

# Revisiting Dietary Cholesterol Recommendations: Does the Evidence Support a Limit of 300 mg/d?

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**Abstract** The perceived association between dietary cholesterol (DC) and risk for coronary heart disease (CHD) has resulted in recommendations of no more than 300 mg/d for healthy persons in the United States. These dietary recommendations proposed in the 1960s had little scientific evidence other than the known association between saturated fat and cholesterol and animal studies where cholesterol was fed in amounts far exceeding normal intakes. In contrast, European countries, Asian countries, and Canada do not have an upper limit for DC. Further, current epidemiologic data have clearly demonstrated that increasing concentrations of DC are not correlated with increased risk for CHD. Clinical studies have shown that even if DC may increase plasma low-density lipoprotein (LDL) cholesterol in certain individuals (hyper-responders), this is always accompanied by increases in high-density lipoprotein (HDL) cholesterol, so the LDL/HDL cholesterol ratio is maintained. More importantly, DC reduces circulating levels of small, dense LDL particles, a well-defined risk factor for CHD. This article presents recent evidence from human studies documenting the lack of effect of DC on CHD risk, suggesting that guidelines for DC should be revisited.

**Keywords** Dietary cholesterol · LDL cholesterol · HDL cholesterol · LDL size · Clinical studies · Epidemiologic data · Eggs

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## Introduction

The American Heart Association (AHA) recommends no more than 300 mg/d of dietary cholesterol (DC) for healthy persons to prevent increased risk for coronary heart disease (CHD) [1]. These recommendations are mostly based on the presence of both saturated fat and cholesterol in many foods and on data derived from animal studies where supraphysiologic doses of cholesterol, ranging from the equivalent of 1,000 mg to 20,000 mg/d for humans, were fed in order to produce atherosclerosis [2].

It is important to note that many other countries do not have the same guidelines for DC. Canada [3•], Korea [4•], New Zealand [5], and India [6], for example, do not set an upper limit for DC, focusing instead on controlling the intake of saturated fat and trans fat, which are the major determinants of blood cholesterol concentrations. Similarly, the European guidelines on cardiovascular disease prevention have the following recommendations regarding healthy food choices: “consume a wide variety of foods, adjust energy intake to maintain a healthy weight, encourage consumption of fruits and vegetables, replace saturated fat with mono or polyunsaturated fatty acids and reduce salt intake” [7]. In contrast to US policies, Europeans have no dietary guidelines for DC [7]. A summary of the dietary recommendations for DC in different countries, including two recent reports from the AHA, is presented in Table 1.

Epidemiologic studies do not support an association between cholesterol intake and CHD [8–12]. This could partly be explained by the fluctuations in response to dietary cholesterol among all individuals, which varies from no changes to large increases in plasma cholesterol. In addition, it is critical to note that for those individuals who have hypercholesterolemic response to dietary cholesterol (about one third of the population), the rise is typically due to

**Table 1** Dietary guidelines for saturated fat and dietary cholesterol in different countries

Author/year	Country/region	Institution/society	Dietary fat guidelines	Cholesterol guidelines
Genest et al. [3••]/2009	Canada	2009 Canadian Cardiovascular Society/ Canadian guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease in the adult	Limit intake of saturated fat to <10% of energy	No recommendation for dietary cholesterol
Task Force Members [7]/2007	Europe	European guidelines on cardiovascular disease prevention in clinical practice: Fourth Joint Task Force of the European Society of Cardiology and Other societies on cardiovascular disease prevention in clinical practice.	Limit intake of saturated fat to <10% of total energy	No recommendation for dietary cholesterol
National Institute of Nutrition [6]/2010	India	National Institute of Nutrition	Limit saturated fat and total fat	No recommendation for dietary cholesterol
Pai et al. [4•]/2008	Korea	Korean Nutrition Society	Total fat <20%	No recommendation for dietary cholesterol
Ministry of Health [5]/2003	New Zealand	Food and Nutrition Guidelines for Healthy Adults	Limit saturated fat to <12% of total energy	No recommendation for dietary cholesterol
Lichenstein et al. [1]/2006	United States	AHA Scientific Committee	Limit intake of saturated fat to <7%	< 300 mg/d
Lloyd-Jones et al. [16••]/2009	United States	AHA Special Report. Defining and setting National goals for cardiovascular health promotion and disease reduction.	Limit intake of saturated fat and trans fat	Dietary cholesterol not mentioned
Gidding et al. [17••]/2010	United States	AHA Pediatric and Adult Nutrition Guidelines: a scientific statement from the AHA Nutrition Committee	Limit intake of saturated fat and trans fat	Adolescents and children. Dietary cholesterol not mentioned

