

Consumption of One Egg Per Day Increases Serum Lutein and Zeaxanthin Concentrations in Older Adults without Altering Serum Lipid and Lipoprotein Cholesterol Concentrations¹

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Abstract

Lutein and zeaxanthin accumulate in the macular pigment of the retina, and are reported to be associated with a reduced incidence of age-related macular degeneration. A rich source of lutein and zeaxanthin in the American diet is the yolk of chicken eggs. Thus, the objective of the study was to investigate the effect of consuming 1 egg/d for 5 wk on the serum concentrations of lutein, zeaxanthin, lipids, and lipoprotein cholesterol in individuals >60 y of age. In a randomized cross-over design, 33 men and women participated in the 18-wk study, which included one run-in and one washout period of no eggs prior to and between two 5-wk interventions of either consuming 1 egg or egg substitute/d. Serum lutein 26% ($P < 0.001$) and zeaxanthin 38% ($P < 0.001$) concentrations increased after 5-wk of 1 egg/d compared with the phase prior to consuming eggs. Serum concentrations of total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides were not affected. These findings indicate that in older adults, 5 wk of consuming 1 egg/d significantly increases serum lutein and zeaxanthin concentrations without elevating serum lipids and lipoprotein cholesterol concentrations. *J. Nutr.* 136: 2519–2524, 2006.

Introduction

Age-related macular degeneration (AMD)⁵ is a progressive eye condition that affects >13 million Americans or 5% of people age 65 and older. This disease attacks the macula of the eye, the area responsible for the sharpest central vision. There are two forms of AMD: dry AMD and wet AMD (1). Although investigators are uncertain of the direct causes of dry AMD, studies suggest that the area of the retina becomes diseased coincident with the accumulation of yellow pigments called *drusen* leading to the slow breakdown of the light-sensing cells in the macula and gradual loss of central vision (2). Whereas the wet form of AMD is not considered to be influenced by dietary manipulation, the dry form is responsive to nutrients such as vitamins and minerals (3,4).

In addition to vitamins and minerals, evidence is now accumulating that dietary and blood concentrations of lutein and zeaxanthin, 2 oxygenated carotenoids that concentrate in the macula of the eye, are associated with reduced risk of AMD

(5–8). There are several studies that suggest that certain fruits and vegetables are good sources of lutein and zeaxanthin, and their consumption has been associated with increased blood concentrations and macular pigment concentration of these carotenoids (6–9). These findings are important insofar as preliminary evidence (10,11) and a more recent clinical trial (12) suggest that lutein supplementation may also slow the progression of AMD.

Lutein and zeaxanthin play a role in the prevention of AMD by aiding in the filtering of damaging blue light and sunlight (13). Because AMD is thought to be associated with long-term oxidative damage to the retina (14,15), the reported findings, that lutein and zeaxanthin are 2 very powerful antioxidants, absorb ultraviolet light, and serve to protect the lens from oxidative damage, would suggest an important role for these nutrients in preventing or treating this disease (14). Human trials with lutein and zeaxanthin supplements indicate that these dietary carotenoids, in addition to fruits and vegetables, can be used to elevate macular pigment concentration (16–20). The chicken egg yolk, a matrix composed of digestible lipids, i.e., cholesterol, triglycerides, and phospholipids, also contains lutein and zeaxanthin dispersed in the matrix along with other fat-soluble micronutrients such as vitamin A, vitamin D, and vitamin E. The lipid matrix of the yolk of chicken eggs provides a readily bioavailable dietary source of lutein and zeaxanthin that is even more bioavailable than lutein supplements and spinach

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⁵ Abbreviations used: AMD, age-related macular degeneration; HDL-C, HDL cholesterol; LDL-C, LDL cholesterol; TC, total cholesterol; TG, triglyceride.

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(21). Two other reports (22,23) examined the same human subjects (mean age 62 y, range: 46–78 y) and showed a 28–50% increase in plasma lutein and a 114–142% increase in plasma zeaxanthin concentrations (22) following a diet supplemented with 1.3 eggs/d for 4.5 wk. However, this increase was associated with an 8–11% increase in plasma LDL cholesterol (LDL-C) concentrations (23). These observations suggest that eggs can be used by individuals who want to increase their circulating concentrations of these carotenoids.

