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Enriched Eggs as a Source of N-3 Polyunsaturated Fatty Acids for Humans[1]

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ABSTRACT Dietary intake of omega-3 fatty acids (n-3 PUFA) decreases the risk of heart disease, inhibits the growth of prostate and breast cancer, delays the loss of immunological functions, and is required for normal fetal brain and visual development. The US has not established a recommended daily intake for n-3 PUFA. However, Canada has established the Canadian Recommended Nutrient Intake (CRNI) at 0.5% of energy. Dietary sources of n-3 PUFA include fish, chicken, eggs, canola oil, and soybean oil. Food consumption studies in the US indicate that the majority of Americans do not meet the CRNI for n-3 PUFA. Mean n-3 PUFA consumption was 78% of the CRNI for Midwestern women during pregnancy. In Midwestern women at risk for breast cancer, the mean n-3 PUFA consumption is approximately 50% of the CRNI. Increased consumption of n-3 PUFA requires identification of a food source that the public would eat in sufficient amounts to meet recommended intake. N-3 PUFA-enriched eggs can be produced by modifying hens diets. When 70 g/kg of cod liver oil, canola oil, or linseed oil are added to a commercial control diet, the n-3 PUFA are increased from 1.2% of egg yolk fatty acids to 6.3, 4.6, and 7.8%, respectively. Feeding flaxseed increases linolenic acid in the egg yolk about 30-fold, and docosahexaenoic acid (DHA) increases nearly fourfold. When individuals are fed four n-3 PUFA-enriched eggs a day for 4 wk, plasma total cholesterol levels and low-density lipoprotein cholesterol (LDL-C) do not increase significantly. Plasma triglycerides (TG) are decreased by addition of n-3 PUFA-enriched eggs to the diet. N-3 PUFA may influence LDL particle size, causing a shift toward a less atherogenic particle. Blood platelet aggregation is significantly decreased in participants consuming n-3 PUFA-enriched eggs. Overall results of studies to date demonstrate positive effects and no negative effects from consumption of n-3-enriched eggs. Three n-3 PUFA-enriched eggs provide approximately the same amount of n-3 PUFA as one meal with fish. It is recommended that n-3 PUFA-enriched eggs be used as one source of n-3 PUFA to increase individual consumption to meet the current Canadian recommendations.

(*Key words*: omega eggs, omega-3 fatty acids, alpha-linolenic acid, docosohexaenoic acid, serum lipids)

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INTRODUCTION

Consumers are increasingly interested in functional foods. One category of functional foods of great interest are products that contain omega-3 fatty acids (n-3 PUFA). Dietary intake of n-3 PUFA decreases risk of heart disease (Temple, 1996), provides an inhibitory effect on the growth of prostate and breast cancer (Pandalai et al., 1996; Rose, 1997), delays the loss of immunological functions (Fernandes, 1995), and is required for normal fetal brain and visual development (Neuringer et al., 1998).

Food Sources and Dietary Intakes

Foods typically considered to be major contributors of n-3 PUFA in the diet include fish and other types of seafood. Other common dietary sources of n-3 PUFA include chicken, eggs, canola oil, and soybean oil (Lewis et al., 1995b). Less common food products that are high in n-3 PUFA and can be obtained in health food stores are wheat grass, fresh liquid lecithin, flaxseed, flaxseed oil, and hemp seed oil. Other foods that provide smaller quantities of n-3 PUFA include legumes, green vegetables, cauliflower, whole milk, and ground beef (Raper et al., 1998).

Although fish is considered to be the primary source of n-3 PUFA, it may not serve as a primary source for people in certain parts of the country. For example, a survey of Nebraskans indicates that fish is consumed only once every 2 wk (Lewis et al., 1995a). In a survey of pregnant women in the Midwest, the primary foods that

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Abbreviation Key: CHD = coronary heart disease; CRNI = Canadian Recommended Nutrient Intake; DHA = docosahexaenoic; EPA = eicosapentaenoic acid; LDL-C = low-density lipoprotein cholesterol; LNA = linolenic acid; MI = myocardial infarction; n-3 PUFA = omega-3 fatty acids; TG = triglycerides.

provided n-3 PUFA were fats, oils, and salad dressing, dairy products, fish, and green vegetables (Lewis et al., 1995b).

The US has not yet set a recommended dietary allowance for n-3 PUFA. However, Canada has established the Canadian Recommended Nutrient Intake (CRNI) at 0.5% of energy, or 1.1 g of n-3 PUFA per day for an individual consuming 2,000 kcal. The recommended ratio of n-6/n-3 PUFA is 4 to 10:1 (Nutrition Recommendations, 1990).