AHA American Heart Association.

increases in both plasma low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C) concentrations, with no alterations in the LDL-C/HDL-C ratio.

### Epidemiologic Studies

Extensive research derived from epidemiologic studies, including Framingham [8], the Nurses' Health study [9], National Health and Nutrition Examination Survey (NHANES) [10], and the Lipid Research Clinics Prevalence Follow-up Study [11], do not support a relationship of increased intake of cholesterol and CHD events [12]. In contrast, Weggermans et al. [13] analyzed 17 clinical studies and concluded that eggs or other cholesterol-rich foods raise the ratio total cholesterol to HDL-C, adversely affecting the associations with CHD risk. However, clinical trials evaluating DC effects from the past three decades show that effects of DC on plasma cholesterol obtained in the short term (eg, 2 weeks) do not reflect the effects of high intakes over longer periods of time [14], and this has led Conti et al. [15•] to caution organizations such as the AHA against publishing clinical guidelines based on a shallow evidence base. In a recent report by the AHA regarding the goals for cardiovascular health promotion, dietary habits with the

strongest evidence for causal effects on cardiovascular events were emphasized [16••]. In this report, the Dietary Approaches to Stop Hypertension (DASH) diet was highly recommended, as was a restriction of saturated fat and trans fat, but there was no mention of DC [16••]. Similarly, Gidding et al. [17••], in their recommendations for the AHA Pediatric and Adult Nutrition guidelines, gave no specific guidelines for DC.

A recent article reviewed the epidemiologic data relating egg consumption and CHD. When multiple regression analysis was used, no relationship was found [18••]. Constance [19] discussed the complexity of atherosclerosis development, indicating that LDL is not the only causal factor and that egg consumption has a low association with atherogenic risk. The relative prohibition of eggs therefore was not considered to be well supported [19]. Nakamura et al. [20] report an inverse association between egg consumption and total cholesterol concentration, possibly due to hypercholesterolemic individuals avoiding eggs. However, they did not find an association between eating eggs and CHD incidence in two cohorts of 90,735 Japanese men and women. Qureshi et al. [21] evaluated egg intake in 9734 adults and divided them into those consuming less than 1 egg per week or more than 6 eggs per week. Egg consumption was not associated with stroke, ischemia, or CHD in these individuals. However, there was

an increase in CHD in patients diagnosed with diabetes at the highest level of egg consumption [21]. Other studies also suggest that individuals with diabetes should limit egg intake [22, 23•]. In summary, the preponderance of the epidemiologic evidence from the past 14 years does not support a relationship between dietary cholesterol (or egg intake) and risk for CHD [4•, 8–14], [15•, 16••, 17••]. Accordingly, neither Europe [7], Canada [3••], nor Asian countries [4•, 5] restrict dietary cholesterol as part of the recommendations for a heart-healthy diet.