Thus, one of the objectives of this study was to investigate, in an elderly population with a mean age of 79 y, whether the serum concentrations of both lutein and zeaxanthin respond in a similar manner to previous reports (22,23) when consuming 1 egg/d or increased cholesterol. Most studies either do not include a source of dietary zeaxanthin or do not analyze serum lutein and zeaxanthin concentrations separately. A second objective was to determine whether the changes in serum lutein and zeaxanthin concentrations are associated with any of the variables measured. A third objective was to determine whether egg consumption in this elderly age group would result in significant increases in serum LDL-C concentrations as reported in younger populations (22).

Subjects and Methods

Subjects. The effects of consuming 1 egg/d on serum lutein and zeaxanthin concentrations, as well as serum lipid and lipoprotein cholesterol concentrations, was examined in 33 subjects (7 men and 26 women) with a mean age of 79 y (range: 60–96 y) who were currently not taking lutein and/or zeaxanthin supplements or cholesterol-lowering medication and who spoke English comfortably. The baseline characteristics of the study participants are described in Table 1. A total of 65 individuals were recruited for the study. Nineteen were excluded from the study due to the onset of illness, death, or nonadherence to protocol. In addition, the data from 13 other subjects who were identified (during the baseline period) as having taken a lutein and/or zeaxanthin supplement prior to baseline were also excluded from the final data analysis because their baseline values of lutein and zeaxanthin were 80% greater than the remaining 33 participants not taking supplements. The data from this group of 13 people who took supplemental lutein and/or zeaxanthin were analyzed separately. Of the 33 participants that completed the study, 22 were recruited from 4 nursing homes, and 11 were from 4 senior citizen centers of the Merrimack Valley and from the University of Massachusetts faculty and staff in Lowell. All but one subject (Asian or Pacific Islander) were Caucasian. Twenty-nine of the 33

graduated from high school or college. Most of the subjects had been diagnosed with at least 1 medical condition, including 22 subjects with hypertension and 6 with diabetes. All subjects were interviewed at baseline using a structured medical medication history and demographic questionnaire. Blood pressure, height, and weight were recorded by a registered nurse upon enrollment into the study. A Mini-Mental State Examination (24) was administered by the registered nurse to each subject to verify mental competence. A 7-d diet record was obtained from each subject once during each of the 4 phases of the study. Nutrient analysis was performed using EvaluaEat, version 1.2 dietary analysis tool (Pearson Education, Benjamin Cummings, 2004). Dietary intakes of energy, protein, carbohydrate, dietary fiber, total fat, monounsaturated fat, polyunsaturated fat, saturated fat, cholesterol, and percent energy from protein, fat, carbohydrate, and alcohol were all assessed. The protocol for the ethical treatment of human subjects was approved by the University of Massachusetts Lowell Institutional Review Board and the Commonwealth of Massachusetts, Executive Office of Elder Affairs, Elder Rights Review Committee, Boston, MA.

Experimental design. This 18-wk randomized, cross-over study consisted of 4 phases. Phase I consisted of a 4-wk baseline period during which 11 senior citizen center and university subjects were instructed to limit their consumption of foods containing lutein and zeaxanthin and to not eat eggs or high egg-content foods. In contrast, the intake of the background diet and the intervention treatments (eggs or egg substitute) of the 22 nursing home subjects were strictly controlled by the nursing home staff. Phase II consisted of a 5-wk intervention period during which subjects consumed either no egg or egg substitute (referred to as no egg) or 1 egg/d in addition to their normal diet. Either a daily visit or phone call to the nursing home facility or to the subject's home by study personnel were used to verify consumption of all interventions. Phase III consisted of a 4-wk washout period similar to phase I. Phase IV consisted of a 5-wk cross-over intervention period during which those subjects who consumed the no-egg or egg-substitute regiment (referred to as no egg) during phase II were switched to 1 egg/d in addition to their normal diet. Those subjects who consumed 1 egg/d during phase II were switched to the no egg or egg substitute in addition to their normal diet.

