Eggs: Friend or Foe?

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Eggs are touted to be bad for the heart as one egg accounts for about two thirds the recommended limit on dietary cholesterol. The benefits of egg intake are often overshadowed by its cholesterol content. There are several publications on the positive and negative effects of egg consumption in humans, the majority of which focus on blood lipids, lipoprotein cholesterol and cardiovascular disease. The purpose of this review is to educate the readers on the nutritional value of eggs and examine the relevant literature. Protein, choline, lutein and zeaxanthin and cholesterol will be discussed at length since these are the nutrients that have generated the most public interest.

INTRODUCTION

"Egg yolk consumption almost as bad as smoking when it comes to atherosclerosis" was the title of an article that appeared in Science Daily on August 13, 2012. Some other egg related stories published in the past few years in Science Daily include "Eggs' antioxidant properties may help prevent heart disease and cancer" – July 6, 2011, "One egg worse than some fast food meals when it comes to cholesterol" – November 1, 2010 and "Egg-Irony: High cholesterol food may reduce blood pressure" – March 1, 2009. The findings on risks versus benefits of egg consumption seem equivocal. The American Heart Association (AHA) has recommended limiting dietary cholesterol intake to less than 300 mg/d [1]. Eggs have been getting a bad reputation because the cholesterol content of one egg yolk (range 141 to 234 mg from small to jumbo egg) accounts for two thirds this limit. However, the saturated fat and trans fat content, the real culprits that raise blood cholesterol, are minimal (1.6 g and 0 g, respectively) in an egg compared to most fast food meals. Besides fast foods are also high in refined carbohydrates, saturated fat and sodium, all of which are known to cause adverse health effects. Apart from the cholesterol-related negative publicity, eggs are actually a good source of high quality protein, a number of essential vitamins and minerals, choline and also two carotenoids, lutein and zeaxanthin, that are associated with eye health. The objective of this review is to dissect the egg by nutrients and evaluate clinical studies that looked at benefits and risks of egg consumption in humans. The nutrients that will be discussed in this review include protein, choline and lutein and zeaxanthin. Cholesterol will also be discussed at length since it is a topic that is most scrutinized.

NUTRITIONAL VALUE OF AN EGG

The nutrients present in two raw large eggs (100 g), egg whites (66 g) and egg yolks (34 g) are listed in Table 1 [2]. Also shown in the table is the percent daily value of nutrients in two large eggs. The percent daily value was calculated based on a 2,000 calorie diet for healthy adults [3]. The purpose of this was to highlight the nutritional value of an egg. The percent daily values will slightly differ for women, women during pregnancy and lactation and children. The majority of the protein, potassium, sodium and

vitamins B2 and B3 are present in the egg white. The yolk contains almost all of the fat, iron, phosphorus, zinc, thiamin, pantothenic acid, vitamins B6 and B12, folate, vitamins A, E, D and K, choline and also lutein and zeaxanthin. Still most people avoid consuming the yolk due to its cholesterol content. Most restaurants and cafes are now offering egg white options for sandwiches, scrambles and omelets. However, looking at the nutrient density (high nutrient to energy ratio) of the yolk, it is evident that discarding the yolk results in a loss of valuable vitamins, minerals and micronutrients. Data from the NHANES III showed that eggs contributed 10% to 20% of dietary folate and 20% to 30% of vitamins A, E and B12 among egg consumers (those who reported consuming at least one egg group product in a 24 hour dietary recall). A greater percentage of egg consumers had adequate intakes (≥Estimate Average Requirement or 70% RDA) of vitamin B12, vitamin A, vitamin E and vitamin C than inadequate intake, while a greater proportion of non egg consumers (no egg products in a 24 hour dietary recall) had inadequate intakes of these vitamins [4]. Overall daily nutrient intake was significantly greater in egg consumers compared to non-egg consumers, with the exception of dietary fiber and vitamin B6 [4]. Eggs are good exogenous, dietary sources of vitamins K and D and choline [5]. Egg yolks are also a highly bioavailable dietary source of lutein and zeaxanthin, two oxygenated carotenoids that are important for eye health [6]. Despite being so nutrient dense only 1.3% of total calories consumed in the American diet can be attributed to eggs [7]. The reason for poor egg consumption in the US may lie in the perceived health risks associated with intake.

