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Original article

Association of egg consumption and calcified atherosclerotic plaque in the coronary arteries: The NHLBI Family Heart Study

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SUMMARY

Background and aims: Eggs are a ubiquitous and important source of dietary cholesterol and nutrients, yet their relationship to coronary heart disease (CHD) remains unclear. While some data have suggested a positive association between egg consumption and CHD, especially among diabetic subjects, limited data exist on the influence of egg consumption on subclinical disease. Thus, we sought to examine whether egg consumption is associated with calcified atherosclerotic plaques in the coronary arteries.
Methods: In a cross-sectional design, we studied 1848 participants of the NHLBI Family Heart Study without known CHD. Egg consumption was assessed by a semi-quantitative food frequency questionnaire and coronary-artery calcium (CAC) was measured by cardiac CT. We defined prevalent CAC using an Agatston score of at least 100 and fitted generalized estimating equations to calculate prevalence odds ratios of CAC.
Results: Mean age was 56.5 years and 41% were male. Median consumption of eggs was 1/week. There was no association between frequency of egg consumption and prevalent CAC. Odds ratios (95% CI) for CAC were 1.0 (reference), 0.95 (0.66–1.38), 0.94 (0.63–1.40), and 0.90 (0.57–1.42) for egg consumption of almost never, 1–3 times per month, once per week, and 2+ times per week, respectively (p for trend 0.66), adjusting for age, sex, BMI, smoking, alcohol, physical activity, income, field center, total calories, and bacon. Additional control for hypertension and diabetes mellitus, or restricting the analysis to subjects with diabetes mellitus or fasting glucose >126 mg/dL did not alter the findings.
Conclusions: These data do not provide evidence for an association between egg consumption and prevalent CAC in adult men and women.

1. Introduction

Coronary heart disease (CHD) remains the leading cause of death in the United States. Elevated serum non-HDL cholesterol is strongly associated with the risk of CHD [1,2], and thus the role of dietary influences on serum cholesterol has fostered much research attention. The role of egg consumption on CHD is of particular interest because of its unique nutritional qualities. Eggs are an important source of protein, minerals, and fat-soluble vitamins, but

Abbreviations: CHD, coronary heart disease; CAC, coronary-artery calcium; CI, confidence interval; CVD, cardiovascular disease; CT, computed tomography; NHLBI FHS, National Heart, Lung, and Blood Institute Family Heart Study; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

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also a source of dietary cholesterol with about 200 mg of cholesterol in an average egg [3].

The relationship between egg consumption and CHD remains unclear [4,5]. A meta-analysis of several large, prospective cohorts did not find an association between egg consumption and CHD, however subgroup analysis showed a positive relationship between egg consumption and CHD in diabetic populations [4]. In contrast, a recent meta-analysis involving twelve studies reported a 19% higher risk of cardiovascular disease (CVD) with higher egg consumption (83% higher risk of CVD with egg intake in diabetic individuals) [5].

Coronary artery calcification (CAC) is a well-described marker for subclinical atherosclerotic disease [6,7]. The extent of CAC can help in risk stratification and can help predict future CHD events [8]. Despite limited studies of egg intake with CVD, no study has investigated whether egg consumption is associated with subclinical CHD.

Hence, the present study sought to determine whether egg consumption was associated with a lower prevalence of CAC in individuals without known coronary heart disease.

2. Materials and methods

2.1. Study population

Participants in this study were members of the National Heart, Lung, and Blood Institute Family Heart Study (NHLBI FHS) in whom coronary calcified plaque was measured by cardiac-gated multi-detector computed tomography (cardiac CT). The NHLBI FHS is a multicenter, population-based study designed to identify and evaluate genetic and non-genetic determinants of CHD, preclinical atherosclerosis, and cardiovascular risk factors, and has been described in detail in previous publications [9,10]. Briefly, families in the study had been chosen randomly (random group) or based on a higher than expected risk of CHD (high-risk group) from previously established population-based cohort studies. A total of 588 families were chosen at random (with 2673 subjects) and 566 families were selected based on higher than expected risk of CHD (3037 subjects). Of the 5710 subjects, 265 were African-American. The high-risk group was defined based on a family risk score, which compares the family's age and sex-specific incidence of CHD to that expected in the general population [10]. All members of these families were invited for a clinical evaluation (between 1993 and 1995). Between 2002 and 2003, about one-third of the families (the largest families available who also had genome-wide anonymous markers typed by the Mammalian Genotyping Service) of the NHLBI FHS were invited to participate in a clinical examination that included measurement of CAC with cardiac CT. In addition to the initial NHLBI FHS study centers, an African-American center – University of Alabama at Birmingham – was recruited from the Hypertension Genetic Epidemiology Network Study, where subjects underwent cardiac CT but did not have dietary assessments. Of the 3360 subjects who had data on cardiac CT, 1084 subjects did not have data on egg consumption at baseline examination (1993–1995), 286 subjects were excluded for prevalent CHD, 68 subjects had missing data on covariates (56 for income; 5 for diabetes; 3 for hypertension; and 4 for physical activity), 18 subjects were non-white, and 56 subjects were excluded for extreme caloric intake ($>$ 4200 and 3500 calories or $<$800 and 600 calories for men and women, respectively). The final sample size for current analyses was 1848. Each participant gave informed consent and the study protocol was reviewed and approved by each of the participating institutions.

