Egg consumption and risk of non-alcoholic fatty liver disease

Zeinab Mokhtari, Hossein Poustchi, Tannaz Eslamparast, Azita Hekmatdoost

Department of Clinical Nutrition and Dietetics, Faculty of Nutrition Sciences and Food Technology, National Nutrition and Food Technology Research Institute, Shahid Beheshti University of Medical Sciences, Tehran 1981619573, Iran

Hossein Poustchi, Liver and Pancreatobiliary Diseases Research Group, Digestive Diseases Research Institute, Tehran University of Medical Sciences, Tehran 14117, Iran

Author contributions: Mokhtari Z, Poustchi H, Eslamparast T and Hekmatdoost A conceptualized and designed the study and wrote the manuscript; Mokhtari Z analyzed data; Eslamparast T and Poustchi H collected data; Hekmatdoost A and Poustchi H interpreted the data, provided professional comments, and critically revised the manuscript for intellectual content and data accuracy.

Institutional review board statement: The study was approved by the ethics committee of National Nutrition and Food Technology Research Institute, Tehran, Iran.

Informed consent statement: All patients signed the informed consent form.

Conflict-of-interest statement: None of the authors had any personal or financial conflicts of interest to report.

Data sharing statement: Technical appendix, statistical code, and dataset available from the corresponding author at a_hekmat2000@yahoo.com.

Open-Access: This article is an open-access article which was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: http://creativecommons.org/licenses/by-nc/4.0/

Manuscript source: Invited manuscript

Correspondence to: Azita Hekmatdoost, MD, PhD, RD, Department of Clinical Nutrition and Dietetics, Faculty of Nutrition Sciences and Food Technology, National Nutrition and Food Technology Research Institute, Shahid Beheshti University of Medical Sciences, West Aghavan St., Farahzadi Blvd., P.O. Box: 19395-4741, Tehran 1981619573, Iran. a_hekmat2000@yahoo.com Telephone: +98-21-22360658 Fax: +98-21-22360657

Received: August 27, 2016 Peer-review started: August 29, 2016 First decision: September 27, 2016 Revised: January 4, 2017 Accepted: March 14, 2017 Article in press: March 17, 2017 Published online: April 8, 2017

Abstract

AIM
To evaluate the association between egg consumption and risk of non-alcoholic fatty liver disease (NAFLD) development.

METHODS
This case-control study was conducted on individuals who were referred to two hepatology clinics in Tehran, Iran in 2015. The study included 169 patients with NAFLD and 782 controls. Egg consumption was estimated using a validated food frequency questionnaire. The participants were categorized according to the frequency of their egg consumption during the previous year: Less than two eggs per week, two to three eggs per week, and four or more eggs per week.

RESULTS
In the crude model, participants who consumed 2 to 3 eggs per week, were 3.56 times more likely to have NAFLD in comparison to those who consumed less than 2 eggs per week (OR: 3.56; 95%CI: 2.35-5.31). Adjustment for known risk factors of NAFLD strengthened
this significant association so that individuals have consumed two to three eggs per week had 3.71 times higher risk of NAFLD than those who have eaten less than two eggs per week (OR: 3.71; 95%CI: 1.91, 7.75).

CONCLUSION
Our data indicate that higher egg consumption in common amount of usage is associated with higher risk of NAFLD.

Key words: Egg; Diet; Non-alcoholic fatty liver disease; Dietary cholesterol

© The Author(s) 2017. Published by Baishideng Publishing Group Inc. All rights reserved.

Core tip: The data indicate that egg consumption in common amount of usage is associated with risk of non-alcoholic fatty liver disease. According to the case-control design of this study, it can not show the causality effect; thus, these findings should be confirmed in future prospective studies with separate parts of eggs to find the etiological relationships.

Mokhtari Z, Poustchi H, Eslamparast T, Hekmatdoost A. Egg consumption and risk of non-alcoholic fatty liver disease. World J Hepatol 2017; 9(10): 503-509 Available from: URL: http://www.wjgnet.com/1948-5182/full/v9/i10/503.htm DOI: http://dx.doi.org/10.4254/wjh.v9.i10.503

INTRODUCTION
Non-alcoholic fatty liver disease (NAFLD) includes a spectrum of liver disorders from simple steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, cirrhosis, and even hepatocellular carcinoma[1]. NAFLD is the most common cause of chronic liver diseases around the world[2] and may be considered as hepatic manifestation of metabolic syndrome[3]. The increasing prevalence of obesity, together with insulin resistance, hypertension, dyslipidemia, and eventually the metabolic syndrome dispose many people to the risk of NAFLD development in the futureyears[4].

