Atherosclerosis 235 (2014) 273–280

Contents lists available at ScienceDirect

Atherosclerosis

journal homepage: www.elsevier.com/locate/atherosclerosis

Egg consumption and carotid atherosclerosis in the Northern Manhattan Study

Sharon Goldberg [a,*,1], Hannah Gardener [b,1], Eduard Tiozzo [c], Cheung Ying Kuen [d], Mitchell S.V. Elkind [e], Ralph L. Sacco [b], Tatjana Rundek [b]

[a] Division of Hospital Medicine, Miller School of Medicine, University of Miami, Miami, FL, USA
[b] Department of Neurology, Miller School of Medicine, University of Miami, Miami, FL, USA
[c] Department of Psychiatry and Behavioral Sciences, Miller School of Medicine, University of Miami, Miami, FL, USA
[d] Department of Biostatistics, Mailman School of Public Health, Columbia University, New York, NY, USA
[e] Department of Neurology, Columbia University College of Physicians and Surgeons, New York, NY, USA

ARTICLE INFO

Article history:
Received 9 January 2014
Received in revised form
4 April 2014
Accepted 14 April 2014
Available online 30 April 2014

Keywords:
Atherosclerosis
Cholesterol
Egg consumption
Myocardial infarction
Stroke

ABSTRACT

Background: The evidence supporting recommendations to limit intake of cholesterol rich foods is inconclusive. We aimed to examine the association between egg consumption and carotid atherosclerosis phenotypes, and the association with clinical vascular events in a prospective, urban, multi-ethnic population.

Methods and results: The Northern Manhattan Study is a population based cohort to determine stroke incidence, risk factors and prognosis. A sub-cohort of 1429 NOMAS participants with both carotid ultrasounds and comprehensive dietary information was evaluated (mean ± SD age of participants 65.80 ± 8.80, 40% male, 18% white, 20% black, 60% Hispanic). The association between egg consumption and carotid intima media thickness (cIMT) was assessed with linear regression. Logistic and quantile regression was used to examine the association between egg consumption and carotid plaque presence, thickness, and area. The relation between egg consumption and clinical vascular events ($N = 2669$) was examined with Cox models. The mean total cIMT was 0.91 ± 0.08 mm and 58% had carotid plaque present. Increasing egg consumption was inversely associated with cIMT, plaque presence, thickness, and area, in models adjusted for demographics, vascular risk factors and diet. For every additional egg consumed per week, the risk of plaque decreased by 11% (95% CI 3%–18%). No association was detected between egg consumption and risk of clinical vascular outcomes, over a mean follow up of 11 years and after adjustment for covariates.

Conclusions: Frequency of egg consumption in the low to moderate range was inversely related to several markers of carotid atherosclerosis. No association with clinical vascular events, including stroke, was detected. Our findings do not support current vascular health guidelines suggesting the extreme limitation or avoidance of egg consumption due to its cholesterol content.

1. Introduction

Diet is a complex and irrefutable risk factor for cardiovascular disease (CVD). In keeping with the diet-heart hypothesis which invoked dietary cholesterol as a major CVD risk factor, dietary guidelines have advised limiting the intake of cholesterol rich foods. Egg is a significant source of cholesterol, containing an average of 213 mg per egg. Since the 1970's, egg consumption in the United States has been particularly discouraged by health stakeholders in the absence of empirical data. The American Heart Association guidelines no longer restrict egg consumption, but the allotted cholesterol allowance of <200 mg/day for individuals at high risk of CVD, and <300 mg/day for otherwise healthy individuals, precludes significant egg intake when guidelines are followed in the context of an omnivorous western diet. European and Canadian guidelines, in contrast, do not restrict cholesterol, as the literature suggests that saturated and trans-fat restriction is a more effective means of CVD risk reduction [1,2].

* Corresponding author. S Goldberg, Division of Hospital Medicine, 1120 NW 14th Street, Suite 1185, Miami, FL 33136, USA. Tel.: +1 305 243 1960; fax: +1 305 243 1538.
E-mail address: sgoldberg@med.miami.edu (S. Goldberg).
[1] Shared first authorship.

http://dx.doi.org/10.1016/j.atherosclerosis.2014.04.019
0021-9150/

Egg is a low glycemic index, whole-food that has been part of the human diet since early mankind. It is an inexpensive source of protein, essential fatty acids, antioxidants, vitamins, and minerals, and is one of the few dietary sources of choline, a potent antioxidant. Benefits of egg consumption are well described in the literature, including: the formation of larger, less atherogenic LDL and HDL particles [3,4], increased HDL-C formation [5,6], and protection against macular degeneration and cataracts [40–42].

A growing body of evidence supports the claim that egg consumption ≤1/day in healthy individuals is not associated with increased risk of CVD, yet inconsistent data remains. A recent study by Spence et al. described increased carotid plaque area, an imaging biomarker of atherosclerosis, in high risk individuals for CVD, consuming 3 or more eggs per week [7]. The study concluded that regular consumption of egg yolk should be avoided by individuals at high risk for CVD. This study contrasts starkly with the findings of other large, well-controlled, population based cohort studies which concluded that consumption of up to one egg per day does not increase CVD risk [8–10].