Table 1: Nutrients found in two fresh, raw, large whole eggs (100 g), two egg whites (66 g) and two egg yolks (34 g). Also shown is the % daily value of nutrients from two whole eggs [2, 3, 8].

Nutrient	Whole egg (100 g)	%Daily value	Egg white (66 g)	Egg yolk (34 g)
Energy, kcal	143	7%	34	109
Protein, g	12.56	22%	7.19	5.39
Total lipid (fat), g	9.51		0.11	9.02
Saturated fat, g	3.126		0	3.247
MUFA, g	3.658		0	3.991
PUFA, g	1.911		0	1.429
Cholesterol, mg	372	124%[a]	0	369
Carbohydrate[b], g	0.72	0.24%	0.48	1.22
Sugars, total, g	0.37	NE	0.47	0.19
Minerals				
Calcium, mg	56	6%	5	44
Iron, mg	1.75	22%	0.05	0.93
Magnesium, mg	12	3%	27	2
Phosphorus, mg	198	28%	10	133
Potassium, mg	138	3%[b]	108	37
Sodium, mg	142	9%[b]	110	16
Zinc, mg	1.29	12%	0.02	0.78
Copper, mg	0.072	9%	0.015	0.026
Manganese, mg	0.028	1%[c]	0.007	0.019
Selenium, µg	30.8	56%	13.2	19.0
Vitamins				
Thiamin, mg	0.04	3%	0.003	0.060
Riboflavin, mg	0.457	35%	0.290	0.180
Niacin, mg	0.075	0.5%	0.069	0.008
Pantothenic acid, mg	1.434	29%[c]	0.125	1.017
Vitamin B6, mg	0.170	13%	0.003	0.119
Folate, µg	47	12%	3	50
Vitamin B12, µg	0.89	37%	0.06	0.66
Vitamin A, RAE, µg	160	18%	0	130
Vitamin E (α-tocopherol), mg	1.05	7%	0	0.88
Vitamin D, µg	2	13%	0	1.8
Vitamin K (phylloquinone), µg	0.3	0.25%[c]	0	0.2
Choline, mg	293.8	53%[c]	0.8	272.4
β-carotene, µg	5	NE	0	5
Lutein & Zeaxanthin, µg	504	NE	0	364

% Daily values are based on a 2000 calorie diet for healthy men

a: Based on the recommended 300 mg/d limitation, b: by difference, c: Values are based on adequate intakes (AI)
MUFA: monounsaturated fatty acids, PUFA: polyunsaturated fatty acids, NE: Not Established