Analysis of serum lipids and lipoprotein cholesterol measurements. During each phase, morning 12-h fasting blood samples were collected from all subjects at wk 2 and wk 4 for phases I and III and wk 3 and wk 5 for phases II and IV. Serum was harvested after centrifugation at $1500 \times g$ at 4°C for 20 min. Serum lipid and lipoprotein cholesterol concentrations were measured using a Cobas Mira Plus Clinical Chemistry Autoanalyzer. Serum total cholesterol (TC) (25) and triglyceride (TG) (26) concentrations were measured enzymatically using the Infinity Cholesterol Reagent (procedure 401) and Triglyceride (GPO-Trinder) Reagent (procedure 337) from Sigma Diagnostics (Sigma-Aldrich). Serum HDL cholesterol (HDL-C) was measured using the EZ HDL Cholesterol Reagent (procedure 354L, Sigma Diagnostics, Sigma-Aldrich). The concentration of serum LDL-C was calculated via the Friedewald equation. The accuracy and precision of the procedures used for the measurements of serum TC, HDL-C, and TG were maintained by participation in the Lipid Standardization Program of the Centers for Disease Control and the National Heart, Blood, and Lung Institute (Atlanta, GA).

Although the results of phases I, III, and the no-egg or egg-substitute intervention of phases II and IV during randomization were not statistically different, the period just before the 1 egg/d was used as the no-egg treatment for comparison with the egg-eating phase. Therefore, results for each subject are reported as egg vs. no egg for serum lutein, zeaxanthin, and lipid and lipoprotein cholesterol concentration.

Analysis of serum lutein and zeaxanthin. Serum aliquots were archived in sealed 1.0 mL cryovials and stored at -80°C for no >6 mo before analysis of serum carotenoids, a duration that has shown carotenoids to remain stable (27). Lutein and zeaxanthin standards were provided by Hoffman-La Roche. The internal standard was β -apo-12'-carotenol-O-t-ethyl-oxime. Cholesterol esterase (sterol esterase, EC 3.1.1.13; *Pseudomonas sp.*) and triacylglycerol lipase (EC 3.1.1.3;

TABLE 1 Baseline characteristics of men and women¹

	Men	Women
<i>n</i>	7	26
Age, y	77 ± 4	81 ± 2
Weight, kg	78 ± 4.5	70 ± 2.7
Height, m	1.7 ± 0.03	1.6 ± 0.01
BMI	26 ± 6	27 ± 6
TC, mmol/L	5.0 ± 0.3	5.2 ± 0.2
HDL-C, mmol/L	1.4 ± 0.2	1.3 ± 0.1
LDL-C, mmol/L	3.0 ± 0.3	3.2 ± 0.2
TG, mmol/L	1.5 ± 0.2	1.5 ± 0.2
MMSE ²	24 ± 3	22 ± 2
Blood pressure, mm Hg		
Systolic	139 ± 9	132 ± 2
Diastolic	77 ± 4	71 ± 2

¹ Values are means ± SEM.

² MMSE, Mini-Mental State Exam.

Rhizopus) were obtained from Calbiochem. High performance liquid chromatography (HPLC) grade solvents were used (Pharmco and Fisher Chemical).

Serum samples (100 μ L) were mixed with 900 μ L of reagent, containing 1 International Unit (IU) cholesterol esterase (Calbiochem) and 160 IU triglyceride lipase (Calbiochem). The enzyme reagent was prepared in a 0.1 mol/L sodium phosphate buffer, pH 7.0, with 0.1% Triton X100. Samples were prepared according to the procedures described by Handelman et al. (27). Calibration was achieved by carrying the standards through the entire protocol parallel to those of the serum samples.

HPLC separation and quantification of lutein and zeaxanthin were carried out using a procedure adapted from Handelman et al. (27). This HPLC assay separates the 2 carotenoids, lutein and zeaxanthin, which were then identified and quantitated as distinct peaks in the chromatogram. Analyses were achieved using an Agilent model 1100 gradient HPLC apparatus with diode array detection. The column used was a 300 mm \times 4.6 mm Adsorbosphere-HS C18 (20% carbon load) (Alltech) with 3 μ m particle size coupled with an identically packed guard column. The flow rate was 1.0 mL/min. The initial mobile phase concentration was 80% acetonitrile:20% methanol:0.4% ammonium acetate (w:v), with a step gradient at 20 min to 30% isopropanol, returning to initial conditions at 40 min, and then finishing with an additional 20 min equilibration period. The column temperature was maintained at 19.5°C.

