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Original Research

Eggs and a Fiber-Rich Diet Are Beneficially Associated with Lipid Levels in Framingham Offspring Study Adults

Xinyi Zhou [1,†], Melanie M Mott [1,2,†], Ioanna Yiannakou [1], M Loring Bradlee [1], Martha R Singer [1], Lynn L Moore [1,*]

[1] *Preventive Medicine and Epidemiology, Department of Medicine, Boston University Chobanian & Avedisian School of Medicine, Boston, MA, United States;* [2] *DynaMed at Elton Bryson Stephens Company (EBSCO) Information Services, Ipswich, MA, United States*

ABSTRACT

Background: For many years, United States' dietary policy recommended limiting egg intake to no more than 3/wk in the belief that restricting dietary cholesterol would lower plasma cholesterol levels and thereby reduce the risk of cardiovascular disease. The evidence supporting these recommendations is controversial.

Objectives: To examine the impact of eggs, a major contributor to dietary cholesterol intake, on lipid levels and to determine whether these egg effects are modified by other healthy dietary factors in adults.

Methods: Males and females aged 30–64 y with available 3-d diet record data, without cardiovascular disease and not taking lipid- or glucose-lowering medications in the prospective Framingham Offspring cohort were included (n = 1852). Analysis of covariance models were used to compare mean follow-up lipid levels adjusting for age, sex, BMI, and dietary factors. Cox proportional hazard's models were used to estimate risk for elevated lipid levels.

Results: Consuming ≥5 eggs/wk was not adversely associated with lipid outcomes. Among men, consuming ≥5 (compared with <0.5) eggs/wk was associated with an 8.6 mg/dL lower total cholesterol level and a 5.9 mg/dL lower LDL cholesterol level, as well as lower triglycerides. Overall, higher egg intake combined with higher dietary fiber (compared with lower intakes of both) was associated with the lowest total cholesterol, LDL cholesterol, and LDL cholesterol–to–HDL cholesterol ratio. Finally, diets with higher (compared with lower) egg intakes in combination with higher total fish or fiber intakes, respectively, were associated with lower risks of developing elevated (>160 mg/dL) LDL cholesterol levels (hazard ratio: 0.61; 95% confidence interval: 0.44, 0.84; and HR: 0.70; 95% confidence interval: 0.49, 0.98, respectively).

Conclusions: Higher egg intakes were beneficially associated with serum lipids among healthy adults, particularly those who consumed more fish and dietary fiber.

Keywords: Eggs, Dietary fiber, Fish, Healthy diet, Lipids, Dyslipidemia, Prospective Study

Introduction

Abnormal lipid levels, particularly elevated levels of LDL cholesterol, are a major risk factor for cardiovascular disease (CVD) [1]. For years, recommendations to limit dietary cholesterol to prevent CVD have been a fundamental component of diet policy in the United States, based on the belief that restricting dietary cholesterol lowers plasma cholesterol levels and LDL cholesterol, thereby reducing the risk of atherosclerotic CVD [2]. These recommendations were based on several lines of evidence [3], including animal studies with supraphysiologic doses of dietary cholesterol [4], epidemiologic studies of dietary cholesterol and ischemic heart disease [5], and some early clinical trials, often involving high doses of dietary cholesterol [6].

These early studies of dietary cholesterol or egg intakes often failed to consider other components of the participants' eating

Abbreviations: CVD, cardiovascular disease; TC, total cholesterol; TMAO, trimethylamine-N-oxide.

* Corresponding author. *E-mail address:* llmoore@bu.edu (L.L. Moore).

† Xinyi Zhou and Melanie M Mott are co-first authors

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patterns. Over time, some investigators concluded that the evidence for dietary cholesterol restriction and lowering of LDL cholesterol was insufficient [7]. As a result, more recent guidelines from the American Heart Association and others have eliminated some of the specific guidance on dietary cholesterol intake [1]. Starting in 2015, the Dietary Guidelines Advisory Committee also determined, based on existing evidence and conclusions from the American Heart Association and the American College of Cardiology, that there was insufficient evidence to retain the existing guidelines restricting dietary cholesterol intake [7–9]. This placed the United States in line with other countries that have removed dietary cholesterol restrictions [10,11].

On the basis of the 2020–2025 Dietary Guidelines for Americans, eggs are considered to be rich in nutrients and may be valuable components in a healthy dietary pattern [12]. It has been shown that some individuals eating more eggs may have higher intakes of total protein and better overall diet quality, including higher intakes of fish and plant protein, total vegetables, total fruit, whole fruit, whole grains, and dairy [13]. These healthier diet patterns can help to lower the risk of diet-related chronic illnesses such as type 2 diabetes, CVD, and certain cancers [14]. Furthermore, analysis of data from the Framingham Offspring Cohort found that higher egg consumption was associated with lower long-term risks of high blood pressure and type 2 diabetes [15]. These effects were strongest among individuals with healthier diet patterns. Finally, it is also possible that eggs may be consumed as a part of a less healthy dietary pattern, and in this situation, there may be an adverse effect on lipid levels and other outcomes [16–18]. Thus, it is important that we consider other dietary factors in determining the effects of eggs on outcomes, such as blood lipids. The goal of this study is to examine the association of egg consumption, alone and as a part of a healthy diet pattern, with fasting lipid levels among adults in the Framingham Offspring Cohort.

Methods

Participants

This study began in 1971 with the enrollment of 5124 offspring (and their spouses) of the Framingham Original Cohort [19]. Participants are assessed at ~4-y intervals for CVD and CVD risk factors, including fasting blood lipids. Three-day diet records were used to assess diet in the third (1984–1988) and the fifth (1991–1995) examination visits. For individuals with dietary records at both visits, mean egg intake was estimated. If individuals only had dietary records at 1 examination, then egg intake from that examination was used. For a majority of the participants, examination 3 served as the baseline visit, whereas examination 5 served as the baseline visit for individuals missing examination 3 dietary data. The present analyses were approved by the Boston University Institutional Review Board and used data from the prospective Framingham Offspring cohort.

For these analyses, 4565 participants survived and were between 30 and 64 y of age at the time of the dietary assessment and returned for follow-up after the baseline dietary examinations. Of the 4565, 3072 (67.3%) completed the dietary records. We further excluded 257 individuals with extreme energy intake values (<1200 or >4000 kcal/d for men; <1000 or >3500 kcal/d for women) or extreme egg intakes (>35 eggs/wk). We further excluded participants who had a history of cancer ($n = 136$), were taking lipid-lowering drugs at baseline ($n = 72$), had a BMI <18.5 kg/m^2 ($n = 22$), had heavy alcohol intakes (>20% of calories/d, $n = 61$), or who were missing data for lipids or important confounders retained in the final multivariable models ($n = 491$). Finally, we excluded 203 participants with prevalent CVD at the beginning of follow-up, leaving 1852 participants for the analyses of continuous lipid outcomes. Additionally, individuals with prevalent high total cholesterol (TC) (>240 mg/dL, $n = 425$), LDL cholesterol (>160 mg/dL, $n = 378$), or triglycerides (>200 mg/dL, $n = 197$) at baseline (even though not taking lipid-lowering medications) were excluded from the respective Cox proportional hazard's models, leaving a final sample of 1427 participants for the evaluation of incident high TC risk, 1474 for the assessment of incident high LDL cholesterol risk, and 1655 for the evaluation of incident high triglyceride levels.