PROTEIN

At only 72 calories, one large egg provides 6 g of high biological value dietary protein. Unlike protein from plant sources, egg protein contains all nine essential amino acids in appropriate ratios making it a 'complete protein' source [8]. Table 2 shows amino acid content in eggs. The amino acid profile of eggs is considered to be a standard against which all other proteins are compared. Although the RDA for protein is 0.8 g/kg/day, research suggests that older adults may require greater amounts of dietary protein in order to maintain nitrogen equilibrium and protein turnover [9, 10]. Intake of essential nutrients may be inadequate in the elderly because of the decreased energy requirements with aging. Studies in older adults have shown an undesired metabolic accommodation effect in response to consumption of diets that provided the RDA of 0.8 g/kg/day of protein [11, 12]. Decreased nitrogen excretion associated with decreased mid-thigh muscle area was observed in one study [12]. In another study that had an additional arm of resistance training, loss of fat-free mass and decreased mid-thigh muscle mass in sedentary individuals was observed. Even though the RDA for protein intake may be sufficient to maintain whole body protein metabolism, the compensatory loss of skeletal muscle may have long term consequences. Chronic skeletal muscle loss observed with aging affects 30% people older than 60 y and may affect >50% of those older than 80 y [13]. Sarcopenia, which is loss of skeletal muscle mass, is associated with increasing body fat mass, decreased basal metabolic rate and daily energy needs, loss of bone mass, reduced strength and functional status [9]. With an increase in the aging population, sarcopenia will become an increasingly important public health concern. Even though studies are suggesting that the RDA for protein may need revisiting, there are between 15% and 38% adult men and between 27% and 41% adult women who have dietary protein intakes below the current RDA [13]. One suggested strategy to combat sarcopenia is moderately increasing dietary protein intake to levels above the RDA to prevent skeletal muscle loss by promoting protein anabolism [11, 12]. Protein quality is also shown to be an important factor in muscle protein synthesis. The essential amino acid composition, specifically the presence of leucine, determines the anabolic potential of a protein and nonessential amino acids do not provide an added effect [13-15]. Protein energy malnutrition could increase older adults' susceptibility to infectious diseases, poor wound healing and negative outcomes to interventions for chronic and acute conditions [10]. Egg protein contains all the essential amino acids – histidine, isoleucine, leucine, lysine, methionine, cystine, phenylalanine, tyrosine, threonine, tryptophan and valine [2]. Unlike other high quality protein sources such as meat, poultry and seafood, eggs are also less expensive [10]. Older adults in America who have fixed incomes and/or limited mobility may have limitations in their ability to purchase or prepare food. Eggs are an affordable source of high quality protein for this population. Eggs are also readily available, easy to prepare, consume, digest and have longer shelf life, making them an ideal dietary protein source for older adults.

Table 2: Amino acid content of two fresh, raw, large whole eggs (100 g), egg whites (66 g) and egg yolks (34 g) [2].

Amino acid	Whole egg (100 g)	Egg white (66 g)	Egg yolk (34 g)
Essential amino acids, g			
Histidine	0.309	0.191	0.141
Tryptophan	0.167	0.082	0.060
Threonine	0.556	0.296	0.234
Isoleucine	0.671	0.436	0.294
Leucine	1.086	0.671	0.476
Lysine	0.912	0.532	0.414
Methionine	0.380	0.263	0.129
Cystine	0.272	0.189	0.090
Phenylalanine	0.680	0.453	0.232
Tyrosine	0.499	0.302	0.231
Valine	0.858	0.534	0.323
Nonessential amino acids, g			
Arginine	0.820	0.428	0.374
Alanine	0.735	0.465	0.284
Aspartic acid	1.329	0.805	0.527
Glutamic acid	1.673	1.023	0.670
Glycine	0.432	0.273	0.166
Proline	0.512	0.287	0.220
Serine	0.971	0.527	0.451

CHOLINE

Eggs are one of the richest dietary sources of choline, which the National Academy of Sciences identified as a required nutrient for humans. The Institute of Medicine recommended adequate intake levels of dietary choline to be 550 mg/d for men, 425 mg/d for women (including pregnancy) and 550 mg/d for lactating women [16]. In the US the average daily intake of choline is estimated to be ~300 mg and intake of betaine (a choline derivative) ~100 mg [17, 18]. Most of the choline found in eggs is in the form of phosphatidylcholine and a small amount in the form of sphingomyelin. Choline is also found in meats, fish, whole grains, breakfast cereals, fruits and vegetables. The plant sources mostly contain betaine, which is important as it spares the use of choline for methyl donation [19]. Humans can synthesize choline endogenously from phosphatidylethanolamine [20]. However, healthy humans fed a choline deficient diet for 3 weeks had depleted tissue stores of choline and developed liver dysfunction [20].

