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심부전(Heart Failure preserved Ejection Fraction), 번역후수식화(Acetylation, Nitrosylation)

[논문]

1. Kim M, Kim YS, Ahn Y, Eom GH, Yoon S*. PSME4 determines mesenchymal stem cell fate towards cardiac commitment through YAP1 degradation. Korean J Physiol Pharmacol. 2023 Jul 1; 27(4): 407-416.
2. Kim YS, Kim M, Cho DI, Yoon S* et al. PSME4 Degrades Acetylated YAP1 in the Nucleus of Mesenchymal Stem Cells. Pharmaceutics. 2022;14(8):1659.
3. Yoon S, Kim M, Lee H, Kang G, Bedi K, Margulies KB, et al. S-Nitrosylation of Histone Deacetylase 2 by Neuronal Nitric Oxide Synthase as a Mechanism of Diastolic Dysfunction. Circulation. 2021; 143(19):1912-1925.
4. Yoon S, Kim M, Min HK, Lee YU, Kwon DH, Lee M, et al. Inhibition of heat shock protein 70 blocks the development of cardiac hypertrophy by modulating the phosphorylation of histone deacetylase 2. Cardiovascular research. 2018.
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Nitric oxide-induced protein modifications in disease

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Nitric oxide (NO) is known as a vasodilator, regulating vascular tone in smooth muscles, and traditionally considered cardioprotective in heart disease. However, recent investigations have uncovered a paradoxical involvement of NO in heart failure pathogenesis. Heart failure with preserved ejection fraction (HFpEF) presents a significant clinical challenge, with current therapeutic strategies, including NO donors, often proving ineffective. Despite the heterogeneous nature of HFpEF, diastolic dysfunction (DD) emerges as a prominent feature. Within DD, we explore the role of neuronal nitric oxide synthase (nNOS) in inducing S-nitrosylation of histone deacetylase 2 (HDAC2). Animal models, including SAUNA and mild transverse aortic constriction mice, revealed impaired diastolic function and exercise tolerance, accompanied by increased S-nitrosylation levels. Enhanced nNOS expression and NO production were observed in heart samples from both mice and patients with left ventricular hypertrophy. In vivo interventions targeting nNOS or HDAC2 S-nitrosylation ameliorated DD development, with nNOS knockout mice exhibiting resistance to SAUNA stress. HDAC2 S-nitrosylation was confirmed at specific cysteine residues (C262 and C274), and HDAC2 C262A/C274A mice maintained diastolic function under DD stimuli. Moreover, gene delivery of NRF2 or pharmacological denitrosylation with dimethyl fumarate attenuated DD in vivo. These findings unveil a novel mechanism underlying DD pathophysiology mediated by nNOS-induced HDAC2 S-nitrosylation, providing insights into the limitations of conventional NO enhancement therapies for HFpEF. Importantly, they propose reducing NO levels or promoting HDAC2 denitrosylation as a promising therapeutic strategy for refractory.