### Eggs and Dietary Cholesterol

The AHA still recommends limiting other food items high in cholesterol if eggs are to be consumed [1] in spite of recent reports that show no association between egg intake and risk for heart disease [8–11, 18••, 19–21]. In fact, there are no studies with substantial evidence supporting the claims of egg consumption involved in CHD risk. In contrast, a recent analysis in which a risk-apportionment approach was used on the risk factors for CHD revealed that egg intake contributes to less than 1% of the risk, and the authors conclude that AHA dietary guidelines possibly should be revised [18••]. Eggs are the only food that is both rich in cholesterol and low in saturated fat, perhaps explaining why eggs are often used to evaluate the effects of dietary cholesterol on plasma lipids and CHD risk [8–11, 24, 25]. Other cholesterol-containing foods, such as dairy products, also contain high concentrations of saturated fat, which is a confounder for dietary cholesterol effects. This might be the reason why controversial results exist regarding the effects of dairy products on CHD risk [26].

Clinical trials conducted in children [27], younger adults [24, 25], and the elderly [28, 29] have clearly demonstrated that although dietary cholesterol provided by eggs significantly increases LDL-C in one third of the population, those individuals considered hyper-responders to a cholesterol challenge exhibit increases in both LDL-C and HDL-C, with

the result of no changes in the LDL-C/HDL-C ratio, a major predictor of CHD [30•]. These results indicate that individuals with initial plasma cholesterol concentrations that place them at a low risk for CHD do not develop an atherogenic lipoprotein profile following the consumption of additional dietary cholesterol, regardless of their response classification.

It is well established that nutritional interventions aimed at managing plasma lipids are known to be less effective in obese and overweight individuals [31]. During a weight loss intervention, intake of 3 eggs per day for 12 weeks was shown to selectively increase plasma HDL-C without increasing LDL-C in overweight men [32••]. Harman et al. [33•] also reported no changes in LDL-C after consuming 2 eggs per day for 12 weeks in a weight loss intervention study. Intake of only 1 egg per day increased HDL-C without increasing LDL-C in men and women aged 40–60 years, resulting in a lower LDL-C/HDL-C ratio [34]. Similarly, in a study in which 56 participants with a mean age of 35 years were given an additional egg per day during 12 weeks, significant increases were reported for HDL-C with no changes in LDL-C [35•]. A summary of plasma LDL-C and HDL-C concentrations as a response to egg intake in recent clinical studies is presented in Table 2.

To evaluate whether insulin resistance, with or without obesity, influences the response to dietary cholesterol, Knopp et al. [36] recruited 197 healthy individuals into a randomized crossover study in which 0, 2, and 4 eggs per day were fed for 4 weeks with a 1-month washout period in between. The participants were classified as insulin sensitive ( $n=65$ ), insulin resistant ( $n=75$ ), and obese insulin resistant (OIR,  $n=58$ ). Insulin-resistant and insulin-sensitive individuals had significant increases in LDL-C of 7.8% and 3.3%, respectively, after consuming 4 eggs per day, whereas OIR individuals had no changes in LDL-C at any intake level. In contrast, HDL-C was significantly increased for all groups even after the consumption of only 2 eggs per day. These studies suggest that dietary management of OIR individuals need not include restrictions on eggs.

**Table 2** Changes in LDL-C, HDL-C, LDL size, and HDL size as a response to dietary cholesterol provided by eggs in various populations

Population	Duration	Additional dietary cholesterol	LDL-C	HDL-C	LDL-C/HDL-C ratio	LDL size	HDL size
Children ( $n=54$ ) [27]	4 wk	518 mg/d	Increase	Increase	No change	Increase	ND
Women ( $n=51$ ) [25]	4 wk	640 mg/d	Increase	Increase	No change	Increase	ND
Men ( $n=28$ ) [32••]	12 wk	640 mg/d	No change	Increase	Decrease	Increase	Increase
Men/women ( $n=42$ ) [34]	12 wk	215 mg/d	No change	Increase	No change	Increase	Increase
Men/women ( $n=34$ ) [28]	4 wk	640 mg/d	Increase	Increase	No change	Increase	Increase
Men/women ( $n=56$ ) [35•]	12 wk	250 mg/d	No change	Increase	Decrease	ND	ND
Men/women ( $n=45$ ) [33•]	12 wk	400 mg/d	No change	No change	No change	ND	ND

HDL-C high-density lipoprotein cholesterol; LDL-C low-density lipoprotein cholesterol; ND not determined.

