Postprandial lipemia in the elderly involves increased incorporation of ingested fat in plasma free fatty acids and small (S_f 20–400) triglyceride-rich lipoproteins

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Puga GM, Meyer C, Everman S, Mandarino LJ, Katsanos CS. Postprandial lipemia in the elderly involves increased incorporation of ingested fat in plasma free fatty acids and small (S_f 20–400) triglyceride-rich lipoproteins. Am J Physiol Endocrinol Metab 301:E356–E361, 2011. First published May 10, 2011; doi:10.1152/ajpendo.00670.2010.—In the elderly, the rise in postprandial plasma triglyceride (TG) concentrations is increased, contributing to their increased risk of cardiovascular disease. We sought to determine the incorporation of ingested fat (whipping cream enriched with [1,1,1-13C]triolein) into plasma lipids during the postprandial period in six healthy elderly (67 ± 1 yr old) and six healthy young (23 ± 2 yr old) subjects. Blood and expired air samples were taken before and at 2-h intervals during the 8-h postprandial period. As expected, the area under the curve of postprandial plasma TG concentrations was larger in the elderly compared with the young subjects (152 ± 38 vs. 66 ± 27 mg·dl⁻¹·h, P < 0.05). The incorporation of [13C]oleate in plasma free fatty acids (FFAs) and TG of the small (S_f 20–400) triglyceride-rich lipoprotein (TRL) fraction was significantly higher in the elderly compared with the young subjects, resulting in increased postprandial contributions of the ingested lipid to plasma FFAs (41 ± 3 vs. 26 ± 6%, P < 0.05) and the small TRL fraction (36 ± 5 vs. 21 ± 3%, P < 0.05) in elderly. Plasma apoB-100 concentration was higher, whereas the rate of oxidation of the ingested lipid was lower (P < 0.05) in the elderly. We conclude that increased postprandial lipemia in the elderly involves increased contribution of ingested lipid to the plasma small TRLs. This appears to be driven at least in part by increased appearance of the ingested fat as plasma FFA and increased availability of apoB-100 lipoproteins in the elderly.

However, the mechanisms that contribute to the age-associated increase in PPL are poorly understood. This is due in part to the selection of subjects in previous studies that makes conclusions on the effects of aging on PPL problematic as well as the lack of isotopic tracer studies that trace the metabolic fate of ingested fat in the elderly.

To elucidate the effects of aging per se on PPL, it is important to control for physiological variables that are not directly linked to aging but are known to modify PPL. Such variables include fasting plasma TG concentrations and fat-free mass/muscle mass. Fasting plasma TG concentrations correlate with PPL (5) because chylomicrons compete with lipoproteins secreted by the liver for hydrolysis/clearance of their TG load by the enzyme lipoprotein lipase. Therefore, the observed increased PPL in the elderly in previous studies (4, 5, 9) can be explained, at least in part, by increased plasma TG concentrations in the fasting state. Furthermore, fat-free mass is a major determinant of energy expenditure, with skeletal muscle representing the tissue with the largest fractional removal of circulating TG (21) and being a major player in fat oxidation and the removal of ingested lipid (23). Hence, loss of fat-free mass/muscle mass as a result of sarcopenia (22) may contribute to increased PPL in the elderly and mask the effects of aging per se on the postprandial lipid metabolism.

Therefore, to determine the effects of aging on PPL following fat ingestion, we matched elderly and young healthy subjects for fasting plasma TG concentrations and fat-free mass and determined the incorporation of isotopically labeled ingested lipid into major plasma lipid and TRL fractions by mass spectrometry analysis.

