Egg consumption and risk of type 2 diabetes in older adults[1–3]

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ABSTRACT

Background: Type 2 diabetes (T2D) remains an important public health issue in the United States. There are limited and inconsistent data on the association between egg consumption and fasting glucose or incident diabetes.

Objectives: We assessed the association between egg intake and incident diabetes in older adults.

Design: In this prospective study of 3898 men and women from the Cardiovascular Health Study (1989–2007), we assessed egg consumption by using a picture-sorted food questionnaire and ascertained incident T2D annually by using information on hypoglycemic agents and plasma glucose. We used Cox proportional hazards models to estimate adjusted relative risks.

Results: During a mean follow-up of 11.3 y, 313 new cases of T2D occurred. Crude incidence rates of T2D were 7.39, 6.83, 7.00, 6.72, and 12.20 per 1000 person-years in people who reported egg consumption of never, <1 egg/mo, 1–3 eggs/mo, 1–4 eggs/wk, and almost daily, respectively. In multivariable-adjusted models, there was no association between egg consumption and increased risk of T2D in either sex and overall. In a secondary analysis, dietary cholesterol was not associated with incident diabetes (*P* for trend = 0.47). In addition, egg consumption was not associated with clinically meaningful differences in fasting glucose, fasting insulin, or measures of insulin resistance despite small absolute analytic differences that were significant.

Conclusion: In this cohort of older adults with limited egg intake, there was no association between egg consumption or dietary cholesterol and increased risk of incident T2D. *Am J Clin Nutr* 2010;92:422–7.

INTRODUCTION

At birth, the lifetime risk of diabetes is 33% for men and 39% for women (1). With the rising obesity epidemic in the United States, type 2 diabetes (T2D) remains a major public health issue with an estimate of $174 billion in direct and indirect costs in 2007 (2–5). Thus, it is critical to identify modifiable risk factors that could help reduce the risk of T2D and its vascular and metabolic consequences. To this end, dietary factors have been shown to influence the risk of T2D (6–12). However, limited data are available on the association between certain dietary components or specific foods and T2D. Eggs are a major source of dietary cholesterol (≈200 mg cholesterol/egg) and contain other important nutrients such as minerals, vitamins, proteins, carotenoids, and saturated (≈1.5 g/egg), polyunsaturated (≈0.7 g/egg), and monounsaturated (≈1.9 g/egg) fatty acids (13, 14). The 2005 Dietary Guidelines (15) and the National Cholesterol Education Program Adult Treatment Panel III (16) recommend a daily intake of <300 mg cholesterol/d for healthy adults and <200 mg cholesterol/d for people with elevated concentrations of LDL. Although several of these nutrients have been associated with an increased risk of T2D [ie, saturated fat and cholesterol (17–19)], other nutrients, such as polyunsaturated fats, may confer a lower risk of T2D (18). In animal experiments, a diet rich in fat was shown to induce hyperglycemia and hyperinsulinemia (20). In addition, a diet enriched with egg yolk, compared with a control diet, was associated with elevated plasma glucose in male, Wistar albino rats (21). However, limited and inconsistent human studies have examined the relation between eggs and glucose metabolism. Data from the Zutphen Study (22) reported a positive association between egg consumption or dietary cholesterol and fasting glucose. A prospective analysis of ≈60,000 men and women suggested a positive association between egg consumption and incident T2D; compared with subjects that did not consume eggs, the multivariable adjusted hazard ratio (95% CI) for T2D was 1.58 (1.25–2.01) in men and 1.99 (1.48–2.67) in women who consumed ≥7 eggs/wk (23). However, in a randomized trial of 28 overweight/obese individuals on a carbohydrate-restricted diet, consumption

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of 3 eggs/d had no effect on fasting glucose concentrations compared with the effect of no intake of eggs (24). Because eggs could serve as a readily available and inexpensive source for vitamins, proteins, and other nutrients in the United States, it is important to elucidate the net effects of egg consumption as a whole food (as opposed to individual component of eggs such as cholesterol) on the risk of T2D. Therefore, the goal of this study was to test the hypothesis that egg consumption is positively associated with incident T2D in older adults. In a secondary hypothesis, we sought to test the hypothesis that dietary cholesterol is positively associated with incident T2D.