Increasing evidence showed that dietary factors contribute to the pathophysiology and treatment of NAFLD[5-7]. Among the known dietary factors that involved in the development of NAFLD, dietary cholesterol has drawn a great deal of attention. Current studies of animal models propose that excess dietary cholesterol is regarded as the key factorrelated to the risk of steatohepatitis and hepatic inflammation[8-10]. Addition of cholesterol to the diet of obese, diabetic mice increased the accumulation of hepatic free cholesterol, hepatocyte apoptosis, and liver fibrosis[11]. Moreover, an association between raised cholesterol intake and the risk or severity of NAFLD has been addressed by epidemiological studies[12-14].

Among single foods, eggs are regarded as a main source of dietary cholesterol, with one large egg containing almost 210 mg of cholesterol; on the other hand, eggs are rich in proteins, and other nutrients[15], which can improve human health. There is limited evidence on the relationship between egg consumption and NAFLD and its risk factors with controversial results[16-18]. Therefore, the present study was designed to examine the association between egg consumption and risk of NAFLD development.

MATERIALS AND METHODS

Participants
The present case-control study was conducted on individuals who were undertaken a liver Ultrasound, and were referred to two Hepatology clinics in Tehran, Iran in 2015. The study included 169 patients with NAFLD and 782 controls. The cases were patients with NAFLD, which was diagnosed by a gastroenterologist according to the presence of hepatic steatosis in Ultrasound exam within previous month, and referred to our clinics to be examined by Fibroscan®, and the Fibroscan results showed a Controlled Attenuation Parameter score of more than 263, and fibrosis score of more than 7. These patients were selected with the convenience-sampling procedure. Controls were randomly selected age- and sex-matched subjects from the same clinic among patients with pancreatobiliary disorders who had been undertaken an Ultrasound showing no hepatic steatosis. The age ranges for matching were 20-40, 40-60 and > 60 years old. Data on each pair of cases and controls were collected at the same time. The participation rate in the study was 94% for cases and 98% for controls. Written informed consent was obtained from all the participants. The study protocol was approved by the local Ethics Review Committee.

Assessment of dietary intake
Dietary intake of patients was assessed using a valid and reliable semi-quantitative food frequency questionnaire (FFQ), which included 168 items of foods with standard servingsizes, as commonly consumed by Iranians[19]. The consumption frequency of each food item was questioned on a daily, weekly or monthly basis and converted to daily intakes. In the case of egg consumption, the participants were categorized according to the frequency of their egg consumption during the previous year: Less than two eggs per week, two to three eggs per week, and four or more eggs per week. Dietary nutrients intakes were calculated using NUTRITIONIST V (First Databank, Hearst Corp, San Bruno, CA, United States). The patients who had completed less than 90% of dietary questionnaires and subjects who reported extremely low or high energy intakes (< 500 or > 5000 kcal/d) were excluded from the study[20].

Assessment of other variables
Physical activity was evaluated using the metabolic
Table 1 Baseline characteristics, biochemical parameters and dietary intakes of study participants based on the patients with non-alcoholic fatty liver disease and control group

