**Egg consumption and risk of Non-alcoholic fatty liver disease (NAFLD)**

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**Running Title**: Egg consumption and NAFLD risk

There is no conflict of interest.

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**Abstract**

The role of dietary intakes in development of Nonalcoholic Fatty Liver Disease

(NAFLD) has benn shown previously; however, there is no study evaluating the role of egg consumption in NAFLD development. This case–control study was conducted to evaluate the association between egg consumption and risk of NAFLD in 169 patients with NAFLD and 782 controls. Egg consumption was estimated using a validated food frequency questionnaire. In the crude model, participants that 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 have eaten less than two eggs per week (OR: 3.71; 95%CI: 1.91, 7.75). In spite of this, egg consumption more than four per week were not significantly associated with the NAFLD odds ratio. In conclusion, our data indicate that higher egg consumption in common amount of usage is associated with higher risk of NAFLD. These findings need to be confirmed in future prospective studies with separate part of eggs to find the etiological relationships.

**INTRODUCTION**

Non-alcoholic fatty liver disease (NAFLD) is defined as liver pathologic spectrum, which is initiated by a lipid accumulation in the liver (steatosis) that may be progressed to Non-alcoholic steatohepatitis (NASH) (1). NAFLD is the most common cause of chronic liver diseases around the world (2) and may be considered the hepatic event in the metabolic syndrome (3). The increasing prevalence of obesity, together with insulin resistance, hypertension, dyslipidemia, and eventually the metabolic syndrome puts a very large people at risk of development of liver failure in the future years (4).

Increasing evidence has shown that dietary factors have been contributed to the pathophysiology and treatment of NAFLD (5-7). Among the known dietary factors that involved in the development of NAFLD, dietary cholesterol has drown a great deal of attention. Current studies of animal models propose that excess dietary cholesterol is regarded as the key factor related to the risk of steatohepatitis and hepatic inflammation (8-10). Addition of cholesterol to the diet of obese, diabetic mice has been 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 individual 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 referred for NAFLD to two clinics in Tehran, Iran in 2015. The study included 169 patients with NAFLD and 782 controls. The cases were patients with NAFLD who were diagnosed with NAFLD by a gastroenterologist within previous month, and referred to our clinics to be examined by Fibroscan, and the Fibroscan results showed a Controlled Attenuation Parameter (CAP) 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 pancreatobiliay disorders who had been undertaken an Ultrasound showing no hepatic steatosis. The age ranges for matching were 20–40, 40–60, and >60 year 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 through a valid and reliable semi-quantitative food frequency questionnaire (FFQ), which included 168 items of foods with standard serving sizes, 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; portion sizes were then converted to grams using household measures. 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. The collected data were analyzed using NUTRITIONIST V (First Databank, Hearst Corp, San Bruno, CA, USA).The patients who had not completed more than 10% of dietary questionnaires and subjects who reported extremely low or high energy intakes (<500 or >5000 kcal /day) were excluded from the study (20).

**Assessment of other variables**

Physical activity was also assessed using the metabolic equivalent task (MET) questionnaire (21-22). Additional covariate information including age, sex, smoking habits, medical history, and current use of medications were obtained by questionnaires. All patients underwent measurements of weight and height. Each individual’s body mass index (BMI) was calculated by using the following formula: BMI=weight (in kg)/height (in m2).

**Statistical analysis**

Baseline characteristics and dietary intakes were compared between those with and without NAFLD using t-test for continuous variables and chi-square 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 chi-square for categorical variables. The relationship between NAFLD and egg consumption was assessed using multiple regression analysis. Estimates were presented in three models; the first model was adjusted for age (continuous), and total energy intake (kcal/d). In the second model, we further controlled for BMI, history of diabetes and smoking (non-smoker, current smoker). Finally, we further adjusted for physical activity (MET), alcohol intake and gender. All models were conducted by treating the first category of egg consumption (<2/week) as a reference. All probability values presented are two-tailed, and probability values below 0.05 were considered statistically significant. All the statistical analyses were done using SPSS for Windows (version 19; SPSS Inc., Chicago, IL).

**Results:**

Baseline characteristics, biochemical parametersand 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 activity, 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).