SUBJECTS AND METHODS

Design and population

Participants in the study were members of the Cardiovascular Health Study (CHS), a prospective cohort consisting of 5888 men and women, aged ≥65 y, who were randomly selected from Medicare-eligibility lists in 4 US communities (Forsyth County, NC; Sacramento County, CA; Washington County, MD; and Pittsburgh, PA). Study participants were not institutionalized or wheelchair dependent, did not require a proxy for consent, were not under treatment of cancer, and were expected to remain in their respective regions for 3 y. The original cohort recruited 5201 participants between 1989 and 1990. In addition, from 1992 and 1993, a total of 687 African American participants were recruited. The baseline examination included standardized questionnaires, physical examination, anthropometric measurements, resting electrocardiography, and laboratory examinations. Participants were followed up every 6 mo, alternating between telephone calls and clinic visits (1989–1999). A detailed description of methods and procedures in the CHS has been published (25). Each participant gave informed consent, and the institutional review board at each center approved the study. The African American cohort (n = 687) was excluded from the analysis because diet was not ascertained at the 1992–1993 examination. Furthermore, we excluded 824 individuals with prevalent diabetes at baseline, 54 individuals with missing information on egg consumption, 349 individuals with implausible energy values, and 76 people who had no follow-up after the baseline visit. Hence, a final sample of 3898 individuals was used for current analyses.

Identification of T2D

Medication use was assessed at baseline and annually by medication inventory (26), and fasting glucose was measured during the examinations in years 1989–1990, 1992–1993, and 1996–1997. Incident diabetes was defined by new use of insulin or oral hypoglycemic agents or a fasting glucose concentration ≥7 mmol/L (126 mg/dL) or a nonfasting glucose concentration of ≥11.1 mmol/L (200 mg/dL). Individuals who met these criteria were excluded at baseline. A detailed description of the diabetes definition in the CHS was published elsewhere (6).

Assessment of egg consumption and dietary cholesterol

Usual dietary habits were assessed at baseline in the original cohort by using a 99-item picture-sort version of the National Cancer Institute food-frequency questionnaire (27). In addition, dietary assessment was completed again during the sixth annual visit by using the Willett food-frequency questionnaire (28). A detailed description of the validity and computation of nutrients (including dietary cholesterol) and energy intake in this cohort was described previously (27, 29). By using a picture-sort questionnaire, we queried about egg consumption at baseline (item 70 on the picture-sort questionnaire). Possible answers included never, <1 egg/mo, 1–3 eggs/mo, 1–4 eggs/wk, and almost every day. Information on egg consumption was updated during the sixth examination by using the food-frequency questionnaire.

Other covariates

Information on demographics, education, income, prevalent chronic diseases, smoking, alcohol consumption, coffee intake, and current medications was obtained during clinic visits. Weight and height were measured by study staff, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Information on usual walking habits including the average pace and distance walked was assessed at baseline and again annually at each follow-up visit. Leisure-time activity [modified Minnesota Leisure Time Activities questionnaire (30)] and exercise intensity [low, medium, or high (31)] were assessed at the baseline, third, and seventh annual visits. Participants were asked to indicate which activities they had engaged in (how often during the past 2 wk, the average time spent per session, and how many months per year they engaged in the activity). Activities included walking, hiking, jogging, biking, exercise cycle, dancing, aerobics, bowling, golf, swimming, and calisthenics/general exercise. The estimate of physical activity was calculated as kilocalories expended in all physical activities but excluding household chores. As described previously, standard laboratory methods were used to measure serum albumin, lipids, fasting glucose, and insulin (32).