Dietary assessment

Roughly 70% of participants completed food records, resulting in ~16,000 d of dietary data. The collected dietary data were entered into the Nutrient Data System, which was developed at the University of Minnesota's Nutrition Coordinating Center for the calculation of mean intakes of macro- and micronutrients [20]. Researchers from Boston University linked the underlying food codes from Nutrient Data System with the USDA food serving data for eggs and other foods using previously described methods [21]. Nutrients and food serving were extracted from whole foods, composite foods, and mixed dishes.

Main outcome measures

Blood specimens were drawn following an overnight fast and analyzed as previously described using standard methods [22]. For these analyses, TC, LDL cholesterol, LDL cholesterol–to–HDL cholesterol ratio, and triglycerides were derived from the baseline visit, and 4 y later. Incident cases of elevated lipid levels were defined using the following standard definitions for high TC (>240 mg/dL) and high LDL cholesterol (>160 mg/dL) [23], as well as high triglycerides, >200 mg/dL [24]. Follow-up for elevated lipid levels started at the time of baseline dietary assessment and continued until the first of the following: *1*) incident high TC (for TC analyses), incident high LDL cholesterol (for LDL analyses), or incident high triglycerides (for triglyceride analyses) for the 3 respective analyses, *2*) start of lipid-lowering medication use, *3*) loss to follow-up, *4*) end of follow-up, or *5*) death.

Potential confounders

There were a number of potential confounders considered in the multivariable models: age, sex, baseline BMI, percent of energy from saturated fat, percent energy from total fat, fruit and nonstarchy vegetables, whole grains, the Healthy Eating Index, and intakes of solid fats, alcohol, and added sugars. Other factors such as physical activity (a composite score of the number of self-reported hours performing moderate and vigorous activities), cigarette smoking, percent energy from carbohydrates, and other dietary factors were explored as potential confounders but not retained in the final models because they changed the effect estimates <5%.

Statistical analyses

Egg intake was classified as follows: 0 to <0.5 eggs/wk (n = 316), 0.5 to <5.0 eggs/wk (n = 1199), and ≥5.0 eggs/wk (n = 337). Dichotomous cutoff values for lower compared with higher intakes of food groups were selected after considering distributions of intake: eggs, <3 compared with ≥3 per week; total dairy, <1.75 compared with ≥1.75 cup-equivalents/d; total fish, <7 compared with ≥7 oz/wk; whole grains, <0.5 compared with 0.5 ounce-equivalents/d; fiber, <17 compared with ≥17 g/d; fruits and nonstarchy vegetables, <3 compared with ≥3 cup-equivalents/d.

Analysis of covariance modeling was used to compare the adjusted mean follow-up lipid values across categories of egg intake as well as categories of egg intake in combination with intakes of other foods reflecting healthy diet patterns such as whole grains, fiber, dairy, fruit and nonstarchy vegetables, and fish. BMI was assessed as a potential intermediate variable and, was additionally examined in stratified analyses (baseline BMI: <25 compared with ≥25 kg/m^2). Sex-stratified analysis of covariance models were also explored.

Cox proportional hazard's models were used to estimate the adjusted hazard ratio and 95% confidence interval for long-term risk of developing high TC, high LDL cholesterol, or high triglycerides associated with egg intake. In stratified analyses, the association between egg intake and each dichotomous lipid outcome was examined independently and in combination with healthy dietary factors. Statistical Analysis Systems software, version 9.4 (SAS Institute), was used to perform all analyses.

Results

Baseline characteristics of participants according to their usual egg intake per week are shown in Table 1. Individuals, who consumed <0.5 eggs/wk, were more likely to be female; they tended to consume a higher percentage of energy from carbohydrates and a lower percentage of intake from total fat, particularly saturated fat intake. Those with an intake of ≥5 eggs/wk had a slightly higher baseline BMI. In addition, this level of egg intake was associated with higher overall energy intake, lower percentages of energy from protein and carbohydrates, lower consumption of fruits and nonstarchy vegetables, and higher intakes of dietary cholesterol.

Table 2 shows that egg intake overall was not associated with lipid outcomes (i.e., TC, LDL cholesterol, LDL cholesterol–to–HDL cholesterol ratio, log of triglycerides). Participants with overweight (BMI ≥25 kg/m^2) generally had higher lipid levels than those with a BMI <25 kg/m^2, but there were no adverse associations with egg consumption, regardless of body weight. Among men, however, higher egg intakes were associated with a TC level that was 8.6 mg/dL lower (P for trend = 0.010) than that of men with the lowest egg intakes. In addition, those with the highest egg consumption had an LDL cholesterol level that was 5.9 mg/dL lower (P = 0.038). Similarly, higher egg intakes were associated with the lowest triglyceride levels among men. There was no association between egg intake and these outcomes in women.

The effects of egg intake as part of healthy dietary patterns on mean lipid outcomes are shown in Table 3. In particular, higher

TABLE 1
Baseline characteristics of participants according to egg consumption

Baseline characteristics	Weekly egg consumption			
	<0.5	0.5 to <5	≥5	
	(n = 316)	(n = 1199)	(n = 337)	
		Mean ± SE[1]		P-trend
Age (y)	49.3 ± 0.49	48.2 ± 0.25	47.9 ± 0.48	0.055
Height (inches)	66.4 ± 0.14	66.4 ± 0.07	66.7 ± 0.14	0.127
BMI (kg/m^2)	26.1 ± 0.25	25.9 ± 0.13	26.8 ± 0.24	0.035
Physical activity index (metabolic-eq/d)	12.6 ± 0.45	12.5 ± 0.23	13.3 ± 0.44	0.299
Dietary intakes				
Energy intake (kcals/d)	1731 ± 25	1926 ± 13	2067 ± 25	<0.0001
Dietary cholesterol (mg)	172 ± 4.42	243.6 ± 2.27	401.9 ± 4.3	<0.0001
% calories from protein	17.6 ± 0.18	16.8 ± 0.09	16.4 ± 0.18	<0.0001
% calories from carbohydrate	47.7 ± 0.44	46.2 ± 0.23	43.9 ± 0.43	<0.0001
% calories from fat	32.8 ± 0.36	35.2 ± 0.18	38 ± 0.35	<0.0001
% calories from saturated fat	10.9 ± 0.16	12.1 ± 0.08	13.3 ± 0.16	<0.0001
Fruit and nonstarchy vegetables (cup-eq/d)	2.7 ± 0.08	2.6 ± 0.04	2.3 ± 0.08	0.002
Whole grains (oz-eq/d)	0.6 ± 0.04	0.6 ± 0.02	0.5 ± 0.04	0.043
Dairy (cup-eq/d)	1.3 ± 0.05	1.4 ± 0.03	1.4 ± 0.05	0.161
Dietary fiber (g/d)	16.3 ± 0.34	16.2 ± 0.17	15.5 ± 0.33	0.077
Baseline lipids				
Total cholesterol (mg/dL)	202.7 ± 1.88	204.9 ± 0.96	202.5 ± 1.83	0.912
LDL cholesterol (mg/dL)	127.9 ± 1.67	131 ± 0.85	127 ± 1.62	0.655
LDL cholesterol–to–HDL cholesterol ratio	2.7 ± 0.05	2.7 ± 0.03	2.6 ± 0.05	0.567
Log of triglycerides	4.6 ± 0.03	4.5 ± 0.01	4.6 ± 0.03	0.800
Percent of subjects		N (column %)		
Male	114 (36.1)	513 (42.8)	198 (58.8)	<0.0001
Smoker (current)	68 (21.5)	252 (21.0)	81 (24.0)	0.493
More than high school	158 (57.3)	690 (64.1)	182 (61.3)	0.102