Food consumption studies in the US indicate that many Americans do not meet the CRNI for n-3 PUFA. Certain population groups who have specific needs for n-3 PUFA are at greater risk of n-3 PUFA deficiency. Lewis et al. (1995b) surveyed 30 low-income pregnant Midwestern women, ages 18 to 37, to determine their n-3 PUFA intake. Mean n-3 PUFA consumption was 78% of the CRNI. About half of the women consumed less than 75% of the CRNI. Five of the 30 women met or exceeded the CRNI for n-3 PUFA. A second survey was of Midwestern women at risk for breast cancer (Bridger et al., 1998). The mean n-3 PUFA consumption was 0.61 ± 0.24 g/d, approximately 50% of the Canadian RNI.

Increased consumption of n-3 PUFA requires identification of food sources that the general public will consume in sufficient amounts to meet recommended intake. Eggs are a potential source of n-3 PUFA because they can be easily enriched with n-3 PUFA by dietary modifications of the laying hens. The per capita consumption of eggs in the US in 1996 was 238 (USDA, 1999), which is approximately five eggs per person per week. If Americans consumed five n-3 PUFA enriched eggs per week, the amount of n-3 PUFA ingested would be approximately equivalent to one five ounce serving of fish per week (Scheideler and Lewis, 1997).

N-3 PUFA and Coronary Heart Disease

Epidemiology studies indicate that increased fish consumption is related to a reduced incidence of myocardial infarct. Daviglus et al. (1997) used data from the Chicago Western Electric study to examine the relation between base-line fish consumption and the 30-yr risk of death from coronary heart disease (CHD). Men who consumed 35 g or more of fish per day had a 42% lower rate of death from myocardial infarction (MI) when compared with men who did not consume fish. Upon adjustment for multiple confounders, a significant inverse relationship prevailed between fish consumption and the risk of a fatal MI. For the men who consumed 35 g or more of fish daily as compared with the nonconsumers, the relative risks of any death from MI and death from CHD was 0.56 and 0.62, respectively (P for trend = 0.02 and 0.04). These trends were accounted for by the data on fatal MI, specifically, by the trend toward a lower risk of nonsudden death from MI with higher fish consumption. The relative risk of death from nonsudden MI for the men who consumed 35 g or more of fish daily was 0.33 (P for trend = 0.077). No significant relationship was found between sudden death from MI and fish consumption.

Siscovick et al. (1995) conducted a case-control study to assess whether the dietary intake of long-chain n-3 PUFA from seafood is associated with a reduced risk of primary cardiac arrest. N-3 PUFA intake was assessed both directly and indirectly through a biomarker. An intake of 5.5 g of n-3 PUFA per month was associated with a 50% reduction in the risk of primary cardiac arrest compared with no dietary intake of n-3 PUFA. A red blood cell n-3 PUFA level of 5.0% of total fatty acids (the mean of the third quartile), compared with a level of 3.3% (the mean of the lowest quartile), was associated with a 70% reduction in the risk of primary cardiac arrest. The results of this study suggest that dietary intake of n-3 PUFA from seafood is associated with a reduced risk of primary cardiac arrest.

The relationship between fish consumption and reduced heart disease incidence may be explained by the effects of the n-3 PUFA on heart rate. Presently CHD is the major disease in which malignant ventricular arrhythmias occur, and malignant ischemia is the most common trigger of the arrhythmias. Kang and Leaf (1996) studied the mechanism of the antiarrhythmic effects of PUFA in vitro with cultured neonatal rat ventricular myocytes. Reversible slowing of the beating rate with no change in the amplitude of contraction invariably occurs with addition of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). Tachyarrhythmias occurred in the myocytes with addition of agents known to cause arrhythmias in patients. These tachyarrhythmias were prevented or abolished by a low concentration of n-3 PUFA added to the perfusing medium.