Statistical analyses

Cox proportional hazards models were used to estimate the relative risk of incident diabetes by using the time at risk until diagnosis of diabetes or the last follow-up visit with information on diabetes status. We evaluated categories of egg consumption and quartiles of dietary cholesterol. Multivariable models were adjusted for age, race (black or nonblack), BMI, smoking status (former, never, or current), alcohol consumption (never, former, <7 drinks/wk, or ≥7 drinks/wk), physical activity (in kcal), cereal-fiber intake (energy-adjusted quintiles), and field center. We examined other dietary covariates including fruits and vegetables, red meat, fish (baked/broiled or fried), whole grains, poultry, and fried potatoes, but these did not appreciably alter our results. Tests for trend were performed by fitting egg consumption and dietary cholesterol as ordinal variables into regression models. A potential sex interaction was evaluated by using both stratified analyses and a multiplicative interaction term with a likelihood ratio test. We also updated information on egg consumption and dietary cholesterol (time-varying exposure by using a cumulative update) by using data from the 1995–1996 examination. To evaluate the association with fasting glucose, fasting insulin, and homeostasis model assessment of insulin resistance, we fitted a generalized linear model by using

the same group of covariates as in the Cox models. Analyses were performed with STATA statistical software (version 10.1; StataCorp LP, College Station, TX)

RESULTS

Of the 3898 participants, 42.8% were men and 96.4% were white. The mean age was 73.2 ± 5.7 y (range: 65–95 y) for men and 72.1 ± 5.2 y (range: 65–98 y) for women. Baseline characteristics according to categories of egg consumption are shown in **Table 1**. Subjects with more frequent egg consumption tended to be men, smokers, and to have lower LDL-cholesterol and triglycerides and a higher intake of saturated fats and dietary cholesterol.

During an average follow-up of 11.3 y, 313 incident cases of T2D occurred. Crude incidence rates of T2D were 6.53, 7.34, 8.55, 7.83, and 15.53 cases/1000 person-years for men who reported egg consumption of never, <1 egg/mo, 1–3 eggs/mo, 1–4 eggs/wk, and almost daily, respectively. Corresponding values for women were 7.82, 6.54, 6.19, 5.78, and 6.92 cases/1000 person-years, respectively. In men, compared with no egg consumption, the risk of T2D was increased by 145% in the highest category of egg consumption in a model adjusting for age and ethnicity (**Table 2**). However, in the multivariable adjusted model, the increased risk was attenuated and was no longer significant. There was no evidence of an association between egg consumption and incident T2D in women (Table 2). We showed no evidence of an interaction between sex and egg consumption on T2D risk ($P = 0.44$). Thus, in a sample of men and women combined, multivariable adjusted hazard ratios (95% CI) were 1.0 (reference), 0.81 (0.51, 1.28), 0.84 (0.58, 1.21), 0.79 (0.54, 1.15), and 0.99 (0.52, 1.88) from the lowest to the highest category of egg consumption, respectively (P for trend = 0.42).

There was a suggestive increase in the incidence rate of T2D across quartiles of dietary cholesterol in men (crude incidence rates of 8.17, 8.53, 8.56, and 9.07 cases per 1000 person-years from the lowest to the highest quartiles of dietary cholesterol) but

TABLE 1
Baseline characteristics of 3898 men and women of the Cardiovascular Health Study according to egg consumption[1]