[1] Adjusted for sex.

TABLE 2
Egg intake and adjusted mean fasting lipid levels, overall and stratified by baseline BMI and sex

Eggs per week (ages 30 to <65 y)	All subjects		BMI < 25		BMI ≥ 25		Females		Males	
	N	Mean ± SE[1]	*N*	Mean ± SE[1]	*N*	Mean ± SE[1]	*N*	Mean ± SE[1]	*N*	Mean ± SE[1]
Total cholesterol (mg/dL)										
<0.5	316	203.1 ± 1.87	145	196.5 ± 2.60	171	208.2 ± 2.62	202	202.4 ± 2.27	114	204.6 ± 3.10
0.5 to <5	1199	203.8 ± 0.95	571	199.0 ± 1.31	628	207.7 ± 1.36	686	203.7 ± 1.23	513	204.1 ± 1.46
≥5	337	200.3 ± 1.82	120	197.2 ± 2.89	217	203.6 ± 2.33	139	205.4 ± 2.75	198	196.0 ± 2.35
P-trend		0.273		0.801		0.168		0.413		0.010
LDL cholesterol (mg/dL)										
<0.5	316	128.5 ± 1.71	145	121.1 ± 2.46	171	134.4 ± 2.34	202	124.5 ± 2.09	114	133.9 ± 2.84
0.5 to <5	1199	129.3 ± 0.87	571	123.4 ± 1.24	628	134.2 ± 1.22	686	125.1 ± 1.14	513	134.8 ± 1.33
≥5	337	126.0 ± 1.67	120	119.7 ± 2.73	217	131.5 ± 2.08	139	125.5 ± 2.54	198	128.0 ± 2.15
P-trend		0.275		0.791		0.326		0.733		0.038
HDL cholesterol (mg/dL)										
<0.5	316	51.4 ± 0.74	145	57.1 ± 1.12	171	46.9 ± 0.92	202	57.3 ± 0.98	114	44.0 ± 1.02
0.5 to <5	1199	52.0 ± 0.38	571	57.5 ± 0.56	628	47.3 ± 0.48	686	57.7 ± 0.53	513	44.7 ± 0.48
≥5	337	51.3 ± 0.72	120	59.4 ± 1.24	217	45.7 ± 0.82	139	58.8 ± 1.19	198	43.8 ± 0.77
P-trend		0.605		0.145		0.439		0.353		0.666
Log-transformed triglycerides										
<0.5	316	4.61 ± 0.03	145	4.39 ± 0.04	171	4.78 ± 0.04	202	4.50 ± 0.03	114	4.76 ± 0.05
0.5 to <5	1199	4.58 ± 0.01	571	4.39 ± 0.02	628	4.74 ± 0.02	686	4.51 ± 0.02	513	4.67 ± 0.02
≥5	337	4.56 ± 0.03	120	4.37 ± 0.04	217	4.73 ± 0.04	139	4.51 ± 0.04	198	4.63 ± 0.04
P-trend		0.179		0.709		0.403		0.852		0.042
LDL cholesterol–to–HDL cholesterol ratio										
<0.5	316	2.72 ± 0.05	145	2.27 ± 0.07	171	3.08 ± 0.08	202	2.34 ± 0.06	114	3.20 ± 0.10
0.5 to <5	1199	2.70 ± 0.03	571	2.31 ± 0.03	628	3.02 ± 0.04	686	2.32 ± 0.03	513	3.18 ± 0.04
≥5	337	2.65 ± 0.05	120	2.14 ± 0.08	217	3.06 ± 0.07	139	2.30 ± 0.07	198	3.09 ± 0.07
P-trend		0.289		0.262		0.971		0.668		0.285

[1] Adjusted for age, sex (except in sex-specific models), whole grains, fruit, and nonstarchy vegetables, and BMI (except in BMI-stratified models).

