Protein Block Stops Vascular Damage in Diabetes

Lund University

Researchers at Lund University in Sweden have discovered how to stop the destructive process that leads to cardiovascular disease in diabetic laboratory animals.

It is well known that high blood sugar levels significantly raise the risk of cardiovascular disease. It is unclear, however, why this happens. An important part of the explanation may be NFAT, a protein activated when blood sugar is raised and which starts a chain of events that damage the blood vessels and accelerate the development of atherosclerosis.

"We have now shown that it is possible to stop the atherosclerosis caused by diabetes despite the fact that the mice continued to have high blood sugar levels", said Maria Gomez, a researcher at the Lund University Diabetes Centre. Over the course of four weeks, the researchers gave diabetic mice a new substance originally developed as an immunosuppressant that prevented the activation of the NFAT protein.

Apart from the blood vessels, the substance did not affect NFAT in any other cells of the body.

"That is important. We don't want to suppress the whole immune system. We also saw that the substance only has an effect when NFAT is active. The plaque formation was only stopped in diabetic mice and not in non-diabetic mice, which had normal blood sugar levels", Anna Zetterqvist points out. "It appears that there are different mechanisms behind plaque formation caused by diabetes and not caused by diabetes", she added.

The process of plaque formation in the blood vessels of diabetic mice resembles the process in humans. The plaques go through the same stages and the damage worsens with age.

"Despite major progress in the treatment of cardiovascular disease in recent decades, there is no treatment that specifically targets the damage to the blood vessels experienced by diabetes patients. Our findings show that blocking the NFAT protein could constitute such a treatment", said Maria Gomez.

For more information, visit Lund University [1].

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