

Galectin-3-centered paracrine network mediates cardiac inflammation and fibrosis upon β-adrenergic insult

December 5 2022



Galectin-3 upregulated both in patients and mice upon acute sympathetic overactivation. IL-18/galectin-3 axis mediates the cardiac inflammatory injuries induced by acute β -adrenergic receptor activation. Galectin-3 inhibitor, but not a β -blocker, treatment one day after β -AR insult can successfully block cardiac



inflammatory injuries upon acute β -adrenergic receptor overactivation. Credit: Science China Press

In a study led by Dr. Han Xiao (Department of cardiology and institute of vascular medicine, Peking university third hospital), researchers found that rapid over-activation of β -adrenergic receptors (β -AR) following acute stress initiates cardiac inflammation and injury. However, the process of inflammation cascades has not been fully illustrated.

Using bioinformatics analysis, the research team discovered galectin-3 as a potential significant downstream effector of β -AR and IL-18 activation. In the heart of mice treated with β -AR agonist (isoproterenol), galectin-3 expression was upregulated markedly later than IL-18 activation.

The serum level of galectin-3 was positively correlated with norepinephrine or IL-18 in ACS (<u>acute coronary syndrome</u>) patients. ISO-induced galectin-3 upregulation was attenuated in Il18-/- mice. It was further revealed that cardiomyocyte-derived IL-18 induced galectin-3 expression in macrophages following ISO treatment.

Moreover, galectin-3 deficiency suppressed ISO-induced cardiac inflammation and <u>fibrosis</u> without blocking ISO-induced IL-18 increase. Treatment with a galectin-3 inhibitor, but not a β -blocker, one day after ISO treatment effectively attenuated cardiac inflammation and fibrosis.

This study demonstrates that following acute sympathetic activation, galectin-3 was upregulated in <u>macrophages</u> by cardiomyocyte-derived IL-18 and subsequently promoted cardiac inflammation cascades leading to fibrosis. Galectin-3 inhibitor effectively blocks cardiac injury one day



after β -AR insult. Galectin-3 mediated cardiac inflammatory amplification and is a potential therapeutic target for cardiac diseases involving β -adrenergic toxicity.

The paper is published in the journal Science China Life Sciences.

More information: Guomin Hu et al, Galectin-3-centered paracrine network mediates cardiac inflammation and fibrosis upon β -adrenergic insult, *Science China Life Sciences* (2022). <u>DOI:</u> <u>10.1007/s11427-022-2189-x</u>

Provided by Science China Press

Citation: Galectin-3-centered paracrine network mediates cardiac inflammation and fibrosis upon β-adrenergic insult (2022, December 5) retrieved 6 May 2024 from https://medicalxpress.com/news/2022-12-galectin-centered-paracrine-network-cardiacinflammation.html

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.