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| **Table 1. Baseline characteristics, biochemical parameters and dietary intakes of study participants based on the patients with NAFLD and control group**  |
|  | Cases (n=169) | Controls (n= 782) | *P* Value a |
| Age; yr, (mean ± SD) | 42.65±12.21 | 43.71±14.52 | 0.373 |
| Male n (%) | 81(47.9) | 314(40.2) | 0.063 |
| BMI(kg m–2), (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(%) | 151(89.9) | 145(18.5) | <0.001 |
| Drank alcohol in past year, n(%) | 22(13.1) | 68(8.7) | 0.077 |
| Diabetes type 2, n(%) | 26(15.6) | 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±43.04 | 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) | 2627.67±61.39 | 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±0.18 | 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/day),( mean ± SEM) | 315.31±11.50 | 263.41±5.35 | <0.001 |
| Saturated Fat (g/day), (mean ± SEM) | 30.62±5.72 | 62.67±2.67 | <0.001 |
| Monounsaturated Fat (g/day) (mg/day), (mean ± SEM) | 29.85±0.48 | 32.00±0.23 | <0.001 |
| Polyunsaturated Fat (g/day) (mg/day), (mean ± SEM) | 18.51±5.74 | 59.58±2.67 | <0.001 |
| Dietary fiber (g/day), (mean ± SEM) | 19.21±0.50 | 14.68±0.23 | <0.001 |
| Red/processed meats (g/day), (mean ± SEM) | 70.95±2.66 | 36.00±1.24 | <0.001 |
| a Independent t-test for quantitative variables and chi-squared test for qualitative variablesDietary intakes (except total energy) were adjusted for age and total energy intake |

Basic characteristics and dietary intakes of the studied participants by categories of egg consumption are presented in Table 2. Compared to egg consumption of lower than two per week, higher egg consumption was associated with a lower average age, male sex, current smoking, higher energy intake, lower percent of total energy from carbohydrate and fat. Additionally, the subjects with higher egg consumption tended to consume more protein, cholesterol, monounsaturated fat and red/processed meats, but less saturated and polyunsaturated fatty acids (Table 2).

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| **Table 2. Basic characteristics and dietary intakes of study participants by frequency of egg consumption n(%)** |
|  | Egg Consumption Categories | *P* Value a  |
|  | <2/week(n=589) | 2-3/week(n=142) | 4≤ /week(n=220) |  |
| Age | 45.65±12.26 | 39.73±13.18 | 40.35±13.30 | <0.001 |
| Male gender | 218(37.0) | 56(39.4) | 121(55) | <0.001 |
| BMI(kg m–2), mean ± SD | 28.58±5.44 | 29.60±7.34 | 28.51±5.87 | 0.150 |
| Physical activity( MET), mean ± SD | 33.99±3.05 | 33.42±3.21 | 33.94±2.95 | 0.136 |
| Current smokers | 155(26.3) | 59(41.8) | 82(37.3) | <0.001 |
| Total energy (kcal), mean ± SEM | 2580.59±30.68 | 2744.94±57.45 | 3101.07±51.20 | <0.001 |
| Carbohydrate (% of total energy), mean ± SEM | 60.44±0.67 | 59.48±0.63 | 58.14±0.85 | 0.001 |
| Protein (% of total energy), mean ± SEM | 14.09±0.10 | 14.71±0.20 | 14.95±0.17 | 0.001 |
| Fat (% of total energy), mean ± SEM | 33.06±0.24 | 32.56±0.49 | 32.97±0.40 | <0.001 |
| Dietary Cholesterol (mg/day) | 226.40±5.75 | 291.95±11.60 | 383.90±9.53 | <0.001 |
| Saturated Fat (g/day) | 56.70±3.16 | 64.70±6.38 | 52.57±5.24 | <0.001 |
| Monounsaturated Fat (g/day) (mg/day), mean ± SEM | 31.20±0.26 | 31.32±0.53 | 32.91±0.44 | <0.001 |
| Polyunsaturated Fat (g/day) (mg/day), mean ± SEM | 53.10±3.20 | 57.26±6.45 | 46.71±5.30 | <0.001 |
| Dietary fiber (g/day) | 15.65±0.28 | 16.25±0.57 | 14.60±0.47 | <0.001 |
| Red/processed meats (g/day) | 37.76±1.53 | 47.79±3.10 | 50.51±2.54 | <0.001 |
| Dietary intakes (except total energy) were adjusted for age and total energy intake |