|                          | Cases (n = 169) | Controls (n = 782) | P value* |
|--------------------------|----------------|-------------------|----------|
| Age (yr), mean ± SD      | 53.9 ± 12.21    | 43.7 ± 14.52      | 0.373    |
| Male n (%)               | 81 (47.9)       | 314 (40.2)        | 0.063    |
| BMI (kg/m²), mean ± SD   | 33.19 ± 8.71    | 27.74 ± 4.495     | < 0.001  |
| Physical activity (MET), mean ± SD | 31.89 ± 3.15 | 34.33 ± 2.85 | < 0.001  |
| Current smokers, n (%)   | 185 (66.7)      | 145 (18.5)        | < 0.001  |
| Drink alcohol in past year, n (%) | 22 (13.1) | 68 (8.7) | 0.077    |
| Diabetes type 2, n (%)    | 31 (18.5)       | 53 (6.8)          | < 0.001  |
| FBS (mg/dL), mean ± SD   | 109.29 ± 39.39  | 90.09 ± 29.24     | < 0.001  |
| Total cholesterol (mg/dL), mean ± SD | 184.79 ± 54.94 | 177.72 ± 38.74 | 0.221    |
| LDL (mg/dL), mean ± SD   | 121.17 ± 45.04  | 104.26 ± 31.65    | < 0.001  |
| HDL (mg/dL), mean ± SD   | 41.26 ± 16.72   | 47.72 ± 10.51     | 0.001    |
| Triglycerides (mg/dL), mean ± SD | 180.40 ± 123.81 | 131.97 ± 81.59 | < 0.001  |
| Total energy (kcal), mean ± SEM | 2627.87 ± 6.13 | 2746.69 ± 27.23 | 0.068    |
| Carbohydrate (% of total energy), mean ± SEM | 58.12 ± 0.95 | 59.82 ± 0.44 | 0.001    |
| Protein (% of total energy), mean ± SEM | 15.84 ± 0.13 | 14.07 ± 0.08 | < 0.001  |
| Fat (% of total energy), mean ± SEM | 29.23 ± 0.30 | 33.78 ± 0.20 | < 0.001  |
| Dietary cholesterol (mg/d), mean ± SEM | 315.31 ± 11.50 | 263.41 ± 5.35 | < 0.001  |
| Saturated fat (g/d), mean ± SEM | 30.62 ± 5.72 | 62.67 ± 2.67 | < 0.001  |
| Monounsaturated fat (g/d) (mg/d), mean ± SEM | 29.85 ± 0.48 | 32.00 ± 0.23 | < 0.001  |
| Polyunsaturated fat (g/d) (mg/d), mean ± SEM | 18.51 ± 5.74 | 59.88 ± 2.67 | < 0.001  |
| Dietary fiber (g/d), mean ± SEM | 19.21 ± 0.50 | 14.68 ± 0.23 | < 0.001  |
| Red/processed meats (g/d), mean ± SEM | 70.95 ± 2.66 | 30.60 ± 1.24 | < 0.001  |

*Independent t-test for quantitative variables and χ² test for qualitative variables. Dietary intakes (except total energy) were adjusted for age and total energy intake. BMI: Body mass index; MET: Metabolic equivalent task; FBS: Fasting blood sugar; LDL: Low-density lipoprotein cholesterol; HDL: High-density lipoprotein cholesterol.

Statistical analysis

Baseline characteristics and dietary intakes were compared between cases and controls using t-test for continuous variables and χ² for categorical variables. Egg consumption was divided into three ascending categories on an ordinal scale. Mean or prevalence of baseline characteristics was computed for each category. Baseline characteristics were also compared using ANOVA for continuous variables and χ² categorical variables. The relationship between NAFLD and egg consumption was assessed using multivariate regression analysis. Estimates were presented in three models; the first model was adjusted for age (continuous) and total energy intake (kcal/d). In these models, we further controlled for body mass index (BMI), history of diabetes and smoking (non-smoker, current smoker). Finally, we further adjusted for physical activity (MET) and gender. All models were conducted by treating the first category of egg consumption (< 2/wk) as a reference. All the statistical analyses were done using SPSS for Windows (version 19; SPSS Inc., Chicago, IL).

RESULTS

Baseline characteristics, biochemical parameters and dietary intakes of the cases and controls are shown in Table 1. Mean age of the total study population was 43.54 ± 14.13 years and 41.5% (395) of participants were male. By design, cases and controls had the similar age and sex distribution. Patients with NAFLD had significantly more BMI, lower physically active, lower consumption of alcohol, and were more likely to be smoker, and have diabetes in comparison to controls. Furthermore, the cases had elevated fasting blood glucose (FBS), low-density lipoprotein cholesterol (LDL), Triglycerides, and reduced high density lipoprotein cholesterol (HDL) levels and increased intake of protein, cholesterol, fiber and red/processed meats compared with the controls (Table 1).

In secondary analysis, there was a similar egg-NAFLD association in women (P-trend 0.001) and men (P-trend 0.048) (Table 3).