Measurement of egg yolk cholesterol and carotenoid composition. For cholesterol analysis, commercial large white chicken eggs were provided by the nursing home facilities or donated by the Aramark Corporation, which is the food service entity for the University of Massachusetts, Lowell. Initially, 12 randomly chosen egg yolks were separated manually from the egg white and placed in a preweighed petri dish to determine weight of yolk. Five hundred μ L of yolk was added to 20 mL of buffer solution and vigorously mixed for 3 min. Two-hundred μ L of the yolk-buffer solution was collected and analyzed for cholesterol content on the Cobas Mira Plus Clinical Chemistry Autoanalyzer using the same methods described for the analysis of serum lipids and lipoprotein cholesterol.

For carotenoid analyses from the same 12 eggs, 250 μ L of yolk was added to 20 mL 0.15 mol/L phosphate buffer, pH 7.0, with 1 mmol/L EDTA and 0.25% Triton \times 100. For analysis, 200 μ L of dilute yolk was mixed with 0.8 mL distilled water, 1 mL 6 mol/L potassium hydroxide, and 2 mL ethanol. After vigorous mixing, the mixture was heated at 60°C for 30 min to saponify the lipids and hydrolyze the carotenoid esters. After saponification, 60 μ L of internal standard was added, mixed, and extracted similarly to serum. For HPLC analysis, the supernatant was collected, dried under N₂, and redissolved in 60 μ L methanol and 25 μ L was injected onto the HPLC.

Statistical analysis. Differences between the 4 phases were determined by repeated measures 1-way ANOVA (SigmaStat, Jandel Scientific). When differences were observed, a Student-Newman-Keuls test was used. A paired *t* test was used to examine the effect of the egg vs. no-egg phases on the different variables measured. All values were expressed as means \pm SEM and significance was set at *P* < 0.05. Pearson correlation coefficient was used to determine significant associations among the variables measured.

Power calculation indicated that a minimum size of 30 was needed to detect a 5% change in LDL-C with a cross-over design at an error rate (α) of 0.001 with a desired power (β) of 80.

Results

Dietary analysis. Using the 7-d diet record for a 1-wk analysis of dietary intake per phase, no significant changes in dietary intake between the nursing home and senior citizen center subjects were observed despite the differences (free-living vs. institutionalized) in the degree of background dietary control (Table 2). The only macronutrient that increased was dietary cholesterol during the egg intervention compared with the no-

TABLE 2 Seven-day diet record analysis of 33 subjects consuming egg vs. no-egg diet¹

	Egg	No egg
Energy intake, kJ/d	6690 \pm 351	6489 \pm 268
Protein, g/d	70 \pm 4	78 \pm 16
Carbohydrate, g/d	199 \pm 14	293 \pm 77
Dietary fiber, g/d	12 \pm 2	14 \pm 3
Total fat, g/d	57 \pm 3	68 \pm 16
Monosaturated fat, g/d	20 \pm 1	23 \pm 5
Polyunsaturated fat, g/d	10 \pm 1	11 \pm 2
Saturated fat, g/d	19 \pm 1	22 \pm 5
Cholesterol, mg/d	451 \pm 24	312 \pm 50*

¹ Values are means \pm SEM, *n* = 33. *Different from egg, *P* < 0.001.

egg/egg substitute period (*P* < 0.001). Although the diet analysis database did not provide the carotenoid content of the various foods, the frequency (number of times carotenoid-containing foods were consumed each wk) did not differ during the egg (10.9 \pm 1.8) vs. no-egg (11.1 \pm 2.0) interventions. These data also showed that the background diet of the participants consisted of only 5 carotenoid-containing foods (orange juice, lettuce, yellow corn, broccoli, and onions) (28) and at mean intakes of 113 g, contributed negligible amounts of dietary carotenoids.

