Original

Serum alkaline phosphatase, body composition, and risk of metabolic syndrome in middle-aged Korean

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Abstract. Some papers have suggested that alkaline phosphatase (ALP) level is a predictor of the metabolic syndrome (MetS) in the general population. However, the association is still controversial, and the mechanisms underlying an association between ALP level and the MetS have not been elucidated. We analyzed the association between serum ALP level and the development of the MetS over a 4-year period. A total of 14,224 subjects who visited the Health Promotion Center for a medical examination in 2005 were followed up after 4 years. Serum ALP level correlated positively with body fat mass and visceral fat mass. The adjusted geometric mean ALP levels were higher in subjects with elevated C-reactive protein level or greater fat mass (P < 0.001). None of the subjects had the MetS at baseline, but 1,179 exhibited the MetS at the 4-year follow-up. After multiple adjustments, the odds ratio (OR) was substantially higher for development of the MetS (OR 1.56, 95% confidence intervals, 1.21-2.01) in subjects in the highest ALP quintile compared with those in the lowest quintile. After adjusting for various covariates, we found significant associations between the quintile of serum ALP level and abdominal obesity, low high-density lipoprotein cholesterol level, and high triglyceride level. Higher serum ALP level was a significant predictor of the MetS in middle-aged Koreans. Serum ALP level correlated positively with body fat mass and independently with a more atherogenic lipid profile in the general population in Korea.

Key words: Alkaline phosphatase, Metabolic syndrome, Body composition, Inflammation

Alkaline phosphatase (ALP) is an enzyme that catalyzes the hydrolysis of organic pyrophosphate, an inhibitor of vascular calcification [1]. Although ALP is expressed in a variety of tissues, its concentration is highest in bone, the liver, and the kidneys. Pathological causes of increased serum ALP level include bone disease and liver disease [2]. Recent data suggest that elevated serum ALP level is associated with increased mortality in the general population [3], survivors of myocardial infarction [2], ischemic or hemorrhagic stroke patients [4], chronic kidney disease (CKD) patients not on dialysis [5], and CKD patients on maintenance hemodialysis [6]. It is thought that, in dialysis patients, the association reflects abnormal bone metabolism, perhaps mediated by vascular calcification, which is common in dialysis patients [1]. However, the mechanism underlying this association is not understood well, especially in the general population [3].

Vascular inflammation has emerged as central to the initiation and progression of atherosclerosis [7]. Obesity and the metabolic syndrome (MetS) are associated with a chronic inflammatory response, which is characterized by abnormal cytokine production, increased acute-phase reactants, and activation of inflammatory signaling pathways. Elevated ALP level is associated with higher C-reactive protein (CRP) concentration [7], the most extensively studied biomarker of low-grade inflammation. Some reports have suggested that ALP level is a predictor of the MetS in the general population, although the association is still controversial [8-10]. Moreover, the mechanisms underlying an association between ALP level and the MetS have not been elucidated.

The aim of our study was to determine whether higher ALP level is associated with an increased risk of
the MetS in the general population. We analyzed the association between serum ALP level and the development of the MetS over a 4-year period in patients within a follow-up cohort selected from a health checkup program in a single health promotion center in Korea. We also investigated the association between body composition and serum ALP level in this study’s population.

**Methods**

**Subjects**

Among subjects who underwent a health checkup at the Health Promotion Center at Kangbuk Samsung Hospital, Seoul, South Korea, in 2005, 16,706 subjects were included in this study at the baseline. The participants were employees of the company or their family members whose health checkup was performed in the health promotion center. At baseline, 2,482 subjects were excluded from enrollment: those who had the MetS at baseline (n = 2,437; see below for definition) or those who had renal failure (serum creatinine concentration > 1.4 mg/dL; n = 45). A total of 14,224 participants were enrolled, and they repeated their health checkup in 2009. The study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori approval by the institution’s Human Research Committee, and all patients provided written informed consent.

