

Online Supplementary Material

Fiscella K, Franks P. Vitamin D, race, and cardiovascular mortality: findings from a national US sample. *Ann Fam Med*. 2009;8(1):11-18.

<http://www.annfammed.org/cgi/content/full/8/1/11/DC1>

Supplemental Appendix. Details on Measures

Data collected from the household survey included sociodemographic, health, morbidity, and physical activity characteristics of the participants. Physical activity was based on the METs (metabolic equivalent tasks) expended per month; total METs were derived by summing across all activities reported, and multiplying the average METs per activity¹ by the number of times that activity was performed per month. Body mass index (BMI) was calculated based on height and weight assessed during the examination (body weight [kilograms]/height [meters] squared). Smoking status was dichotomized into current smoker or not. Poverty was assessed based on percentage of federal poverty (5 categories). This measure is based on household income and family size. Total cholesterol was assayed enzymatically by a central laboratory certified by the Lipid Standardization Program of the Centers for Disease Control and Prevention. Serum creatinine was measured by the modified kinetic method of Jaffe using a Roche Hitachi 737 analyzer (Roche Diagnostics Corporation, Indianapolis, Indiana). Systolic blood pressure was measured based on a standard protocol.² Serum albumin was measured using an albumin test system (Boehringer Mannheim Diagnostics, Indianapolis, Indiana) and C-reactive protein (CRP) was measured based on a modification of the Behring latex-enhanced CRP assay (Behring Diagnostics, Westwood, Massachusetts). Serum albumin is an established measure of nutritional status and CRP is measure of inflammation. Both low albumin and high CRP have been associated with higher cardiovascular risk.^{3,4}

We assessed albuminuria based on the urinary albumin-creatinine ratio (ACR). We estimated Glomerular filtration rate (eGFR) based on serum creatinine. To do so, we first corrected serum creatinine values in NHANES III for differences in assays between NHANES III and the Modification of Diet in Renal Disease (MDRD) Laboratories.⁵ Second, we used the MDRD prediction formula to derive the eGFR: $eGFR \text{ (mL/min per } 1.73 \text{ m}^2) = 175 * \text{serum creatinine (exp}[-1.154]) * \text{Age (exp}[-0.203]) * (0.742 \text{ if Female}) * (1.21 \text{ if Black})$.⁶ Third, we truncated eGFR values greater than 200 mL/min per 1.73 m².⁷

We assessed diabetes based on self-report, a fasting glucose >126 mg/dL, or glycohemoglobin >6.0%. We assessed history of cardiovascular disease based on self-report (heart failure, myocardial infarction, or stroke).

Supplementary Measures

We also examined the contribution of additional variables, including dietary measures and serum high-density lipoprotein cholesterol (HDL-C). These variables were not included in the main analyses, because up to 5% of these variables had missing data, and they did not affect the adjusted relationship between 25(OH)D and subsequent cardiovascular mortality. Dietary measures were based on a single 24-hour dietary recall, administered by a trained dietary interviewer. Derived dietary variables included total average daily calories, total protein (and percentage of total calories from protein), total saturated fats (and percentage of total calories from saturated fat), total dietary calcium, and total dietary vitamin D.

Results of Supplementary Analyses

In a series of supplementary analyses (details available from the authors), we found that the dietary measures (total dietary protein, total dietary saturated fats, percentage of calories from protein, percentage of calories from saturated fats, and total dietary calcium) exhibited no adjusted relationship with cardiovascular mortality and did not affect the adjusted risk associated with 25(OH)D. Persons with a caloric intake below the median had higher cardiovascular mortality than those with a higher caloric intake, but the risks associated with 25(OH)D were unchanged. Finally, oral vitamin D intake was inversely associated with cardiovascular mortality in an analysis excluding serum 25(OH)D. When 25(OH)D was included in the analysis together with oral vitamin D, however, the effect associated with oral vitamin D was no longer statistically significant, whereas the effect associated with 25(OH)D remained statistically significant, and little changed from earlier analyses.

References

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