

# Compound identified that improves heart function in rats

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Heart attack survivors may think the worst is behind them. But many later develop heart failure, a progressive disease marked by shortness of breath and swelling in the legs. Symptoms can prevent patients from working, exercising—even picking up grandchildren.

Heart failure occurs after a [heart attack](#) when enough of the [heart muscle](#)

dies, causing the rest of the heart to overwork, which leads to more damage. To protect an overworked, failure-prone heart, cardiologists typically prescribe medications that encourage the heart to take it easy, said Daria Mochly-Rosen, Ph.D., professor of chemical and [systems biology](#) and the George D. Smith Professor in Translational Medicine.

Mochly-Rosen is hoping to tackle heart failure at the molecular level. She and her colleagues developed a compound that in preliminary tests appeared to improve [heart function](#) in rats with heart failure caused by a heart attack.

The study was published Jan. 18 in *Nature Communications*. Julio Ferreira, Ph.D., a professor at the University of Sao Paulo, is the lead author.

One contributor to heart failure following a heart attack is the accumulation of broken or dysfunctional mitochondria, the small organelles in cells that produce energy. The researchers identified a pair of proteins that, when bonded, gum up the normal activity of mitochondria and contribute to heart failure. One of those proteins, protein kinase C beta 2, is found in higher levels in failing human and rodent hearts.

The researchers tapped their chemistry know-how to develop a compound called SAM $\beta$ A (pronounced "samba"), which can prevent these proteins from bonding, thereby improving mitochondrial function and providing more energy for the heart.

In tests, post-heart-attack rats that developed [heart failure](#) and were treated with SAM $\beta$ A had better cardiac function—measured by how well their left heart ventricles pumped blood with each [heart beat](#)—than rats that weren't treated with SAM $\beta$ A.

"We greatly improved their hearts," Mochly-Rosen said. "If humans are going to be like rats, perhaps we can treat them with a drug that prevents this deterioration."

She added that they also gave healthy rats doses of SAM $\beta$ A "and it had absolutely no effect," an indication that the compound is nontoxic.

Mochly-Rosen and Ferreira suspect that SAM $\beta$ A will also be effective in humans. If so, it has the potential to be developed into a drug for human heart attack patients, they believe.

"I'm hopeful SAM $\beta$ A will be accepted by the industry for [drug development](#) because it appears very promising," Mochly-Rosen said.

**More information:** Julio C. B. Ferreira et al. A selective inhibitor of mitofusin 1- $\beta$ IIPKC association improves heart failure outcome in rats, *Nature Communications* (2019). [DOI: 10.1038/s41467-018-08276-6](https://doi.org/10.1038/s41467-018-08276-6)

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