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Dear Editor

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We thank Dr. Eyüboğlu for his interest in our article entitled "*Intake of polyunsaturated fat in relation to mortality among statin users and non-users in the Southern Community Cohort Study*" [1]. In his letter to the editor [2], Dr. Eyüboğlu wondered whether use of angiotensin converting enzyme inhibitors (ACEI) and angiotensin receptor blockers (ARBs), known to lower all-cause mortality [3], could explain the inverse association we observed between intake of polyunsaturated fatty acids (PUFA) and all-cause mortality among non-statin users. While we agree with Dr. Eyüboğlu that ACEI and ARBs lower all-cause mortality, we do not believe that these medications explain the inverse association we observed between intake of PUFA and all-cause mortality. In order for ACEI or ARBs to confound the association between PUFA and all-cause mortality, use of these medications must be simultaneously associated with intake of PUFA and all-cause mortality.

In our cohort, collection of data on use of blood pressure medications was initiated after enrollment had already began and medications have been categorized only for participants recruited from Community Health Centers. Thus, such data are only available for 27,976 participants who also have data on PUFA intake. Although we do not have data on use of blood pressure medications for the whole cohort, we explored whether use of ACEI or ARBs explains the inverse association between PUFA intake and all-cause mortality in the subset with medication use data. Use of ARBs was not associated with PUFA intake (P = 0.73) and thus cannot be a confounder. The proportions of ACEI users across quintiles of PUFA were 10.5%, 11.7%, 11.5%, 11.9% and 13.0% for the 1st (lowest), 2nd, 3rd, 4th and 5th quintile (highest) of PUFA. While use of ACEI was significantly associated with PUFA intake (P = 0.01), the differences in frequency of ACEI use across quintiles of PUFA were very small (e.g., 2.5% for the highest versus lowest quintile), suggesting that use of ACEI is

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unlikely to explain the 10-20% reduction in all-cause mortality we observed for total, n3- and n6-PUFA. Indeed when our final models, albeit with smaller sample sizes, were adjusted for ACEI use, the inverse associations remained. For instance, the hazard ratio (95% confidence intervals) for all-cause mortality when comparing the highest to the lowest quintile of n6-PUFA in the subset with medication data (n=27,976) was 0.83 (0.64, 1.07) in the fully-adjusted model and remained unchanged (HR=0.83; 95% CI: 0.64, 1.07) after additionally adjusting for ACEI use. While the association was slightly attenuated and the confidence interval widened in this smaller sample, the hazard ratio remained close to that (HR=0.80; 95% CI: 0.70-0.92) reported in the article when a larger sample size (n=69,559) was used; suggesting the observed inverse association for PUFA cannot be explained by use of ACEI or ARBs. Further adjustment for diuretics, a fairly common class of medications that showed an association with PUFA intake, did not abrogate the association either (HR=0.82; 95% CI: 0.64-1.07).

Taken together, our data are consistent with an independent protective effect of PUFA on all-cause mortality in individuals not taking statins.

Thank you.

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