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Serum Lipid Response to n-3 Fatty Acid Enriched Eggs in Persons with Hypercholesterolemia

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Consumption of long-chain n-3 polyunsaturated fatty acids (n-3 PUFA) is low among certain population subgroups in the United States, especially in people living in Midwestern states and those who dislike fish (7,8). Therefore, there is a need to identify acceptable food sources of n-3 PUFA. Vegetable oils, such as canola and soy, provide some n-3 fatty acids. However, these sources are high in n-6 fatty acids and there is concern that they may have detrimental side effects (9). Eggs enriched in n-3 PUFA can be produced by incorporating sources of these fatty acids into poultry rations (10,11). In acceptability studies, US consumers responded positively to n-3 enriched eggs (12). However, Americans may be reluctant to consume eggs as a source of n-3 PUFA because of their cholesterol content.

Dietary intake of n-3 PUFA is associated with a reduced risk of primary cardiac arrest and sudden and non-sudden death (1–4). The plantbased form of n-3 PUFA, α linolenic acid (LNA, 18:3n-3), may also reduce coronary heart disease (5). Harris (6) suggested that the effects of LNA and n6 fatty acids on serum lipids are similar, whereas marine n-3 PUFAs appear to raise low-density lipoprotein (LDL) and high-density lipoprotein levels and reduce serum triglycerides. The n-3 PUFAs may have positive effects on serum lipids. Consumption of a large dose (20 g/day) of n-3 PUFA from fish oil significantly reduced both serum triglycerides and total serum cholesterol in patients with hyperlipidemia (13). Oh et al (14) reported that persons with normal lipid levels who consumed 4EPA and DHA-enriched eggs per day reduced their serum triglyceride and blood pressure levels, but there was no effect on total serum cholesterol. People with normal lipid levels who consumed 4 LNA-enriched eggs per day experienced no significant effects on either serum triglycerides or total serum cholesterol (15). There is a need to further assess the role of eggs as a source of n-3 PUFA in the US diet. The purpose of this study was to evaluate the effects of the addition of LNA- and DHA-enriched eggs on serum lipids of people with hyperlipidemia who were consuming a low-fat diet.

Methods

Twenty-five volunteers with hypercholesterolemia (serum cholesterol >5.2 mmol/L) were recruited from the community. The participants—13 men and 12 women—ranged in age from 26 to 73 years. None of the volunteers had been diagnosed with any disease (including heart disease). The project was approved by the Institutional Review Board at the University of Nebraska, Lincoln. Volunteers were randomly assigned to 3 experimental treatments: a low fat, self selected diet with no eggs (no egg); a low-fat self-selected diet with 12 n-3 PUFA-enriched eggs per week (omega egg); and a low-fat, self-selected diet with 12 control eggs per week (control egg). A low-fat diet was defined as <30% of energy from fat and <10% of energy from saturated fat. A Latin square design was used so that all volunteers received each treatment during the 38-week study. A 2-week baseline period was followed by a 6-week, low-fat, lead-in period before the first 6-week diet treatment began. The order of the diet treatments was randomized. Six-week, low-fat, washout periods—during which no eggs were consumed—were used between diet treatments.

After the baseline period, volunteers received nutrition counseling on the low-fat diet by a registered dietitian once every 2 weeks. Body weight was also monitored at each 2-week appointment. Volunteers received detailed instruction on estimating serving sizes and keeping complete food records. They provided 3-day food records twice during each period (weeks 3 and 6). Week 3 food records were used for monitoring and week 6 records were included in the data analysis. Food records were checked for completeness and analyzed using the Food Processor Plus computer program (ESHA version 6.0, Salem, Ore). Mean daily energy, fat, and cholesterol consumption were determined for each period. Blood samples were drawn from fasting subjects twice during each period (weeks 4 and 6) and serum lipid levels were determined immediately at a certified medical laboratory (Nickels Institute, Lincoln, Neb) using standardized laboratory procedures.

The Department of Animal Science at the University of Nebraska, Lincoln, produced the eggs consumed during the omega-egg and control-egg treatment periods. The n-3 PUFA-enriched eggs were produced by including flaxseed in the poultry ration (12). The n-3 PUFA content of the control egg was 65 mg/egg and this increased to 412 mg/egg in the n-3 PUFA-enriched egg. The majority of the increase in n-3 PUFA in the enriched egg was from LNA, which increased from 17 mg/egg in the control egg to 277 mg/egg in the n-3 PUFA-enriched egg. The DHA content of the n-3 PUFA-enriched egg also increased from 35 mg/egg in the control egg to 114 mg/egg in the n-3 PUFA-enriched egg.

