Polymorphism exon 1 variant at the locus of the scavenger receptor class B type I gene: influence on plasma LDL cholesterol in healthy subjects during the consumption of diets with different fat contents1–3

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ABSTRACT

Background: The association between polymorphisms in the scavenger receptor class B type I (SRB-I) gene and variations in basal plasma concentrations of cholesterol in humans has recently been described.

Objective: The objective of the study was to determine whether the exon 1 variant (G→A) at the SRB-I gene is associated with the lipid response to the content and quality of dietary fat in healthy subjects.

Design: We studied 97 healthy volunteers with exon 1 polymorphism (65 homozygous for allele 1 (1/1) and 32 heterozygous for allele 2 (1/2)). Both groups consumed 3 diets lasting 4 wk each. The first was a saturated fatty acid (SFA)–rich diet (38% fat, 20% SFA), which was followed by a carbohydrate (Cho)–rich diet (30% fat, < 10% SFA, 55% carbohydrate) or a monounsaturated fatty acid (MUFA), olive oil–rich diet (38% fat, 22% MUFA) according to a randomized crossover design. At the end of each dietary period, plasma concentrations of triacylglycerol and of total, LDL, and HDL cholesterol were measured.

Results: Carriers of the 1/2 genotype had a trend toward higher concentrations of LDL cholesterol (P < 0.11) after the SFA–rich diet than did those who were homozygous for 1/1. Carriers of the mutation showed a significantly greater (P = 0.007) decrease in LDL-cholesterol concentrations (−23%) in changing from an SFA–rich diet to a Cho–rich diet than did noncarriers of the mutation (−16%).

Conclusion: Carriers of the minority allele, 1/2, are more susceptible to the presence of SFA in the diet because of a greater increase in LDL cholesterol.

KEY WORDS Scavenger receptor class B type I, SRB-I, dietary intervention, LDL cholesterol, genetic polymorphism, cardiovascular risk

INTRODUCTION

The scavenger receptor class B type I (SRB-I) gene is an HDL receptor that mediates cholesterol uptake from cells (1, 2). In rodents, SRB-I has a critical influence on plasma HDL-cholesterol concentration and structure as well as on cholesterol delivery to steroidogenic tissues, female fertility, and biliary cholesterol concentration (3, 4). SRB-I can also serve as a receptor for non-HDL lipoproteins and appears to play an important role in reverse cholesterol transport. SRB-I binds HDL with high affinity and is expressed primarily in liver and nonplacental steroidogenic tissues. It mediates selective cholesterol uptake by a mechanism that is distinct from the classic LDL receptor pathway (5). SRB-I has clearly been shown to be important to HDL metabolism in mice. Its importance in humans is still unknown, although recent studies showed that it may play a role in the metabolism of LDL and HDL cholesterol (6, 7). The SRB-I gene, located in 12q24, reveals that this locus is polymorphic in whites (8). There is evidence of significant sex-specific associations between several single-nucleotide polymorphisms (SNPs) and plasma lipids and anthropometric measures. The exon 1 variant (G→A) has been significantly associated with higher HDL-cholesterol and lower LDL-cholesterol concentrations in men, but not in women (8). In contrast, the exon 8 variant (C→T) has been related to lower LDL-cholesterol concentrations compared with homozygosity for the common allele in women. Women carriers of the intron 5 variant (C→T) showed a higher body mass index than women who are homozygous for the common allele. No associations for these variants were observed in men (8). Recent studies involving the manipulation of SRB-I expression in mice indicate that the expression of SRB-I protects against atherosclerosis (9–11).

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If SRB-I has a similar activity in humans, it may become an attractive target for therapeutic intervention. Our aim was to determine whether the exon 1 polymorphism of the SRB-I gene modifies the lipid response to the quantity and quality of dietary fat in healthy subjects.

