

Abstract

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Association of small low-density lipoprotein particles with the incidence of coronary artery disease in men and women.

Gardner CD, Fortmann SP, Krauss RM.

Stanford Center for Research in Disease Prevention and the Department of Medicine, Stanford University School of Medicine, Calif, USA.

OBJECTIVE: To investigate the prospective association of low-density lipoprotein (LDL) particle diameter with the incidence of fatal and nonfatal coronary artery disease (CAD).

DESIGN: A nested case-control study.

SETTING: Cases and controls were identified from a population-based sample of men and women combining all of the 5 cross-sectional surveys conducted from 1979 to 1990 of the Stanford Five-City Project (FCP).

PARTICIPANTS: Incident CAD cases were identified through FCP surveillance between 1979 and 1992. Controls were matched by sex, 5-year age groups, survey time point, ethnicity, and FCP treatment condition. The sample included 124 matched pairs: 90 pairs of men and 34 pairs of women.

MAIN OUTCOME MEASURES: LDL peak particle diameter (LDL size) was determined by gradient gel electrophoresis on plasma samples collected during the cross-sectional surveys (stored at 70 degrees C for 5-15 years). Established CAD risk-factor data were available from FCP baseline measurements.

RESULTS: LDL size was smaller among CAD cases than controls (mean $\pm$ SD) (26.17 $\pm$ 1.00 nm vs 26.68 $\pm$ 0.90 nm; $P < .001$). The association was graded across control quintiles of LDL size. The significant case-control difference in LDL size was independent of levels of high-density lipoprotein cholesterol (HDL-C), non-HDL cholesterol (non-HDL-C), triglyceride, smoking, systolic blood pressure, and body mass index, but was not significant after adjusting for the ratio of total cholesterol (TC) to HDL-C (TC:HDL-C). Among all the physiological risk factors, LDL size was the best differentiator of CAD status in conditional logistic regression. However, when added to the physiological parameters above, the TC:HDL-C ratio was found to be a stronger independent predictor of CAD status.

CONCLUSION: LDL size was significantly smaller in CAD cases than in controls in a prospective, population-based study. These findings support other evidence of a role for small, dense LDL particles in the etiology of atherosclerosis.

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