Medical Xpress

Study closes debate on folic acid and heart disease

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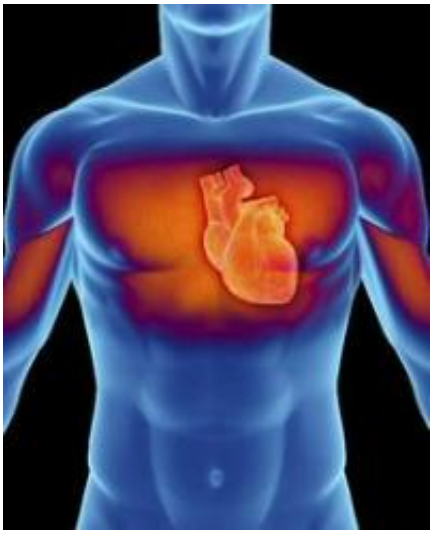

Taking folic acid doesn't lower risk of heart disease

(Medical Xpress) -- Taking folic acid supplements is not going to have any meaningful effect on your risk of coronary heart disease.

That's the conclusion of a comprehensive study led by Oxford University researchers that pretty much closes the door on this debate once and for all.

The possibility that folic acid might help prevent heart disease has attracted considerable interest over the last two decades – understandably, because folic acid pills would have been an attractive,

simple and cheap measure to reduce people's heart disease risk. But studies have often provided conflicting evidence on whether it helps.

Folic acid lowers levels of the amino acid, homocysteine, in the blood. It was thought that this would be beneficial because raised levels of homocysteine have been linked with coronary heart disease.

However, the Oxford researchers recently reported that taking folic acid supplements has no effect on reducing heart disease risk, through a large meta-analysis of data from randomised clinical trials.

Now, in a large-scale genetics study published in PLoS Medicine, they and their colleagues demonstrate that the reasoning behind the interest in folic acid doesn't hold either: there is no link between high homocysteine levels and your likelihood of developing coronary heart disease.

With the evidence from genetic studies and clinical trials both arguing against the use of folic acid supplements, the researchers say the debate is over.

"After two decades of research, this report provides a definitive answer to this question and refutes the relevance of use of folic acid for primary prevention of coronary heart disease," states Dr. Robert Clarke of the Clinical Trial Service Unit at Oxford University, who led the study.

The international group of researchers involved in the new study were interested to understand whether people whose genes lead them to have slightly higher levels of homocysteine throughout life have any extra risk of coronary heart disease.

The MTHFR gene encodes an enzyme which metabolises homocysteine, removing it from the body. But a single change in the DNA code, inserting a T for a C, reduces the enzyme's efficiency.

Individuals with two copies of the altered gene have homocysteine levels that are about 20% higher than those with the more common gene variant.

This makes the gene a very good tool for studying whether homocysteine levels influence coronary heart disease risk.

However, the research team found that among 48,000 individuals with coronary heart disease and 68,000 controls, people who had the MTHFR gene variant did not have an increased risk of developing coronary heart disease.

The major new step taken by the researchers was to include 19 previously unpublished datasets involving 48,000 additional coronary heart disease cases in their analysis.

It was these unpublished data that drove the conclusion that high homocysteine levels do not have any effect on coronary heart disease.

The researchers suggest that publication bias (where more positive results tend to get published in scientific journals over negative findings) played a role in previous suggestions linking homocysteine with coronary heart disease risk, together with methodological problems in some studies.

Homocysteine is produced in the body from methionine, an essential amino acid found in large amounts in meat, eggs and milk.

"Some believed that elevated homocysteine causes damage to the inner lining of blood vessels," explains Dr. Clarke. "However, elevated homocysteine levels are closely correlated with known risk factors, such as smoking, high blood pressure and high cholesterol. In retrospect, elevated homocysteine levels are probably a marker of cardiovascular

disease rather than being causal."

The study was funded by the British Heart Foundation and UK Medical Research Council.

Provided by Oxford University