**SUBJECTS AND METHODS**

**Subjects**

A group of 97 subjects (70 men, 27 women) was recruited from among 250 students at the University of Cordoba. The subjects had a mean (± SD) age of 21 ± 0.4 y. Of these subjects, 65 were homozygous for 1/1 and 32 were heterozygous carriers of the minority allele, 1/2. There were no homozygotes for 2/2. The frequencies were similar to those in previous studies. Forty-seven subjects had participated in a previous study (12). Written informed consent was obtained from all participants. All subjects underwent a comprehensive medical history, physical examination, and clinical chemistry analysis before enrollment. Subjects showed no evidence of any chronic disease (hepatic, renal, thyroid, or cardiac dysfunction), obesity, or unusually high levels of physical activity (eg, sports training). None of the subjects had a family history of premature coronary artery disease or had taken medications or vitamin supplements in the 6 mo before the study. Physical activity and diet, including alcohol consumption, were recorded in a personal log for 1 wk, and the data were used to calculate individual energy requirements. Mean (± SD) body mass index (BMI; in kg/m²) was 22.86 at the onset of the study and 22.90 ± 1.02 at the end of the study. Both the beginning and the end of the study did not differ in BMI.

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**Diets**

The study design included an initial 28-d period during which all subjects consumed a saturated fatty acid (SFA)–rich diet, with 15% protein, 47% carbohydrate, and 38% fat [20% SFA, 12% monounsaturated fatty acid (MUFA), and 6% polyunsaturated fatty acid (PUFA)]. After this period, volunteers were randomly assigned to 1 of 2 diet sequences. Forty-eight subjects consumed a MUFA–rich diet containing 15% protein, 47% carbohydrate, and 38% fat (<10% SFA, 6% PUFA, and 22% MUFA) for 28 d. This diet was followed for 28 d by consumption of a carbohydrate (CHO)–rich diet containing 15% protein, 55% carbohydrate, and <30% fat (<10% SFA, 6% PUFA, and 12% MUFA). The other 49 subjects consumed the CHO diet before the MUFA diet. The cholesterol content remained constant (<300 mg/d) during the 3 dietary periods.

Eighty percent of the MUFA diet was provided by virgin olive oil, which was used for cooking, in salad dressing, and as a spread. The carbohydrate intake on the CHO diet was based on the consumption of biscuits, jam, and bread. Butter and palm oil were used during the SFA dietary period.

The composition of the experimental diets was calculated from the US Department of Agriculture food tables (13) and Spanish food-composition tables for local foodstuffs (14). All meals were prepared in the hospital kitchen and were supervised by a dietitian.

Lunch and dinner were eaten in the hospital dining room, and breakfast and an afternoon snack were eaten in the medical school cafeteria. Fourteen menus with ordinary solid foods were prepared and rotated during the experimental period. Duplicate samples from each menu were collected, homogenized, and stored at −70°C. The protein, fat, and carbohydrate contents of the diet were analyzed by standard methods (15). Dietary compliance was verified by analyzing the fatty acids in each subject’s LDL-cholesterol esters at the end of each dietary period (16). The study took place during January, February, and March to minimize seasonal effects and academic stress.

**Lipid analysis and biochemical determinations**

Venous blood samples were collected into tubes containing EDTA (1g/L) from all subjects after a 12-h overnight fast at the beginning of the study and at the end of each dietary period. Plasma was obtained by low-speed centrifugation at 2500 × g for 15 min at 4°C within 1 h of venipuncture. To reduce interassay variation, plasma was stored at −80°C and analyzed at the end of the study. Plasma cholesterol and triacylglycerol concentrations were measured by enzymatic techniques (17, 18). HDL-cholesterol concentrations were measured after precipitation with phosphotungstic acid (19). Apolipoprotein A-I (apo A-I) and apo B were measured by immunoturbidimetry (20). LDL-cholesterol concentrations were calculated with the use of the Friedewald formula (21).