Egg yolk lutein, zeaxanthin, and cholesterol contents. The cholesterol content for the 12 randomly sampled egg yolks was 210 \pm 9 mg/yolk (individual data not shown). Analyses of the lutein and zeaxanthin content of the 12 egg yolks randomly sampled were 143 \pm 28 and 94 \pm 18 μ g/yolk, respectively (individual data not shown).

Serum carotenoids and lipids. Serum lutein (Fig. 1A) and zeaxanthin (Fig. 1B) concentrations during the 4-wk phase prior to consuming 1 egg/d increased 26 and 38%, respectively, by the end of 5 wk of 1 egg/d intervention (*P* < 0.05).

Serum TC, LDL-C, HDL-C, and TG concentrations during the no-egg and egg interventions did not differ. (Fig. 2).

Association between serum carotenoids, lipids, and lipoprotein cholesterol. Serum lutein and zeaxanthin concentrations during the no-egg and egg interventions were only significantly associated (*r*) with HDL-C (Table 3). However, these correlations, although significant, only explained \leq 21% of the variability (*r*²). Absent from the table are the elevated values of serum lutein and zeaxanthin after egg consumption (*r* = 0.835, *P* < 0.001), which were associated with their values before egg consumption (*r* = 0.850, *P* < 0.001).

Discussion

There appear to be only 4 published studies that have used egg yolks as a bioavailable source of the oxygenated carotenoids, lutein and zeaxanthin. Three of these studies used carotenoid-enriched eggs (21,29,30) and only 1 of these studies measured lutein and zeaxanthin separately (22). In this present study, consumption of 1 noncarotenoid-enriched egg/d for 5 wk significantly increased serum lutein and zeaxanthin concentrations by 26 and 38%, respectively, in this population with a mean age of 79 y consuming dietary egg yolk lutein and zeaxanthin concentrations of 143 and 94 μ g/yolk, respectively.

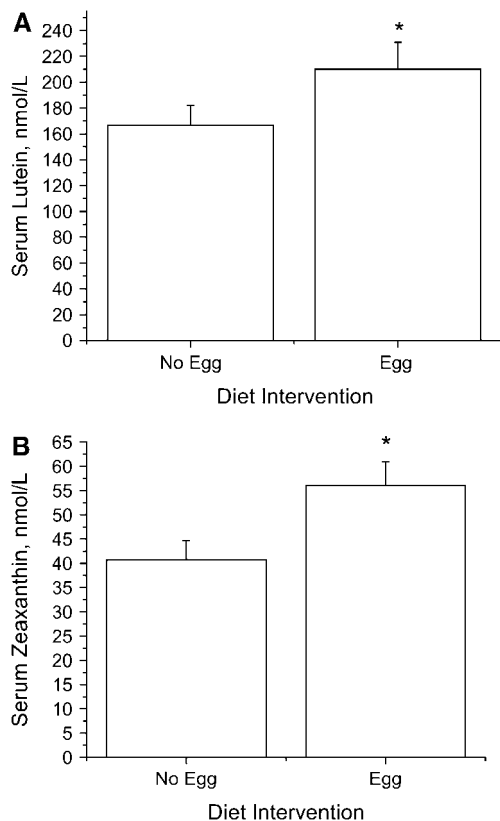


Figure 1 Serum lutein (A) and zeaxanthin (B) concentrations in older adults when they consumed 1 egg/d for 5 wk and when they consumed no eggs or egg substitute. Values are means + SEM, $n = 33$. *Different from no egg, $P < 0.001$.