Choline or its metabolites are required for the structural integrity and signaling functions of cell membranes, it is the major source of methyl groups in the diet, and it directly affects cholinergic neurotransmission, transmembrane signaling and lipid transport and metabolism [21]. Furthermore, choline derived from phosphatidylcholine may be especially important when extracellular choline is in short supply [22]. A number of animal studies have shown choline to be essential for normal brain development [23]. Choline is also required to methylate homocysteine. Humans depleted of choline have diminished capacity to methylate homocysteine and developed elevated plasma homocysteine after a methionine loading test [24]. Elevated plasma homocysteine is one of the risk factors for cardiovascular disease and stroke [25, 26].

Choline concentrations in amniotic fluid are 10 fold higher than in maternal blood

indicating active transfer of choline across the placenta [27]. Also, serum choline concentrations are 6 to 7 fold higher in the fetus and neonate than in adults suggesting choline's importance during the critical period of infant growth and development [28]. Choline concentration in breast milk is dependent on mother's choline intake in the diet. Adequate choline intake is critical during pregnancy and lactation because it influences fetal brain development and is important for maintaining maternal homocysteine concentrations. High maternal homocysteine is associated with increased incidence of birth defects [27]. To date only one double-blinded, randomized controlled trial has explored the effects of maternal choline supplementation on cognitive development in infants aged 10 to 12 months. In this study supplementation with 750 mg of phosphatidylcholine from 18 weeks of gestation to 90 days postpartum did not increase measures of global development, language development, short-term visuospatial memory or long-term episodic memory in infants compared to placebo [29]. The null findings of this study were attributed to a possible influence of betaine, diets of the US population contain good sources of choline and short follow up time. Studies of choline supplementation in adults have yielded inconsistent results. Choline supplementation was shown to improve different measures of memory in normal subjects and also those with memory deficits and dementia [30-32]. However, a null effect of choline supplementation was also reported in normal subjects and patients with dementia [33-35]. Genetic variation has a strong influence on dietary choline requirements. Several pathways control how much choline is required from the diet and single nucleotide polymorphisms in these pathways might be of importance in determining dietary requirements, which could probably be a separate review topic [27]. In the meantime, it is noteworthy that almost 50% of the recommended adequate levels of choline can be obtained from 2 large eggs.

LUTEIN AND ZEAXANTHIN

Lutein and zeaxanthin are oxygenated carotenoids or xanthophylls that are solely obtained from the diet. Of the 700 carotenoids identified in nature, only 13 carotenoids and their 12 isomers are found in the serum and only two of those, lutein and zeaxanthin, exclusively accumulate in the macula of the human eye [36]. The macula lutea is a yellowish region 5-6 mm in diameter in the posterior pole of the human retina responsible for the central 15 to 20 degrees of vision [37, 38]. The concentration of lutein and zeaxanthin in the macula is 1000 fold higher than in serum [39, 40]. Lutein and zeaxanthin protect the retina from damaging short wavelength blue light and oxidative stress [41]. Lutein and zeaxanthin are also present in all other ocular structures with the exception of the vitreous, cornea, and sclera [38]. In the iris, lutein and zeaxanthin most likely act as blue light filters, while in the ciliary body, an area of high metabolic activity, they most likely act as antioxidants [42]. In the lens as well, lutein and zeaxanthin most likely have an antioxidant function, with the constant exposure to oxygen and UV radiation. Because of their exclusive accumulation and specific function in the macula, lutein and zeaxanthin have been extensively studied for the prevention and treatment of age-related macular degeneration (AMD), a major cause of visual impairment in the elderly [43]. Studies have shown that lutein

and zeaxanthin in the diet, serum and macula (known as macular pigment) are significantly associated with reduced risk of AMD [44-50].