**DNA amplification and genotyping**

Genotyping of the SRB-I exon 1 (G→A) was carried out as previously described (22). The primer and probe sequences used were as follows: forward primer, 5′-GTCCCGGTCTCTGCGCA-3′; reverse primer, 5′-CCCAGCACAGCGAGAGATGA-3′; G-allele probe, 5′-FAM-AGACATGGGCTGCTCCGGCA-TAMRA-3′; and A-allele probe, 5′-VIC-CAGACATGAGCTGCTCCGCCA-3′. The bases in boldface type represent point mutations. Polymerase chain reaction was performed in a 10-μL final volume for each SNP. The reaction mixture contained 5 μL TaqMan 2X Universal PCR Master Mix (Applied BioSystems, Foster City, CA); 200 nmol FAM-labeled probe/L, 150 nmol VIC-labeled probe/L, 900 nmol reverse primer/L, and 900 nmol forward primer/L (all: Epoch Biosciences, Bothell, WA); and 2–20 ng genomic DNA. The thermal cycler program included one cycle at 50°C for 2 min to activate uracil-DNA glycosylase (Trevigen Inc, Gaithersburg, MD), which is added to prevent carryover contamination; one cycle at 95°C for 10 min to activate AmpliTaq Gold Polymerase (Applied BioSystems); and then 40 cycles at 95°C for 15 s for denaturing and at 62°C for 60 s for annealing and extending. Allelic discrimination was performed on the post-polymerase chain reaction product. Fluorescence data were collected with the use of the 7700 Sequence Detection System (Perkin-Elmer/Applied BioSystems) on the samples for 5 s and analyzed with the use of SDS software, version 6.0 (Applied BioSystems), which could be visualized in graph form (22).

**Statistical analysis**

We used repeated-measures analysis of variance to test for effects of the exon 1 polymorphism of the SRB-I gene on plasma total cholesterol, LDL-cholesterol, HDL-cholesterol, triacylglycerol, apo A-I, and apo B concentrations in each dietary phase. When statistical significance was found, Tukey’s post hoc comparison test was used to identify group differences. Values were

\[
\text{SRB-I}
\]

\[
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considered significant at $P < 0.05$. Statistical analyses were carried out with the use of SPSS statistical software, version 8.0 (SPSS Inc, Chicago).

RESULTS

Significant differences were not found in any of the variables studied when we compared the basal characteristics of subjects who were homozygous for allele 1 (1/1) and of carriers of the minority allele 2 (1/2) in the exon 1 SNP. No differences exist between sexes in terms of the genotype in basal conditions (Table 1) or after the consumption of the different diets (data not shown). The actual composition of the mean daily intake of the participants in shown in Table 2. Analysis of LDL-cholesterol concentrations in the exon 1 SNP were observed to

<table>
<thead>
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<th>TABLE 1</th>
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Characteristics at baseline of men and women with 1/1 and 1/2 genotypes

<table>
<thead>
<tr>
<th></th>
<th>1/1 (n = 46)</th>
<th>1/2 (n = 24)</th>
<th>1/1 (n = 19)</th>
<th>1/2 (n = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>20.09 ± 0.38</td>
<td>21 ± 0.47</td>
<td>21.03 ± 0.4</td>
<td>21.01 ± 0.5</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.59 ± 0.38</td>
<td>24.18 ± 0.5</td>
<td>19.84 ± 0.25</td>
<td>20.41 ± 0.35</td>
</tr>
<tr>
<td>Total cholesterol (mmol/L)</td>
<td>4.03 ± 0.61</td>
<td>4.19 ± 0.71</td>
<td>4.10 ± 0.57</td>
<td>4.32 ± 0.75</td>
</tr>
<tr>
<td>LDL cholesterol (mmol/L)</td>
<td>2.41 ± 0.54</td>
<td>2.56 ± 0.68</td>
<td>2.35 ± 0.62</td>
<td>2.47 ± 0.73</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/L)</td>
<td>1.21 ± 0.28</td>
<td>1.17 ± 0.27</td>
<td>1.44 ± 0.37</td>
<td>1.56 ± 0.50</td>
</tr>
<tr>
<td>Triacylglycerol (mmol/L)</td>
<td>0.85 ± 0.38</td>
<td>0.99 ± 0.58</td>
<td>0.66 ± 0.29</td>
<td>0.63 ± 0.21</td>
</tr>
<tr>
<td>Apo A-I (g/L)</td>
<td>1.22 ± 0.24</td>
<td>1.28 ± 0.23</td>
<td>1.54 ± 0.11</td>
<td>1.63 ± 0.18</td>
</tr>
<tr>
<td>Apo B (g/L)</td>
<td>0.62 ± 0.14</td>
<td>0.65 ± 0.16</td>
<td>0.73 ± 0.12</td>
<td>0.71 ± 0.13</td>
</tr>
</tbody>
</table>