**Measurements**

All participants were required to fast for 12 h before undergoing a physical examination by trained staff and physicians using standard protocols. Body weight and height were measured with the subject barefoot and wearing light clothing, and the values were used to calculate body mass index (BMI). Waist circumference was measured with the subject in the standing position and at the level of the umbilicus by a single examiner. Blood pressure was measured twice in seated subjects after a 5 min rest using a mercury sphygmomanometer according to the Hypertension Detection and Follow-up Program protocol. The mean of these measurements was used in the analyses. The presence of fatty liver was defined as abnormal hepatic features seen by ultrasonography (Logic Q700 MR; GE, Milwaukee, WI, USA) suggestive of fatty infiltration using standardized criteria [11].

Body composition was measured using segmental bioelectric impedance analysis with eight tactile electrodes according to the manufacturer’s instructions (InBody 3.0; Biospace, Seoul, Korea). Lean mass (kg), fat mass (kg), percent fat mass (%), and waist-to-hip ratio (WHR), a marker of visceral obesity, were measured.

Serum levels of glucose, total cholesterol, triglycerides, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, gamma-glutamyl transferase (GGT), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and ALP were measured using Bayer Reagent Packs on an automated chemistry analyzer (Advia 1650 Autoanalyzer; Bayer Diagnostics, Leverkusen, Germany; ALP normal adult range 45 to 129 IU/L). High sensitivity C-reactive protein (hsCRP) concentration was measured by particle-enhanced immunonephelometry with the BN II System (Dade Behring, Marburg, Germany). Apolipoprotein B and apolipoprotein AI concentrations were measured with the nephelometric method using a BN II system (Dade Behring Co., Marburg, Germany).

Serum insulin concentration was measured using an immunoradiometric assay (INS-IRMA; BioSource, Nivelles, Belgium). The homeostasis model assessment of insulin resistance (HOMA-IR) was calculated using the formula: fasting plasma glucose (mmol/L) × fasting insulin (mIU/L)/22.5 [12].

**Definition of metabolic syndrome**

For the definition of MetS, we adopted the 2005 revision of the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III) criteria and the Asian specific cutoff point for abdominal obesity, as recommended [13]. MetS was diagnosed when the subject had three or more of the following criteria: 1) a waist circumference ≥ 90 cm in men and ≥ 80 cm in women (International Obesity Task Force criteria for the Asia-Pacific population were used to determine waist circumference criteria); 2) SBPs and/or DBPs ≥ 130/85 mmHg or being on antihypertensive drug treatment in a patient with a history of hypertension; 3) fasting glucose ≥ 100 mg/dL or if on drug treatment for elevated glucose; 4) fasting serum triglycerides ≥ 150 mg/dL; and 5) HDL-cholesterol < 40 mg/dL in men and < 50 mg/dL in women.

**Statistical analysis**

All data were analyzed using the SPSS statistical package (SPSS Inc., Chicago, IL, USA). Data are presented as the mean ± SD unless otherwise stated. If
necessary, logarithmic transformation was performed to achieve a normal distribution. Spearman correlation coefficients were determined for bivariate associations of ALP with other covariates. Participant characteristics were compared according to the MetS status using independent sample Student’s t tests for continuous measures and χ² tests for categorical measures. Participants were classified into five groups according to serum ALP concentration quintiles. Analysis of covariance was used to determine the association of ALP with MetS components after adjustment for potential confounders. A Tukey test was then used to analyze the data post hoc. The relationships between the development of the MetS and ALP concentration after 4 years were analyzed by logistic regression analyses after adjusting for confounding factors. P < 0.05 was accepted as significant.

Results

General characteristics of the study population

The mean age of the study participants was 41.5 years. Of the 14,224 subjects who did not have the MetS at baseline, 1,179 were found to have the MetS at the 4-year follow-up. The mean ALP level was significantly higher in those with the MetS (60.8 ± 16.2 vs. 64.7 ± 18.0 IU/L, P < 0.001; Table 1). Serum calcium level was significantly higher in subjects with the MetS than in those without (9.33 ± 0.35 vs. 9.37 ± 0.36 mg/dL, P < 0.001). Serum phosphorus levels did not differ significantly between the two groups (3.46 ± 0.44 vs. 3.44 ± 0.43 mg/dL, P = 0.178).