Egg yolks are a highly bioavailable source of lutein and zeaxanthin. Bioavailability of lutein was found to be significantly greater from lutein enriched eggs compared to spinach and also lutein and lutein ester supplements in healthy men [6]. The concentration of lutein and zeaxanthin in non-enriched eggs is variable and dependent on the chicken feed. The lutein and zeaxanthin concentration in 2 large eggs based on the USDA nutrient database is shown in Table 1. The mean concentrations of lutein and zeaxanthin reported in clinical studies that used large non-enriched eggs were 143 μg and 94 μg [51], 243 μg and 230 μg [52] and 197 μg and 133 μg [53] per yolk, respectively. The concentration of lutein and zeaxanthin in egg yolks is not as high as in plant sources such as spinach (cooked 13 mg/100g) and kale (cooked 9 mg/100 g) [2]. However, the cholesterol (~200 mg/yolk), phospholipids (1 g/yolk) and triglycerides (~4 g/yolk) in the egg yolk provides an ideal lipid matrix for optimal absorption of these xanthophylls [54]. Carotenoids in the plant matrix are generally associated with chloroplasts or chromoplasts, which have to be broken down for absorption, thus affecting their bioavailability.

A number of studies have evaluated serum and macular lutein and zeaxanthin responses to egg consumption. Handelman et al were the first to show significant plasma lutein and zeaxanthin responses with consumption of 1.3 egg yolks/d for 5 weeks [54]. They found plasma lutein increases of 28% and 50% and plasma zeaxanthin increases of 142% and 114% when egg yolks were consumed with beef tallow and corn oil, respectively. Goodrow et al reported serum lutein and zeaxanthin. Additionally, there were also differences in study population, egg dosage and intervention duration. Increased egg consumption of 2 egg yolks/d and 4 egg yolks/d for 5 weeks resulted in serum zeaxanthin increases of 36% and 82%, and serum lutein increases of 16% and 24%, respectively [52]. Significant increases in plasma lutein and zeaxanthin were also observed with consumption of 3 eggs/d for 30 days and also 12 weeks [55, 56].

Two studies have shown that macular pigment optical density (MPOD) can be manipulated by consumption of eggs. Consumption of 1 egg/d for 12 weeks significantly increased MPOD in women aged 29 to 54 years [53]. Consumption of 2 egg yolks/d for 5 weeks and 4 egg yolks/d for 5 weeks significantly increased MPOD in subjects $\geq$60 years of age who had low baseline MPOD [52]. The percent increases in MPOD with 2 and 4 egg yolks (~946 μg and 2 mg lutein and zeaxanthin, respectively) were greater than increases observed in lutein supplementation studies that used 10 mg and 30 mg lutein supplements [57, 58]. Although levels of lutein and zeaxanthin are relatively lower in egg yolks compared to other sources, evidence suggests that they are highly bioavailable to the macula. The fact that macula is neural tissue makes these findings more compelling as it indicates eggs are able to influence lutein and zeaxanthin in neural tissue. It has been shown that the concentration of lutein and zeaxanthin in the macula is positively related to the concentrations in brain tissue in non-human primates [59]. Furthermore, recent findings

of a significant association between lutein in the cortex and pre-mortem cognitive function measures in older adults suggest that these carotenoids may be important for brain function [60].

CHOLESTEROL

One egg yolk has about 200 mg of cholesterol. The AHA recommends limiting dietary cholesterol intake to less than 300 mg/d for reducing the risk of cardiovascular disease (CVD) in a healthy population [1]. According to the AHA an egg can fit within heart healthy guidelines only if other dietary sources of cholesterol – meats, poultry and dairy products are limited. Because an egg contains two thirds the recommended upper limit for dietary cholesterol, it is often recommended that egg intake be limited. However, there is substantial evidence to show that egg consumption does not adversely affect blood lipids and lipoprotein cholesterol concentrations. Two meta analyses showed that saturated fat in the diet and not cholesterol increases blood cholesterol in the general population [61, 62].

