Cyclic Amp-Incompetent Adenylyl Cyclase Gene Transfer For Heart Failure

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BACKGROUND

Heart failure is the most common cause of non-elective admission to the hospital in subjects 65 years and older. Despite optimal drug and device therapy, prognosis in heart failure is dismal. Many clinical trials of drugs that increase heart function (“inotropes”) have failed, possibly due to the deleterious effects of agents that increase cAMP. An alternative strategy is to alter myocardial calcium handling or myofilament response to calcium using agents that do not affect cAMP. Expression of a catalytically impaired adenylate cyclase type 6 mutant molecule (AC6mut), one that markedly reduces cAMP production, is associated with normal cardiac function in response to β-adrenergic receptor stimulation. The mechanism is through enhanced effects of AC6mut on Ca2+ handling - effects that do not require cAMP. These data are important in clinical settings for two reasons: 1) the results provide additional insight regarding the interplay between Ca2+ handling and βAR signaling vis-à-vis LV function; and 2) AC6mut may provide inotropic support free from the potentially deleterious effects of increased cAMP.

TECHNOLOGY DESCRIPTION

Scientists at VA San Diego Healthcare System and UC San Diego and have described a novel AC6mut molecule that increases LV function through promoting enhanced calcium handling, while reducing cAMP generation. This strategy appears to protect the heart from programmed cell death.

APPLICATIONS

When AC6mut is expressed in the failing heart, it is likely to restore heart function and reduce symptoms of heart failure in the absence of increased levels of cAMP.

PATENT STATUS

Country	Type	Number	Dated	Case
United States Of America	Published Application	20160101164	04/14/2016	2013-027

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OTHER INFORMATION

KEYWORDS

cAMP, heart failure, cardiomyopathy, gene therapy, cardiovascular

CATEGORIZED AS

- **Medical**
  - Disease: Cardiovascular and Circulatory System
  - Gene Therapy

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