The degree of serum carotenoid responsiveness in this communication is similar to the study by Handelman et al. (22) in subjects consuming 1.3 egg yolks/d but is not consistent with the results from the study of Surai et al. (30) where only individuals consuming the designer eggs (1910 μg of lutein/egg) and not the control eggs (120 μg lutein/egg) increased their plasma lutein concentrations. The reason for the discordant findings of Surai et al. (30) compared with the studies of Handelman et al. (22) and the present communication is not known. The studies of Yeum et al. (29) suggest that baseline values may influence serum

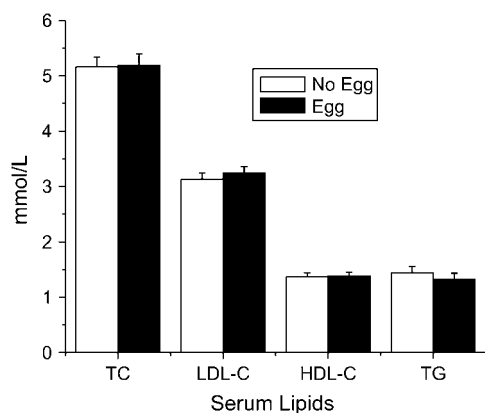


Figure 2 Serum lipids and lipoprotein cholesterol concentrations in older adults when they consumed 1 egg/d for 5 wk and when they consumed no eggs or egg substitute. Values are means + SEM, $n = 33$. *Different from no egg, $P < 0.001$.

TABLE 3 Association between serum concentrations of lutein and zeaxanthin before and after egg consumption with serum lipids and lipoprotein cholesterol concentrations¹

	No egg			Egg		
	<i>r</i>	<i>r</i> ²	<i>P</i> -value	<i>r</i>	<i>r</i> ²	<i>P</i> -value
TC						
Lutein	0.200	0.040	NS	0.121	0.015	NS ²
Zeaxanthin	0.025	0.001	NS	0.133	0.018	NS
LDL-C						
Lutein	0.159	0.025	NS	-0.02	0.001	NS
Zeaxanthin	-0.075	0.006	NS	0.015	0.001	NS
HDL-C						
Lutein	0.410	0.168	<0.017	0.453	0.205	0.008
Zeaxanthin	0.325	0.106	NS	0.424	0.180	0.014
TG						
Lutein	-0.233	0.054	NS	-0.133	0.018	NS
Zeaxanthin	-0.150	0.023	NS	-0.144	0.021	NS

¹ The *r* is Pearson correlation coefficient; the *r*² is the proportion of variance explained uniquely by a particular variable.

² NS, not significant.

carotenoid responsiveness because men ingesting controlled diets high in fruits and vegetables, and who had higher baseline values of lutein, did not increase their serum concentrations of lutein in response to increased dietary lutein. With the 13 people in the present study who consumed supplements containing lutein, baseline serum lutein concentrations (300 ± 43 nmol/L) were ~80% higher than the 33 individuals who had not consumed supplements (167 ± 16 nmol/L). Similar to the findings of Yeum et al. (29), these 13 people did not show significant serum carotenoid increases in response to increased dietary lutein and zeaxanthin after consuming 1 store-bought egg/d (data not shown). Surprisingly, in the randomization process in which participants did not consume eggs for up to 13 wk (4 wk baseline + 5 wk of Egg Beaters + 4 wk of washout) the baseline concentrations of serum lutein and zeaxanthin of the 13 individuals taking supplements remained greater than individuals not on supplements (286 ± 40 nmol/L and 71 ± 9 nmol/L vs. 210 ± 21 nmol/L and 56 ± 5 nmol/L). These findings suggest that 13 wk of not taking supplemental lutein and zeaxanthin is not a sufficient duration to reduce serum lutein and zeaxanthin to previous concentrations. This may explain why participants recruited into the study of Chung et al. (21) were required to stay off of supplemental lutein for 6 mo. This observation, plus the nonresponsive carotenoid findings of Yeum et al. (29) in individuals with high baseline lutein values, was the rationale for analyzing separately the data from the 13 people who consumed supplements prior to the baseline period in the present study. In addition to baseline values of circulating carotenoids, it is also possible that the concentrations of carotenoids consumed may affect carotenoid responsiveness. In a study of individuals who consumed designer eggs enriched with 1910 μg of lutein for 8 wk, plasma lutein concentrations increased 88% (240 to 450 nmol/L) (30). The >2-fold (88 vs. 26%) increase in plasma lutein concentrations in the designer-egg study of Surai et al. (30), compared with the present study, was probably associated with the 12-fold greater concentrations (1910 μg /yolk vs. 143 μg /yolk) of lutein in the designer eggs than in the store-bought eggs. The possibility that dietary fat may also influence serum carotenoid concentration is suggested by the findings of Chung et al. (21) in subjects that consumed comparable concentrations