Multivariate adjusted odds ratios for NAFLD based on egg consumption categories are indicated in Figure 1. In the crude model, participants that consumed 2 to 3 eggs per week, were 3.56 times more likely to have NAFLD in...
The role of diet and dietary supplements on the pathogenesis of NAFLD have been shown previously\[23-36\], however, to our knowledge, no study has yet evaluated the association of egg consumption and NAFLD risk. It is well established that eggs contain a wide variety of essential nutrients and bioactive compounds that can affect human health. Their high quality protein, fats and micronutrients and low price make them an important part of many people’s diet\[23\]. Despite the nutritional benefits of egg consumption, there are concerns about their high content of cholesterol and saturated fat and their influences on metabolic disorders\[38\]. Thus, one possible explanation for the inverse association between egg consumption and risk of NAFLD development may be due to the high cholesterol content of egg. Previous studies have shown that a higher consumption of cholesterol is associated with NAFLD and its exacerbation\[12,13,39,40\]. In addition, the presence of high amount of cholesterol in diet is necessary for development of NAFLD\[41\]. Baumgartner et al\[39\] have shown that daily egg consumption increases serum cholesterol and LDL-C concentrations in women; however, there was no effect on markers for inflammation, endothelial activity, and liver function. Interestingly, the consumption of egg white hydrolyzed with pepsin considerably improved hepatic liver function. Interestingly, the consumption of egg white hydrolyzed with pepsin considerably improved hepatic steatosis\[42\]. Thus, it seems that the association between egg consumption and NAFLD is mainly due to high cholesterol content of it, and might not be seen when people consume only the white part of it. Therefore, more studies are recommended to evaluate the effects of consumption of different parts of egg on NAFLD risk\[13\].

An unexpected finding of the present study was that more than 4 eggs consumption per week was not significantly associated with risk of NAFLD. This may be explained by the fact that nutritional factors are correlated with each other and determining of the effect of particular nutrients or particular foods on a risk factor is difficult. The effects of egg cholesterol on serum cholesterol concentrations depends on the content of individuals’ diet specially the fiber content of it\[13,44\]. It is possible that those who ate more than 4 eggs per week, consumed it in mixed dishes containing vegetables, which reduces the absorption of cholesterol. Thus, we compared to those who consumed less than 2 eggs per week (OR: 3.56; 95%CI: 2.35-5.31). After controlling for age and total energy intake, consuming 2 to 3 eggs per week was positively associated with the risk of NAFLD (OR: 3.83; 95%CI: 2.49-5.89). These associations remained significant even after additionally controlling for BMI, history of diabetes and smoking (OR: 3.83; 95%CI: 2.49-5.89). Further adjustment for physical activity, and gender strengthened this significant association so that individuals who have consumed two to three eggs per week had 3.71 times higher risk of NAFLD than those who have eaten less than two eggs per week (OR: 3.71; 95%CI: 1.91-7.75). Egg consumption more than four per week was not significantly associated with the NAFLD risk.

**DISCUSSION**

The results of the present study showed that the egg consumption increases the risk of NAFLD in common range of its consumption (two to three eggs per week). This relationship was also significant after adjustment for age, gender, BMI, history of diabetes, smoking, and physical activity.

The role of diet and dietary supplements on the pathogenesis of NAFLD have been shown previously\[23-36\], however, to our knowledge, no study has yet evaluated the association of egg consumption and NAFLD risk.
suggest that future studies assess the type of dishes with egg to find the possible interactions of different constituent of them.

It has been reported that dietary intake of patients with NAFLD was richer in saturated fat, cholesterol and was poorer in polyunsaturated fat [12]. Subramanian et al [40] have concluded that dietary cholesterol confers in progression of NAFLD to NASH. Furthermore, Zelber-Sagi et al [18] found that NAFLD patients have a higher intake of meat, which is another source of dietary cholesterol; however, some other studies only found a significant association between NAFLD and high dietary intake of carbohydrate and simple sugars [45,46], and some studies did find an association only between NAFLD and low intake of n-3 fatty acids and some antioxidants [16]. These dietary habits may accelerate the development of NAFLD by directly affecting steatosis of liver and oxidative injury [12].