METHODS

Subjects. The postprandial fate of ingested lipid in six healthy elderly males was compared with that of six healthy young males, who served as controls. Exclusion criteria included smoking, body mass index (BMI) > 30 kg·m⁻², hypertension, diabetes, heart disease, peripheral vascular disease, use of either prescription or over-the-counter medications, and history of liver or kidney disease. Subjects included in the study were determined to be healthy based on medical history report, physical examination, resting electrocardiogram, and routine blood and urine tests. Those who qualified for the study were instructed to abstain from any form of exercise for 3 days before the study, maintain their regular diet during the same period, and avoid alcohol consumption. Groups of elderly and young subjects were matched for fat-free mass, determined by bioimpedence, and fasting plasma TG concentrations. Subject characteristics for the two groups are presented in Table 1. All study participants were Caucasians. The purpose, procedures, and the risks associated with the experiments were explained to each subject before informed written consent was obtained. The study protocol was approved by the Institutional Review Board at Arizona State University.
Table 1. Subject characteristics

<table>
<thead>
<tr>
<th></th>
<th>Young</th>
<th>Elderly</th>
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<tbody>
<tr>
<td>Age, yr</td>
<td>22.7 ± 2.4</td>
<td>66.7 ± 1.1*</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>72.5 ± 2.7</td>
<td>85.5 ± 2.4*</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>23.4 ± 0.4</td>
<td>26.0 ± 0.7*</td>
</tr>
<tr>
<td>Fat-free mass, kg</td>
<td>60.9 ± 2.7</td>
<td>63.9 ± 1.8</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>15.0 ± 2.2</td>
<td>25.8 ± 0.7*</td>
</tr>
<tr>
<td>Plasma lipids, mg/dl</td>
<td></td>
<td></td>
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<tr>
<td>Triglycerides</td>
<td>72.3 ± 10.3</td>
<td>69.5 ± 10.7</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>140.0 ± 5.2</td>
<td>179.7 ± 21.8</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>43.0 ± 2.1</td>
<td>55.0 ± 5.7</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>85.0 ± 3.9</td>
<td>87.2 ± 19.4</td>
</tr>
<tr>
<td>Plasma glucose, mg/dl</td>
<td>92.5 ± 2.2</td>
<td>90.5 ± 3.2</td>
</tr>
<tr>
<td>Plasma insulin, µIU/ml</td>
<td>6.1 ± 0.9</td>
<td>6.0 ± 1.7</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>1.4 ± 0.2</td>
<td>1.4 ± 0.4</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>120.8 ± 2.3</td>
<td>126.7 ± 3.0</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>78.2 ± 3.9</td>
<td>74.7 ± 3.9</td>
</tr>
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</table>

Values are means ± SE. BMI, body mass index; HOMA-IR, homeostasis model assessment-estimated insulin resistance; SBP, systolic blood pressure; DBP, diastolic blood pressure. *Significantly different from the young (P < 0.05).

Experimental protocol. Subjects reported to the Clinical Research Unit at Arizona State University on the morning of the study, at ~7 AM, after an overnight fast starting no later than 10 PM. After verifying compliance with the instructions, subjects were laid in bed for the entire duration of the experiment, and a catheter was inserted into an antecubital vein for blood sampling. After ~30 min of rest a baseline blood sample was collected, and subjects ingested fat in the form of whipping cream (0.7 g fat/kg body wt). [1,1,1-13C]triolein (Cambridge Isotope Laboratories, Andover, MA) was incorporated (sonicated) into the whipping cream to achieve an isotopic enrichment of 4%. Subjects were allowed to drink only water during the course of the study.

Blood and breath samples were collected at baseline and every 2 h after ingestion of the fat and for a total period of 8 h. Blood samples were collected into EDTA Vacutainer tubes and placed on ice, followed by immediate separation of plasma by centrifugation (1,500 g for 15 min at 4°C). The concentration of plasma C-terminal pro-BNP (n = 10) and intact leptin (n = 10) was measured by enzyme-linked immunosorbent assay (ELISA) (Acmi, Chandler, AZ) and ultrasensitive (n = 10) (Quintel Diagnostics, Waltham, MA), respectively.