The mean of the 2 serum samples obtained from each volunteer during each treatment period was used for data analysis. The general linear model procedure of SAS (version 6, 1994, SAS Institute, Cary, NC) was used to compare treatment means using a repeated-measures analysis of variance. Statistical analyses were also used to identify if any treatment order-effect was present. Mean change from

* To convert mmol/L cholesterol to mg/dL, multiply mmol/L by 38.7. To convert mg/dL cholesterol to mmol/L, multiply mg/dL by 0.026. Cholesterol of 5.00 mmol/L = 193 mg/dL.
### Table 1. Mean (±SEM) serum lipids<sup>a</sup> of volunteers consuming a no egg, n-3 PUFA<sup>c</sup>-enriched egg, or control egg low-fat<sup>d</sup> diet treatment

<table>
<thead>
<tr>
<th>Serum lipids</th>
<th>No egg</th>
<th>n-3 egg&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Control egg&lt;sup&gt;f&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Nonresponders (n=23)</td>
<td>Nonresponders and responders (n=25)</td>
<td>Nonresponders (n=23)</td>
</tr>
<tr>
<td>Cholesterol (mmol/L)&lt;sup&gt;g&lt;/sup&gt;</td>
<td>5.92±0.07</td>
<td>5.90±0.07</td>
<td>6.00±0.06</td>
</tr>
<tr>
<td></td>
<td>0.08±0.20&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.16±0.20</td>
<td>4.16±0.06**</td>
</tr>
<tr>
<td></td>
<td>0.21±0.17</td>
<td>0.29±0.17</td>
<td>0.10±0.17</td>
</tr>
<tr>
<td>LDL</td>
<td>3.88±0.06</td>
<td>3.87±0.06</td>
<td>4.09±0.06</td>
</tr>
<tr>
<td></td>
<td>0.00±0.06</td>
<td>0.01±0.06</td>
<td>0.02±0.06</td>
</tr>
<tr>
<td>HDL</td>
<td>1.24±0.02</td>
<td>1.22±0.02</td>
<td>1.24±0.02</td>
</tr>
<tr>
<td></td>
<td>0.00±0.06</td>
<td>0.01±0.06</td>
<td>0.02±0.06</td>
</tr>
<tr>
<td>Triglycerides (mmol/L)&lt;sup&gt;i&lt;/sup&gt;</td>
<td>1.73±0.09</td>
<td>1.76±0.08</td>
<td>1.46±0.09</td>
</tr>
<tr>
<td></td>
<td>0.27±0.25</td>
<td>0.26±0.23</td>
<td>0.14±0.25</td>
</tr>
</tbody>
</table>

<sup>a</sup> SEM=standard error of the mean.
<sup>b</sup> Mean of 2 serum samples obtained at weeks 4 and 6 of each period.
<sup>c</sup> PUFA=polyunsaturated fatty acid.
<sup>d</sup> Low-fat diet=<30% of energy as fat, <10% of energy as saturated fat.
<sup>e</sup> 12 n-3 fatty acid-enriched eggs per week.
<sup>f</sup> 12 standard eggs per week.
<sup>g</sup> To convert mmol/L cholesterol to mg/dL, multiply mmol/L by 38.7. To convert mg/dL cholesterol to mmol/L, multiply mg/dL by 0.026.
<sup>h</sup> Cholesterol of 5.00 mmol/L=193 mg/dL. LDL=low-density lipoprotein. HDL=high-density lipoprotein.
<sup>i</sup> To convert mmol/L triglyceride to mg/dL, multiply mmol/L by 88.6. To convert mg/dL triglyceride to mmol/L, multiply mg/dL by 0.0113.
<sup>j</sup> Triglyceride of 1.80 mmol/L=159 mg/dL.

### Table 2. Dietary fat intake<sup>a</sup> (mean±SEM)<sup>b</sup> of volunteers consuming a self-selected low-fat<sup>c</sup> diet with no eggs, n-3 fatty acid-enriched eggs or control eggs added (n=25)

<table>
<thead>
<tr>
<th>Dietary fat</th>
<th>No egg</th>
<th>n-3 egg&lt;sup&gt;d&lt;/sup&gt;</th>
<th>Control egg&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total fat (% of energy)</td>
<td>21.5±0.64&lt;sup&gt;xz&lt;/sup&gt;</td>
<td>24.1±0.68</td>
<td>24.5±0.67</td>
</tr>
<tr>
<td>Saturated fatty acids (% of energy)</td>
<td>6.60±0.25&lt;sup&gt;y&lt;/sup&gt;</td>
<td>7.30±0.26</td>
<td>7.86±0.26</td>
</tr>
<tr>
<td>Monounsaturated fatty acids (% of energy)</td>
<td>7.19±0.29&lt;sup&gt;xz&lt;/sup&gt;</td>
<td>8.97±0.32</td>
<td>9.32±0.31</td>
</tr>
<tr>
<td>Polyunsaturated fatty acids (% of energy)</td>
<td>5.07±0.26</td>
<td>5.56±0.28</td>
<td>4.93±0.28</td>
</tr>
<tr>
<td>n-3 Fatty acids (mg/d)</td>
<td>620±71&lt;sup&gt;x&lt;/sup&gt;</td>
<td>1,420±76&lt;sup&gt;f&lt;/sup&gt;</td>
<td>658±75</td>
</tr>
<tr>
<td>Cholesterol (mg/d)</td>
<td>142±17.8&lt;sup&gt;xz&lt;/sup&gt;</td>
<td>468±19.1</td>
<td>481±18.8</td>
</tr>
</tbody>
</table>