	Egg consumption					
Characteristics	Never	<1/mo	1–3/mo	1–4/wk	Almost daily	*P*
Subjects [*n* (%)]	527 (13.5)	476 (12.2)	1378 (35.4)	1367 (35.1)	150 (3.9)	—
Age at baseline (y)	73.0 ± 5.4[2]	72.0 ± 5.2	72.5 ± 5.3	72.7 ± 5.6	73.1 ± 6.4	0.036
BMI (kg/m^2)	25.5 ± 4.5	25.5 ± 4.0	26.3 ± 4.4	26.3 ± 4.3	26.2 ± 4.7	0.001
Male sex [*n* (%)]	196 (37.2)	181 (38.0)	522 (37.9)	669 (48.9)	101 (67.3)	0.001
Black race [*n* (%)]	22 (4.2)	17 (3.6)	35 (2.5)	54 (4.0)	14 (9.3)	0.001
Smoking status [*n* (%)]						
Never	251 (47.8)	221 (46.4)	638 (46.3)	612 (44.8)	55 (36.7)	0.001
Former	227 (43.2)	198 (41.6)	600 (43.5)	576 (42.2)	62 (41.3)	
Current	47 (9.0)	57 (12.0)	140 (10.2)	178 (13.0)	33 (22.0)	
Pack-years of cigarette smoking	15.9 ± 25.2	16.4 ± 23.2	17.6 ± 25.4	19.6 ± 28.2	23.4 ± 28.3	0.003
Alcohol consumption [*n* (%)]						
Never	240 (45.5)	193 (40.6)	498 (36.2)	449 (32.9)	60 (40.0)	<0.001
Former	33 (6.3)	30 (6.3)	96 (7.0)	106 (7.8)	16 (10.7)	
<7 drinks/wk	184 (34.9)	187 (39.3)	577 (42.0)	563 (41.3)	47 (31.3)	
≥7 drinks/wk	70 (13.3)	66 (13.9)	204 (14.8)	247 (18.1)	27 (18.0)	
Education [*n* (%)]						
<High school	142 (27.0)	98 (20.6)	338 (24.6)	332 (24.4)	62 (41.6)	<0.001
High school or GED	151 (28.7)	137 (28.8)	393 (28.5)	387 (28.4)	35 (23.5)	
>High school	233 (44.3)	240 (50.5)	646 (46.9)	643 (47.2)	52 (34.9)	
Use of blood pressure medication [*n* (%)]	250 (47.4)	187 (39.3)	586 (42.5)	569 (41.6)	44 (29.3)	0.001
Hypertension [*n* (%)]	345 (65.6)	276 (58.2)	838 (60.8)	818 (59.8)	82 (54.7)	0.057
LDL (mg/dL)	135.7 ± 36.5	132.0 ± 32.8	131.2 ± 35.6	128.4 ± 34.1	122.9 ± 32.9	<0.001
HDL (mg/dL)	55.7 ± 16.4	54.8 ± 16.1	55.6 ± 16.3	54.8 ± 15.4	54.1 ± 15.7	0.524
LDL:HDL ratio	2.6 ± 1.0	2.6 ± 1.0	2.5 ± 1.0	2.5 ± 1.0	2.5 ± 1.0	0.085
Triglycerides (mg/dL)	138.1 ± 65.0	143.7 ± 71.7	138.4 ± 72.5	132.8 ± 65.4	120.3 ± 54.3	<0.001
Use of lipid-lowering drugs [*n* (%)]	62 (11.8)	41 (8.6)	53 (3.9)	35 (2.6)	1 (0.7)	<0.001
Physical activity (kcal)	1183 ± 1518	1365 ± 1665	1263 ± 1607	1245 ± 1586	1968 ± 2426	<0.001
Energy intake (kcal/d)	1567 ± 499	1517 ± 493	1604 ± 481	1933 ± 521	2131 ± 612	<0.001
Cereal fiber (g/d)	4.9 ± 1.7	5.0 ± 1.6	5.1 ± 1.6	5.2 ± 1.6	6.0 ± 1.9	<0.001
Dietary cholesterol (mg/d)	168.5 ± 90.3	172.6 ± 80.6	213.2 ± 82.1	446.0 ± 98.0	757.4 ± 117.2	<0.001
Saturated fat (g/d)	17.4 ± 9.0	17.4 ± 8.3	20.5 ± 8.7	28.1 ± 10.3	35.1 ± 12.9	<0.001
Fruit and vegetables (servings/d)	4.3 ± 3.0	4.2 ± 2.6	4.3 ± 2.8	4.3 ± 2.7	4.1 ± 2.6	0.783
Dairy intake [*n* (%)][3]	486 (92.8)	461 (97.1)	1339 (97.3)	1334 (97.7)	145 (96.7)	<0.001
Beef/pork intake [*n* (%)][3]	491 (93.2)	466 (97.9)	1364 (99.0)	1357 (99.3)	150 (100.0)	<0.001
Coffee intake [*n* (%)][3]	446 (84.6)	429 (90.1)	1247 (90.5)	1221 (89.3)	133 (88.7)	0.006

[1] GED, General Educational Development. Values were derived by chi-square tests for categorical variables and ANOVA tests for continuous variables.
[2] Mean ± SD (all such values).
[3] *n* represents number of subjects who consumed dairy, beef/pork, and coffee.