TABLE 3
Egg-related dietary patterns and adjusted mean fasting lipid levels

Dietary patterns[1]	Total cholesterol (mg/dL)			LDL cholesterol (mg/dL)		HDL cholesterol (mg/dL)		Log (triglycerides)		LDL cholesterol–to–HDL cholesterol ratio	
	N	Mean ± SE[2]	*P* value	Mean ± SE[2]	*P* value	Mean ± SE[2]	*P* value	Mean ± SE[2]	*P* value	Mean ± SE[2]	*P* value
Eggs/dairy											
Lower/lower	887	204.7 ± 1.12	—	130.0 ± 1.02	—	51.9 ± 0.43	—	4.60 ± 0.02	—	2.73 ± 0.03	—
Lower/higher	305	201.2 ± 1.90	0.109	127.3 ± 1.73	0.179	51.1 ± 0.72	0.357	4.59 ± 0.03	0.852	2.70 ± 0.05	0.697
Higher/lower	453	202.0 ± 1.56	0.152	127.3 ± 1.42	0.120	52.1 ± 0.59	0.727	4.57 ± 0.02	0.378	2.66 ± 0.04	0.195
Higher/higher	207	200.5 ± 2.33	0.106	127.2 ± 2.13	0.239	51.9 ± 0.89	0.996	4.54 ± 0.03	0.154	2.63 ± 0.07	0.191
Eggs/total fish											
Lower/lower	614	203.6 ± 1.34	—	129.0 ± 1.23	—	51.2 ± 0.51	—	4.63 ± 0.02	—	2.74 ± 0.04	—
Lower/higher	578	204.0 ± 1.38	0.837	129.6 ± 1.26	0.713	52.1 ± 0.53	0.202	4.56 ± 0.02	0.008	2.70 ± 0.04	0.402
Higher/lower	340	202.0 ± 1.80	0.473	127.6 ± 1.65	0.488	51.6 ± 0.69	0.657	4.59 ± 0.03	0.189	2.65 ± 0.05	0.153
Higher/higher	320	201.1 ± 1.86	0.266	127.0 ± 1.70	0.332	52.5 ± 0.71	0.131	4.54 ± 0.03	0.005	2.65 ± 0.05	0.135
Eggs/whole grains											
Lower/lower	660	206.4 ± 1.29	—	131.7 ± 1.18	—	51.5 ± 0.49	—	4.62 ± 0.02	—	2.78 ± 0.04	—
Lower/higher	532	200.7 ± 1.43	0.003	126.4 ± 1.31	0.003	51.9 ± 0.55	0.636	4.57 ± 0.02	0.088	2.65 ± 0.04	0.013
Higher/lower	388	202.4 ± 1.69	0.064	127.4 ± 1.54	0.029	52.8 ± 0.64	0.101	4.56 ± 0.02	0.052	2.63 ± 0.05	0.010
Higher/higher	272	200.3 ± 2.01	0.012	127.1 ± 1.84	0.037	50.9 ± 0.77	0.513	4.57 ± 0.03	0.210	2.68 ± 0.06	0.131
Eggs/fiber											
Lower/lower	745	205.0 ± 1.23	—	130.5 ± 1.12	—	51.4 ± 0.47	—	4.61 ± 0.02	—	2.77 ± 0.03	—
Lower/higher	447	202.0 ± 1.58	0.134	127.4 ± 1.44	0.098	52.1 ± 0.60	0.311	4.57 ± 0.02	0.165	2.65 ± 0.04	0.031
Higher/lower	421	202.7 ± 1.62	0.257	128.1 ± 1.48	0.192	52.2 ± 0.62	0.271	4.57 ± 0.02	0.198	2.68 ± 0.05	0.117
Higher/higher	239	199.5 ± 2.18	0.030	125.8 ± 1.99	0.043	51.7 ± 0.83	0.697	4.54 ± 0.03	0.075	2.60 ± 0.06	0.015
Eggs/fruit and nonstarchy vegetables											
Lower/lower	800	204.5 ± 1.17	—	129.9 ± 1.07	—	51.5 ± 0.45	—	4.61 ± 0.02	—	2.75 ± 0.03	—
Lower/higher	392	202.4 ± 1.68	0.312	128.2 ± 1.54	0.371	52.0 ± 0.64	0.514	4.57 ± 0.02	0.161	2.66 ± 0.05	0.124
Higher/lower	475	201.5 ± 1.53	0.119	126.6 ± 1.40	0.062	52.2 ± 0.58	0.331	4.58 ± 0.02	0.246	2.64 ± 0.04	0.047
Higher/higher	185	201.8 ± 2.44	0.313	129.1 ± 2.23	0.762	51.6 ± 0.93	0.910	4.53 ± 0.04	0.045	2.67 ± 0.07	0.288

[1] Lower vs. higher egg intake: <3 vs. ≥3 per week; lower vs. higher dairy intake: <1.75 vs. ≥1.75 cup-eq/d; lower vs. higher total fish intake, <7 vs. ≥7 oz/wk; lower vs. higher whole grain intake, <0.5 vs. ≥0.5 oz-eq/d; lower vs. higher fiber intake, <17 vs. ≥ 17 g/d; lower vs. higher fruit and nonstarchy vegetable intake, <3 vs. ≥3 cups/d.
[2] Adjusted for age, sex, and BMI.

egg intake combined with higher intakes of dietary fiber was associated with a 5.5 mg/dL ($P = 0.030$) lower TC level, a 4.7 mg/dL ($P = 0.043$) lower LDL cholesterol level, and a 0.17 ($P = 0.015$) lower LDL cholesterol–to–HDL cholesterol ratio than those with lower intakes of both. In addition, higher intakes of eggs, with or without higher intakes of whole grains, were associated with lower TC and LDL cholesterol compared with the referent group (those with lower egg and lower whole grain intakes). Finally, consumption of more eggs combined with higher intakes of either fish or fruits and nonstarchy vegetables were associated with statistically significantly lower triglyceride levels.

During a follow-up period of ~11 y, the occurrences of elevated TC, LDL cholesterol, and triglyceride levels in association with egg intake are shown in Table 4. There was no increased risk of developing elevated levels of TC (>240 mg/dL), LDL cholesterol (>160 mg/dL), or triglycerides (>200 mg/dL) associated with consuming ≥5 eggs/wk. There were also no statistically significant increased or decreased risks of elevated lipid levels in either males or females. Table 5 then examines these same associations of egg-related dietary patterns with elevated lipid levels. In particular, selected healthy eating patterns that included eggs were inversely associated with risk of developing elevated LDL cholesterol levels. Specifically, higher intakes of eggs, when consumed as part of a diet that included higher amounts of total fish or dietary fiber, led to 30%–39% reductions in risk of developing an elevated LDL cholesterol level. Higher intakes of dietary fiber alone (but not eggs) were associated with a lower risk of developing elevated triglyceride levels.

TABLE 4
Risk of elevated lipid levels associated with weekly egg intake

Eggs/week (ages 30 to <65 y)	N	PY	Cases	IR/10^5 PY	HR (95% CI)
All participants					
Total cholesterol >240 (mg/dL)[1]					
<0.5	236	2432.1	56	23.03	1.00 (Ref.)
0.5 to <5	926	10,262.0	255	24.85	1.07 (0.80, 1.43)
≥5	265	2855.1	59	20.67	0.91 (0.62, 1.32)
LDL cholesterol >160 (mg/dL)[2]					
<0.5	256	2560.0	67	26.17	1.00 (Ref.)
0.5 to <5	946	10,342.0	245	23.69	0.93 (0.70, 1.22)
≥5	272	2898.6	60	20.70	0.83 (0.58, 1.20)
Triglycerides >200 (mg/dL)[2]					
<0.5	276	2911.5	46	15.80	1.00 (Ref.)
0.5 to <5	1086	12,249.4	198	16.16	1.06 (0.76, 1.46)
≥5	293	3185.5	62	19.46	1.30 (0.87, 1.94)
Females					
Total cholesterol >240 (mg/dL)[1]					
<0.5	160	1631.6	38	23.29	1.00 (Ref.)
0.5 to <5	540	5934.2	159	26.79	1.09 (0.77, 1.56)
≥5	108	1121.7	34	30.31	1.16 (0.72, 1.87)
LDL cholesterol >160 (mg/dL)[2]					
<0.5	170	1693.4	42	24.80	1.00 (Ref.)
0.5 to <5	562	6259.8	132	21.09	0.88 (0.62, 1.25)
≥5	114	1190.9	28	23.51	1.08 (0.66, 1.78)
Triglycerides >200 (mg/dL)[2]					
<0.5	187	1958.3	26	13.28	1.00 (Ref.)
0.5 to <5	640	7298.6	103	14.11	1.09 (0.71, 1.69)
≥5	126	1372.4	25	18.22	1.38 (0.78, 2.45)
Males					
Total cholesterol >240 (mg/dL)[1]					
<0.5	76	800.5	18	22.49	1.00 (Ref.)
0.5 to <5	386	4327.8	96	22.18	0.99 (0.59, 1.64)
≥5	157	1733.5	25	14.42	0.66 (0.36, 1.23)
LDL cholesterol >160 (mg/dL)[2]					
<0.5	86	866.6	25	28.85	1.00 (Ref.)
0.5 to <5	384	4082.2	113	27.68	0.98 (0.63, 1.51)
≥5	158	1707.6	32	18.74	0.70 (0.41, 1.20)
Triglycerides >200 (mg/dL)[2]					
<0.5	89	953.2	20	20.98	1.00 (Ref.)
0.5 to <5	446	4950.8	95	19.19	1.01 (0.62, 1.65)
≥5	167	1813.1	37	20.41	1.22 (0.69, 2.16)

