Walnut inclusion in a palm-based high-fat and high-cholesterol diet without changing total energy supply stabilises advanced atheroma plaque through an anti-inflammatory mechanism in ApoE-deficient mice

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BACKGROUND AND AIM

The North-to-South gradient in cardiovascular disease in Europe could be partially explained by the low prevalence of lipid-rich atheroma plaques (responsible for most culprit lesions) in Southern Europe. Consumption of certain foods delays atherosclerosis progression.

In a mouse model of accelerated atherosclerosis we investigated whether the inclusion of walnuts within an atherogenic diet stabilizes advanced atheroma plaque

RESULTS

No differences in lesion extension and maximum lesion area

Inclusion of walnuts partially reverts features of unstable plaque

Stary method:

Grade I: foam cells
Grade II: foam cells, VSMC, few cholesterol clefts
Grade III: foam cells, VSMC, numerous cholesterol clefts
Grade IV: lipid core

EXPERIMENTAL DESIGN

Walnut inclusion mitigates inflammatory gene expression

The effects of walnuts are likely explained by changes in NFκB and Nrf2 activation

The effect of walnuts in plaque lipid content is beyond changes in circulant c-LDL

CONCLUSION

In an experimental model of atherosclerosis, isocaloric inclusion of walnuts in an unhealthy high-fat diet stabilizes advanced atheroma plaque. This contributes novel mechanistic evidence for the cardiovascular benefits of walnuts.