Association between serum ALP concentration and body composition and metabolic parameters

Partial Spearman correlation analysis showed that ALP correlated weakly but significantly with waist circumference (r = 0.079), body fat mass (r = 0.113), WHR (r = 0.109), HOMA-IR (r = 0.045), and the concentrations of hsCRP (r = 0.109), fasting blood glucose (r = 0.039), triglycerides (r = 0.068), and apolipoprotein B (r = 0.073) (all P < 0.001). ALP correlated negatively with lean mass (r = -0.083), HDL cholesterol level (r = -0.049), and apolipoprotein A1 level (r = -0.036) (all P < 0.001). All correlation coefficients were calculated after correcting for age, sex, and BMI.

We examined the combined effects of body composition and serum hsCRP level on serum ALP level by creating a four-category variable. Percent fat mass and serum hsCRP level were designated as high or low based upon the median levels. Crude, age-, sex-, and BMI-adjusted geometric means of serum ALP levels were calculated for each of the four categories, and differences between categories were tested using analysis of covariance. Serum ALP levels were highest among subjects with high hsCRP level and fat mass.

### Table 1 Baseline characteristics of participants according to the subsequent development of metabolic syndrome

<table>
<thead>
<tr>
<th></th>
<th>MetS (-) (n=13,045)</th>
<th>MetS (+) (n=1,179)</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ALP (IU/L)</td>
<td>60.8±16.2</td>
<td>64.7±18.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Age (years)</td>
<td>41.9±7.2</td>
<td>43.9±8.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>8,491/4,582</td>
<td>868/318</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>22.9±2.7</td>
<td>25.4±2.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Waist Circumference (cm)</td>
<td>78.1±8.7</td>
<td>85.0±8.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>eGFR (mL/min/1.73m²)</td>
<td>80.8±14.5</td>
<td>87.5±17.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Serum calcium (mg/dL)</td>
<td>9.33±0.35</td>
<td>9.37±0.36</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Serum phosphorus (mg/dL)</td>
<td>3.46±0.44</td>
<td>3.44±0.43</td>
<td>0.178</td>
</tr>
<tr>
<td>Serum AST (IU/L)</td>
<td>23.5±13.3</td>
<td>26.0±12.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Serum ALT (IU/L)</td>
<td>24.2±21.0</td>
<td>30.6±18.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Serum GGT (IU/L)</td>
<td>28.7±30.5</td>
<td>44.0±45.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Fasting glucose (mg/dL)</td>
<td>94.2±11.0</td>
<td>100.7±17.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>2.0±0.8</td>
<td>2.5±0.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Serum C-reactive protein (mg/L)</td>
<td>0.13±0.37</td>
<td>0.20±0.49</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

MetS, metabolic syndrome; ALP, alkaline phosphatase; BMI, body mass index; eGFR, estimated glomerular filtration rate; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, γ-glutamyl transferase; HOMA-IR, homeostasis model assessment-insulin resistance
Kim et al. (64.4-66.1) in subjects with high hsCRP and the presence of fatty liver (P for trend < 0.001).

Serum ALP level and 4-year incidence of the MetS
Serum ALP level showed a significant positive relationship with incident MetS. Multiple logistic regression analyses revealed a significant association of ALP with the incidence of the MetS (Q1 reference; Q5 odds ratios, OR 1.56; 95% CI, 1.21–2.01, P for trend = 0.007; model 3, Table 2). Further adjustment for ALT, GGT, and hsCRP levels, and HOMA-IR (model 6) reduced the magnitude of the ORs for the MetS, but they remained significant (OR 1.40; 95% CI, 1.05-1.87).