Abbreviations: CI, confidence interval; HR, hazard ratio; IR, incidence rate; PY, person-years; Ref., reference group.
[1] Adjusted for age, sex, percent energy from total fat, and BMI.
[2] Adjusted for age, sex, percent energy from saturated fat, and BMI.

Discussion

In these analyses, consuming ≥5 eggs/wk was not associated with adverse increases in blood lipids. In fact, eggs when combined with other eating patterns were inversely associated with some lipids. For example, in combination with >17 g/d of dietary fiber, higher egg consumption was linked with lower levels of TC, LDL cholesterol, triglycerides, and an LDL cholesterol–to–HDL cholesterol ratio after 4 y of follow-up than either dietary factor alone. Furthermore, healthy egg-related eating patterns that included higher intakes of fish or dietary fiber were associated with a 30% lower risk of developing an elevated LDL cholesterol level. Furthermore, among men, those who consumed the most eggs had lower levels of TC, LDL cholesterol, and triglycerides. In previous analyses in this same cohort, we also found that egg consumption was associated with lower long-term risks of incident high blood pressure and diabetes [15]. Collectively, our results are consistent with the conclusion that egg consumption is not adversely associated with blood lipids or other cardiometabolic risk factors in adults [25].

A number of prospective cohort studies have examined the effects of egg consumption on TC and LDL cholesterol levels. However, the results of these studies have been considerably heterogeneous, leading some to conclude that more well-conducted studies are needed [26]. Recently, however, an analysis of egg consumption in 21 countries (from Europe, Canada, South Asia, Southeast Asia, China, and the Middle East) in the Prospective Urban Rural Epidemiology study found that there was no association between egg intake (1 or more eggs/d compared with <1 egg/wk) and blood lipid levels or CVD risk [27]. Both observational studies and clinical trials have examined the association between egg consumption and triglyceride levels, another important marker of CVD risk. As found in the current study, most previous reviews and meta-analyses have found little or no association between egg consumption and serum triglycerides [26,28–30], even among adults with hyperlipidemia [31]. One clinical trial of 10 ordinary eggs per week compared with DHA-enriched eggs found that both interventions substantially lowered triglycerides and raised HDL [32]. In contrast, another trial of 2 eggs/d for 3 mo found no difference in triglycerides or HDL levels in the high versus low egg group [33]. In the current study, we found beneficial associations with fasting triglycerides and LDL cholesterol only among males. This finding might be due to the somewhat higher egg intakes in the highest category of intake in males compared with females.

The current analysis adds additional information to evidence published over the past 2–3 decades. In a 2017 meta-analysis of

TABLE 5
Egg-related dietary patterns and risks of elevated lipid levels

Dietary patterns[1] (ages 30 to <65 y)	Total cholesterol >240 (mg/dL)[2]		LDL cholesterol >160 (mg/dL)[3]		Triglycerides >200 (mg/dL)[4]	
	N	HR (95% CI)	*N*	HR (95% CI)	*N*	HR (95% CI)
Eggs/dairy						
Lower/lower	659	1.00 (Ref.)	697	1.00 (Ref.)	793	1.00 (Ref.)
Lower/higher	239	0.96 (0.71, 1.30)	240	1.08 (0.80, 1.44)	268	0.84 (0.58, 1.20)
Higher/lower	359	0.87 (0.67, 1.13)	368	0.82 (0.63, 1.07)	399	1.12 (0.85, 1.48)
Higher/higher	170	1.03 (0.73, 1.47)	169	0.91 (0.63, 1.30)	195	1.13 (0.78, 1.64)
Eggs/total fish						
Lower/lower	477	1.00 (Ref.)	495	1.00 (Ref.)	545	1.00 (Ref.)
Lower/higher	421	1.00 (0.77, 1.29)	442	0.80 (0.62, 1.03)	516	0.91 (0.67, 1.22)
Higher/lower	273	1.08 (0.81, 1.44)	278	0.91 (0.68, 1.22)	300	1.18 (0.85, 1.63)
Higher/higher	256	0.78 (0.57, 1.07)	259	0.61 (0.44, 0.84)	294	1.07 (0.77, 1.48)
Eggs/whole grains						
Lower/lower	494	1.00 (Ref.)	514	1.00 (Ref.)	591	1.00 (Ref.)
Lower/higher	404	0.66 (0.51, 0.87)	423	0.77 (0.59, 1.00)	470	0.85 (0.63, 1.14)
Higher/lower	308	0.72 (0.55, 0.96)	316	0.65 (0.48, 0.87)	344	1.06 (0.77, 1.44)
Higher/higher	221	0.86 (0.64, 1.17)	221	0.88 (0.65, 1.19)	250	1.14 (0.82, 1.59)
Eggs/fiber						
Lower/lower	571	1.00 (Ref.)	588	1.00 (Ref.)	667	1.00 (Ref.)
Lower/higher	327	0.76 (0.57, 1.02)	349	0.75 (0.57, 0.98)	394	0.63 (0.45, 0.88)
Higher/lower	334	0.86 (0.66, 1.12)	342	0.78 (0.59, 1.02)	382	1.10 (0.83, 1.45)
Higher/higher	195	0.83 (0.59, 1.18)	195	0.70 (0.49, 0.98)	212	0.91 (0.63, 1.32)
Eggs/fruit and nonstarchy vegetables						
Lower/lower	618	1.00 (Ref.)	633	1.00 (Ref.)	710	1.00 (Ref.)
Lower/higher	280	0.79 (0.59, 1.05)	304	0.78 (0.59, 1.04)	351	0.73 (0.53, 1.02)
Higher/lower	381	0.88 (0.69, 1.14)	387	0.79 (0.61, 1.02)	423	1.17 (0.89, 1.53)
Higher/higher	148	0.81 (0.56, 1.18)	150	0.73 (0.50, 1.07)	171	0.87 (0.58, 1.30)

Abbreviations: CI, confidence interval; HR, hazard ratio; Ref., reference group.

