In addition, nascent VLDL is primarily released by the liver and becomes intermediate-density lipoprotein, LDL, and VLDL remnants in blood circulation accompanying the concentrated cholesterol contents. To our knowledge, the effect of calcium supplementation on VLDL synthesis and decomposition in postmenopausal women is still unclear. According to our data, an alternative possibility is that calcium supplementation affects the metabolism of VLDL cholesterol in postmenopausal women, which needs more evidence in future study.

Beyond this, we apologize for the typographical errors in the article. The word “estrol” in Table 1 should be “estradiol.” The statement in the Results section (page 1355) that “Serum estradiol concentrations in postmenopausal women were significantly higher than those in premenopausal women (147.44 ± 28.29 compared with 33.08 ± 8.41 ng/L; \( P < 0.01 \))” should be corrected to “Serum estradiol concentrations in premenopausal women were significantly higher than those in postmenopausal women (147.44 ± 28.29 compared with 33.08 ± 8.41 ng/L; \( P < 0.01 \)).”

Neither of the authors had a conflict of interest.

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**REFERENCES**


Association between plasma phospholipids and insulin-related variables with special reference to statistical validity

Dear Sir:

In a recent study, “Specific plasma lipid classes and phospholipid fatty acids indicative of dairy food consumption associate with insulin sensitivity,” Nestel et al (1) reported that 3 indicators of insulin resistance were inversely associated with specific plasma phospholipids, especially with the adjustment of waist-hip ratio as an obesity index. They conducted a cross-sectional study in 86 overweight or obese nonsmokers without diabetes mellitus. I have some concerns about their statistical validation.

First, the authors used the Matsuda insulin sensitivity index (ISI) in combination with 3 variables of insulin resistance, such as fasting plasma insulin, HOMA-IR, and plasma insulin with AUC (AUC\(_{0-120}\). The Matsuda ISI and AUC\(_{0-120}\) were calculated by using a standard oral-glucose-tolerance test. The Quantitative Insulin Sensitivity Check Index (QUICKI) is a marker of insulin sensitivity, which can be calculated by fasting plasma glucose and insulin. It is clear that there is no different meaning between HOMA-IR and QUICKI from the definition of each indicator (2, 3). Because the HOMA-IR and the Matsuda ISI were highly correlated in subjects with a wide variety of glucose tolerance (4), there is no advantage of using the Matsuda ISI as an ISI instead of the QUICKI from the viewpoint of simplicity.

Second, I suppose that the Matsuda ISI shows a normal distribution after logarithmic transformation, which is the same distribution with fasting plasma insulin and HOMA-IR. Independent and dependent variables for multivariable regression analysis should be converted as they become normal distributions, and I recommend presenting the transformation procedure in the authors’ “Statistical analysis” section.

As a third concern, the authors presented \(\beta\)-coefficients and 95% CIs for each insulin-related biomarker in their tables. I assume that these values are nonstandardized. I would recommend presenting standardized regression coefficients for the purposes of multiple comparisons.

Finally, coefficients of determination, calculated by the square value of multiple regression coefficients, should be presented in the authors’ tables, because lipid profiles are regulated by several factors other than insulin-related biomarkers.

Although a cause-effect relation between insulin-related biomarkers and phospholipid class or fatty acids can be determined by longitudinal follow-up study, cross-sectional study also presents valuable information if adequate statistical procedures can be applied. There is a recent report that sex is related to insulin sensitivity (5), and further study is required to confirm the association.

The author had no conflicts of interest to disclose.

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Note: The authors of the original article chose not to submit a reply.

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Erratum


Two errors appear in the article. In Table 1 (page 1356), the word esterol should be estradiol. In the Results section on page 1355, the second sentence under “Descriptive and biochemical characteristics and dietary intakes” should read as follows: “Serum estradiol concentrations in premenopausal women were significantly higher than those in postmenopausal women (147.44 ± 28.29 compared with 33.08 ± 8.41 ng/L; P < 0.01).”


Erratum


The references in the original online supplemental data were incorrect. A corrected version of the online supplemental data has been posted.