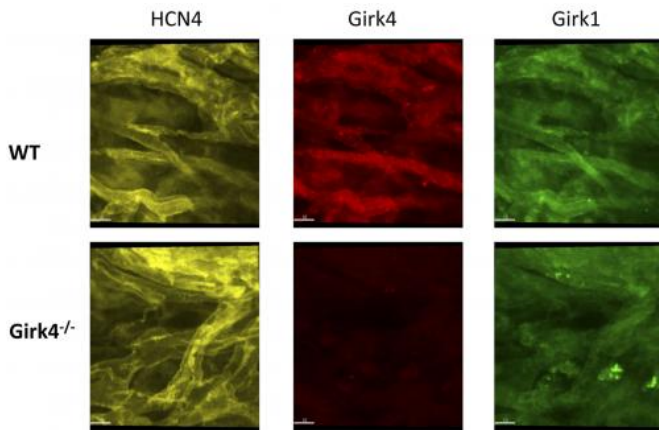


# Understanding the role of $I_{KACH}$ in cardiac function

15 July 2013



specific gene required for  $I_{KACH}$  to investigate the consequences of its loss. The mice showed a moderate increase in [resting heart rate](#) compared with that in a control group, and they displayed a significant delay in the recovery of resting heart rate after stress, exercise, or administration of a drug that simulated activation of the fight or flight response. The results indicate that  $I_{KACH}$  plays a critical role in both of these parasympathetic cardiac functions.

**More information:** Mesirca, P., et al. 2013. *J. Gen. Physiol.* [doi:10.1085/jgp.201310996](https://doi.org/10.1085/jgp.201310996)

This is a close-up view of cardiac pacemaker cells within the sinoatrial node. A study in *The Journal of General Physiology* shows a novel role for  $I_{KACH}$  in cardiac pacemaker activity and heart rate regulation. Credit: Mesirca et al., 2013

Provided by Rockefeller University

Researchers have uncovered a previously unknown role for the acetylcholine-activated inward-rectifying potassium current ( $I_{KACH}$ ) in cardiac pacemaker activity and heart rate regulation, according to a study in *The Journal of General Physiology*.

The heart rate increases in response to fear or exercise, when the body's [sympathetic nervous system](#) activates the "fight or flight" stress response. After sympathetic stimulation, the heart rate is brought back to normal by the [parasympathetic nervous system](#), which regulates the body at rest. Parasympathetic regulation of the heart rate is initiated when acetylcholine released from the vagus nerve spurs a chain of events that activate  $I_{KACH}$  in the sinoatrial node—the pacemaker of the heart—to reduce the heart rate. However, the precise role of  $I_{KACH}$  is not fully understood.

To find out more, researchers used mice lacking a

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