[1] Lower vs. higher egg intake: <3 per wk vs. ≥3 per wk; lower vs. higher dairy intake, <1.75 vs. ≥1.75 cups/d; lower vs. higher total fish intake, <7 vs. ≥7 oz/wk; lower vs. higher whole grain intake, <0.5 vs. ≥0.5 oz/d; lower vs. higher fiber intake, <17 vs. ≥17 g/d; lower vs. higher fruit and nonstarchy vegetable intake, <3 vs. ≥3 cups/d.

[2] Adjusted for age, sex, percent energy from fat, and BMI.

[3] Adjusted for age, sex, percent energy from saturated fat, and BMI.

[4] Adjusted for age, sex, BMI, solid fat, alcohol and added sugars.

randomized clinical trials, investigators found that the consumption of 1–3 eggs/d led to higher levels of TC, LDL cholesterol, and HDL cholesterol but not to higher triglycerides or a higher LDL cholesterol–to–HDL cholesterol ratio [28]. In that study, higher egg consumption was associated with a 4.71 mg/dL increase in LDL cholesterol. A later meta-analysis with more trials included in the summary estimates found higher egg intake to lead to a 3.08 mg/dL increase in LDL [30]. Because both meta-analyses also found eggs to be associated with increased HDL cholesterol, the LDL cholesterol–to–HDL cholesterol ratio was unchanged. One meta-analysis found a small (0.14) increase in this ratio [34], while in our own analyses, higher egg intakes in combination with other healthy eating patterns were associated with an LDL cholesterol–to–HDL cholesterol ratio that was 0.10–0.17 units lower than that of the reference group. Because higher egg intakes were associated with lower LDL cholesterol levels, but not higher HDL cholesterol levels, the lower LDL cholesterol–to–HDL cholesterol ratio is most likely attributable to the lower LDL cholesterol levels. Even in the absence of other healthy dietary factors, higher egg intake in the current study was associated with a slightly lower LDL cholesterol–to–HDL cholesterol ratio.

Other components in the usual diet have been shown to impact the association between eggs and dietary cholesterol as well as CVD risk [35]. In the current study, the intake of dietary fiber, in particular, was shown to modify the relationship between eggs and lipid levels. In addition, both total fat and saturated fat were found to confound the observed associations between eggs and certain lipids. Thus, our careful examination of confounding and effect modification has helped to disentangle the effects of egg intake from those of other dietary factors.

It has been shown that the dietary cholesterol content of eggs is not consistently well absorbed, which may explain the absence of an effect on blood cholesterol levels [36]. The majority of plasma cholesterol is produced endogenously, and when dietary cholesterol intake is increased, a compensatory decrease in absorption or biosynthesis has been shown [37]. Furthermore, it has been shown that dietary cholesterol is less well absorbed in individuals with insulin resistance and/or obesity [38]. It has also been shown that a small number of individuals are hyper-responsive to the consumption of dietary cholesterol [39,40], perhaps owing to genetic polymorphisms in the *ABCG5* and *ABCG8* genes impacting cholesterol absorption [41], which have been shown to interact with other dietary patterns [42].

There are several potential mechanisms by which eggs may benefit or have neutral effects on lipid outcomes. Eggs are relatively low in carbohydrates and calories and contain other important nutrients [43]. For example, lutein has been shown to reduce cholesterol accumulation, decrease LDL cholesterol oxidation, and reduce cytokine production in the aortas of guinea pigs, thus potentially playing a role in the prevention of atherosclerosis [44].

Eggs and other animal-based foods are significant sources of dietary choline, an essential nutrient that has an important role in regulating memory and muscle function as well as maintaining a healthy liver [45]. Both choline and carnitine from animal-source foods are converted in the gut to trimethylamine, which is, in turn, metabolized to trimethylamine-N-oxide (TMAO), a known independent risk factor for CVD [46]. The extent to which dietary choline from eggs is converted to TMAO is unclear, but it appears that it is not readily metabolized to TMAO [47]. The phospholipids in eggs, particularly sphingomyelin, have been shown to inhibit the absorption of dietary cholesterol, which could lower serum lipid levels [48]. Another possible beneficial effect from eggs may relate to observed increases in lipid particle sizes and higher concentrations of HDL particles [49,50] associated with egg consumption.

Previous studies evaluating the effects of consuming eggs as part of dietary patterns on lipid outcomes often use data-derived patterns (i.e., factor analysis) [18,51]. As a result, eggs are often linked with less healthy eating patterns, such as those with higher saturated fat. However, when eggs were consumed as part of a healthier eating pattern, as was shown in the present study, they were not associated with increased cardiometabolic risk [13]. It has been previously shown that egg consumers in the United States tend to also consume more dietary fiber [13], a finding that may in part explain some of the benefits of a diet higher in eggs because dietary fiber lowers both TC and LDL cholesterol by reducing the absorption of cholesterol and fat, increasing bile acid synthesis and excretion, and producing short chain fatty acids which also may reduce cholesterol synthesis [52]. Fish intake may lower triglycerides by displacing carbohydrates in the diet with protein [53] or by direct effects of omega-3 fatty acids [54].

This study had several strengths. For instance, the dietary data in this study included nearly 16,000 d of diet records, which were carefully collected following standardized protocols, including portion size estimation as well as collection of brands and recipe data. Furthermore, all participant records were routinely debriefed by a staff nutritionist. Egg consumption is likely to be more complete in this study as intake from all sources, including mixed dishes, was derived from the dietary records. In addition, data on potential confounders were collected systematically at each examination visit in Framingham. Nonetheless, we cannot rule out errors in the ascertainment of the exposure or potential confounding variables that were self-reported. Another limitation of this study is that only ~70% of participants provided dietary record data. In addition, we had a somewhat limited range of egg intake, perhaps due to the perceived harmful effects of eggs on CVD risk at that time in the United States' history. In addition, the somewhat low numbers of participants consuming ≥5 eggs/wk limited our power to carry out subgroup analyses (e.g., males compared with females). Finally, the homogenous racial and ethnic backgrounds of the participants in this study mean that we were not able to address these questions in a more diverse population group.

In conclusion, this longitudinal study finds that higher egg intakes are not associated with elevated serum lipids in healthy adults. In fact, when combined with healthy eating patterns that include more dietary fiber or fish, eggs may be beneficially associated with lipid outcomes. Future randomized clinical trials are needed to further evaluate the influence of egg consumption as part of a healthy dietary pattern on blood lipid levels. Overall, these results provide no evidence that egg intake should be limited when consumed as a part of a fiber-rich, heart-healthy diet.

Author contributions

The authors' responsibilities were as follows – LLM, MMM: designed the research; MMM, MRS: analyzed data; MMM, LLM, XZ: wrote the paper; LLM: oversaw the conduct of the research and takes primary responsibility for final content; and all authors (LLM, XZ, MMM, IY, MLB, and MRS): read, edited, did background reading and research, and approved the final manuscript.

