Growth Factor Treatment of Myocardial Infarction
Tech ID: 21477 / UC Case 2010-769-0

SUMMARY

UCLA investigators have invented a method of decreasing myocardial injury and infarct size through the intravenous administration of a growth hormone, netrin-1, during a cardiac event. This method confers powerful cardioprotective effects, and represents a novel and effective therapy for myocardial infarction.

BACKGROUND

Myocardial infarction (heart attack) affects millions of people in the United States each year. New and more effective therapies for myocardial infarction are in urgent demand.

Currently, there are a few treatments available for myocardial infarction. However, these treatments do have undesirable consequences. Drug-eluting stents are often used after angioplasty, which works acutely but often lead to restenosis. Stem cell therapy is theoretically less toxic and adequately repairs damaged heart muscles, but its mechanisms are unclear and does not always work. Other treatments may help reduce heart load or inhibit processes such as thrombosis and pulmonary edema, but are not directly helpful in reducing infarct size.

INNOVATION

The invention is the use of netrin-1 to reduce infarct size during myocardial infarction events, and a method of administering netrin-1 to confer powerful cardioprotective effects. Netrin-1 is a secreted molecule that is largely known to play a role in guiding vertebrate commissural axons in neuronal development. It also has a critical role in endothelial cell proliferation, migration, and angiogenic signaling in addition to morphogenesis of epithelial cells.

However, UCLA investigators have now demonstrated that the pre-perfusion of netrin-1 can also directly reduce infarct size and myocardial injury in an animal model. In fact, experimental evidence shows that netrin-1 pre-perfusion of mouse hearts reduced ischemia-reperfusion induced infarct size dramatically from 42.5% (±3.6%) to 21.8% (±4.9%). This shows that netrin-1 has strong cardioprotective effects.

Further, netrin-1 perfusion may also inhibit coronary restenosis via its production of nitric oxide to scavenge reactive oxidative species. It has also been shown to activate angiogenic pathways, facilitating the formation of new blood vessels and cardiac repair. Thus, this invention has advantages over current therapies, and may even be used in conjunction with some of those therapies as well.

APPLICATIONS

▶ Direct intravenous injection of netrin-1 into patients at the first sign of a cardiac event to reduce infarct size and myocardial injury.

ADVANTAGES

▶ None of the existing treatments for myocardial infarction are as simple as an intravenous perfusion of netrin-1. Current intravenous drugs serve a primarily supportive role, and angioplasty is an invasive procedure.

STATE OF DEVELOPMENT

In vivo experiments on mouse hearts have been completed.

PATENT STATUS

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<th>Country</th>
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<td>9,050,298</td>
<td>06/09/2015</td>
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RELATED MATERIALS

▶ Netrin-1 prevents ischemia-reperfusion-induced myocardial infarction via a DCC/ERK1/2/eNOS s1177/NO/DCC feed-forward mechanism
▶ Netrin-1 induces angiogenesis via a DCC-dependent ERK1/2-eNOS feed-forward mechanism

ADDITIONAL TECHNOLOGIES BY THESE INVENTORS
Gateway to Innovation, Research and Entrepreneurship

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