<sup>a</sup> Mean of 3 days obtained at week 6 of each period.
<sup>b</sup> SEM=standard error of the mean.
<sup>c</sup> Low-fat diet=<30% of energy as fat, <10% of energy as saturated fat.
<sup>d</sup> 12 n-3 fatty acid-enriched eggs/week.
<sup>e</sup> 12 standard eggs/week.
<sup>x</sup> Different from n-3 egg P<.05
<sup>y</sup> Different from control egg P<.0001
<sup>z</sup> Different from control egg P<.05
no egg to either n-3 egg or standard egg was calculated.

Results and Discussion

Analysis of diet records indicated that all 25 volunteers maintained a low-fat diet throughout the treatment and washout periods. Overall mean daily total consumption and saturated fat consumption for the 36 weeks were 22% and 7% of energy, respectively. Mean daily cholesterol consumption was 142±18 mg/day on the no-egg treatment and 475±19 mg/day on the omega-egg and control-egg treatments combined. Initial body mass index of the volunteers was 25.7±2.9 (mean±SD) and results of weight monitoring indicated that this did not change throughout the study. Analysis indicated that the order in which the treatments were given had no effect. Two subjects did not have blood samples for one treatment period. The statistical analysis used accounts for these missing data. Two volunteers responded to the egg treatments with an increase in total serum cholesterol that was beyond 2 standard deviations of the group mean. Data from these 2 volunteers were omitted from the column labeled “Nonresponders (n=25)” in Table 1 but were included in the column labeled “Non responders and responders (n=25).”

There were no significant differences in the effects of the dietary treatments on any of the serum lipids in the nonresponders. However, when data from responders were added to the analysis, there was a significant increase in LDL cholesterol with the egg treatments (P<0.01). There was a 7% increase in LDL cholesterol when omega eggs were added and a 5% increase when control eggs were added. For the 2 responders with the high total serum cholesterol increase alone, corresponding increases in LDL cholesterol were 26% for omega eggs and 22% for control eggs (data not shown). Other researchers have also found that there is a certain subset of the population that is responsive to dietary cholesterol and will therefore respond to an increase in dietary cholesterol with an increase in total and LDL serum cholesterol levels (16).

The most substantial change in serum lipid levels was in serum triglyceride levels, which were 16% lower on the omega-egg diet treatment than on the no-egg treatment. When responders (n=25) were added to the data analysis, the difference in the decline in serum triglycerides on the 2 types of eggs became significant (P<0.05). The drop in serum triglycerides from the no-egg treatment was 15% on the omega-egg treatment and 9% on the control-egg treatment.

Table 2 shows the effect on fat intake of the addition of the 12 eggs per week. When eggs were added to the low-fat diet, total fat, monounsaturated fatty acids, and cholesterol significantly increased (P<0.05). Saturated fat intake increased significantly on the control-egg treatment but not on the omega-egg-treatment. When volunteers consumed the omega-egg dietary treatment, the intake of n-3 PUFA more than doubled.

Applications

Results of this research indicate that the majority of people with hypercholesterolemia who are consuming a step 1 diet can add 12 eggs per week without experiencing a significant increase in serum total or LDL cholesterol levels. However, a subset of this population is sensitive to dietary cholesterol and their serum total and LDL cholesterol levels will increase with egg consumption.

- The addition of 2 n-3 PUFA-enriched eggs per day as part of a low-fat diet increases the total n-3 PUFA intake to approximately 1.4 g/day. If n-3 PUFA-enriched eggs are recommended as a mechanism for increasing n-3 PUFA in the diet, serum cholesterol must be checked to ensure that the patient does not respond to the additional dietary cholesterol. Dietetics professionals can monitor the response of their patients to the addition of eggs by taking a pre and postmeasurement of serum cholesterol approximately 1 month apart.

- The n-3 enriched egg is an affordable food source of n-3 PUFAs that is acceptable and easy to prepare for most people. Three n-3 PUFA-enriched eggs provide approximately the same amount of n-3 fatty acids as one fish meal (equivalent to 3 oz fish). The number of n-3 PUFA-enriched eggs recommended for a person will vary with his or her current n-3 PUFA intake. It is recommended that n-3 PUFA-enriched eggs be used as one source of n-3 PUFA to increase individual consumption, particularly in people who seldom eat fish.

References