Commercially available procedures were used for the determination of the concentrations of plasma TGs (Sigma-Aldrich, St. Louis, MO), FFAs, 3-HB (Wako Chemicals, Richmond, VA), and apoB-100 (Kamiya Biomedical, Seattle, WA) as well as insulin (Alpco Diagnostics, Windham, NH). Isolation of TGs in the large and small TRL fractions as well as FFAs in the plasma for the determination of [13C]oleate enrichment was based on procedures described in detail elsewhere (27). Briefly, lipids were extracted with a mixture of hydrochloric acid-n-heptane-2-propanol (1:10:40, vol/vol/vol), and the upper layer was removed and dried under nitrogen. After chloroform-methanol (2:1, vol/vol) was added, lipids were isolated by thin-layer chromatography (TLC; Silica gel plates, 250 μm thickness; Whatman) using an n-heptane-ethyl ether-glacial acetic acid (80:20:2, vol/vol/vol) solvent. The TG and FFA location on the TLC plate was visualized using primuline (5 mg in 80 ml of acetone, 20 ml of water). The bands on the TLC plate corresponding to the TGs (TRL subfractions) and FFAs (plasma samples) were cut and transferred into tubes. The isolated lipids were methylated using a mixture of boron trifluoride methanol. Isotopic enrichment, expressed as tracer/tracee ratio (TTR) of methyl oleate, was determined using gas chromatography-mass spectrometry and by selected ion monitoring of mass-to-charge ratios 296 and 297. Breath samples were analyzed for isotopic enrichment of 13CO2 by Metabolic Solutions (Nashua, NH) using a Finnigan BreathMat gas isotope ratio mass spectrometer.

Calculations. The fractional contributions of ingested FAs to the plasma TG of the small TRL fraction and FFA pool were calculated at each time point as follows (2): %plasma TG of the small TRL fraction originating from the ingested fat = [13C]oleate TTR in plasma TG of the small TRL fraction/[13C]oleate TTR in the ingested fat) × 100 and %plasma FFAs originating from the ingested fat = [13C]oleate TTR in plasma FFA/[13C]oleate TTR in the ingested fat) × 100.

The [13C]oleate TTR in the ingested fat was calculated on the basis of the exact weighted amounts of whipping cream (9.3% triolein) and the [1,1,1-13C]triolein incorporated into the whipping cream.

The rate of oxidation of ingested lipid was calculated from 13C enrichment in the expired CO2 and the rate of VCO2 (l/min) production, using the following formula:

Oxidation of ingested lipid (g/min) = 

\[((\text{ECO}2(t) - \text{ECO}2(t_0)) / \text{ETINGCO}2 - \text{ECO}2(t_0)) \times V_{\text{CO}2} / (1.43 \times 0.6)\]

where ECO2(t) is the enrichment of expired CO2 at time t (the end of the 2-h period), ECO2(t0) is the enrichment of expired CO2 at time t0, ETINGCO2 is the 13C enrichment of the ingested fat, 1.43 is the amount (L/g) of CO2 associated with the oxidation of fat, and 0.6 is the acetate correction factor (24). ETINGCO2 was calculated on the basis of the exact weighted amounts of the ingested whipping cream/fat and the incorporated tracer.

Statistical analyses. Data were tested for normality according to the Ryan-Joiner test. Logarithmic transformation was performed for variables that were not normally distributed (i.e., plasma FFA concentrations, [13C]oleate enrichment in TGs of the large TRL fraction), and the normality of the transformed variables was confirmed before statistical analysis. Data from the two groups were analyzed using two-factor (age × time) ANOVA. The incremental area under the curve (AUC; area under the curve after subtracting the fasting value) for the variables of interest was calculated using the trapezoidal method. AUC values were calculated for the entire postprandial period as well as for the first (0–4 h) and last half (4–8 h) of the postprandial period to evaluate relative responses during these particular periods. AUC values were compared between groups by unpaired Student’s t-test. Results are expressed as means ± SE. Pearson correlation analyses were performed to examine relationships between

AJP-Endocrinol Metab • VOL 301 • AUGUST 2011 • www.ajpendo.org
variables. Statistical significance was set at $P \leq 0.05$. All the statistical analyses were performed using the Minitab 15.1 statistical software (Minitab, State College, PA).

RESULTS

Unlabeled biochemical parameters. At baseline, plasma TG concentrations were not different between elderly and young subjects. Following the ingestion of fat, plasma TG concentrations increased to a peak at $\sim 4$ h and subsequently decreased during the next $4$ h to values below baseline in both groups. However, in the elderly subjects, the increase in plasma TG concentrations at $4$ h was more pronounced and remained markedly elevated during the $4$- to $8$-h postprandial period. Hence, the AUC of postprandial TG concentrations was $\sim 130\%$ larger in the elderly subjects (Fig. 1), due mainly to the increased plasma TG concentrations in the $4$- to $8$-h postprandial period (Table 2). Fat ingestion had no significant effect on plasma insulin concentrations in either group (data not shown).