Elevated LDL cholesterol (LDL-C) is a major cause of coronary heart disease [63]. However, 30% of patients diagnosed with premature coronary heart disease (CHD) have lipoprotein values that are within the National Cholesterol Education Program (NCEP) defined normal range [64]. Serum cholesterol response to dietary cholesterol largely varies from person to person. A normal response is defined as a 2.2 mg/dL or 1% increase in total cholesterol (1.9 mg/dL change in LDL-C and 0.4 mg/dL increase in HDL cholesterol (HDL-C) so as to maintain LDL/HDL ratio) for every 100 mg/d increase in dietary cholesterol. A hyper-response is defined as an increase in total cholesterol of >2.2 mg/dL and a hypo-response is defined as an increase in total cholesterol of <2.2 mg/dL for every 100 mg/d dietary cholesterol consumed [65, 66]. In hyperresponders dietary cholesterol raises not only LDL-C, but also HDL-C, keeping the LDL:HDL ratio intact, which is an important marker for CHD risk [67, 68]. Greene et al found significant increases in plasma LDL-C and HDL-C and no change in LDL:HDL ratio in elderly men and women who consumed 3 eggs/d for 30 days [69]. They showed that the observed increases in plasma LDL-C and HDL-C were due to increases in lipoprotein particle size. Hyperresponders had larger, less atherogenic LDL particle ($\geq$21.2 nm) and larger HDL particle (>8.8 nm) with no significant difference in the total number of LDL and HDL particles [55, 69]. Hyperresponders also had higher concentration of carotenoids [55]. Herron et al also reported increase in less atherogenic LDL-1 particle with egg consumption in hyperresponders [70]. LDL particles are heterogeneous with regard to size, density, composition, charge and atherogenicity. LDL subclass, as opposed to plasma LDL-C, is related to risk of CHD [71-73] as small and dense LDL particles can easily penetrate arterial walls [74]. The increase in LDL and HDL particle size observed during the 3 egg intervention was positively related to plasma lutein concentration [75]. No correlation was observed with number of HDL and LDL particles. Lutein and zeaxanthin are said to be associated with the surface layer of the lipoproteins [76]. Larger HDL and LDL particles have greater surface area and can thus carry more lutein and zeaxanthin [75]. Lutein and zeaxanthin are also beneficial in reducing the risk of CHD due to their antioxidant potential [77]. Furthermore, in

a hyperlipidemic population taking cholesterol lowering medications, addition of 2 egg yolks/d for 5 weeks, 3 eggs/d for 12 weeks and 4 egg yolks/d for 5 weeks to the normal diet was shown to significantly increase only serum HDL-C and not serum LDL-C [52, 78]. Data from the NHANES III showed that mean serum cholesterol concentrations was significantly lower in subjects who consumed ≥4 eggs per week compared to those who consumed ≤1 egg per week [4]. More frequent egg consumption was negatively associated with serum cholesterol concentration [4].

So far we have discussed studies that evaluated effects of egg consumption on blood lipids and lipoprotein cholesterol concentrations, which are predictors of CHD. We will now examine the evidence on egg consumption and incidence of CHD. Hu et al were among the first to report that consumption of 1 egg a day did not increase the risk of CHD and stroke in the Nurses' Health Study and Health Professionals Follow up study cohorts [79]. One study that evaluated CHD risk using risk apportionment model found that all modifiable lifestyle risk factors, which included smoking, overweight or obesity, poor diet, minimal exercise, alcohol intake and egg consumption accounted for 30-40% CHD risk in the US population aged 25 years and older. Consuming one egg a day accounted for only <1% of CHD mortality risk [80]. Two prospective studies found no association between egg consumption and CHD risk [81, 82]. One cross-sectional study reported that egg consumption was not associated with increased risk of mortality from CHD or stroke [83]. Also, consumption of 2 eggs a day for 6 weeks did not adversely affect endothelial function, a reliable index of CVD risk, in healthy adults [84].