of lutein in which a background diet, consisting of 60% of energy from fat compared with 17% in the present communication, was associated with 11-fold greater increase in (323 vs. 26%) blood lutein concentrations. Their (21) dramatic increase in plasma lutein concentrations may have also resulted from the greater consumption of lutein (6 mg/d) compared with the concentrations consumed (148 $\mu\text{g}/\text{d}$) in the present study. Other factors that can influence the absorption of lutein and zeaxanthin include digestion of the food matrix, the formation of lipid micelles, and uptake of the carotenoids by mucosal cells and transport of the carotenoids to the lymphatic or portal circulations (31). The substantial contribution that the digestibility of the food matrix can make to absorption is suggested by the studies of Chung et al. (21) in which lutein from eggs was nearly 3 times more bioavailable than spinach. Competition for absorption from other carotenoids besides lutein and zeaxanthin has also been suggested (31), a possibility that could not be evaluated in the present study because other carotenoids were not measured. It is also possible that a population's response to dietary lutein and zeaxanthin may be age dependent, although the studies of Johnson et al. (18), in a population aged 33–54 y, and Yeum et al. (29), in a population ranging in age from 20 to 80 y, did not report age-dependent differences in plasma lutein response.

The finding that concentrations of serum lutein and zeaxanthin during the no-egg and egg interventions were associated with serum HDL-C, is not unexpected, insofar as these oxygenated carotenoids are predominantly transported in this lipoprotein fraction (32). Due to the small number of subjects ($n = 11$) compared with the current study ($n = 33$), Handelman et al. (22) were unable to determine whether the lutein concentration was associated with any serum lipid measurements such as HDL-C.

In the current study, the carotenoid responsiveness is similar to the findings of Handelman et al. (22), despite the differences in the mean age of the study populations (62 vs. 79 y in the current study), which suggests that age is not related to the degree of diet responsiveness.

A difference between our study and the results of Handelman et al. (22) concerning serum LDL-C changes (11 and 3.2%, respectively), may be a result of the number of subjects in our study (33) vs. the number (11) in the study by Handelman et al. (22). Another possibility for the difference in serum LDL-C response may be gender related, insofar as Handelman et al. (22) had 54% males, vs. 21% males the current study.

Our results demonstrated that increases in serum lutein and zeaxanthin concentrations were not significantly associated with a similar increase in serum lipids and lipoprotein cholesterol concentrations in this population with a mean age of 79 y. The actual 0.6 and 3.2% increase in serum TC and LDL-C concentrations, respectively, is consistent with the meta-analysis of studies of dietary cholesterol effects on plasma TC and LDL-C conducted by Clarke et al. (33), Howell et al. (34), and Weggemans et al. (35). Greene et al. (36) showed that dietary cholesterol concentrations from consuming 3 eggs/d significantly increased plasma LDL-C and HDL-C in subjects 29–60 y of age, and there were no significant alterations in the ratios of LDL-C:HDL-C or the TC:HDL-C, which is often associated with increased atherogenicity. Our study did not find significant increases in serum LDL-C or HDL-C, possibly because our participants consumed only 1 egg/d. This may also be related to the greater age of the population in this study. There are several studies (37–39) that indicate serum LDL-C concentrations are reduced in older populations, and, in one study, the suggested mechanism may be reduced.

In conclusion, compared with the study of Handelman et al. (22), our study demonstrated that consuming only 1 store-bought egg/d in a population with a mean age of 79 y can significantly increase both serum lutein and zeaxanthin concentrations without elevating serum TC and LDL-C concentrations. The study also revealed that the degree of carotenoid response in an older population was similar to those reported for younger populations (22), despite the fact that the work by Handelman et al. (22) was a metabolic ward study (personal communication, Dr. Garry Handelman, University of Massachusetts, Lowell), environmentally different from the relatively free-living study reported here. Finally, this study showed a significant association between serum concentrations of carotenoids with only HDL-C, a finding that could not be observed in the study of Handelman et al. (22) with only 11 participants.

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