$x \pm SD$. Apo, apolipoprotein. There were no significant differences between the sexes or genotypes, $P < 0.05$ (ANOVA).

in the different intervention stages. During the SFA diet period, we observed a significantly greater ($P < 0.005$) increase in palmitic acid in the LDL-cholesterol esters than were observed during the Cho and MUFA diets: 27.3% compared with 19.8% and 15.2%, respectively. A significantly greater ($P < 0.05$) increase in oleic acid in the cholesterol esters was also seen during the MUFA diet period than during the Cho diet period: 50.3% compared with 38.8%.

The concentrations of total, LDL, and HDL cholesterol; triacylglycerol; apo A-I; and apo B after the 3 diets are shown in Table 3. The effect of the isolated genotype could not be seen in any of the variables studied. Changes in diet were associated with significant ($P = 0.001$) decreases in the concentrations of total, LDL, and HDL cholesterol; apo A-I; and apo B after the Cho diet and the MUFA diet, both in volunteers who were homozygous for 1/1 and in the carriers of the minority allele, 1/2, in the exon 1 SNP. Significant differences were not observed in triacylglycerol concentrations after the different diets ($P = 0.437$). In addition, a significant effect on total and LDL-cholesterol plasma concentrations ($P = 0.002$ and $P = 0.023$, respectively) was observed for the interaction between the presence of this mutation and diet (Table 3). Carriers of 1/2 in the exon 1 SNP were observed to have a trend toward significantly ($P < 0.11$) higher concentrations of LDL cholesterol after the SFA diet than were subjects who were homozygous for 1/1. Thus, the decrease in LDL-cholesterol concentrations in the 1/2 subjects was significantly ($P = 0.007$) greater when they changed from an SFA diet to a Cho diet ($−0.58$ mmol/L, $−23\%$) than it was in subjects who were homozygous for 1/1 ($−0.37$ mmol/L, $−16\%$). Likewise, when the SFA diet was compared with the MUFA diet, a significant ($P = 0.035$) difference in these concentrations was seen with a lesser decrease in LDL-cholesterol concentrations for carriers of the 1/1 genotype ($−0.36$ mmol/L, $−14\%$) than for carriers of the 1/2 genotype ($−0.54$ mmol/L, $−20\%$) (Table 3). The greater decrease in LDL-cholesterol concentrations in carriers of the 1/2 genotype were observed in both males and females. There is no significant sex $\times$ diet interaction by genotype. When the MUFA diet was compared with the Cho diet, no differences were found for any of the variables analyzed.

<table>
<thead>
<tr>
<th>TABLE 2</th>
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Daily intake during each experimental dietary period

<table>
<thead>
<tr>
<th></th>
<th>SFA diet</th>
<th>Cho diet</th>
<th>MUFA diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein (% of energy intake)</td>
<td>15</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>Analyzed</td>
<td>18.0</td>
<td>17.5</td>
<td>17.7</td>
</tr>
<tr>
<td>Fat (% of energy intake)</td>
<td>20</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Saturated</td>
<td>22.2</td>
<td>9.1</td>
<td>9.1</td>
</tr>
<tr>
<td>Calculated</td>
<td>12</td>
<td>12</td>
<td>22</td>
</tr>
<tr>
<td>Analyzed</td>
<td>11.1</td>
<td>13.2</td>
<td>24.1</td>
</tr>
<tr>
<td>Monounsaturated</td>
<td>6</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Calculated</td>
<td>5.1</td>
<td>5.2</td>
<td>4.9</td>
</tr>
<tr>
<td>Analyzed</td>
<td>47</td>
<td>57</td>
<td>47</td>
</tr>
<tr>
<td>Carbohydrate (% of energy intake)</td>
<td>44.2</td>
<td>54.5</td>
<td>44.1</td>
</tr>
<tr>
<td>Complex</td>
<td>27.1</td>
<td>33.3</td>
<td>27.5</td>
</tr>
<tr>
<td>Simple</td>
<td>17.1</td>
<td>21.2</td>
<td>16.6</td>
</tr>
<tr>
<td>Cholesterol (mg/d)</td>
<td>285</td>
<td>285</td>
<td>285</td>
</tr>
<tr>
<td>Analyzed</td>
<td>272</td>
<td>275</td>
<td>277</td>
</tr>
<tr>
<td>Fiber (g/d)</td>
<td>30</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>Analyzed</td>
<td>25.8</td>
<td>26.0</td>
<td>24.6</td>
</tr>
<tr>
<td>Energy (MJ)</td>
<td>10.2</td>
<td>10.2</td>
<td>10.2</td>
</tr>
</tbody>
</table>