Similar to the TG concentrations, plasma FFA concentrations were not different at baseline and increased to peak concentrations $\sim 4$ h after fat ingestion in both groups; plasma FFA concentrations subsequently declined but remained above baseline levels until the end of the $8$-h experiment (Fig. 2A). However, in the elderly, the excursion of postprandial plasma FFA concentration was more pronounced, resulting in an AUC that was $\sim 40\%$ larger in the elderly compared with the young subjects ($2.5 \pm 0.6$ vs. $1.8 \pm 0.3$ mmol·l$^{-1}$·h), but this did not reach statistical significance ($P > 0.05$). A similar pattern was observed for plasma 3-HB concentrations. Levels were similar at baseline, increased to a peak at $\sim 4$–$6$ h after fat ingestion, and subsequently declined modestly in both groups (Fig. 2B). However, the postprandial excursion in plasma 3-HB concentration was higher in the elderly subjects such that the AUC for plasma 3-HB was $56\%$ larger in the elderly compared with the young subjects ($1447 \pm 133$ vs. $929 \pm 179$ mmol·l$^{-1}$·h, $P < 0.05$).

Table 2. Incremental AUC (after subtracting the postabsorptive value) calculated for the first $4$ (0–$4$ h AUC) and last $4$ h (4–$8$ h AUC) of the postprandial period

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<thead>
<tr>
<th></th>
<th>0-4 h AUC</th>
<th>4-8 h AUC</th>
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<tbody>
<tr>
<td></td>
<td>Young</td>
<td>Elderly</td>
</tr>
<tr>
<td>TG, mg·dl$^{-1}$·h</td>
<td>$60 \pm 14$</td>
<td>$85 \pm 15$</td>
</tr>
<tr>
<td>FFA$t$ , mmol·l$^{-1}$·h</td>
<td>$0.7 \pm 0.1$</td>
<td>$1.0 \pm 0.3$</td>
</tr>
<tr>
<td>FFA$i$, mmol·l$^{-1}$·h</td>
<td>$0.6 \pm 0.3$</td>
<td>$1.0 \pm 0.2$</td>
</tr>
<tr>
<td>3-HB, $\mu$mol·l$^{-1}$·h</td>
<td>$274 \pm 90$</td>
<td>$500 \pm 121$</td>
</tr>
</tbody>
</table>

Values are means ± SE. AUC, area under the curve; TG, plasma triglycerides; FFA$t$, total plasma free fatty acids; FFA$i$, plasma free fatty acids originating from the ingested fat; 3-HB, plasma 3-hydroxybutyrate.
At baseline, the average plasma apoB-100 concentration in the elderly was twice that of the young (132 ± 28 vs. 66 ± 11 mg/dl, P = 0.06). Plasma apoB-100 concentrations did not change following fat ingestion in either group (P > 0.05), with the plasma apoB-100 concentrations in elderly remaining higher than those in the young throughout the postprandial period (P < 0.05; ANOVA). When data from all subjects were analyzed together, a significant positive correlation was found between the baseline plasma apoB-100 concentration and the AUC describing the postprandial plasma TG response (r = 0.68, P < 0.05).

Incorporation of ingested lipid into plasma TG of large and small TRL fractions and plasma FFAs. As expected, [13C]oleate from the ingested fat rapidly enriched the pool of TG of plasma large TRLs during the postprandial period in both groups (P < 0.05). The [13C]oleate enrichment in TG of plasma large TRLs peaked later in the elderly compared with the young subjects. The peak [13C]oleate enrichment in TG of plasma large TRLs was observed at 6.3 ± 0.6 h in the elderly vs. 5.2 ± 1.0 h in the young. However, this difference was not statistically significant (P > 0.05). Similarly, ANOVA indicated no significant differences in the enrichment of TG of plasma large TRLs during the 8-h postprandial period between groups (P > 0.05; Fig. 3A).