Conflict of interest

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Data availability

These data were obtained from the Framingham Heart Study and may be requested from (https://www.framinghamheartstudy.org/fhs-for-researchers/data-available-overview/) (accessed on June 22, 2023).

References

[1] S.M. Grundy, N.J. Stone, A.L. Bailey, C. Beam, K.K. Birtcher, R.S. Blumenthal, et al., AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA guideline on the management of blood cholesterol: a report of the American College of Cardiology/American Heart Association task force on clinical practice guidelines, Circulation 139 (2018) e1082–e1143, 2019.

[2] Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III), JAMA 285 (2001) 2486–2497.

[3] R.M. Krauss, R.H. Eckel, B. Howard, L.J. Appel, S.R. Daniels, R.J. Deckelbaum, et al., AHA dietary guidelines: revision 2000: a statement for healthcare professionals from the Nutrition Committee of the American Heart Association, Circulation 102 (2000) 2284–2299.

[4] L.L. Rudel, Genetic factors influence the atherogenic response of lipoproteins to dietary fat and cholesterol in nonhuman primates, J. Am. Coll. Nutr. 16 (1997) 306–312.

[5] R.B. Shekelle, J. Stamler, Dietary cholesterol and ischaemic heart disease, Lancet 1 (1989) 1177–1179.

[6] R.M. Weggemans, P.L. Zock, M.B. Katan, Dietary cholesterol from eggs increases the ratio of total cholesterol to high-density lipoprotein choles terol in humans: a meta-analysis, Am. J. Clin. Nutr. 73 (2001) 885–891.

[7] D.K. Arnett, R.S. Blumenthal, M.A. Albert, A.B. Buroker, Z.D. Goldberger, E.J. Hahn, et al., 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: a report of the American