Because of conflicting evidence, guidelines, and the declining overall health and nutritional status of North Americans, it is important to understand the effect of egg consumption in a healthy, ethnically diverse population. The relationship between egg consumption and atherosclerosis in particular requires further study. Stroke and other vascular events are etiologically heterogeneous and atherosclerosis is likely an important pathway linking diet with clinical vascular events. Therefore, in this cross sectional study we examined the association between egg consumption with carotid atherosclerosis phenotypes, including carotid plaque cIMT, and with clinical vascular events in an urban, multi-ethnic population-based cohort.

2. Methods

2.1. Study population

NOMAS is a prospective cohort study designed to determine stroke incidence, risk factors, and prognosis in a multi-ethnic urban population. Study details have been published previously [11].

Eligible subjects: a) had never been diagnosed with ischemic stroke; b) were >40 years old; and c) resided in Northern Manhattan for ≥3 months, in a household with a telephone. Subjects were identified by random-digit dialing, and interviews were conducted by trained bilingual research assistants. The telephone response rate was 91%. Subjects were recruited from the telephone sample to have an in-person baseline interview and assessment. The enrollment response rate was 75%, the overall participation rate was 69%, and a total of 3298 subjects were enrolled with an average annual contact rate of 95%. For this study we excluded participants with missing information on egg consumption ($N = 423$) and additionally those with a myocardial infarction prior to baseline ($N = 206$). Of the 3298 NOMAS subjects, 1788 had ultrasound measurements of IMT and carotid plaque. Of these participants, dietary information was lacking on 359 participants, so the study population for the analysis of carotid IMT and plaque included 1429 NOMAS participants with both carotid ultrasounds and diet measured. The study was approved by the Columbia University and University of Miami IRBs and all subjects provided written informed consent.

2.2. Baseline evaluation

Data were collected through interviews with trained bilingual research assistants in English or Spanish. Physical and neurological examinations were conducted by study neurologists. Race-ethnicity was based upon self-identification through a series of questions modeled after the US census and conforming to standard definitions outlined by Directive 15 [12]. Standardized questions were adapted from the Behavioral Risk Factor Surveillance System by the Centers for Disease Control regarding hypertension, diabetes, smoking, and cardiac conditions [13]. The questionnaire included a question about history of stroke and MI among brothers and sisters. Hypertension was defined as a blood pressure ≥140/90 mmHg (based on the average of two measurements during one sitting), the patient's self-reported hypertension, or use of antihypertensive medications. Diabetes mellitus was defined as fasting glucose ≥126 mg/dl, the patient's self-reported diabetes, or use of insulin or oral anti-diabetic medication. Fasting lipid profile was measured at enrollment as previously described [14]. Body mass index (BMI) was examined continuously in kg/m^2. Smoking was categorized as never smoking, former smoking, and current (within the past year) smoking. Physical activity was defined as the frequency and duration of 14 different recreational activities during the 2-week period before the interview, as described previously [15]. Moderate alcohol use was defined as current drinking of >1 drink per month and ≤2 drinks per day.

2.3. Diet

At baseline, participants were administered a modified Block National Cancer Institute food frequency questionnaire by trained research assistants, in English or Spanish [16]. This food frequency questionnaire listed 207 foods (HHHQ version Full87, Form A, Form B) and is intended to represent typical food consumption over the previous year. The questionnaire contained questions regarding the average consumption of eggs, with a medium portion size identified as 2 eggs. The possible responses were: never or <1/month, 1/month, 2–3/month, 1/week, 2/week, 3–4/week, 5–6/week, 1/day, 2+/day.

In order to account for confounding by overall dietary habits, we also included as a covariate a Mediterranean-style diet score, with a higher score on a 0–9 scale representing increasing adherence to a Mediterranean-style diet. In NOMAS, we have previously shown this score to be inversely associated with the risk of vascular events, and details about the calculation of the score have been described [17].

2.4. Carotid ultrasound

High-resolution B-mode ultrasounds (GE LogIQ 700, 9- to 13-MHz linear-array transducer) were performed by trained and certified sonographers as described previously [18]. Presence of plaque is defined as a focal wall thickening or protrusion in the lumen more than 50% greater than the surrounding thickness. Carotid plaque area (mm^2) and thickness (mm) were measured using an automated computerized edge tracking software M'Ath (Paris, France) [19]. Total plaque area (TPA) was defined as the sum of all plaque areas measured in any of the carotid artery segments within an individual. IMT in all carotid segments was measured in areas without plaque. IMT was calculated as a composite measure of IMT in the near and the far walls of the CCA, bifurcation and ICA of both sides of the neck, and examined continuously as a mean of the maximum measurements of the 12 carotid sites. [18]

2.5. Prospective follow-up and clinical outcomes

Annual telephone screening was conducted to determine changes in vital status, detect neurologic events, document interval hospitalizations, and review risk factor status, medication changes, and changes in functional status. Persons who screened positive