Wnt1 for the Treatment of Peripheral Vascular Disease and the Repair of Heart
Tech ID: 24785 / UC Case 2013-782-0

SUMMARY

UCLA researchers in the Department of Medicine have developed a novel therapeutic agent for cardiac repair after acute cardiac injury.

BACKGROUND

Heart disease is a leading cause of mortality and morbidity and an emerging public health problem in the developing world. The inability of the mammalian heart to regenerate cardiac muscles after acute myocardial injury remains a fundamental biological problem to the therapy of heart disease. Therefore, there is a clear need for novel therapeutic strategies.

INNOVATION

Dr. Arjun Deb and colleagues in the Department of Medicine have demonstrated that Wnt1 is an agent critical for cardiac repair. After acute cardiac injury, Wnt1 is expressed in the epicardium of the heart and cardiac fibroblasts, and orchestrates a critical early repair response. Interruption of downstream Wnt signaling leads to the development of heart failure and death within a few days of injury. In addition, the researchers discovered that Wnt1 can also enhance human endothelial progenitor function.

APPLICATIONS

▶ Can be used as a novel therapy for cardiac repair after acute cardiac injury
▶ Can be used as a therapeutic agent to increase blood flow to ischemic extremities
▶ Can be used to enhance the function of endothelial progenitor cells prior to cell based therapy in clinical trials

ADVANTAGES

▶ This is the first time that the role of Wnt1 in enhancing cardiac repair after acute cardiac injury has been described and demonstrated
▶ It has been demonstrated that Wnt1 can also enhance function of human endothelial progenitors

STATE OF DEVELOPMENT

Researchers have experimentally demonstrated the physiological role and therapeutic effect of Wnt1 in the treatment of cardiovascular disease.

PATENT STATUS

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RELATED MATERIALS


CATEGORIZED AS

▶ Medical
▶ Disease: Cardiovascular and Circulatory System
▶ Therapeutics

RELATED CASES

2013-782-0

INVENTORS

▶ Deb, Arjun

OTHER INFORMATION

KEYWORDS

Cardiovascular disease, Wnt1, Cardiac repair, Endothelial progenitors