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New research shows how heart cells communicate to regulate heart activity

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New research from Western University (London, Canada) is leading to a better understanding of what happens during heart failure; knowledge that could lead to better therapeutics or a more accurate predictor of risk. The research led by Robarts Research Institute scientists Robert Gros, PhD, and Marco Prado, PhD, along with graduate student Ashbeel Roy found the heart is regulated not only by nervous systems but also by heart cells sending messages to each other through the release of the neurotransmitter acetylcholine (ACh). The research has been published online by *The FASEB Journal*.

As Gros explains, heart activity is regulated by two nervous systems: the sympathetic and the parasympathetic. The sympathetic acts like an accelerator, speeding up the heart and the parasympathetic acts like a brake, decreasing the heart rate. When these systems get dysregulated or out of whack, it can lead to heart failure.

"But the heart is not well innervated or in other words, there are very few nerves to control the heart. So we wanted to know how the signal from the nerve is communicated throughout the heart. A neuronal system is nerve-based but now we're talking about a non-neuronal system, which means it's not in any nerve tissue but found in the heart cells themselves," says Gros, an associate professor in the Departments of Physiology & Pharmacology and Medicine at Western's Schulich School of Medicine & Dentistry and a scientist in the Vascular Biology Research Group at Robarts. "We've shown how the nerve sends a signal and individual heart cells pick up that signal; they can transduce that

signal by the release of ACh from one cell to the next. It's the propagation of this signal that regulates the heart. Now we need to look at how this system changes in heart failure."

In collaboration with Robarts' scientist Vania Prado, PhD, Gros tested the theory using mice which were engineered so that their heart cells exclusively, could not release ACh. Under non-stressful conditions the mutant mice had normal heart rates. But when they exercised, these mice had a far greater increase in their heart rate, and it took longer for them to return to their pre-exercise heart rate, as compared to control mice. The results suggest the heart cell derived ACh may boost parasympathetic signaling to counterbalance sympathetic activity.

Gros calls the research a kick start, because if this non neuronal source of ACh is playing such an important role in the heart, it's probably important in other organs as well. The research was supported by the Heart and Stroke Foundation of Ontario, the Canadian Institutes of Health Research and the Canada Foundation for Innovation.

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