

QUICK SEARCH:		[advanced]
	Author:	Keyword(s):
Go		
Year:	Vol:	Page:

This Article

Alert me when this article is cited Alert me if a correction is posted

Similar articles in this journal Similar articles in PubMed

Download to citation manager

Citing Articles via Google Scholar Google Scholar

Articles by Rainwater, D. L

Search for Related Content

Articles by Rainwater, D. L Articles by Wang, X. L.

Articles by Rainwater, D. L

Articles by Wang, X. L.

PubMed

Agricola

Articles by Wang, X. L.

PubMed Citation

C Get Permissions

Alert me to new issues of the journal

▶ Full Text Full Text (PDF)

Purchase Article View Shopping Cart

Citation Map

## HOME HELP FEEDBACK SUBSCRIPTIONS ARCHIVE SEARCH TABLE OF CONTENTS

American Journal of Clinical Nutrition, Vol. 86, No. 3, 597-603, September 2007 © 2007 American Society for Nutrition

ORIGINAL RESEARCH COMMUNICATION

## Vitamin E dietary supplementation significantly affects multiple risk factors for cardiovascular disease in baboons<sup>1,2,3</sup>

David L Rainwater, Michael C Mahaney, John L VandeBerg and Xing Li Wang

<sup>1</sup> From the Department of Genetics (DLR, MCM, JLV, and XLW) and the Southwest National Primate Research Center (MCM and JLV), Southwest Foundation for Biomedical Research, San Antonio, TX, and the Division of Cardiothoracic Surgery, Texas Heart Institute, Michael E. DeBakey Department of Surgery, Baylor College of Medicine, Houston, TX (XLW)

Background: Oxidative stress is a widely accepted risk factor for cardiovascular disease (CVD), but the CVD benefit of dietary antioxidants, such as vitamin E, is controversial.

Objective: Therefore, we have investigated, in the baboon model, the effects of dietary vitamin E supplementation on risk factors for CVD.

Design: Pedigreed baboons (n = 251) were fed 2 atherogenic diets, high in fat and cholesterol, that differed in vitamin E concentrations. After 7 wk on each diet, blood samples were taken, and a panel of CVD risk factor traits (ie, indicators of lipoprotein metabolism and oxidative stress) were measured.

Results: Vitamin E supplementation caused significantly higher total antioxidant status (TAS) and lower oxidized LDL as expected. In addition, vitamin E caused 2 paradoxical effects on HDL metabolism: higher apolipoprotein A-I (apo A-1) concentrations and lower HDL sizes. We calculated a difference ( $\Delta$ ) variable for each trait as the value on the high-vitamin E diet minus that on the low-vitamin E diet and determined that several HDL concentration  $\Delta$  variables were significantly correlated with  $\Delta$  TAS, but only one,  $\Delta$  apo A-L, was independently correlated. Genetic analyses showed that 2  $\Delta$  variables,  $\Delta$  paraoxonase and  $\Delta$  HDL<sub>2</sub>, were significantly heritable, but that neither  $\Delta$  TAS nor  $\Delta$  apo

A-I were heritable.

Conclusions: Thus, our data show that dietary vitamin E improves TAS and LDL quality. They also show 2 apparently paradoxical effects on HDL metabolism: lower HDL<sub>2</sub>, which is mediated by genes, and higher apo A-I, which is not.

These effects have contrasting associations with CVD risk and may help account for the mixed results from clinical trials of dietary vitamin E.

Key Words: Vitamin E • HDL • high-density lipoprotein • antioxidants • paraoxonase • apo A-I • apolipoprotein A-I • baboons