This study was the first study that examined the relationship between egg consumption and risk of NAFLD in newly diagnosed patients who have not probably changed their diet due to the disease diagnosis; other strengths of this study includes its relatively large sample size, the high participation rate of participants, and socioeconomic differences of participants, which affects their dietary intakes.

Although we used a validated FFQ for measurement of dietary intakes, measurement error, and recall bias are unavoidable errors. Moreover, there might be some unknown risk factors that affect our results. Therefore, we recommend this analysis to be done in other populations.

In conclusion, our data indicate that egg consumption in common amount of usage is associated with risk of NAFLD. According to the case-control design of this study, it can not show the causality effect; thus, these findings should be confirmed in future prospective studies with separate parts of eggs to find the etiological associations.

ACKNOWLEDGMENTS

This work was supported by a grant from “National Nutrition and Food Technology Research Institute” of Shahid Beheshti University of Medical Sciences, and Digestive Disease Research Institute, Tehran University of Medical Sciences, Tehran, Iran.

COMMENTS

Background
Among the known dietary factors that affect the pathogenesis of non-alcoholic fatty liver disease (NAFLD), dietary cholesterol has drawn a great deal of attention. Current studies propose that excess dietary cholesterol is regarded as the key factor related to the risk of steatohepatitis and hepatic inflammation. Among individual foods, eggs are regarded as a main source of dietary cholesterol; on the other hand, eggs are rich in proteins, and other nutrients. Limited research has assessed the relationship between egg consumption and risk of (NAFLD) development.

Research frontiers
Understanding of the association between egg consumption and risk of NAFLD development can contribute to clarify how intake of special food groups correlate with the disease and could lead to more particular guidelines for NAFLD prevention.

Innovations and breakthroughs
This study showed that egg consumption in common amount of usage is associated with risk of NAFLD. It seems that this association is mainly due to

Figure 1 Multivariate-adjusted odds ratio for non-alcoholic fatty liver disease according to egg consumption. A: Crude model; B: Model 2, multivariate adjusted for age and energy intake; C: Model 3, further controlled for, body mass index, history of diabetes and smoking; D: Model 4, additionally adjusted for physical activity, and gender. Data are presented as the odds ratio (95% CI).
high cholesterol content of it, and might not be seen when people consume only the white part of it.

**Applications**

According to the results of this study, the authors recommend low intake of eggs specially the yolk part of it for prevention of NAFLD; however, further studies are recommended to reach to a consensus in this regard.

**Peer-review**

This is an interesting paper evaluating the association between egg consumption and NAFLD.

