Nuclear Localized Therapeutics for Heart Disease and Skeletal Muscle Pathologies

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Technology description

Dr. Song and his lab have discovered a protein that, when truncated, is able to penetrate the nucleus of cells and transcribe the genetic coding, allowing for consistent calcium emission, which stabilizes the calcium dependent enzyme calpain.

Background Information

Heart disease is the leading cause of death in the US, occurring in 1 of 16 adults. This field has a very high commercial value due to this, costing the US economy $444 billion yearly. Cardiac hypertrophy and heart failure are dangerous and all too common myocardium diseases. Mortality rates from heart disease has increased by 41% from 1990 to 2013, and has been continuing on that same trajectory. Common treatments for heart disease currently aim to lower the work load of the hearts rather than disease development. Skeletal muscle diseases and abnormalities, such as muscle fatigue and muscular dystrophy, are the leading cause of physical disability in the US. Musculoskeletal conditions such as arthritis and back pain affect 1.7 billion people worldwide and has the 4th greatest impact on the overall health of the world population when considering both death and disability.

Application area

This truncated protein is able to act as a cardiac protector against hypertrophy and heart failure by way of preventing, inhibiting, or treating the issue. Since the protein also exists in skeletal muscle, the altered protein is also applicable to prevent, inhibit, or treat skeletal muscle diseases, such as muscle fatigue, muscular dystrophy, and the like.

Advantages

Reverses effects of heart failure by altering the genetic code in the JP2 protein
Reverses effects of skeletal diseases by the same method
Can be locally or systematically administered to prevent, inhibit, or treat issues

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