After adjustment for age, sex, and BMI, compared with the subjects in Q1, those in Q5 had an OR of 1.25 (95% CI, 1.06-1.49; P = 0.010) for abdominal obesity, 1.50 (95% CI, 1.26–1.78; P < 0.001) for low HDL

Table 2 Unadjusted prevalence and adjusted odds ratios (95% CI) of incident metabolic syndrome by quintiles of serum alkaline phosphatase (ALP) at baseline

<table>
<thead>
<tr>
<th>Quintiles of ALP (IU/L)</th>
<th>Q1 (≤47)</th>
<th>Q2(48-55)</th>
<th>Q3(56-63)</th>
<th>Q4(64-72)</th>
<th>Q5(≥73)</th>
<th>P for trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>2692</td>
<td>3008</td>
<td>3036</td>
<td>2582</td>
<td>2906</td>
<td></td>
</tr>
<tr>
<td>Unadjusted prevalence</td>
<td>5.9 %</td>
<td>7.5 %</td>
<td>9.5 %</td>
<td>10.8 %</td>
<td>&lt;0.001</td>
<td></td>
</tr>
<tr>
<td>Model 1</td>
<td>1(ref)</td>
<td>1.21 (0.98, 1.50)</td>
<td>1.22 (0.99,1.52)</td>
<td>1.48 (1.19,1.84)</td>
<td>1.64 (1.33, 2.02)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Model 2</td>
<td>1(ref)</td>
<td>1.13 (091, 1.41)</td>
<td>1.10 (0.89,1.38)</td>
<td>1.26 (1.01,1.58)</td>
<td>1.41 (1.14, 1.75)</td>
<td>0.010</td>
</tr>
<tr>
<td>Model 3</td>
<td>1(ref)</td>
<td>1.21 (093, 1.57)</td>
<td>1.18 (0.91,1.53)</td>
<td>1.34 (1.03,1.75)</td>
<td>1.56 (1.21, 2.01)</td>
<td>0.007</td>
</tr>
<tr>
<td>Model 4</td>
<td>1(ref)</td>
<td>1.21 (0.93, 1.57)</td>
<td>1.15 (0.88,1.49)</td>
<td>1.31 (1.01,1.71)</td>
<td>1.51 (1.17, 1.95)</td>
<td>0.016</td>
</tr>
<tr>
<td>Model 5</td>
<td>1(ref)</td>
<td>1.07 (0.80, 1.44)</td>
<td>1.16 (0.87,1.55)</td>
<td>1.27 (0.95,1.71)</td>
<td>1.46 (1.10, 1.94)</td>
<td>0.047</td>
</tr>
<tr>
<td>Model 6</td>
<td>1(ref)</td>
<td>1.09 (0.81, 1.46)</td>
<td>1.15 (0.86,1.54)</td>
<td>1.23 (0.91,1.66)</td>
<td>1.40 (1.05, 1.87)</td>
<td>0.148</td>
</tr>
</tbody>
</table>

Data represent %. Model 1; adjusted for age and sex   Model 2; adjusted for age, sex and BMI   Model 3; adjusted for age, sex, BMI, serum calcium, serum phosphorus, and eGFR   Model 4; adjusted for age, sex, BMI, serum calcium, serum phosphorus, eGFR, and HOMA-IR   Model 5; adjusted for age, sex, BMI, serum calcium, serum phosphorus, eGFR, and hsCRP   Model 6; adjusted for age, sex, BMI, serum calcium, serum phosphorus, eGFR, HOMA-IR, hsCRP, and serum alanine transaminase and GGT.

Fig. 1  Adjusted geometric means of serum ALP levels according to serum hsCRP level and body fat mass (A) or the presence of fatty liver (B).

Fig. 1A. The adjusted geometric mean ALP was 58.2 IU/L (95% confidence intervals,CI, 57.5-58.8) in subjects with low hsCRP and fat mass; 59.8 IU/L (95% CI, 59.0-60.5) in subjects with low hsCRP and high fat mass; 63.0 IU/L (95% CI, 62.3-63.7) in subjects with high hsCRP and low fat mass; and 65.0 IU/L (95% CI, 64.4-65.7) in subjects with high hsCRP and fat mass (P for trend < 0.001).