## Effects of Eggs on Atherogenic Lipoproteins

The association between elevated LDL-C and an increased risk for CHD has been well documented [37]. However, it is now well established that LDL particles are heterogeneous with regard to size, density, composition, charge, and atherosclerotic potential [38]. These characteristics need to be taken into consideration when we evaluate the effects of any dietary component on plasma lipoprotein levels and atherogenic potential. The Adult Treatment Panel III acknowledged the predominance of small, dense LDL particles as an emerging CHD risk factor [37]. The increase in cardiovascular risk, attributable to plasma lipids, is significantly modulated by variations in LDL particle size and number [39]. Utilizing nuclear magnetic resonance spectroscopy, Blake et al. [40] reported that median baseline values of LDL particle concentration were higher and LDL particle size was lower among women who subsequently experienced a coronary event. In a cohort of men from the Quebec Cardiovascular Study, the association between LDL particle size and the incidence of ischemic heart disease yielded similar results [41]. Thus, concentrations of LDL-C alone do not provide a comprehensive evaluation of CHD in the general population. Egg intake has been shown to increase the number of both large LDL and HDL particles while decreasing the concentrations of small LDL subfractions [42••]. Other studies have also shown a shift of the atherogenic pattern B to pattern A following egg intake in children [27] and in adults [43], and these larger LDL particles showed no increased susceptibility to oxidation. In addition, individuals having a lower number of the large HDL particles as well as increased number of small HDL particles are considered to be at increased risk for heart disease [38], possibly because of the existing relationship between large HDL and more efficient reverse cholesterol transport. Increases in large HDL as measured by nuclear magnetic resonance spectroscopy have been reported in adults consuming 3 eggs per day for 4 weeks [34] and in individuals following a weight loss intervention and consuming 3 eggs per day for 12 weeks [42••].

## Other Beneficial Effects of Eggs

The current recommendations for dietary cholesterol pose a challenge to those individuals who might benefit from including eggs in their regular diets. Eggs have numerous nutritional benefits that are often overlooked when eggs are viewed as nothing more than a cholesterol-containing food. Eggs contain lutein and zexanthin, two major carotenoids that are known to protect against macular

degeneration [44] and the development of cataracts [45••]. These carotenoids are highly bioavailable in eggs compared with other dietary sources [46], and egg consumption has been associated with increases in macular pigment density [34, 41], which protects against increasing visual range by absorbing blue light and decreasing oxidative stress [47]. Because egg intake results in formation of larger HDL particles [48], the transport of these carotenoids in a larger particle becomes more efficient. Lutein may also protect against inflammation by decreasing C-reactive protein and other inflammatory markers [49]. Eggs are also very good sources of highly bioavailable and affordable protein [50]. Eggs in combination with a low-carbohydrate diet have also been shown to decrease insulin resistance and leptin in overweight individuals [51]. Finally, a recent study that compared two types of breakfasts (bagel based vs egg based) reported consumption of 400 less kcal in the same day after the egg-based compared with the bagel-based breakfast [52•]. In addition, the egg-based breakfast was associated with decreases in ghrelin, the hormone associated with increased appetite, indicating a potential role of eggs in weight loss interventions. In agreement with these observations, more efficient weight loss was seen when egg-based breakfasts were consumed as part of an energy-deficient diet as compared with bagel-based breakfasts [53]. The only cautionary note regarding egg intake is that, as mentioned earlier, diabetic individuals may benefit from limiting egg intake [22]. However, for healthy populations across all ages, eggs can be part of an overall heart-healthy diet, as stated by Eckel [54•].

## Conclusions

The epidemiologic studies and clinical trial results reviewed here suggest that compelling evidence is lacking for limiting cholesterol intake to 300 mg/d. Accordingly, the current US dietary guidelines should be re-evaluated. Based on recent evidence, the current restrictions on cholesterol have largely and inappropriately been translated into a reduced consumption of eggs, a highly nutritious food. The fact that most other countries have chosen to not only not limit eggs, but dietary cholesterol itself, indicates that the overall evidence cannot sustain such limitations. Why not follow their example and accept the challenge of modifying these guidelines?

