

Vitamin K intake reduces mortality in people with chronic kidney disease

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Background: Cardiovascular disease (CVD) is the leading cause of death in patients with chronic kidney disease (CKD), partly due to increased vascular calcification. Emerging evidence suggests that vitamin K plays a key role in preventing vascular calcification in CKD. However, the relationship between vitamin K intake and mortality in people with CKD remains unknown. The objective of this study was to examine the association of vitamin K intake with all-cause and CVD mortality in a nationally representative sample aged 20 years or above.

Methods: A total of 3401 participants with CKD from the Third National Health and Nutrition Examination Survey were included. Dietary intake was assessed during nutritional examination based on 24-hour dietary recall. Vitamin K intake was used in multivariate Cox regression analysis to predict all-cause and CVD mortality.

Results: During a median follow-up of 13.3 years (37 408 person-years), 1815 and 876 participants died from all-cause and CVD causes, respectively. The majority of participants had vitamin K intake lower than the recommended intake levels. In multivariable Cox-regression analysis, participants in higher quintiles (quintile 4-5) of vitamin K intake had significantly lower risk of all-cause (hazard ratio [HR] = 0.86; 95% confidence interval [CI], 0.75-1; P = 0.046) and CVD mortality (HR = 0.79; 95% CI, 0.64-96; P = 0.021) when compared with quintiles 1 through 3. Participants with vitamin K intake higher than recommended adequate intake value for vitamin K were associated with lower risk of all-cause and CVD mortality.

Conclusion: These findings suggest that adequate-to-high level of vitamin K intake reduces CVD and all-cause mortality in people with CKD.

Adiponectin gene variant +276G>T independently predicts incident coronary heart disease in men: a 16-year prospective study

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Introduction: Adiponectin has been suggested to play a protective role in the development of coronary heart disease (CHD). However, recent prospective studies suggested that high adiponectin levels are associated with a higher risk of cardiovascular mortality in an established CHD cohort. Study of genetic variants of the adiponectin gene (*ADIPOQ*) may provide more insights into the primary role of adiponectin in CHD development. Our objective was to examine the prospective relationship between the genetic variants of *ADIPOQ* and incident CHD in a 16-year prospective population-based cohort of southern Chinese.

Methods: Nine *ADIPOQ* genetic variants with potential functional relevance or shown to be associated with adiponectin levels and/or CHD were genotyped in 2196 subjects from the Hong Kong Cardiovascular Risk Factors Prevalence Study (CRISPS), who were free of CHD at baseline. Among these subjects, 184 had developed CHD over the 16-year follow-up period.

Results: The *ADIPOQ* +276G>T variant was found to be independently associated with incident CHD in men but not in women, even after adjustments for different sets of conventional cardiovascular risk factors ($P_{\text{adjusted}} = 5.5 \times 10^{-3}$ to 0.023; hazard ratio = 1.39 to 1.54). Moreover, the T allele of +276G>T was found to be significantly associated with reduced plasma adiponectin level (P = 0.027) in 1676 subjects with available plasma samples for analysis.

Conclusion: This study supports a protective role of adiponectin in the development of CHD in the general population.