Two groups of researchers, Djousse et al and Spence et al, have reported egg consumption to be associated with increased risk of heart failure, mortality in diabetics, type 2 diabetes and increased carotid plaque burden. Djousse et al analyzed the Physician's Health Study I (PHS I) cohort and reported increased risk of heart failure in physicians who consumed ≥7 eggs/week, but not in those who consumed up to 6 eggs/week [85]. Interestingly in a separate report on the same cohort, Djousse et al reported no association between egg consumption and incident myocardial infarction and stroke. But they reported an increased risk of all cause mortality with consumption ≥7 eggs/week in diabetics [86]. The relative risk models did not adjust for some important confounders of CVD including, intakes of saturated fat, cholesterol from other sources, total energy, fruits and vegetables, which could have overestimated the results. Other limitations include use of food frequency questionnaires (subjects had to recall egg intake in the past year) and the subject population who were all physicians (not representative of an average healthy individual). As Kritchevsky pointed out the lack of association with myocardial infarction and stroke in the second report by Djousse et al suggests high egg consumption is associated with noncardiovascular causes of death, which has no biological plausibility [87]. In a recent publication Spence et al report that egg consumption is significantly related to carotid plaque area, a strong predictor of CVD risk [88]. In this report, the observed exponential increase in plaque area with

egg consumption was similar to cigarette smoking. Similar to the Djousse et al report, this observational study fails to adjust for important covariates that are linked to CVD risk such as intake of saturated fat, other cholesterol sources, physical activity and fruits and vegetables. Also, egg consumption and smoking history were self reported. The strength of these observational studies is thus questionable.

FINAL REMARKS

Apart from the nutrients discussed above the effects of egg intake has also been studied in diabetics, metabolic syndrome and cancer patients. In 2009, Djousse et al reported that daily egg consumption was associated with increased risk of type 2 diabetes in men and women from the PHS I and the Women's Health Study [89]. But in 2010 they found no such association when they prospectively analyzed egg intake and incidence of type 2 diabetes in Cardiovascular Health Study cohort [90]. In a randomized controlled trial consumption of 3 eggs/d in conjunction with a carbohydrate restricted diet (CRD) did not increase plasma fasting glucose concentrations in overweight/obese men. Egg intervention increased plasma HDL cholesterol and lowered plasma triglycerides in these subjects, 58% of whom had metabolic syndrome, showing eggs do not modify the beneficial effects of CRD in metabolic syndrome [91]. CRD coupled with intake of 3 whole eggs also increased HDL and LDL particle size, reduced atherogenic large VLDL particles and small, dense LDL particles and increased lutein and zeaxanthin without affecting intake of other carotenoids with the exception of lycopene [56]. Furthermore, egg intake coupled with CRD improved insulin resistance in men and women with metabolic syndrome and decreased plasma C-reactive protein, an important marker of inflammation [92, 93]. Consuming eggs for breakfast was also shown to suppress plasma glucose, insulin and ghrelin response suggesting that eggs can effectively promote satiety and reduce subsequent energy intake [94]. Evidence on egg consumption and cancer risk are equivocal. Egg consumption was not associated with the development of colorectal polyps in a meta-analysis of 11 case-control and 2 cohort studies, while a positive association was observed with colon and rectal cancer risk and also with mortality from these cancers in a population-based study and [95-97]. A modest positive association was observed with egg consumption and breast cancer risk, while two studies showed no association [98-100]. Studies on prostate cancer risk have also yielded mixed results [101-104]. The effects of egg consumption on cancer risk warrants further investigation.

CONCLUSION

Based on the variety of evidence presented in this review the recommendation on limiting egg consumption may need to be reconsidered. The evidence that egg intake can increase CHD risk is weak. Studies have shown that dietary cholesterol from eggs increased LDL and HDL lipoprotein size and not number, which is suggestive of a protective effect due to their decreased atherogenicity. Larger LDL and HDL particles can accommodate more carotenoids, especially lutein and zeaxanthin, which are highly bioavailable from eggs. Egg intake causes increased accumulation of lutein and zeaxanthin in the macula and greater protection of the retina from AMD. The choline and high quality protein in an egg

make it a valuable addition to a nutritious diet. Recent studies also suggest that metabolic syndrome patients who are on a CRD may benefit from egg consumption. The nutrient density and affordability of eggs make them a highly attractive whole food. An egg symbolizes the start of life and thus nutrients that are incorporated in an egg are those that are essential for life. When consumed as part of a healthy, well balanced diet, the benefits of egg consumption can outweigh any risks. Eat your eggs minus the sausage and bacon.

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