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## References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. Lichtenstein AH, Appel LJ, Brands M, et al.: Diet and Lifestyle recommendations Revision 2006. A scientific statement from the American Heart Association Scientific Committee. *Circulation* 2006, 114:82–96.
2. McNamara DJ: Dietary cholesterol and atherosclerosis. *Biochim Biophys Acta* 2000, 1529:310–320.
3. •• Genest J, McPherson R, Frohlich J, et al.: 2009 Canadian Cardiovascular Society/Canadian guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease in the adult—2009 recommendations. *Can J Cardiol* 2009, 25:567–579. *This is an updated guideline for diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease in adults. The article discusses well-established risk factors for CHD, including elevated concentrations of LDL-C and emerging factors such as inflammatory markers and lipoprotein(a). Nutrition recommendations focus on maintenance of healthy weight and reduction of sugars, saturated fat and trans fat, and salt. There is no recommendation for dietary cholesterol.*
4. • Pai HY, Kim CI, Moon HK, et al.: 2008 Dietary goals and dietary guidelines for Korean adults. *Korean J Nutr* 2008, 41:887–899. *This article describes the Nutritional health plan for Korea in 2010. Recommendations that are emphasized are balance of energy intake and physical activity, food safety, and limitations of salt intake and total fat. There are no recommendations for dietary cholesterol.*
5. Ministry of Health: Food and Nutrition Guidelines for Healthy Adults. New Zealand: Ministry of Health; 2003:18–21.
6. National Institute of Nutrition. Available at <http://www.invinindia.org>. Accessed June 5, 2010.
7. Task Force Members, Graham I, Atar D, Borsch-Johnsen K, et al.: European guidelines on cardiovascular disease prevention in clinical practice: executive summary. Fourth Joint Task Force of the European Society of Cardiology and Other societies on cardiovascular disease prevention in clinical practice. *Eur Heart J* 2007, 28:2375–2414
8. McNamara DJ: Cholesterol intake and plasma cholesterol, an update. *J Am Coll Nutr* 1997, 16:530–534.
9. Hu FB, Stampfer MJ, Rimm EB, Manson JE, et al.: A prospective study of egg consumption and risk of cardiovascular disease in men and women. *JAMA* 1999, 281:1387–1394.
10. Song WO, Kerver JM: Nutritional contribution of eggs to American diets. *J Am Coll Nutr* 2000, 5(Suppl):556S–562S.
11. Esrey KL, Joseph L, Grover SA: Relationship between dietary intake and coronary heart disease mortality: lipid research clinics prevalence follow-up study. *J Clin Epidemiol* 1996, 49:211–216.
12. Howell WH, McNamara DJ, Tosca MA, et al.: Plasma lipid and lipoprotein responses to dietary fat and cholesterol. *Am J Clin Nutr* 1997, 65:1747–1764.
13. Weggermans RM, Zock PL, Katan MB: Dietary cholesterol from eggs increases the ratio of total cholesterol to high-density lipoprotein cholesterol in humans: a meta-analysis. *Am J Clin Nutr* 2001, 73:885–891.
14. Okuyama H, Ichikawa Y, Sun Y, et al.: The cholesterol hypothesis, its basis and its faults. *World Rev Nutr Dietetics* 2007, 96:1–17.
15. • Conti AA, Dilaghi B, Modesti PA, Nozzoli C: New evidence in cardiovascular medicine, general practice and public health. *Intern Emerg Med* 2009, 4:343–345. *In this article, the authors assess all dietary interventions using DC or eggs and derive their own conclusions. They point out that short-term effects where there is a rise in plasma cholesterol with DC should be interpreted with caution because they differ from long-term effects where there is no relationship between DC and plasma cholesterol.*
