



Deleting a non-coding region leads to narrowing of arteries in mice.

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The risk of developing a form of heart disease has been linked to a region of junk DNA.

Researchers have made headway in working out why a section of junk DNA — the 98% or so of the genome that does not code for proteins — raises the risk of at least one form of heart disease.

About one in five deaths in the United States results from excessive build-up of fatty plaques inside arteries supplying blood to the heart — known as coronary artery disease (CAD). In 2007, genome-wide association studies on thousands of participants linked a non-coding stretch of chromosome 9p21 with the disease, and showed that people who carry certain single nucleotide mutations in this stretch of DNA have an increased chance of developing CAD.

The latest work, published online today in *Nature*, builds on these studies by knocking out this area of the equivalent chromosome in mice. "We were really interested in understanding how this purely non-coding interval leads to CAD, so we thought, 'Let's delete it and see what happens'," says geneticist Len Pennacchio of the Lawrence Berkeley National Laboratory in Berkeley, California, who led the study.

"We did, and found that the expression of two genes nearly 100,000 base pairs away from the deletion dramatically decreased in mice," Pennacchio explains. In addition, many of the mice without the non-coding DNA died earlier than normal and some developed tumours.

Published online 21 February 2010 | *Nature* | (Abstract from *Nature*, License Number 2257020292199)

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