N-3 PUFA-Enriched Eggs and Their Effects on Serum Lipids

N-3 PUFA-enriched eggs can be produced by modifying hen diets. Scheideler and Froning (1996) conducted a study to determine how modifying hen diets would change the fatty acid composition of the egg yolks. The hens were assigned to control, 15% fish oil, or 5, 10, or 15% flaxseed dietary rations. The n-3 PUFA were significantly higher in eggs produced from hens fed fish oil or flaxseed as compared with the control. The amount of n-3 PUFA in the egg yolks of the control and treatment groups were 1.2% of fatty acids, or 2.0 to 7%, respectively. Scheideler et al. (1997) studied consumer acceptance of n-3 fatty acid-enriched eggs and found little taste differentiation among scrambled control versus n-3-enriched eggs. Ferrier et al. (1995) utilized flax seed to increase the n-3 PUFA in eggs. They fed a control diet or linolenic acid (LNA) diet that contained 10 or 20% flaxseed. In the modified eggs, the n-3 PUFA, LNA, increased from 28 mg per egg in the control to 261 and 527 mg per egg in the modified eggs, respectively. The DHA content increased from 51 mg per egg to 81 and 87 mg per egg.

Scheideler and Froning (1996) have also produced n-3 PUFA-enriched eggs from hens fed flaxseed. The modified egg contains 350 mg of n-3 PUFA compared with the standard egg that contains 60 mg of n-3 PUFA. The

LNA content is 250 mg and the DHA content is 100 mg in the modified egg compared with the standard egg that contains 40 mg and 20 mg, respectively. The ratio of n-6/n-3 PUFA is 13.0 in a standard egg compared with a ratio of 2.6 in the modified egg. Cholesterol content in the modified egg has also been reduced to 180 mg per egg, compared with the standard egg value of 210 mg per egg. Nutrient content in other aspects such as energy, protein, and carbohydrate are similar to a standard egg (Scheideler and Lewis, 1997).

One of the barriers to increasing n-3 PUFA consumption with n-3 PUFA-enriched eggs is consumer perception that increasing egg consumption and, therefore, increasing cholesterol consumption is unhealthy. The American Heart Association (1999) recommends consuming no more than 3 to 4 egg yolks per week. Therefore, initial studies with n-3 PUFA-enriched eggs have been conducted to assess the effects of these eggs on serum lipids profiles as well as other health factors.

Ferrier et al. (1995) compared the effects of four n-3 PUFA eggs per day or four standard eggs per day on serum lipids in 28 normolipidemic subjects. After 2 wk, no significant changes were observed in total cholesterol, high-density lipoprotein cholesterol, or plasma triglyceride (TG) concentrations. However, consumption of modified eggs resulted in a significant increase in DHA and total n-3 PUFA in the blood platelet phospholipids.

Lewis et al. (2000) fed 12 regular or 12 n-3 PUFA-enriched eggs per week to hypercholesterolemic participants who were consuming a low-fat diet. After 6 wk, the majority of the participants showed no response in total or low-density lipoprotein cholesterol (LDL-C) to either of the egg treatments. However, 2 of the 25 participants responded to the increase in dietary cholesterol with a significant increase in both serum total and LDL-C. These two participants were identified as responders, and the data were analyzed both with and without the responders. In the nonresponders, no significant effects of the diets were found on any of the serum lipids with the n-3 PUFA-enriched eggs or the standard eggs added compared with the no-egg diet. The largest effect in the nonresponders was a 14% reduction in TG when the n-3 PUFA-enriched egg was consumed. When the data from the responders were added to the analysis, there was a significant increase in LDL-C with the egg treatments ($P < 0.01$) and a significant decrease in serum TG during the dietary period that included the n-3 PUFA-enriched eggs ($P < 0.05$). For the majority of the population, consumption of two eggs per day does not appear to have negative effects on blood lipid patterns. However there is a certain subset of the population (i.e., responders) for whom increasing n-3 PUFA via n-3 PUFA-enriched eggs would not be appropriate.

Another mechanism that may explain the relationship between n-3 fatty acid and CHD is the effect of n-3 PUFA on platelet aggregability. Van Elswyk et al. (1998) fed volunteers four n-3 PUFA enriched eggs per week or four regular eggs per week for 6 wk. Blood platelet aggregability was measured at baseline and following 6 wk of dietary treatment. After the treatment period, there was a significant decrease in the platelet aggregation when the volunteers consumed the n-3 PUFA-enriched eggs.

CONCLUSION

In conclusion, consumption of n-3 PUFA in the Midwest is approximately half of that recommended by the CRNI. Because this population is already eating more eggs than fish, n-3 PUFA-enriched eggs are an alternative source of n-3 PUFA. The n-3 PUFA-enriched eggs do not appear to have negative effects on serum lipids in the majority of the population. Future research needs to focus on the relationship of consumption of n-3 PUFA-enriched eggs and risk of heart disease. It is recommended that n-3 PUFA-enriched eggs be used as one source of n-3 PUFA to increase individual consumption to levels that will meet the current CRNI.

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