16. •• Lloyd-Jones DM, Hung Y, Labarthe D, Mozaffarian D: AHA Special Report. Defining and setting National goals for cardiovascular health promotion and disease reduction. *Circulation* 2010, 121:586–613. *This is a comprehensive report on national goals for cardiovascular health promotion in the United States in which emphasis is made on lifestyle factors and health for adolescents and children. Among the dietary guidelines, trans fat and saturated fat are recommended to be restricted, but there is no specific mention of dietary cholesterol.*
17. •• Gidding SS, Lichtenstein AH, Faith MS, et al.: Implementing AHA Pediatric and Adult Nutrition Guidelines: a scientific statement from the AHA Nutrition Committee of the Council on Nutrition, Physical Activity and Metabolism. *Circulation*, 2009:119:1161–1175. *These are important dietary guidelines crafted by the AHA Nutrition Committee. Emphasis is placed on reducing salt intake to decrease hypertension and the DASH diet is recommended. There are recommendations against consuming saturated fat and trans fat and there are no specific guidelines regarding dietary cholesterol.*
18. •• Barraj L, Tran N, Mink P, et al.: Comparison of egg consumption with other modifiable coronary heart disease lifestyle risk factors: a relative risk apportionment study. *Risk Anal* 2009, 29:401–415. *This study supports the lack of relationship between eggs and CHD. The authors created a model to evaluate risk factors for CHD using data from the Nurse's Health Study and the Health Professionals Follow-up Study plus National Health and Nutrition Examination Survey data. They concluded that 1 egg per day contributes to less than 1% of the CHD mortality*
19. Constance C: The good and the bad: what researchers have learned about dietary cholesterol, lipid management and cardiovascular disease risk since the Harvard Egg Study. *Intern J Clin Pract* 2009, 63(Suppl):9–14.
20. Nakamura Y, Iso H, Kita Y, et al.: Egg consumption, serum total cholesterol concentrations and coronary heart disease incidence: Japan Public Health Center-based prospective study. *Br J Nutr* 2006, 96:921–928.
21. Qureshi AI, Suri FK, Ahmed S, et al.: Regular egg consumption does not increase the risk of stroke and cardiovascular diseases. *Med Sci Monit* 2007, 13:CR1–8.
22. Djousee L, Gaziano JM: Egg consumption in relation to cardiovascular disease and mortality. *The Physicians Health Study. Am J Clin Nutr* 2008, 87:964–969.
23. • Houston DK, Ding J, Lee JS, et al.: Dietary fat and cholesterol and risk of cardiovascular disease in older adults: The Health ABC Study. *Nutr Met Card Dis* 2010 (in press). *The associations between dietary fat, DC, and eggs were analyzed in 1941 adults aged 70 to 79 years. Dietary cholesterol and egg consumption were associated with increased CHD risk only in individuals with type 2 diabetes.*
24. Herron KL, Vega-Lopez S, Ramjiganesh T, et al.: Men classified as hypo- or hyper-responders to dietary cholesterol feeding exhibit differences in lipoprotein metabolism. *J Nutr* 2003, 133:1036–1042.
25. Herron KJ, Vega-Lopez S, Conde K, et al.: Pre-menopausal women classified as hypo- or hyper-responders, do not alter their

