RE: Plasma Phospholipid Fatty Acids and Prostate Cancer Risk in the SELECT Trial

Brasky and colleagues (1) report that, within the Selenium and Vitamin E Cancer Prevention Trial, plasma phospholipid levels of long-chain omega-3 fatty acids measured in blood samples collected at baseline are positively associated with subsequent risk for low-grade, high-grade, and total prostate cancer (1). They then perform a meta-analysis incorporating previous pertinent prospective case-control studies that appears to sustain this conclusion. In their discussion, the authors indicate that their findings strongly suggest that long-chain omega-3s play a causative role in prostate cancer induction and caution that “general recommendations to increase long-chain ω-3 PUFA intake should consider its potential risks” (1). Subsequent media coverage of this study has created the impression that dietary omega-3 fatty acids may be dangerous.

Whether a positive correlation between plasma omega-3 and prostate cancer risk will be sustained in future research remains to be seen. As the authors note, previous pertinent studies have had variable results. In fact, a meta-analysis published earlier this year—before the availability of Brasky et al.’s (1) new data—concluded that plasma omega-3 levels are not associated with risk for total or advanced prostate cancer, aside from an inverse association with docosapentaenoic acid (2). (A positive association between omega-3 status and risk for advanced prostate cancer emerged only after results from the Physician’s Health Study were excluded.)

Furthermore, even if subsequent research does confirm a positive correlation between plasma omega-3s and prostate cancer risk, it will not be clear that this association is causative. Arguably, some peculiarity of metabolism might affect the distribution and oxidation of omega-3s while concurrently modulating prostate cancer risk. The notion that long-chain omega-3s play a pathogenic role in prostate cancer induction is not supported by the many studies that have analyzed fish ingestion and prostate cancer risk. In a recent meta-analysis of these studies, Szymanski et al. observed a modest inverse association between fish consumption and prostate cancer risk in case-control studies (odds ratio = 0.85; 95% confidence interval [CI] = 0.72 to 1.00; P = .05), albeit no association was observed in cohort studies; in the four included studies that reported prostate cancer-specific mortality, fish consumption was linked to a strong reduction in this mortality (relative risk = 0.37; 95% CI = 0.18 to 0.74; P = .005) (3). Moreover, epidemiology also suggests that frequent fish consumption improves survival in men who already have prostate cancer (4,5). And diets enriched in fish oil, or in the terrestrial omega-3 stearidonic acid (readily converted to eicosapentaenoic acid within the body), have slowed the growth of human prostate cancers in nude mice (6).

The traditional diets of the Japanese and of Inuits are notable for their exceptionally high omega-3 content; in past decades, the traditional diets of the Japanese and of Inuits are notable for their exceptionally high omega-3 content; in past decades, these peoples has been very low compared Western rates (6)

In light of the physiological role that myocardial omega-3s play in the prevention of arrhythmias and of suggestive evidence that replete omega-3 status may slow age-related cognitive decline, it is inappropriate to suggest that these fatty acids may increase prostate cancer risk while failing to cite the considerable evidence that appears to contradict this position.

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References

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