TABLE 2
Type 2 diabetes according to egg consumption

	No. of events		Incidence rate		Hazard ratio (95% CI)[1]		Hazard ratio (95% CI)[2]	
Egg intake	Men (n = 1669)	Women (n = 2229)	Men (n = 1669)	Women (n = 2229)	Men (n = 1669)	Women (n = 2229)	Men (n = 1669)	Women (n = 2229)
Never	13	31	6.53	7.82	1.0	1.0	1.0	1.0
<1/mo	15	24	7.34	6.54	1.05 (0.50, 2.21)	0.82 (0.48, 1.40)	0.95 (0.45, 2.01)	0.77 (0.43, 1.38)
1–3/mo	46	64	8.55	6.19	1.28 (0.69, 2.37)	0.79 (0.51, 1.21)	1.14 (0.60, 2.15)	0.73 (0.47, 1.14)
1–4/wk	54	48	7.83	5.78	1.15 (0.63, 2.10)	0.73 (0.47, 1.15)	0.96 (0.50, 1.82)	0.76 (0.47, 1.23)
Almost daily	14	4	15.53	6.92	2.45 (1.15, 5.22)	0.78 (0.27, 2.20)	1.81 (0.77, 4.22)	0.38 (0.10, 1.37)
P for trend	—	—	—	—	—	—	0.57	0.16

[1] Adjusted for age and race by using Cox proportional hazards regression.
[2] Adjusted for age, race (black or nonblack), BMI, smoking status (former, never, or current), alcohol consumption (never, former, <7 drinks/wk, or ≥7 drinks/wk), physical activity (in kcal), cereal-fiber intake (energy-adjusted quintiles), and field center by using Cox proportional hazards regression.

not in women (corresponding crude rates of 7.71, 5.67, 6.15, and 6.31) (**Table 3**). However, after adjustment for potential confounders, there was no evidence for an association between dietary cholesterol and incident T2D in either sex (Table 3) or combined data [multivariable adjusted hazard ratios (95% CI) were 1.0 (reference), 0.90 (0.62, 1.31), 0.88 (0.60, 1.28), and 0.84 (0.53, 1.34) from the lowest to the highest quartiles of dietary cholesterol].

With the use of egg consumption and dietary cholesterol obtained during the sixth examination to update each exposure over time, similar findings were yielded: multivariable adjusted hazard ratios (95% CI) for T2D in a combined sample of men and women were 1.0 (reference), 1.03 (0.74–1.45), 1.11 (0.80, 1.53), and 1.27 (0.74, 2.19) from the lowest to the highest categories of egg consumption, respectively, and 1.0 (reference), 0.97 (0.69, 1.38), 1.11 (0.78, 1.56), and 1.13 (0.77, 1.65) from the lowest to the highest quartiles of dietary cholesterol, respectively.

Last, in cross-sectional analyses, egg consumption was not associated with clinically meaningful differences in fasting glucose, fasting insulin, or measures of insulin resistance despite small absolute analytic differences that were significant (**Table 4**).

DISCUSSION

In this prospective cohort of ≈4000 older adults, we showed no evidence of an association between dietary cholesterol and incident T2D. When stratified by sex, egg consumption was not associated with incident T2D in women. However, in a multivariable adjusted Cox regression model, there was an increased risk of T2D (albeit not significant) in men who reported an almost daily consumption of eggs compared with men who did not consume eggs.

