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A company of the LifeScience Foundation

REFERENCE NUMBER TO 15-00663

lncRNA FOXO6OS/AC093151.3 AS THERAPEUTIC TARGET IN CARDIO-PROTECTION AND REGENERATION

Keywords: lncRNA, cardioprotection, cardiac regeneration, heart failure, cardiac hypertrophy, RNA therapeutics, gene therapy, AAV

INVENTION NOVELTY

The invention discloses the previously uncharacterized long non-coding RNA (lncRNA) Foxo6os and its human orthologue AC093151.3 as important regulators in both heart development and heart disease. Murine Foxo6os is highly expressed during physiological heart development and specifically enriched in cardiomyocytes, yet its expression declines markedly across multiple models of cardiac disease, including pressure overload-induced heart failure, ischemia-reperfusion injury, and drug-induced cardiotoxicity. The human transcript AC093151.3 as a conserved functional counterpart exhibits highly similar regulation patterns. Expression of AC093151.3 increases during cardiomyocyte differentiation but decreases under hypertrophic and hypoxic stress conditions and is significantly reduced in heart tissue from heart failure patients.

VALUE PROPOSITION

Current therapies primarily slow disease progression but do not directly address the molecular mechanisms underlying cardiac degeneration nor do they promote meaningful regeneration of damaged myocardium. This novel technology offers a highly differentiated approach to cardiovascular therapy by targeting endogenous regenerative and survival mechanisms rather than merely treating symptoms or hemodynamic consequences of disease. The technology is compatible with multiple therapeutic modalities and can be implemented through direct administration of RNA molecules, vectors encoding therapeutic transcripts such as recombinant adeno-associated viruses, or advanced genome-editing platforms designed to activate endogenous expression of the target lncRNAs.

TECHNOLOGY DESCRIPTION

Foxo6os is cardiomyocyte-associated, strongly induced during heart development, and consistently downregulated in cardiac disease models. Therapeutic overexpression via AAV9 gene therapy improves cardiac function in a mouse model of myocardial infarction. The human transcript AC093151.3 exhibits similar expression dynamics and is significantly reduced in heart tissue from patients with heart failure. Functional studies demonstrated that silencing Foxo6os or AC093151.3 impairs cell viability and promotes adverse cellular responses. Conversely, overexpression increases cardiomyocyte survival and activates gene programs associated with cardiac development and function.

COMMERCIAL OPPORTUNITY

Both licensing and co-development are possible.

DEVELOPMENT STATUS

AAV9-mediated Foxo6os therapy improves cardiac function in a mouse model of myocardial infarction. Likewise, AAV6-mediated overexpression of AC093151.3 improved viability of heart tissue from explanted human hearts and of human iPSC-derived cardiomyocytes exposed to hypoxia, altogether highlighting its therapeutic relevance for ischemic cardiac injury.

PATENT SITUATION

Applications in Europe and USA based on WO2022/023413 with priority of 2020 are pending.

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