2.2. Assessment of egg consumption

Dietary information was collected through a staff-administered semi-quantitative food frequency questionnaire developed by Willett et al. [11]. The reproducibility and validity of the food frequency questionnaire have been documented elsewhere [12,13]. Each subject was asked the following question: "In the past year, how often on average did you consume eggs?" (Item #21 in the questionnaire forms). Possible responses were: almost never, 1–3/month, 1/week, 2–4/week, 5–6/week, 1/day, 2–3/day, 4–6/day, and $>$6/day. Due to sparse data, we collapsed adjacent categories while creating final exposure categories of almost never, 1–3/month, 1/week, and 2+/week for stable estimates.

2.3. Measurement of calcified atherosclerotic plaque in the coronary arteries

Cardiac CT examinations were obtained using General Electric Health Systems LightSpeed Plus and LightSpeed Ultra, Siemens Volume Zoom, or Philips MX 8000 machines. Examinations were performed using the same protocol as employed in the NHLBI's Multi-Ethnic Study of Atherosclerosis [14]. The scans were performed using prospective ECG gating at 50% of the cardiac cycle, 120 kV, 106 mAs, 2.5 mm slice collimation, 0.5 s gantry rotation and a partial scan reconstruction resulting in a temporal resolution of between 250 and 300 ms. Images were reconstructed using the standard algorithm into a 35 cm display field-of-view. All subjects were imaged with a calcium calibration standard within the imaging field (Image Analysis, Columbia, KY). The scan through the heart was repeated after a 1-min pause during the same examination, resulting in two sequential scans for measurement of CAC. The effective radiation exposure for the average participant of each coronary scan was 1.5 mSv for men and 1.9 mSv for women. CT images from all sites were sent electronically to the central CT reading center located at Wake Forest University Health Sciences, Winston Salem, NC. Trained CT analysts using dedicated hardware (GE Advantage Windows Workstation) and software (GE SmarScore) identified CAC in the epicardial coronary arteries and an Agatston score modified to account for slice thickness was calculated using a 130 CT number threshold and a minimum lesion size of 0.9 mm (i.e., 2 pixel connectivity filter). Agatston score refers to the amount of calcium detected by the scan and is based on the area and the density of the calcified plaques [15]. In this report, the sum of the vessel plaque is reported as the total CAC score. Total CAC scores from the first and second measured were then averaged.

2.4. Blood collection and assays

All participants were asked to fast for 12 h before their arrival at the study center. Evacuated tubes without additives were used to collect samples for lipids. Triglyceride concentrations were measured using triglyceride GB reagent on the Roche COBAS FARA centrifugal analyzer (Boehringer Mannheim Diagnostics, Indianapolis). Serum total cholesterol was measured using a commercial cholesterol oxidase method on a Roche COBAS FARA centrifugal analyzer (Boehringer Mannheim Diagnostics, Indianapolis). HDL-cholesterol quantification was performed with the above described cholesterol method after precipitation of non-HDL-cholesterol with magnesium/dextran. For samples with triglyceride concentrations less than 4.5 mmol/L (400 mg/dL), LDL-cholesterol was calculated using the Friedewald formula [16]. For subjects with higher levels of triglycerides, LDL-cholesterol quantitation was performed on EDTA plasma by ultracentrifugation.

2.5. Other variables

Information on cigarette smoking, alcohol intake, and education was obtained by interview during the clinic visit. Resting blood pressure was measured three times on seated participants after a 5-