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

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| **Table 3. Odds ratio for NAFLD according to egg consumption stratified by gender** |
| Multivariate adjusted Model a |
| Egg Consumption | Female | Male  |
| <2/week | 1.00 | 1.00 |
| 2-3 /week | 5.55(2.30-13.37) | 1.90(0.50-7.16) |
| 4≤ /week | 1.67(0.68-4.10) | 0.25(0.06-1.01) |
| *P* for trend | 0.001 | 0.048 |
| a Adjusted for age, energy intake, BMI, history of diabetes, smoking, physical activity and alcohol intake.  |

Multivariate adjusted odds ratios for NAFLD based on egg Consumption categories are indicated in Table 4. In the crude model, participants that 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). 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: 3.57; 95%CI: 1.89, 6.75). Further adjustment for physical activity, alcohol intake and gender 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 have eaten less than two eggs per week (OR: 3.71; 95%CI: 1.91, 7.75). In spite of this, egg consumption more than four per week were not significantly associated with the NAFLD odds ratio.

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| **Table 4. Multivariate-adjusted odds ratio for NAFLD according to egg consumption** a |
|  | Egg Consumption Categories | P-value for trend |
|  | <2/week | 2-3/week | 4≤ /week |
| Crude | 1.00 | 3.56(2.35-5.31) | 1.32(0.87-2.02) | <0.001 |
| Model 1b | 1.00 | 3.83(2.49-5.89) | 1.54(0.99-2.41) | <0.001 |
| Model 2c | 1.00 | 3.57(1.89-6.75) | 0.94(0.52-1.7) | <0.001 |
| Model 3d | 1.00 | 3.71(1.91-7.25) | 1.10(0.58-2.11) | <0.001 |
| aData are odds ratio (95% confidence interval).b Adjusted for age and energy intake.c Further controlled for, BMI, history of diabetes and smoking.d Additionally adjusted for physical activity, alcohol intake and gender. |

**Discussion**

The role of diet and dietary supplements on the pathogenesis of NAFLD have been shown previously (23-36); however, to our knowledge, this is no study evaluating the association of egg consumption and NAFLD risk. Eggs contain a wide variety of essential nutrients and bioactive compounds that can impact human health. Their high quality protein, fats and micronutrients and low price make them as an important part of many people’s diet (37); however, there are concerns on their high content of cholesterol and its impact on metabolic disorders. The results of current study have shown that egg consumption increases the risk of NAFLD in common range of its consumption. This finding is consistent with previous reports which had shown that NAFLD and its exacerbation are associated with high consumption of cholesterol (12-13, 38-39). Moreover, the presence of high amount of cholesterol in diet is necessary for development of NAFLD (40). Baumgartner et al (38) have shown that daily egg consumption for 12 wk increases serum cholesterol and LDL-C concentrations in women; however, there was no effects on markers for inflammation, endothelial activity, and liver function. On the other hand, it has been shown that dietary cholesterol intakes were superabundant in non obese patients with NAFLD (13), and dietary intake of NASH patients was richer in saturated fat, cholesterol and was poorer in polyunsaturated fat (12). Subramanian et al (39) 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 soft drinks and 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 (41-42), and some studies did find an association only between NAFLD and low intake of n-3 fatty acids and some antioxidants(16). 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 white part of egg on NAFLD risk.

This study has many advantages; one of the most important strengths of the current study is that it is the first study evaluating the association between egg consumption and risk of NAFLD in newly diagnosed patients who have not probably changed their diet due to the disease diagnosis. Another advantage of this study is its relatively large sample size of cases and controls which gave us the opportunity to evaluate the association between egg consumption and risk of NAFLD. Moreover, the high participation rate of patients in is another advantage of the current study which reduces the inter-individual response bias. Furthermore, this study was conducted in a developing country where economic resources are limited, so even small economic differences directly influence the diet. This relevance might

increase discrepancy among people.

This study had some limitations. Although we used a validated FFQ for measurement of dietary intakes, measurement error, and recall bias cannot be completely ruled out in this study. Although we adjusted all analysis for the known risk factors for NAFLD, some unknown or unmeasured risk factors were not adjusted for in this study. Thus, it is necessary to replicate the results of our study in other populations. There is the possibility that cases might recall their diets differently after disease diagnosis; although the recall bias is unlikely because we included incident cases, and trained dieticians collected dietary data which assessed dietary intakes of cases one year before diagnosis.

In conclusion, our data indicate that egg consumption in common amount of usage is associated with risk of NAFLD. Since case–control studies are weak to show the causality effect, these findings need to be confirmed in future prospective studies with separate part of eggs to find the etiological relationships.

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