide extra energy to the body, a process called lipolysis. Dyck's team found that during heart failure, the fat cells in mice were also becoming inflamed throughout the body, mobilizing and releasing fat faster than normal and causing inflammation in the heart and rest of the body. This inflammation put additional stress on the heart, adding to the cascade effect, increasing damage and reducing heart function.

"Our research began by looking at how the function of one organ can affect other organs, so I thought it was very fascinating to find that a fat cell can influence cardiac function in heart failure," Dyck said. "Fortunately, we had a drug that could inhibit fat mobilization from [fat cells](#) in mice, which actually protected the hearts from damage caused by inflammation."

Dyck points out that although his results are promising, more work is needed to better understand the exact mechanisms at play in the process and develop a drug that could work in humans.

"This work is a proof-of-concept showing that

abnormal fat-cell function contributes to worsening heart failure, and now we're working on understanding the mechanisms of how the drug works to limit lipolysis better," he said. "Once we get that, that's the launchpad for making sure it's safe and efficacious, then advancing it to our chemists, and then maybe some early trials in humans."

Dyck said the findings—and a better understanding of how organ functions affect other organs—could be used to develop new approaches to several other diseases.

"We know that people have high rates of lipolysis when they have heart failure, so I presume this approach would benefit all types of [heart failure](#)," he said. "But if you consider that inflammation is associated with a wide variety of different diseases, like cancer, diabetes or other forms of [heart](#) disease, then this approach could have a much wider benefit."

**More information:** Shingo Takahara et al, Inhibition of ATGL in adipose tissue ameliorates isoproterenol-induced cardiac remodeling by reducing adipose tissue inflammation, *American Journal of Physiology-Heart and Circulatory Physiology* (2020). DOI: [10.1152/ajpheart.00737.2020](https://doi.org/10.1152/ajpheart.00737.2020)

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