**REFERENCES**

1. **Angulo P.** Nonalcoholic fatty liver disease. *N Engl J Med* 2002; 346: 1221-1231 [PMID: 11961152 DOI: 10.1056/NEJMra011775]
2. **Vernon G, Baranova A, Younossi ZM.** Systematic review: the epidemiology and natural history of non-alcoholic fatty liver disease and non-alcoholic steatohepatitis in adults. *Aliment Pharmacol Ther* 2011; 34: 274-285 [PMID: 21623852 DOI: 10.1111/j.1365-2036.2011.04724.x]
3. **Marchesini G, Brizi M, Bianchi G, Tomassetti S, Bugianesi E, Lenzi M, McCullough AJ, Natale S, Forlani G, Melchionda N.** Nonalcoholic fatty liver disease: a feature of the metabolic syndrome. *Diabetes 2001; 50: 1844-1850* [PMID: 11473047 DOI: 10.2373/diabetes.50.8.1844]
4. **Marchesini G, Bugianesi E, Forlani G, Cerrelli F, Lenzi M, Marinini N, Santei V, Villanova E, Melchionda N, Rizzetto M.** Nonalcoholic fatty liver, steatohepatitis, and the metabolic syndrome. *Hepatology 2003; 37: 917-923* [PMID: 12668987 DOI: 10.1053/hep.2003.50161]
5. **Papandreou A, Andreou E.** Role of diet on non-alcoholic fatty liver disease: An updated narrative review. *World J Gastroenterol 2010; 16: 775-582* [PMID: 25848481 DOI: 10.4245/wjg.v7.i3.575]
6. **Nsei W, Narras F, ASSY.** Soft drinks consumption and nonalcoholic fatty liver disease. *World Gastroenterol 2010; 16: 2579-2588* [PMID: 20518077 DOI: 10.3748/wig.v16.i21.2579]
7. **Zelber-Sagi S, Nitzan-Kaluski D, Goldsmith R, Webb M, Blends L, Halpern Z, Oren R.** Long term nutritional intake and the risk for non-alcoholic fatty liver disease (NAFLD): a population based study. *J Hepatol 2007; 47: 711-717* [PMID: 17850914 DOI: 10.1016/j.jhep.2007.06.020]
8. **Esfahani FH, Asghari G, Mirrmon P, Azizi F.** Reproducibility and relative validity of food group intake in a food frequency questionnaire developed for the Tehran Lipid and Glucose Study. *J Epidemiol 2010; 20: 150-158* [PMID: 20154450]
9. **Jaceldo-Siegk K, Knutsen SF, Sabaté J, Beeles WL, Chan J, Herring RP, Butler TL, Haddad E, Bennett H, Montgomery S, Sharma SS, Oda K, Fraser GE.** Validation of nutrient intake using an FFQ and repeated 24 h recalls in black and white subjects of the Adventist Health Study-2 (AHS-2). *Public Health Nutr 2010; 13: 812-819* [PMID: 19968897 DOI: 10.1017/s1368980009992072]
10. **Aadahl M, Jørgensen T.** Validation of a new self-report instrument for measuring physical activity. *Med Sci Sports Exerc 2003; 35: 1196-1202* [PMID: 12840642 DOI: 10.1249/01.mss.0000074446.02192.14]
11. **Kelishadi R, Kabiri K, Khorasani A, Famouri F, Sadeghi M, Rouhaifa H, Shirani S.** Assessment of physical activity of adolescents in Isfahan. *2001*
12. **Askari F, Rashidkhani B, Hekmatdoost A.** Cinnamon may reduce inflammation and nonalcoholic fatty liver disease: An updated narrative review. *World J Gastroenterol 2010; 16: 800-803* [PMID: 20143458]
13. **Song WO, Kerver JM.** Nutritional contribution of eggs to American diets. *J Am Coll Nutr 2000; 19: 556S-562S* [PMID: 11023007 DOI: 10.1080/07317527.2000.10718980]
14. **Han JM, Jo AN, Lee SM, Bae HS, Jun DW, Cho YK, Suk KT, Yoon JH, Ahn SB, Cho YJ, Kim SW, Jang EC.** Associations between intake of individual nutrients or whole food groups and non-alcoholic fatty liver disease among Korean adults. *J Gastroenterol Hepatol 2014; 29: 1265-1272* [PMID: 24955455 DOI: 10.1111/jgh.12520]
15. **Shi L, Liu ZW, Li Y, Gong C, Zhang H, Song LJ, Huang CY, Li M.** The prevalence of nonalcoholic fatty liver disease and its association with lifestyle/dietary habits among university faculty and staff in Chengdu. *Biomed Environ Sci 2012; 25: 383-391* [PMID: 23026517 DOI: 10.3967/0895-3988.2012.04.002]
16. **Zelber-Sagi S, Nitzan-Kaluski D, Goldsmith R, Webb M, Blends L, Halpern Z, Oren R.** Long term nutritional intake and the risk for non-alcoholic fatty liver disease (NAFLD); a population based study. *J Hepatol 2007; 47: 711-717* [PMID: 17850914 DOI: 10.1016/j.jhep.2007.06.020]
17. **Eslamparast T, Mokhtari Z, Babazehi P.** Dietary habits and their relation to insulin resistance and postprandial lipemia in non-alcoholic steatohepatitis. *Hepatology 2003; 37: 909-916* [PMID: 12668986 DOI: 10.1053/jhep.2003.50132]
18. **Yasatuka K, Nakamura M, Shima Y, Ohyama A, Masuda K, Haruta N, Fujimoto T, Aoyagi Y, Fukuzumi K, Yoshimoto T, Takemoto R, Miyahara T, Harada N, Hayata F, Nakashima M, Enjoji M.** Nutritional intervention of non-obese patients with non-alcoholic fatty liver disease: the significance of dietary cholesterol. *Scand J Gastroenterol 2009; 44: 471-477* [PMID: 19058085 DOI: 10.1080/00365520802588133]
19. **Enjoji M, Nakamura M.** Is the control of dietary cholesterol intake sufficiently effective to ameliorate nonalcoholic fatty liver disease? *World J Gastroenterol 2010; 16: 204-212* [PMID: 20529475 DOI: 10.4245/wjg.v7.i2.204]
20. **Eslamparast T, Poustchi H, Zarrabi H, Malekzadeh R, Hekmatdoost A.** Syntrophic supplementation in nonalcoholic fatty liver disease patients. *J Nutr Sci Food Technol 2013; 59: 129-136* [PMID: 24829682]
21. **Eslamparast T, Eslamparast A, Poustchi H.** Probiotics and Nonalcoholic Fatty liver Disease. *Middle East J Dig Dis 2013; 5: 129-136* [PMID: 24829682]
Mokhtari Z et al. Egg consumption and NAFLD risk