College of Cardiology/American Heart Association task force on clinical practice guidelines, Circulation 140 (2019) e596–e646.
[8] J.A.S. Carson, A.H. Lichtenstein, C.A.M. Anderson, L.J. Appel, P.M. Kris-Etherton, K.A. Meyer, et al., Dietary cholesterol and cardiovascular risk: a science advisory from the American Heart Association, Circulation 141 (2020) e39–e53.
[9] S. McGuire, Scientific report of the 2015 dietary guidelines advisory committee. Washington, DC: US Departments of Agriculture and Health and Human Services, Adv. Nutr. 7 (2016) 202–204.
[10] M.L. Fernandez, Rethinking dietary cholesterol, Curr. Opin. Clin. Nutr. Metab. Care. 15 (2012) 117–121.
[11] M.J. Puglisi, M.L. Fernandez, The health benefits of egg protein, Nutrients 14 (2022) 2904.
[12] L.G. Snetselaar, J.M. de Jesus, D.M. DeSilva, EE. Stoody, Dietary guidelines for Americans, 2020–2025: understanding the Scientific Process, Guidelines, and Key Recommendations, Nutr. Today. 56 (2021) 287–295.
[13] Y. Papanikolaou, V.L. Fulgoni, Patterns of egg consumption can help contribute to nutrient recommendations and are associated with diet quality and shortfall nutrient intakes, Nutrients 13 (2021) 4094.
[14] M.L. Neuhouser, The importance of healthy dietary patterns in chronic disease prevention, Nutr. Res. 70 (2019) 3–6.
[15] M.M. Mott, X. Zhou, M.L. Bradlee, M.R. Singer, I. Yiannakou, L.L. Moore, Egg intake is associated with lower risks of impaired fasting glucose and high blood pressure in Framingham Offspring Study Adults, Nutrients 15 (2023) 507.
[16] M. Amini, A. Esmaillzadeh, S. Shafaeizadeh, J. Behrooz, M. Zare, Relationship between major dietary patterns and metabolic syndrome among individuals with impaired glucose tolerance, Nutrition 26 (2010) 986–992.
[17] P.R. Deshmukh-Taskar, C.E. O'Neil, T.A. Nicklas, S.J. Yang, Y. Liu, J. Gustat, et al., Dietary patterns associated with metabolic syndrome, sociodemographic and lifestyle factors in young adults: the Bogalusa Heart Study, Public Health Nutr 12 (2009) 2493–2503.
[18] C. Heidemann, C. Scheidt-Nave, A. Richter, G.B.M. Mensink, Dietary patterns are associated with cardiometabolic risk factors in a representative study population of German adults, Br. J. Nutr. 106 (2011) 1253–1262.
[19] W.B. Kannel, M. Feinleib, P.M. McNamara, R.J. Garrison, W.P. Castelli, An investigation of coronary heart disease in families. The Framingham Offspring Study, Am. J. Epidemiol. 110 (1979) 281–290.
[20] S.F. Schakel, Y.A. Sievert, I.M. Buzzard, Sources of data for developing and maintaining a nutrient database, J. Am. Diet. Assoc. 88 (1988) 1268–1271.
[21] L.L. Moore, M.R. Singer, M.L. Bradlee, L. Djoussé, M.H. Proctor, L.A. Cupples, et al., Intake of fruits, vegetables, and dairy products in early childhood and subsequent blood pressure change, Epidemiology 16 (2005) 4–11.
[22] J.L. Jenner, J.M. Ordovas, S. Lamon-Fava, M.M. Schaefer, P.W. Wilson, W.P. Castelli, et al., Effects of age, sex, and menopausal status on plasma lipoprotein(a) levels. The Framingham Offspring Study, Circulation 87 (1993) 1135–1141.
[23] S.M. Abdullah, L.F. Defina, D. Leonard, C.E. Barlow, N.B. Radford, B.L. Willis, et al., Long-term association of low-density lipoprotein cholesterol with cardiovascular mortality in individuals at low 10-year risk of atherosclerotic cardiovascular disease, Circulation 138 (2018) 2315–2325.
[24] M. Miller, N.J. Stone, C. Ballantyne, V. Bittner, M.H. Criqui, H.N. Ginsberg, et al., Triglycerides and cardiovascular disease: a scientific statement from the American Heart Association, Circulation 123 (2011) 2292–2333.
[25] Z.S. Clayton, E. Fusco, M. Kern, Egg consumption and heart health: a review, Nutrition 37 (2017) 79–85.
[26] S. Berger, G. Raman, R. Vishwanathan, P.F. Jacques, E.J. Johnson, Dietary cholesterol and cardiovascular disease: a systematic review and meta-analysis, Am. J. Clin. Nutr. 102 (2015) 276–294.
[27] M. Dehghan, A. Mente, S. Rangarajan, V. Mohan, S. Lear, S. Swaminathan, et al., Association of egg intake with blood lipids, cardiovascular disease, and mortality in 177,000 people in 50 countries, Am. J. Clin. Nutr. 111 (2020) 795–803.
[28] M.H. Rouhani, N. Rashidi-Pourfard, A. Salehi-Abargouei, M. Karimi, F. Haghighatdoost, Effects of egg consumption on blood lipids: a systematic review and meta-analysis of randomized clinical trials, J. Am. Coll. Nutr. 37 (2018) 99–110.
[29] M.X. Wang, C.H. Wong, J.E. Kim, Impact of whole egg intake on blood pressure, lipids and lipoproteins in middle-aged and older population: a systematic review and meta-analysis of randomized controlled trials, Nutr. Metab. Cardiovasc. Dis. 29 (2019) 653–664.
[30] M. Khalighi Sikaroudi, S. Soltani, R. Kolahdouz-Mohammadi, Z.S. Clayton, M.L. Fernandez, F. Varse, et al., The responses of different dosages of egg consumption on blood lipid profile: an updated systematic review and meta-analysis of randomized clinical trials, J. Food Biochem. 44 (2020) e13263.
[31] T. Klangjareonchai, S. Putadechakum, P. Sritara, C. Roongpisuthipong, The effect of egg consumption in hyperlipidemic subjects during treatment with lipid-lowering drugs, J. Lipids. 2012 (2012) 672720.
[32] K.C. Maki, M.E. Van Elswyk, D. McCarthy, M.A. Seeley, P.E. Veith, S.P. Hess, Lipid responses in mildly hypertriglyceridemic men and women to consumption of docosahexaenoic acid-enriched eggs, Int. J. Vitam. Nutr. Res. 73 (2003) 357–368.
[33] N.R. Fuller, I.D. Caterson, A. Sainsbury, G. Denyer, M. Fong, J. Gerofi, et al., The effect of a high-egg diet on cardiovascular risk factors in people with type 2 diabetes: the Diabetes and Egg (DIABEGG) study-a 3-mo randomized controlled trial, Am. J. Clin. Nutr. 101 (2015) 705–713.
[34] M.Y. Li, J.H. Chen, C. Chen, Y.N. Kang, Association between egg consumption and cholesterol concentration: a systematic review and meta-analysis of randomized controlled trials, Nutrients 12 (2020) 1995.
[35] D.J. McNamara, Dietary cholesterol and atherosclerosis, Biochim. Biophys. Acta. 1529 (2000) 310–320.
[36] J.E. Kim, W.W. Campbell, Dietary cholesterol contained in whole eggs is not well absorbed and does not acutely affect plasma total cholesterol concentration in men and women: results from 2 randomized controlled crossover studies, Nutrients 10 (2018) 1272.
[37] D.J. McNamara, R. Kolb, T.S. Parker, H. Batwin, P. Samuel, C.D. Brown, et al., Heterogeneity of cholesterol homeostasis in man. Response to changes in dietary fat quality and cholesterol quantity, J. Clin. Invest. 79 (1987) 1729–1739.
[38] J. Pihlajamäki, H. Gylling, T.A. Miettinen, M. Laakso, Insulin resistance is associated with increased cholesterol synthesis and decreased cholesterol absorption in normoglycemic men, J. Lipid Res. 45 (2004) 507–512.
[39] K.L. Herron, S. Vega-Lopez, K. Conde, T. Ramjiganesh, S. Roy, N.S. Shachter, et al., Pre-menopausal women, classified as hypo- or hyperresponders, do not alter their LDL/HDL ratio following a high dietary cholesterol challenge, J. Am. Coll. Nutr. 21 (2002) 250–258.
[40] K.L. Herron, S. Vega-Lopez, K. Conde, T. Ramjiganesh, N.S. Shachter, M.L. Fernandez, Men classified as hypo- or hyperresponders to dietary cholesterol feeding exhibit differences in lipoprotein metabolism, J. Nutr. 133 (2003) 1036–1042.
[41] H. Gylling, M. Hallikainen, J. Pihlajamäki, J. Ågren, M. Laakso, R.A. Rajaratnam, et al., Polymorphisms in the ABCG5 and ABCG8 genes associate with cholesterol absorption and insulin sensitivity, J. Lipid Res. 45 (2004) 1660–1665.
[42] E. Viturro, M. de Oya, M.A. Lasunción, L. Gorgojo, J.M. Moreno, M. Benavente, et al., Cholesterol and saturated fat intake determine the effect of polymorphisms at ABCG5/ABCG8 genes on lipid levels in children, Genet. Med. 8 (2006) 594–599.
[43] W.O. Song, J.M. Kerver, Nutritional contribution of eggs to American diets, J. Am. Coll. Nutr. 19 (2000) 556S–562S.
[44] J.E. Kim, J.O. Leite, R. deOgburn, J.A. Smyth, R.M. Clark, M.L. Fernandez, A lutein-enriched diet prevents cholesterol accumulation and decreases oxidized LDL and inflammatory cytokines in the aorta of guinea pigs, J. Nutr. 141 (2011) 1458–1463.
[45] S.H. Zeisel, K.A. da Costa, Choline: an essential nutrient for public health, Nutr. Rev. 67 (2009) 615–623.
[46] W.H.W. Tang, Z. Wang, B.S. Levison, R.A. Koeth, E.B. Britt, X. Fu, et al., Intestinal microbial metabolism of phosphatidylcholine and cardiovascular risk, N. Engl. J. Med. 368 (2013) 1575–1584.
[47] C.N. Blesso, Egg phospholipids and cardiovascular health, Nutrients 7 (2015) 2731–2747.
[48] C.N. Blesso, M.L. Fernandez, Dietary cholesterol, serum lipids, and heart disease: are eggs working for or against you? Nutrients 10 (2018) 426.
[49] C.N. Blesso, C.J. Andersen, J. Barona, J.S. Volek, ML. Fernandez, Whole egg consumption improves lipoprotein profiles and insulin sensitivity to a greater extent than yolk-free egg substitute in individuals with metabolic syndrome, Metabolism 62 (2013) 400–410.
[50] C.M. Greene, D. Waters, R.M. Clark, J.H. Contois, M.L. Fernandez, Plasma LDL and HDL characteristics and carotenoid content are positively influenced by egg consumption in an elderly population, Nutr. Metab. (Lond). 3 (2006) 6.

[51] T.T. Fung, E.B. Rimm, D. Spiegelman, N. Rifai, G.H. Tofler, W.C. Willett, et al., Association between dietary patterns and plasma biomarkers of obesity and cardiovascular disease risk, Am. J. Clin. Nutr. 73 (2001) 61–67.
[52] C.E. Smith, K.L. Tucker, Health benefits of cereal fibre: a review of clinical trials, Nutr. Res. Rev. 24 (2011) 118–131.
[53] D.K. Layman, P. Clifton, M.C. Gannon, R.M. Krauss, F.Q. Nuttall, Protein in optimal health: heart disease and type 2 diabetes, Am. J. Clin. Nutr. 87 (2008) 1571S–1575S.
[54] D. Mozaffarian, J.H.Y. Wu, Omega-3 fatty acids and cardiovascular disease: effects on risk factors, molecular pathways, and clinical events, J. Am. Coll. Cardiol. 58 (2011) 2047–2067.