$^{1}$x, SFA, saturated fatty acids–rich diet; Cho, low-fat, high-carbohydrate diet; MUFA, monounsaturated fatty acid–rich diet.

<table>
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<th>TABLE 3</th>
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INTERACTION OF DIET AND EXON 1 AT THE SRB-I GENE

Our results show that the presence of 1/2 in the exon 1 SNP of the SRB-I gene enhances the response of LDL cholesterol to the presence of SFA in the diet of healthy subjects. Previous studies suggest that one of the most intriguing aspects of the response of
plasma cholesterol to diet is how it varies among individuals. The influence of the genetic loci of the principal apolipoproteins such as the apo A-I–apo CIII–apo A-IV complex, the apo A-I gene, and apo E has been studied. The allelic variables of these genes influence the degree of response by both HDL and LDL cholesterol (23, 24). Thus, the hyperresponse of LDL-cholesterol concentrations associated with the E4 allele occurred only when the fat content in the diet varied, independent of cholesterol content, or when the cholesterol content in the diet was modified (25). Something similar occurs in the present study with carriers of allele 2 in the exon 1 SNP of the SRB-I gene. Recent studies showed that the genetic expression of SRB-I modifies the metabolism of HDL cholesterol in animals. Overexpression in the liver lowers plasma values for HDL cholesterol and increases the concentration and biliary secretion of cholesterol. In contrast, the total suppression of its expression increases plasma HDL cholesterol and lowers the cholesterol content in bile and the suprarenal gland (26, 27). However, lipoprotein metabolism in model animals is different from that in humans. Whereas HDL is the principal circulating lipoprotein in rats, VLDL and LDL lipoproteins play an important role in humans. Some studies have suggested that the SRB-I gene could have an antiatherogenic effect by stimulating plasma clearance of LDL cholesterol. This has been shown in knockout mice fed an atherogenic diet rich in cholesterol, fat, and colic acid, in whom fewer atherosclerotic lesions were seen than were seen in nontransgenic control animals. This reduction was associated with a decrease in LDL-cholesterol concentrations (28, 29). In addition, studies on SRB-I function in cultured cells showed that this receptor can also interact with lipoproteins such as LDL and VLDL and mediate the selective uptake of LDL cholesterol (30–32), which suggests that SRB-I could participate in the metabolism of lipoproteins containing apo B (7). The association between polymorphisms in the SRB-I gene and variations in lipid concentrations in humans has been described, although the cause-and-effect relation is unknown (8). Thus, men who are carriers of I/2 in the exon 1 SNP have an increase in HDL-cholesterol concentrations and a decrease in LDL-cholesterol concentrations. This study was carried out in a population under normal daily conditions without dietary intervention. In our study, subjects were randomly assigned to a dietary intervention study, which means that the results obtained are more reliable. LDL cholesterol was seen to increase when an SFA diet was consumed, although no effects were observed on HDL cholesterol. In accordance with data from previous studies, this would appear to suggest that it is more likely for carriers of I/2 to develop a lipid profile while consuming an SFA–rich diet, regardless of sex. One of the limitations to genetic association studies is the difficulty in corroborating the findings observed in populations with different characteristics. We must be cautious therefore when extrapolating the results to a more general population. We do not know the mechanism of action that explains the effect of fat on SRB-I expression and lipoprotein metabolism. One possibility is that the changes in LDL cholesterol seen in our study may modify the hepatic expression of LDL receptors. This would explain the greater decrease in LDL cholesterol concentrations with a change from SFA in the diet in carriers of I/2 than in subjects who are homozygous for allele 1, as a consequence of changes in the expression of the LDL receptor.