Very few studies have examined the association between egg consumption and incident T2D in free-living populations. In older women, we observed an adjusted hazard ratio (95% CI) of 0.38 (0.10, 1.37) in the highest category of egg intake (almost daily) and 0.70 (0.36, 1.35) in the highest quartile of dietary cholesterol; in contrast, a report from 36,295 female health professionals (23) showed an increased risk of T2D with higher consumption of eggs [hazard ratio = 1.77 (95% CI: 1.28, 2.43) when the highest category of egg intake was compared with lowest category of egg intake] and dietary cholesterol [hazard ratio = 1.28 (95% CI: 1.10, 1.50) when the highest quintile of dietary cholesterol was compared with the lowest quintile of dietary cholesterol]. Because 95% CIs overlapped between the 2 groups of women, we could not exclude a small increased risk of T2D in our female sample. We had only 4 events in the highest category of egg intake in women. A suggestive increased risk of T2D in men who consumed eggs almost every day in the current study is in line with an elevated risk of T2D observed in US male physicians in the highest category of egg consumption (23). Unfortunately, data on dietary cholesterol were not available in the Physicians' Health Study (23) to compare with the current findings. The different results between the 2 studies merit some considerations.

TABLE 3
Type 2 diabetes according to sex-specific quartiles of dietary cholesterol

	No. of events		Incidence rate		Hazard ratio (95% CI)[2]		Hazard ratio (95% CI)[3]	
Quartiles of dietary cholesterol[1]	Men (n = 1466)	Women (n = 1974)	Men (n = 1466)	Women (n = 1974)	Men (n = 1466)	Women (n = 1974)	Men (n = 1466)	Women (n = 1974)
124.9 (18.9–163.3)	22	57	8.17	7.71	1.0	1.0	1.0	1.0
205.4 (163.4–262.1)	31	36	8.53	5.67	1.02 (0.59, 1.76)	0.71 (0.47, 1.08)	1.17 (0.63, 2.17)	0.76 (0.47, 1.25)
345.2 (262.2–414.9)	33	36	8.56	6.15	1.03 (0.60, 1.76)	0.78 (0.51, 1.18)	1.16 (0.62, 2.17)	0.78 (0.47, 1.27)
501.2 (415.1–1133.3)	44	26	9.07	6.31	1.10 (0.66, 1.84)	0.78 (0.49, 1.24)	1.12 (0.56, 2.26)	0.70 (0.36, 1.35)
P for trend	—	—	—	—	—	—	0.82	0.30

[1] Values are medians; ranges in parentheses.
[2] Adjusted for age and race by using Cox proportional hazards regression.
[3] Adjusted for age, race (black or nonblack), BMI, smoking status (former, never, or current), alcohol consumption (never, former, <7 drinks/wk, or ≥7 drinks/wk), physical activity (in kcal), cereal-fiber intake (energy-adjusted quintiles), and field center by using Cox proportional hazards regression.

TABLE 4
Multivariable-adjusted means of fasting glucose, fasting insulin, and homeostasis model assessment of insulin resistance (HOMA-IR) according to egg consumption[1]

	Egg consumption					
	Never (n = 527)	<1/mo (n = 476)	1–3/mo (n = 1378)	1–4/wk (n = 1367)	Almost every day (n = 150)	P
Fasting glucose (mg/dL)	99.5 ± 3.5	99.6 ± 3.2	99.6 ± 3.4	100.6 ± 3.4	100.6 ± 3.5	<0.001
Fasting insulin (IU/mL)	13.9 ± 3.4	13.0 ± 2.9	13.6 ± 3.3	14.3 ± 3.2	14.0 ± 3.8	<0.001
HOMA-IR	3.5 ± 1.0	3.2 ± 0.8	3.4 ± 0.9	3.6 ± 0.9	3.5 ± 1.1	<0.001

[1] All values are means ± SDs adjusted for age, sex, race (black or nonblack), BMI, smoking status (former, never, or current), alcohol consumption (never, former, <7 drinks/wk, or ≥7 drinks/wk), physical activity (in kcal), cereal-fiber intake (energy-adjusted quintiles), and field center by using linear regression. P values were derived by ANOVA.

