Histamine H3 receptor agonists to treat/prevent arrhythmias

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Technology description

Myocardial ischemia is marked by a pathologic release of norepinephrine (NE) from cardiac sympathetic nerves; the norepinephrin in turn causes severe arrhythmias and sudden cardiac death.

Investigators from the Weill Medical College of Cornell University have discovered that activation of histamine H3 receptors in cardiac sympathetic nerve endings inhibits the activity of the Na+/H+ exchanger, which plays a pivotal role in excessive norepinephrine release in the ischemic heart. Cornell holds a recently issued patent covering methods to use histamine H3R agonists to reduce cardiac dysfunction related to excessive NE release.
Activation of H3R significantly inhibits carrier-mediated NE release and alleviates reperfusion arrhythmias. Unlike other presynaptic negative modulatory receptors (e.g., adenosine A1-receptors) H3R activation is devoid of negative chronotropic and dromotropic effects. Furthermore, although 2-adrenoceptor stimulation reduces NE exocytosis, it actually enhances carrier-mediated NE release. Because H3R stimulation decreases carrier-mediated NE release in the human heart, selective H3R agonists represent a new therapeutic frontier in myocardial ischemia.

Institution

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