- LDL/HDL ratio following a high dietary cholesterol challenge. *J Am Coll Nutr* 2002, 21:250–258.
26. German JB, Gibson RA, Krauss RM, et al.: A reappraisal of the impact of dairy foods and milk fat on cardiovascular disease risk. *Eur J Nutr* 2009, 48:191–204.
  27. Ballesteros MN, Cabrera RM, Saucedo MS, Fernandez ML: Dietary cholesterol does not increase biomarkers for chronic disease in a pediatric population at risk from Northern Mexico. *Am J Clin Nutr* 2004, 80:855–861.
  28. Greene CM, Zern TL, Wood R, et al.: Maintenance of the LDL cholesterol: HDL cholesterol ratio in an elderly population given a dietary cholesterol challenge. *J Nutr* 2005, 135:2793–2798.
  29. Homma Y, Kobayashi T, Yamaguchi H, et al.: Apolipoprotein-E phenotype and basal activity of low-density lipoprotein receptor are independent of changes in plasma lipoprotein subfractions after cholesterol ingestion in Japanese subjects. *Nutrition* 2001, 17:310–314.
  30. • Fernandez ML, Webb D: The LDL to HDL cholesterol ratio as a valuable tool to evaluate coronary heart disease risk. The impact of dietary cholesterol. *J Am Coll Nutr* 2008, 27:1–5. *This review provides evidence on the importance of the LDL-C/HDL-C ratio in assessing the risk for CHD, with data derived from several large clinical studies. In addition, the effect of dietary cholesterol in maintaining the ratio across different populations is discussed.*
  31. Hill AM, Rousell MA, Kris-Etherton P: Nutritional management of lipids for overweight and obesity: what can we achieve? *Future Lipidol* 2008, 3:573–584.
  32. •• Mutungi G, Ratliff J, Puglisi M, et al.: Dietary cholesterol from eggs increases HDL cholesterol in overweight men consuming a carbohydrate restricted diet. *J Nutr* 2008, 138:272–276. *This study is a weight loss intervention in which participants followed a carbohydrate-restricted diet. Half of the participants were consuming 3 whole eggs whereas the other half were consuming 3 egg substitutes for a period of 12 weeks. At the end of the intervention, although all participants had significant decreases in plasma triglycerides and no changes in LDL-C, only those individuals in the egg group had a significant increase in plasma HDL-C.*
  33. • Harman NL, Leeds AR, Griffin BA: Increased dietary cholesterol does not increase plasma low density lipoprotein when accompanied by an energy-restricted diet and weight loss. *Eur J Nutr* 2008, 47:287–293. *In this study, a combination of increasing dietary cholesterol and weight loss was tested in people consuming 2 eggs per day (n=24) or no eggs (n=21). Energy intake was reduced and participants lost weight. There were no changes in LDL-C for any of the participants in the study. The authors concluded that cholesterol-rich foods should not be excluded from dietary advice in those patients losing weight*
  34. Ata S, Barona J, Kopec R, et al.: Consumption of one regular egg or a lutein-enriched egg per day increases HDL cholesterol, reduces apolipoprotein B and the number of small LDL particles while increasing plasma carotenoids and macular pigment density in adult subjects. *FASEB J* 2010, 24:A92.4.
  35. • Mayurasakorn K, Srisura W, Sitphahul P, Hongto PO: High-density lipoprotein cholesterol changes after continuous egg consumption in healthy adults. *Med Assoc Thai* 2008, 91:400–407. *In this study, 56 individuals 35 years of age consumed 1 additional egg per day. There were no changes in LDL-C after 12 weeks and HDL-C increased.*
  36. Knopp RH, Retzlaff B, Fish B, et al.: Effects of insulin resistance and obesity on lipoproteins and sensitivity to egg feeding. *Arterioscler Thromb Vasc Biol* 2003, 23:1437–1443.
  37. 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). Final report. *Circulation* 2002, 106:3143–3421.
  38. Siri PW, Krauss RM: Influence of dietary carbohydrate and fat on LDL and HDL particle distribution. *Curr Atheroscler Rep* 2005, 7:455–459.
  39. Berneis K, Rizzo M: LDL size: does it matter? *Swiss Med Wkly* 2004, 134:720–724.
  40. Blake GJ, Otvos JD, Rifai N, Ridker PM: Low-density lipoprotein particle concentration and size as determined by nuclear magnetic resonance spectroscopy as predictors of cardiovascular disease in women. *Circulation* 2002, 106:1930–1937.
