

New drug could reduce heart attack damage

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LONDON (Reuters) – A new drug that blocks a protein could reduce the damage and scarring caused by heart attacks, British scientists said on Wednesday.

The drug interferes with C reactive protein (CRP) which is produced in large amounts in the body after a heart attack.

"By blocking the protein we reduce the extent of damage in a heart attack, and that in turn reduces the size of the scar," Professor Mark Pepys, of University College London, who headed the research team, said in an interview.

Heart attack is a leading causes of death in developed countries. Patients often survive an attack but if there is extensive damage they are more likely to suffer heart failure.

"Reducing the immediate damage is thus critically important," said Pepys, who reported the findings in the journal Nature.

In animal studies, Pepys and his team found the drug was effective in reducing the damage caused by the protein and produced no serious side effects.

The researchers are in discussions with pharmaceutical companies and hope to begin human trials within a year or two.

Everyone has CRP but it is normally at low levels. When a person has a heart attack there is a dramatic rise in the protein. The higher the level of the protein, the poorer the prognosis of the patient.

"People whose CRP goes very high after a heart attack are more likely to die immediately, or in the first week or first year after the heart attack," said Pepys.

"You find CRP deposited in and around the damaged tissue in everyone who dies of a heart attack."

CRP levels also rise sharply during trauma, strokes, infection and chronic illnesses such as rheumatoid arthritis.

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"The drug would be given as soon as patients arrived in hospital. If effective, it would reduce the amount of damage in the heart, thus limiting early mortality and the size of the scar left in the heart," Pepys explained.

He said there was a window of opportunity because CRP concentrations did not increase until about six hours after the start of the attack. It reaches a peak about 50 hours afterwards.

The drug prevents the protein from binding to the damaged tissue.