The CHS enrolled participants who were ≥65 y old at baseline, whereas the average age at enrollment in the Physicians' Health Study or Women's Health Study was ≈54 y (23). It is possible that, because of a natural selection process, CHS participants were more likely to have been depleted from subjects susceptible to adverse effects of eggs/cholesterol on T2D risk and, thus, represented healthy survivors. It is known that the individual response to dietary cholesterol is not uniform (33–35). For example, because hyperresponders to dietary cholesterol are more likely to develop fatal atherosclerotic events, the proportion of hyperresponders could have been drastically reduced in the CHS sample than in a younger cohort. Unfortunately, we do not have additional information on the response pattern to dietary cholesterol to test such hypothesis in the current study. Furthermore, the median egg consumption in the CHS sample was <1 egg/wk compared with 1 egg/wk in the Physicians' health Study and the Women's Health Study (23). Because an increased risk of T2D in these cohorts was mostly observed with consumption of ≥7 eggs/wk, our cohort may not have contained adequate numbers of individuals with such high exposure to egg consumption to observe adverse effects. Nevertheless, our findings do not support adverse effects on T2D or glucose-insulin homeostatis at lower amounts of egg consumption. It is less likely that the suggestive increased risk of T2D with egg consumption in men compared with women in the current study was due to sex-specific preferences with diet because additional adjustment for elements of poor diet that may be associated with egg consumption (eg, intakes of bacon, sausages, burgers, french fries, and dairy) did not alter the results. Furthermore, we did not observe a sex-by-egg interaction in our study. Although subjects who reported frequent egg consumption tended to have lower LDL-cholesterol and a lower percentage of lipid-lowering treatment than did subjects with infrequent egg consumption, additional adjustment for lipid treatment did not alter the results.

Insofar as our results do not contradict the previously reported association between daily egg consumption and T2D, little is yet known about potential biological mechanisms that may explain such an association. Animal experiments have reported that a diet rich in fat can induce hyperglycemia and hyperinsulinemia (20). In addition, feeding animals egg yolk was associated with hyperglycemia (21). A high-fat diet was associated with increased plasma glucose and insulin concentrations in rats after 66 d of intervention (36). However, in a randomized trial of overweight individuals, consumption of 3 eggs/d had no effect on plasma concentrations of fasting glucose (24). These findings are consistent with the current study in which egg consumption was not associated with clinically meaningful differences in fasting glucose or insulin concentrations or measures of insulin resistance despite small absolute analytic differences that were significant.

Our study has some limitations. We could not specifically evaluate the potential effects of high amounts (daily or more) of egg consumption because few people consumed such amounts in our cohort. Despite careful control of confounding by multiple other risk factors, we could not exclude residual confounding or chance as an explanation for the current results. We did not have information on other foods usually consumed with eggs by study participants. Furthermore, details on the methods of egg preparation (eg, boiled, fried, and poached) were not available in this study. Dietary and/or diabetes misclassification may have occurred and could have biased the results toward the null. Our findings may not be generalizable to younger populations if the biologic effects of egg or dietary cholesterol consumption differ at older ages.

Our investigation also has several strengths. Our cohort was a well-established prospective cohort that used standardized procedures to collect data on exposure and outcomes and, thus, minimized recall bias. We had sufficient numbers of events to provide a reasonable statistical power to detect clinically meaningful associations. Cross-sectional analyses of glucose-insulin biomarkers and prospective analyses of incident diabetes consistently support the lack of a major adverse effect of limited egg consumption on glucose/insulin metabolism. The long-term follow up, the robustness of the finding when egg/cholesterol intake was updated over time, and the availability of numerous covariates to minimize confounding are additional strengths of our study.

In conclusion, we observed no evidence of a significant association between occasional or almost daily egg consumption or dietary cholesterol and incident T2D in older adults.

We are indebted to the participants and staff of the CHS. A full list of participating CHS investigators and institutions can be found at http://www.chs-nhlbi.org.

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