P. Rodriguez JA, Lamas A, Franco CM, Cepeda A. Egg and egg-derived foods: effects on human health and use as functional foods. *Nutrients* 2015; 7: 706-729 [PMID: 25608941 DOI: 10.3390/nutrients7050706]

Weggemans 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 [PMID: 11338411]

Baumgartner S, Kelly ER, van der Made S, Berendschot TT, Husche C, Lütjohann D, Plat J. The influence of consuming an egg or an egg-yolk buttermilk drink for 12 wk on serum lipids, inflammation, and liver function markers in human volunteers. *Nutrition* 2013; 29: 1237-1244 [PMID: 23911216 DOI: 10.1016/j.nut.2013.03.020]

Subramanian S, Goodspeed L, Wang S, Kim J, Zeng L, Ioannou GN, Haigh WG, Yeh MM, Kowdle KV, O’Brien KD, Pennathur S, Chait A. Dietary cholesterol exacerbates hepatic steatosis and inflammation in obese LDL receptor-deficient mice. *J Lipid Res* 2011; 52: 1626-1635 [PMID: 21690266 DOI: 10.1194/jlr.M016246]

Emamati H, Noori M, Foroughi F, Rismanchi M, Eini-Zinab H, Hekmatdoost A. An Accessible and Pragmatic Experimental Model of Nonalcoholic Fatty Liver Disease. *Middle East J Dig Dis* 2016; 8: 109-115 [PMID: 27252817 DOI: 10.15171/mejd.2016.15]

Garcés-Rimón M, González C, Uranga JA, López-Miranda V, López-Fandiño R, Miguel M. Pepsin Egg White Hydrolysate Ameliorates Obesity-Related Oxidative Stress, Inflammation and Steatosis in Zucker Fatty Rats. *PLoS One* 2016; 11: e0151193 [PMID: 26985993 DOI: 10.1371/journal.pone.0151193]

Cohn JS, Kamili A, Wat E, Chung RW, Tandy S. Reduction in intestinal cholesterol absorption by various food components: mechanisms and implications. *Atheroscler Suppl* 2010; 11: 45-48 [PMID: 20439167 DOI: 10.1016/j.atherosclerosis.2010.04.004]

Gunness P, Gidley MJ. Mechanisms underlying the cholesterol-lowering properties of soluble dietary fibre polysaccharides. *Food Funct* 2010; 1: 149-155 [PMID: 21774645 DOI: 10.1039/c0fo00080a]

Solga S, Alkhuraishie AR, Clark JM, Torbenson M, Greenwald A, Diehl AM, Magnuson T. Dietary composition and nonalcoholic fatty liver disease. *Dig Dis Sci* 2004; 49: 1578-1583 [PMID: 15573908 DOI: 10.1023/B:DDAS.0000004336.6970.87]

Toshimitsu K, Matsuura B, Okubo I, Niyi T, Furukawa S, Hiasa Y, Kawamura M, Ebihara K, Onji M. Dietary habits and nutrient intake in non-alcoholic steatohepatitis. *Nutrition* 2007; 23: 46-52 [PMID: 17140767 DOI: 10.1016/j.nut.2006.09.004]

P- Reviewer: Bellanti F, Tziomalos K S- Editor: Kong JX L- Editor: A E- Editor: Li D
