

Dietary Cholesterol and Diabetes, What do We know About this Relationship?

Maria Luz Fernandez *

Department of Nutritional Sciences, University of Connecticut Storrs, CT 06269, USA

*Corresponding author: Maria Luz Fernandez, Department of Nutritional Sciences, University of Connecticut Storrs, CT 06269, USA Tel: +860 486 5547; E-mail: maria-luz.fernandez@uconn.edu

Received date: January 29, 2016; Accepted date: February 01, 2016; Published date: February 10, 2016

Copyright: © 2016 Fernandez ML. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Editorial

The new USDA dietary guidelines, released on January 7, 2016, removed the upper limits for dietary cholesterol [1] on what would appear to be a bold and unprecedented decision. However, US is not the only nation recognizing that dietary cholesterol does not affect blood cholesterol levels since similar dietary guidelines have been supported by a number of countries for the past few years [2]. In support of these guidelines, Eckel et al. [3] had already published in 2014 a report on life style and heart disease in which they clearly stated that there was not sufficient evidence to sustain that reducing dietary cholesterol would reduce LDL cholesterol.

Although the lack of effect of dietary cholesterol [or eggs, that have been used as a high cholesterol-containing food] on coronary heart disease risk has been demonstrated both in epidemiological analysis and well-controlled clinical trials for healthy populations [4], there is still a level of uncertainty on the association between dietary cholesterol and diabetes. Most of the controversy raises from epidemiological data in which dietary cholesterol has been correlated to either have an increased risk for heart disease [5,6], or no effect [7,8] in diabetic individuals. Therefore, a possibility that dietary cholesterol could have a negative effect on diabetic patients appears to be a legitimate concern according to epidemiological analysis although the presence of confounding variables cannot be ruled out.

In contrast to epidemiological analysis, clinical interventions assessing the effects of eggs (dietary cholesterol) on diabetic patients have failed to find detrimental effects on plasma lipids, glucose metabolism or inflammation [9-11]. In contrast, these studies reported beneficial effects of eggs in these patients. For example, in a clinical trial in which high and low cholesterol diets were provided to 65 individuals with type-2 diabetes mellitus [9], authors reported no differences in plasma lipids between groups while the high cholesterol diet was more effective in improving glycemic control and the plasma lipid profile by raising HDL cholesterol [9]. Ballesteros et al. [10] reported that in a randomized cross-over intervention in 29 diabetic patients, consumption of 1 cup of oatmeal versus 1 egg for breakfast did not change plasma lipids, lipoprotein size distribution, glucose, insulin or insulin resistance but resulted in improvements in inflammatory markers and liver enzymes [10]. In another study, 140 patients with diabetes were assigned to consume either a high egg diet (2/d) or a low egg diet (2/wk.) for 6 weeks [11]. No adverse effects on the lipid profile was observed in those individuals with the high egg intake; this groups also reported to be less hungry and to have a greater satiety after breakfast.

It is then clear that the few clinical interventions, which have been conducted in diabetic patients, have reported similar findings [9-11], that is a lack of detrimental effects of eggs on lipoprotein or glucose metabolism. However, there is controversy in terms of epidemiological data on this regard [5-8], In order to have a better understanding between the relationship of dietary cholesterol and risk for heart disease in conditions of diabetes, more prospective studies should focus on the evaluation of these relationships and increased number of clinical interventions should be conducted before a solid conclusion can be reached.

References

1. <http://www.npr.org/sections/thesalt/2016/01/07/462160303/new-dietary-guidelines>
2. Fernandez ML, Calle MC (2010) Revisiting dietary cholesterol recommendations: does the evidence support a 300 mg/d limit? *Curr Atherosclerosis Rep* 12: 377-383.
3. Eckel RH, Jakicic JM, Ard JD, Miller NH, Hubbard BS, et al. (2014) 2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk. *J. Am Coll Cardiol* 63: 2960-84.
4. Fernandez ML (2010) Effects of eggs on plasma lipoproteins in healthy populations. *Food & Function* 1: 156-160.
5. Radzeviciene L, Ostrauskas R (2012) Egg consumption and the risk of type 2 diabetes mellitus: a case-control study. *Public Health Nutr* 15: 1437-1441.
6. Djousse L, Gaziano Jm, Buring JE, Lee IM (2009) Egg consumption and risk of type 2 diabetes in men and women. *Diabetes Care* 32: 295-300.
7. Agrawal S, Ebrahim S (2011) Prevalence and risk factors for self-reported diabetes among adult men and women in India: findings from a national cross-sectional survey. *Pub Health Nutr* 15: 1065-1077.
8. Chagas P, Caramori P, Galdino TP, de Barcellos dC, Gomez I, et al. (2013) Egg consumption and coronary atherosclerotic burden. *Atherosclerosis* 229: 381-384.
9. Pearce KL, Clifton PM, Noakes M (2011) Egg consumption as part of an energy-restricted high-protein diet improves blood lipid and blood glucose profiles in individuals with type 2 diabetes. *Brit J Nutr* 105: 584-892.
10. Ballesteros MN, Valenzuela F, Robles AE, Artalejo E, Aguilar D, et al. (2015) One egg per day improves inflammation when compared to an oatmeal-based breakfast without increasing other cardiometabolic risk factors in diabetic patients. *Nutrients* 7: 3449-3463.
11. Fuller NR, Caterson ID, Sainsbury A, Denyer G, Fong M, et al. (2015). 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: 705-713