  41. Lamarche B, St-Pierre AC, Ruel IL, et al.: A prospective, population-based study of low density lipoprotein particle size as a risk factor for ischemic heart disease in men. *Can J Cardiol* 2001, 17:859–865.
  42. •• Mutungi G, Waters D, Ratliff J, et al.: Eggs distinctly modulate plasma carotenoid and lipoprotein subclasses in adult men following a carbohydrate restricted diet. *J Nutr Biochem* 2010, 21:261–267. *In this study, participants (n=30) followed a carbohydrate-restricted diet. Half of the participants were randomly assigned to consume 3 eggs per day and the other half 3 egg substitutes per day. Individuals consuming the whole eggs had significant increases in HDL-C, large HDL, and large LDL compared with those fed the egg substitutes. In addition, they also experienced a greater decrease in small LDL, indicating that egg consumption beneficially altered all atherogenic lipoproteins. Further, there were significant decreases in apolipoprotein B, indicating a decreased number of overall atherogenic particles.*
  43. Herron KL, Lofgren IE, Sharma M, et al.: A high intake of dietary cholesterol does not result in more atherogenic LDL particles in men and women independent of response classification. *Metab Clin Exp* 2004, 53:823–830.
  44. Subczynski WK, Wisniewska A, Widomska J: Location of macular xanthophylls in the most vulnerable regions of photoreceptor outer-segment membranes. *Arch Biochem Biophys* 2010 (in press).
  45. •• Vishwanathan R, Goodrow-Kotyla EF, Wooten BR, et al.: Consumption of 2 and 4 egg yolks/d for 5 wk increases macular pigment concentrations in older adults with low macular pigment taking cholesterol-lowering statins *Am J Clin Nutr* 2009, 90:1272–1279. *This study was conducted in older adults. The key findings are that participants did not experience an increase in LDL-C after consuming 2 to 4 egg yolks during a 5-week period and that there were increases in macular pigment density due to the high content of lutein in the eggs.*
  46. Chung HY, Rasmussen HM, Johnson EJ: Lutein bioavailability is higher from lutein-enriched eggs than from supplements and spinach in men. *J Nutr* 2004, 134:1887–1893.
  47. Ribaya-Mercado JD, Blumberg JB: Lutein and zeaxanthin and their potential roles in disease prevention. *J Am Coll Nutr* 2004, 23:567S–587S.
  48. Greene CM, Waters D, Clark RM, et al.: Plasma LDL and HDL characteristics and carotenoid content are positively influenced by egg consumption in an elderly population. *Nutr Metab* 2006, 3:6
  49. Ratliff J, Mutungi G, Puglisi M, et al.: Eggs modulate the inflammatory response to carbohydrate restricted diets in overweight men. *Nutr Metab (Lond)* 2008, 5:6
  50. Drewnowski A: The Nutrient Rich Foods Index helps to identify healthy, affordable foods. *Am J Clin Nutr* 2010, 91:1095S–1101S.
  51. Ratliff J, Puglisi M, Mutungi G, et al.: Carbohydrate restriction (with or without additional dietary cholesterol provided by eggs) reduces insulin resistance and plasma leptin without modifying appetite hormones in adult men. *Nutr Res* 2009, 29:262–268.
  52. • Ratliff JC, Leite JO, DeOgurn R, et al.: Consuming eggs for breakfast influences plasma glucose and ghrelin, while reducing caloric intake during the next 24 hours in adult men. *Nutrition Res* 2010, 30:96–103. *In this study, a comparison was made in the*

- same participants of an egg-based versus a bagel-based breakfast on kilocalorie intake in the next 24 hours on appetite and on plasma ghrelin concentrations. Participants consumed a lighter lunch after the egg breakfast. They also consumed 400kcal less in the next 24 hours and had lower levels of plasma ghrelin.*
53. Vander Wal JS, Gupta A, Khosla P, Durandhar MV: Egg breakfast enhances weight loss. *Int J Obes (London)* 2008, 32:1545–1551.
54. • Eckel RH: Egg consumption in relation to cardiovascular disease and mortality: the story gets more complex. *Am J Clin Nutr* 2008, 87:799–800. *This editorial reviews the effects of eggs on cardiovascular disease from data coming from the Physicians' Health study. It concluded that diabetic individuals could benefit from restricting egg intake. However, the author pointed out that eggs can be part of a healthy diet.*