We also examined the combined effects of the presence of fatty liver and hsCRP level on serum ALP levels by creating a four-category variable (Fig. 1B). The adjusted geometric mean ALP was 58.3 IU/L (95% CI, 57.8-58.8) in subjects with low hsCRP and the absence of fatty liver; 60.7 IU/L (95% CI, 59.4-62.0) in subjects with low hsCRP and the presence of fatty liver; 63.3 IU/L (95% CI, 62.8-63.9) in subjects with high hsCRP and the absence of fatty liver; and 65.2 IU/L (95% CI, 64.4-66.1) in subjects with high hsCRP and the presence of fatty liver (P for trend < 0.001).
level, and 1.43 (95% CI, 1.25-1.65; \( P < 0.001 \)) for hypertriglyceridemia (Table 3). The quintiles of serum ALP level did not correlate significantly with hyperglycemia or high blood pressure.

Mean ALP levels increased significantly as the number of MetS components increased (\( P < 0.001 \); Fig. 2). The post hoc analysis showed that subjects without any MetS components had significantly lower mean serum ALP levels compared with subjects with one or more MetS components (\( P < 0.001 \); Fig. 2). These relationships remained significant after adjusting for age, sex, and BMI (\( P < 0.001 \)).

### Prevalence of and risk for the MetS according to serum ALP level in subjects with normal liver transaminase levels

After excluding the subjects with abnormal AST or ALT levels (\( n = 874 \)), the adjusted ORs for having the MetS in the first quintile compared with the other four quintiles of serum ALP level were 1.29 for quintile 2 (95% CI: 0.98-1.71), 1.13 for quintile 3 (0.85-1.48), 1.38 for quintile 4 (1.05-1.81), and 1.58 for quintile 5 (1.21-2.06) (\( P \) for trend = 0.006). Among the components, there were significant associations between the quartile of serum ALP concentration and abdominal obesity (\( P = 0.022 \)), low HDL concentration (\( P < 0.001 \)), and hypertriglyceridemia (\( P = 0.001 \)) after adjusting for age, sex, and BMI; serum concentrations of calcium, phosphorus, ALT, GGT, and hsCRP; estimated glomerular filtration rate (eGFR); and HOMA-IR.

### Discussion

We found that higher total (tissue-nonspecific) ALP level was a significant predictor of the MetS in middle-aged Koreans. This association was significant even after adjusting for baseline ALT and GGT levels, markers of liver cell damage. Excluding the subjects with abnormal liver function tests did not alter this significant association. Our data suggest that independent of liver dysfunction, higher ALP level is associated with
the MetS in middle-aged people.

The mechanisms underlying the association of ALP level with the MetS have not been elucidated, but there are at least two possible mechanisms: ALP is a marker of visceral obesity or hepatic steatosis (fatty liver), and ALP is a marker of subclinical inflammation. ALP activity is present in the human preadipocyte population that resides within adipose tissue, and its enzyme activity increases during adipogenesis [14]. Intracellular lipid accumulation and ALP activity increase in parallel during adipogenesis in human preadipocytes. The treatment of preadipocytes with inhibitors of tissue-nonspecific ALP blocks lipid accumulation [15]. The identification of ALP in human preadipocytes suggests that adipose tissue may also contribute to serum ALP level. The relationship between measures of body fat mass and serum ALP concentration has not been analyzed specifically, and the possible contribution of adipose-derived ALP to total serum ALP concentration is not known [14]. Our study showed that serum ALP levels correlated positively with body fat mass and visceral fat mass (as indicated by the WHR). ALP levels predicted serum concentrations of triglycerides (positively) and HDL-C (negatively) independent of age, sex, and BMI, suggesting an atherogenic characteristic of ALP. Inflammation might also be an explanation for the association of elevated ALP with the MetS and mortality. Higher ALP level may be a marker for an underlying subclinical inflammatory state.