Following the ingestion of fat, [13C]oleate enrichment in TG of the small TRLs increased less rapidly and to a lesser extent than the [13C]oleate enrichment of the large TRLs in both groups. However, throughout the postprandial period the enrichment was increased consistently in the elderly subjects, and the pattern differed. In the young subjects, the enrichment peaked at 4–6 h and subsequently decreased to ~50% of the peak level by the end of the experiment, whereas in the elderly subjects, the enrichment continued to increase throughout the postprandial period (P < 0.05; Fig. 3B). Consequently, the percent contribution of ingested lipid to plasma TG of the small TRLs during the entire 8-h postprandial period was significantly increased in the elderly subjects (36 ± 5 vs. 21 ± 3%, P < 0.05).

[13C]oleate enrichment in plasma FFAs peaked at 4 h after the ingestion of fat and subsequently decreased slightly in both groups. However, as for the small TRL fraction, the enrichments of plasma FFA were consistently higher in the elderly than in the young subjects (P < 0.05; Fig. 3C) such that the percent contribution of ingested lipid to the plasma FFA pool during the 8-h postprandial period was increased ~1.6-fold in the former group (Fig. 4). This resulted in plasma FFA concentrations originating from the ingested fat that were considerably higher in the elderly compared with the young subjects during the course of the 8-h postprandial period (AUC: 2.8 ± 0.4 vs. 1.6 ± 0.5 mmol·l⁻¹·h, P = 0.08). This difference was more pronounced and statistically significant between groups during the last part (4–8 h) of the postprandial period (P < 0.05; Table 2).

Oxidation of ingested lipid. The rate of oxidation of the ingested lipid increased progressively throughout the 8-h postprandial period in both groups. However, the postprandial oxidation of ingested lipid was significantly lower (P < 0.05) in the elderly subjects whether expressed per unit of body weight (data not shown) or expressed per unit of fat-free mass (Fig. 5). Overall, the elderly subjects oxidized 32 ± 4% of the ingested lipid during the 8-h postprandial period compared with 45 ± 3% oxidized by the young subjects (P < 0.05).

DISCUSSION

The present study was designed to elucidate mechanisms of increased PPL in the elderly for the first time by measuring the
circulation (17). Therefore, the increased plasma FFA concentrations during the 8-h postprandial period in young and elderly subjects. *P < 0.05.

The contribution of isotope-labeled ingested fat into plasma FFA and TG of TRL fractions in elderly and young subjects. We found that in these well-matched groups PPL was ~130% increased in the elderly compared with that in the young subjects, consistent with previous reports (4). This response in the elderly was associated with 1) ~40% increased plasma FFA excursions, solely due to FFA originating from the ingested fat, 2) an increased contribution of ingested fat to TG of small but not large TRL, 3) increased plasma apoB-100 concentrations, and 4) decreased rate of oxidation of the ingested fat.

In the present study, elderly and young subjects were of the same sex and matched for physical activity, fat-free mass, fasting plasma TG concentrations, and insulin sensitivity, the latter based on fasting plasma glucose and insulin concentrations. Furthermore, subjects were excluded for taking any medications to minimize the influence of possible confounders on our data. However, the elderly subjects had a significantly greater fat mass compared with the young subjects. This increased overall fat mass might have not only been associated with increased adipose tissue but also increased ectopic fat accumulation in muscle and liver, which might have affected our data. For example, intramuscular (6) and intermuscular (18) lipid content are increased in elderly, a metabolic condition that has been associated with decreased capacity for fat oxidation (8). Although possible differences in muscle lipid content between groups may explain the decrease in the rate of oxidation of ingested fat in the elderly, its direct effects on PPL remain to be determined. On the other hand, increased liver fat content is associated with increased PPL and specifically with an increase in the concentrations of plasma TGs originating from the liver (16). Although we did not measure liver fat content, it can be expected that it was higher in the elderly than in the young subjects, in line with previous observations (6) and because of increased body fat in our elderly subjects. In any case, it may have contributed to the increased PPL in the elderly by increasing the concentration of plasma TGs in the small TRL particles (16).