In conclusion, allelic variability in the SRB-I gene could partially explain differences in individual responses to diet. Furthermore, carriers of the minority allele, I/2, are more susceptible to the presence of SFA in the diet because of a greater increase in LDL cholesterol.

We appreciate the assistance of Beatriz Pérez in the translation of the manuscript. PP-M, JMO, JL-M, and FP-J were responsible for the conception and design of the study. RAFeIP, CM, JM, JL-M, PP-M, and FP-J were responsible for the provision of study materials or subjects. PP-M, JM, CM, PG, RAFeIP, and FF were responsible for the collection and assembly of data. PP-M, JL-M, PG, FF, CM, JM, and FP-J were responsible for the analysis and interpretation of the data. JL-M, PG, and FP-J provided statistical expertise. PP-M, JL-M, JMO, RAFeIP, and FP-J were responsible for drafting the manuscript. JL-M, CM, JMO, and FP-J were responsible for the critical review of the manuscript for important intellectual content. PP-M, PG, JMO, FF, JL-M, and FP-J were responsible for final approval of the manuscript. JL-M, CM, and FP-J obtained funding. JM, CM, PG, RAFeIP, and FF provided administrative, technical, or logistic support. None of the authors had any conflict of interest.

REFERENCES


### TABLE 3

Plasma lipids and apolipoproteins (Apo) at the end of each dietary period according to genotype and the percentage change in LDL cholesterol between the saturated fatty acid–rich (SFA) and low-fat, high-carbohydrate (Cho) diets and between the SFA and monounsaturated fatty acid–rich (MUFA) diets by genotype.

<table>
<thead>
<tr>
<th>Genotype and diet</th>
<th>Total</th>
<th>LDL</th>
<th>HDL</th>
<th>TG</th>
<th>Apo A-I</th>
<th>Apo B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I/1 (n = 65)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>SFA</td>
<td>4.14 ± 0.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.52 ± 0.5&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.21 ± 0.2</td>
<td>0.83 ± 0.4</td>
<td>1.33 ± 0.2</td>
<td>0.67 ± 0.1</td>
</tr>
<tr>
<td>Cho</td>
<td>3.67 ± 0.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.15 ± 0.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.12 ± 0.2</td>
<td>0.82 ± 0.4</td>
<td>1.24 ± 0.2</td>
<td>0.58 ± 0.1</td>
</tr>
<tr>
<td>MUFA</td>
<td>3.70 ± 0.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.16 ± 0.5&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.17 ± 0.2</td>
<td>0.76 ± 0.3</td>
<td>1.27 ± 0.2</td>
<td>0.59 ± 0.1</td>
</tr>
<tr>
<td>I/2 (n = 32)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>SFA</td>
<td>4.36 ± 0.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.70 ± 0.6&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.28 ± 0.3</td>
<td>0.80 ± 0.3</td>
<td>1.37 ± 0.2</td>
<td>0.67 ± 0.1</td>
</tr>
<tr>
<td>Cho</td>
<td>3.63 ± 0.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.12 ± 0.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.13 ± 0.2</td>
<td>0.80 ± 0.4</td>
<td>1.26 ± 0.2</td>
<td>0.56 ± 0.1</td>
</tr>
<tr>
<td>MUFA</td>
<td>3.76 ± 0.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.16 ± 0.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.22 ± 0.3</td>
<td>0.79 ± 0.3</td>
<td>1.30 ± 0.2</td>
<td>0.57 ± 0.1</td>
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<tr>
<td>Genotype</td>
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<tr>
<td>Diet</td>
<td>0.001</td>
<td>0.001</td>
<td>0.001</td>
<td>0.437</td>
<td>0.001</td>
<td>0.001</td>
</tr>
<tr>
<td>Interaction</td>
<td>0.002</td>
<td>0.023</td>
<td>0.198</td>
<td>0.710</td>
<td>0.631</td>
<td>0.222</td>
</tr>
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</table>

<sup>a</sup> ± SD; percentage change in brackets. I/1 represents homozygosity for the most common allele; I/2 represents heterozygosity for the less common allele. TG, triacylglycerol. Values within a genotype with different superscript letters are significantly different, P < 0.05 (repeated-measures ANOVA).
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