Another possible explanation for the observed association in our study is a link between higher ALP level and lower vitamin D levels, which themselves are associated with the MetS and mortality [2]. Higher ALP level increases the hydrolysis of pyrophosphate, a potent inhibitor of vascular calcification [1]. The balance of inorganic pyrophosphate to serum phosphate regulates vascular calcification. There are excess risks associated with higher ALP levels, and a higher serum phosphate level is additive [2]. Fibroblast growth factor-23 (FGF-23), which regulates phosphate balance and vitamin D metabolism, is associated with higher mortality in patients with CKD [16]. A recent study suggested that, in addition to its key role in the pathogenesis of calcium–phosphorus disorders, FGF-23 may also be involved in many other metabolic processes [17]. There are complex interactions between higher ALP level, phosphate homeostasis, increased FGF-23 and parathyroid hormone levels, and decreased vitamin D levels.

In CKD patients without liver disease, ALP can be elevated in high-turnover bone disease [18]. Higher ALP level is associated with mortality and coronary artery calcification in CKD patients [19] and in patients without CKD [2]. Similar findings were reported for bone-specific ALP in a small study of patients with CKD stages 1-5 [18]. However, in renal transplant recipients, ALP is associated with the MetS and all-cause and cardiovascular mortality [20]. In separate analyses of the bone and nonbone parts of ALP activity, the association with all-cause and cardiovascular mortality was present for only nonbone ALP; nonbone ALP is usually considered equivalent to liver ALP [20]. According to the study about the relationship between anthropometry and serum concentrations of ALP isoenzymes [21], total and liver ALP serum levels are higher in obese than in lean subjects. It is possible that the higher level of liver ALP in obese than in lean subjects is a result of ALP release from adipose tissue. Obesity is associated with hyperparathyroidism and increased bone turnover. Severely obese women had higher parathyroid hormone, FGF-23, and bone ALP and lower vitamin D levels than control. Bone turnover markers are increased in obesity [22]. The most significant difference between severely obese women and controls was found in the bone formation marker bone ALP [22]. In our study, the association between ALP and the MetS persisted after adjusting for liver enzyme levels and was similar after excluding the subjects with abnormal liver enzyme levels, indicating an effect that was independent of the hepatic pathology. Moreover, the putative mechanism of action whereby ALP could be instrumental in causing vascular calcification is the same irrespective of the source of ALP [20].

Our findings are consistent with earlier findings showing that higher ALP levels predicted cardiovascular disease, mortality, and development of the MetS in the general population. Higher serum ALP levels are strongly associated with increased prevalence of the MetS and a subsequent increase in all-cause mortality in the US general population [3]. However, in that study, the association between serum ALP and mortality persisted after adjusting for the MetS and other covariates. The strongest association between serum ALP level and mortality was observed in those with none of the MetS components. The authors concluded that the MetS does not appear to explain the increased mortality risk associated with higher serum ALP level [3]. In our study, higher ALP level was a significant predic-
tor of the MetS. Abdominal obesity and high triglyceride and low HDL levels were the strongest contributors to this relationship. This suggests that the MetS might be the link between higher serum ALP levels and increased mortality in the general population.

Our study had several limitations. First, associations of serum ALP with body composition and metabolic parameters were weak. However, our analyses took into account many potential covariates that might confound the observed associations. The persistence of the serum ALP-MetS relationship after adjustment for multiple potential confounders indicates that serum ALP can be viewed as an independent predictor of MetS in middle-aged Korean. Second, data on serum vitamin D levels were not available. And most (88%) of our participants were younger than 50 years, and all of the participants were volunteers or employees required to undertake a comprehensive health examination. Thus, our study subjects do not represent the Korean population as a whole.

In conclusion, the present study suggests that higher serum ALP level is strongly associated with development of the MetS in healthy adults. Serum ALP level correlated positively with body fat mass and was independently associated with a more atherogenic lipid profile. Because a moderately high ALP level does not usually trigger a change in routine patient care, the ALP level is likely to represent a less biased tool for assessing risk of the MetS in epidemiological studies [20]. This study provides additional support for trials of therapies to lower ALP levels. Future studies examining interventions targeting ALP directly are needed to delineate the complex actions of this molecule.

Conflict of Interests

The authors have no financial conflicts or anything of interest to disclose.

References