TG in plasma chylomicrons is subject to hydrolysis by lipoprotein lipase, and the liberated fatty acids are taken up by tissues, mainly skeletal muscle and adipose tissue. However, some of these fatty acids escape and spill over in the systemic circulation (17). Therefore, the increased plasma FFA concentrations together with the increased contribution of the ingested fat to plasma FFA in the elderly subjects suggest that the immediate uptake of fatty acids derived from chylomicrons becomes less efficient in the elderly, resulting in a greater spillover into the systemic circulation. Possible mechanisms include impairments in fatty acid transport and tissue utilization, including oxidation and storage, consistent with the reduced utilization of the ingested fat in our elderly subjects, and reduced capacity of dietary fat storage in adipose tissue (13) and fatty acid uptake in skeletal muscle (3) with aging.

The incremental AUC representing the postprandial concentrations of 3-HB, an indirect measurement of hepatic lipid oxidation, was 56% larger in the elderly compared with that observed in the young subjects, a response mediated probably by the increased postprandial plasma FFA concentrations in the elderly. In addition to the upregulation of hepatic lipid oxidation, exposure of the liver to increased plasma FFA concentrations stimulates hepatic TG production (15). Greater [13C] oleate enrichment in the plasma FFA pool of the elderly in the present study (Fig. 3C) was accompanied by greater [13C] oleate enrichment in the plasma TG of the small TRLs (Fig. 3B), which include TRLs secreted by the liver that readily incorporate dietary plasma FFA during the postprandial period (2). Therefore, greater enrichment of the plasma FFA pool with ingested lipid during the postprandial period in the elderly could have mediated increased incorporation of the ingested fat into lipoproteins secreted by the liver. Furthermore, there was a significant positive correlation between the plasma apoB-100 concentrations (a marker of the number of plasma lipoproteins from hepatic origin) and PPL in the present study, pointing to a possible role of hepatic lipoproteins in determining the PPL. Despite comparable fasting plasma TG concentrations, there were increased fasting plasma apoB-100 concentrations in the elderly, which are attributed to the greater rate of secretion of apoB-100 with aging (26). Under these circumstances, increased delivery of dietary lipid into the liver during the postprandial period combined with increased capacity for apoB-100 secretion with aging can facilitate increased incorporation of dietary lipid into hepatic TG and secretion into plasma (i.e., recycling) in the form of TRLs in elderly.

Fig. 5. Rate of oxidation of ingested lipid following fat ingestion (whipping cream ingested at time 0) in young and elderly. *Significant effect for age (P < 0.05), FFM, fat-free mass.
The elderly subjects in the present study had lower rates of oxidation of ingested lipid during the postprandial period (Fig. 5). This observation extends previous findings describing reduced rates of lipid oxidation with aging in the basal state (25) to the postprandial state and specifically in association with the oxidation of the ingested fat. It is noted that the subjects in the present study were not matched for aerobic capacity. Given that high aerobic capacity is associated with improved capacity for lipid oxidation (7), a possible lower aerobic capacity in elderly may have resulted in decreased capacity for oxidation of the ingested fat during the postprandial period. However, although differences in the fitness levels between groups may have contributed, it is less likely that they have played a major or direct role in the age-associated increase in PPL, which was the main end point of the present study. This is because PPL decreases as a result of the acute effects of exercise (i.e., in relation to the timing of the last exercise session) rather than as a result of increased aerobic capacity per se (11). Nevertheless, we cannot exclude the possibility that decreased uptake and oxidation of ingested lipid across tissues in the elderly increased the availability of this lipid within the liver, which has an increased capacity for lipid uptake during the postprandial period (14), thus contributing to an increased hepatic lipid accumulation and TG secretion during the postprandial period in the elderly.

In conclusion, there are no differences in the contribution of ingested lipid into plasma TG of large (S₁ > 400) TRLs during the postprandial period between healthy elderly and young individuals. Increased contribution of ingested lipid into plasma FFAs coupled with reduced capacity for oxidation of the ingested lipid in the elderly may upregulate the secretion of hepatic TG into plasma during the postprandial period, thus increasing the magnitude of postprandial lipemia in the elderly. The specific roles of these two mechanisms in the regulation of postprandial lipemia with aging remain to be determined.

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GRANTS

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

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