‘Exercise’ protein offers hope for heart patients

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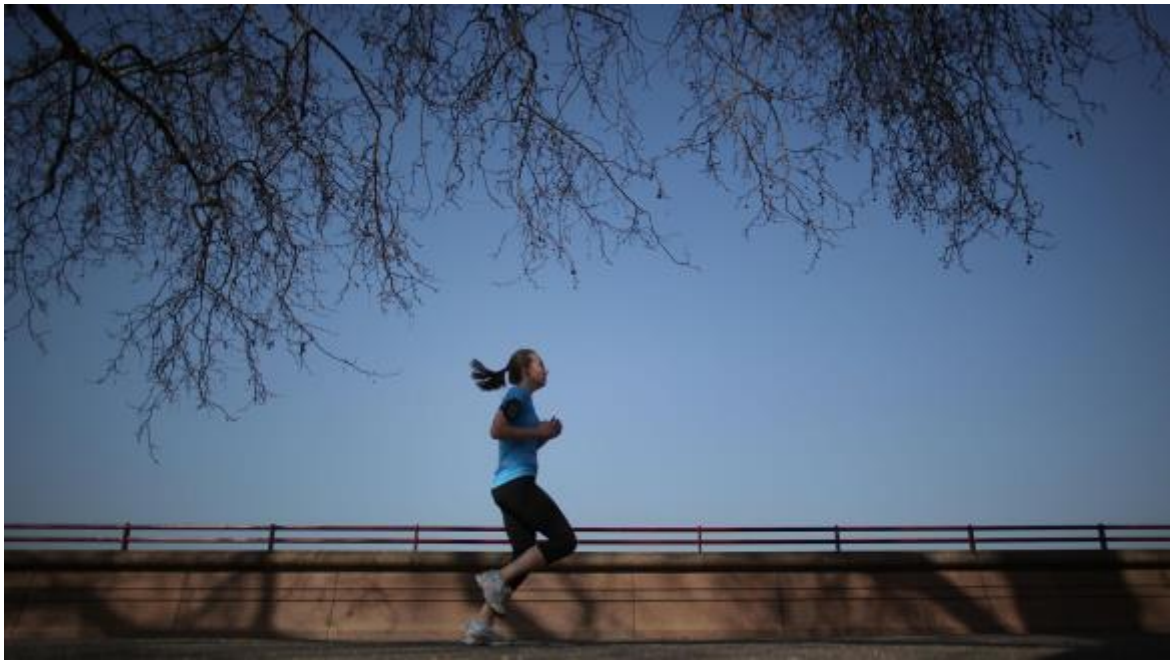

Cardiotrophin 1 can cause the heart to become bigger and healthier, similar to what happens after exercise
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A molecule that mimics the effect of exercise could treat heart failure by repairing damaged cardiac tissue.
The protein, which animal experiments found tricks the heart into responding as if it has been exercised, offers hope for people who currently can only be treated using transplants.
Heart failure, which happens when the organ lacks the strength to pump blood throughout the body, is a huge and growing health problem in Britain, with more than half a million sufferers.

It is most often the result of a heart attack damaging the

heart muscle. The heart can fail either in the left side, which pumps blood out to the body, or the right side, where the blood returns to the heart.

A study published in Cell Research reveals that the protein cardiotrophin 1 can cause the heart to grow bigger and healthier, in a similar way to exercise or pregnancy. Just as with abandoned exercise regimes, this growth is completely reversible.

Treatment with cardiotrophin 1 significantly improved heart function in animal models of both left and right-heart failure.

Duncan Stewart from the University of Ottawa, one of the senior authors of the study, said: “Currently, the only treatment for right-heart failure is a transplant. Although we have drugs that can reduce the symptoms of left-heart failure, we can’t fix the problem, and left-heart failure often leads to right-heart failure over time.”

Lynn Megeney, the other lead author, said: “If the heart attack is large enough and you’ve lost a substantial amount of heart muscle, you will over time move toward a heart failure-like outcome. You could imagine cardiotrophin 1 might be able to put the brakes on that transition.”

The experiments, on rats and mice, together with preliminary experiments in pigs, were performed with the human version of the cardiotrophin 1 protein — a good sign for success in treating humans with the same protein.

“We’re hoping for there to be a human trial within three years,” said Dr Megeney. “If it was targeted as a therapy for right-heart failure, and the initial trial works well, within two to three [further] years it could be a standard therapeutic intervention.”

There is also a danger of abuse, however. Because it boosts heart function cardiotrophin 1 could be used for doping in sport.

“I’m sure that people will have an interest from [the doping] perspective,” Dr Megeney said. Apart from doping, however, cardiotrophin 1 could be “a way to get the benefits of exercise for people who cannot otherwise get those benefits”, whether due to heart failure or other conditions.

Metin Avkiran, associate medical director at the British Heart Foundation, welcomed the research.

“More than half a million people in the UK are living with heart failure,” he said. “There is no cure for this debilitating condition and for patients with severe heart failure the chances of surviving for more than five years are worse than most forms of cancer, so the need to find new treatments is urgent. This is an encouraging discovery, suggesting that the CT1 protein could help improve the function of a failing heart. However, much more research is needed to find out if CT1 is safe and effective in humans with or at risk of developing heart failure.”

comments

SteveTolcher

This is something very close to my heart (pardon the pun). My father suffers from Heart Failure and we lost my Father in Law due to it last year. As someone who suffers from Coronary Heart Disease (being just 35 when diagnosed) I applaud this research and hope that human trials begin in earnest - anything that can be done to treat this condition is surely a good thing.

Dana Andrews
@SteveTolcher Hear hear! Sorry for your recent loss and hoping very much that you benefit from this development, Steve.

ThoughtIdRetired

@SteveTolcher To add to your support for this research, as many NHS services become "consolidated" in larger hospitals, a significant portion of the population have poorer access to some types of care. In areas of low population density, this can mean a long journey to percutaneous coronary intervention (fitting a stent), with the result of more heart muscle being damaged. This will lead to these geographically disadvantaged groups to have a higher rate of heart failure. Let us hope that a successful therapy is up and running soon, and made available early to those otherwise disadvantaged by poor access to services.

(And, yes, I am aware that the model of providing specialist PCI labs in specialised centres does make sense - but a side effect of that is that some people can be a very long distance from that care.)