The authors establish the context of the research article in the "Background" section. In addition, they emphasize the *shared knowledge* in the research area.

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Vitamin D and cause-specific vascular disease and mortality: a Mendelian randomisation study involving 99,012 Chinese and 106,911 European adults

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To create a research space, the authors highlight a gap in the previous research. The use of the word "however" illustrates the transition from known knowledge to unknown knowledge, and hence the *knowledge gap*.

This *move* outlines the purpose/objective of the study and forms the basis of the research being reported. Note the use of "we", a form of *self-mention*.

Abstract

Background: Randomised control trials and genetic analyses have demonstrated that vitamin D or 25-hydroxyvitamin D (25[OH]D) levels may not play a causal role in the development of cardiovascular disease. However, it is unclear if 25(OH)D is causally associated with cause-specific vascular disease and lipids. Therefore, we examined the causal association of 25(OH)D with myocardial infarction, stroke, ischaemic heart disease, ischaemic stroke, subarachnoid haemorrhage, intracerebral haemorrhage, and lipid levels among both Chinese and Europeans.

Methods: We used a Mendelian randomisation (MR) design in the China Kadoorie Biobank, the Copenhagen City Heart Study, and the Copenhagen General Population Study. The 25(OH)D-related genetic variants in the *CYP2R1* and *DCHR7* genes were genotyped in 99,012 Chinese adults and 106,911 Danish adults.

Results: In Chinese adults, plasma 25(OH)D levels were not significantly associated with cause-specific vascular disease or mortality, with the exception of intracerebral haemorrhage (HR, 1.09 [95% CI, 1.01,1.18] per 25 nmol/L higher plasma 25(OH)D). In Europeans, plasma 25(OH)D levels were inversely associated with all types of vascular diseases and mortality. However, MR analysis did not demonstrate causal associations of genetically increased 25(OH)D levels with cause-specific vascular diseases, or mortality in both Chinese and European adults. In addition, each 25 nmol/L higher 25(OH)D was observationally associated with lower total cholesterol and low-density lipoprotein cholesterol levels, but higher high-density lipoprotein cholesterol levels. Likewise, MR analysis showed that 25(OH)D levels were not causally associated with lipids in both Chinese and European adults after Bonferroni correction.

Conclusions: We found no evidence to support that genetically increased 25(OH)D was associated with a lower risk of ischaemic stroke, intracerebral haemorrhage, subarachnoid haemorrhage, and lipid levels in both Chinese and European adults. These results suggest that the inverse associations of vitamin D with vascular disease could be the result of confounding.

Keywords: Mendelian randomisation, Vitamin D, Cardiovascular diseases, Lipids, Causal effect

The authors report the core findings for each of the outcome variables stated earlier. In this *move* the authors present the findings from the statistical analysis.

In the abstract of the research article, the methodology explains the approach used to examine the research problem. It provides a brief description of the study design (Mendelian randomisation), target population (Chinese and Danish adults), sample size, and data collection.

In the "Conclusions" section, the authors interpret results and draw inferences. In the final sentence "could be" operates as a *hedge*.

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