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[The dangers of over-analyzing too much data in prostate study](#)

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In the last week there has been a constant buzz about an online pre-publication of a new research article that **suggests that high concentrations of omega-3 fatty acids promote aggressive prostate cancer (1)**. **Well, that really isn't the case**, in spite of the press reports. That's why **you have to carefully read the article before jumping to conclusions**.

Prostate cancer, like all cancers, is driven by cellular inflammation. The level of cellular inflammation is defined by the AA/EPA ratio of isolated serum phospholipids. When you analyze the data correctly in that article, you find that there was no difference in the AA/EPA ratio between the low-aggressive, high-aggressive, or control group. In fact, all the groups had the same elevated AA/EPA ratio of 18.8. Since I like to have individuals try to maintain an AA/EPA ratio of less than 3, all of these groups could be considered to be inflamed.

Not surprisingly, when you look at either EPA or AA levels separately in each group, they are identical. It's only when you look at the DHA levels, do you see a small difference statistically, but it's meaningless clinically. There was a 2.5 percent increase in the DHA levels in the high-aggressive group compared to the control group. In the paper, authors state their error in measuring DHA is ± 2.4 percent. Call me crazy, but I don't see the big difference between the reported results and their error measurements. To further cloud the results, the authors also find that the levels of trans-fatty acids are lower in the aggressive prostate cancer patients than the controls. So I guess if you wanted to take their data at face value, DHA makes prostate cancer more aggressive and trans-fatty acids found in junk foods make prostate cancer less aggressive.

I believe this is simply a case of over-interpretation of massive amounts of collected data. If you get enough data points, you can always make some type of correlation, but that's all it is. At some point you also have to allow common sense to enter the final analysis.

Nonetheless, let's say their data might be correct. How could excess DHA increase the aggressiveness of any cancer? Well, it might decrease the levels of dihomo gamma linolenic acid (DGLA) as I have explained in many of my books (2-5). DGLA is the building block for a powerful group of anti-inflammatory eicosanoids, and its formation is inhibited by DHA. Depressing DGLA levels would reduce the body's ability to hold back the inflammation that drives the tumor. Unfortunately, with all the data they accumulated, they forgot to publish the changes in the DGLA levels in the various groups. Oops.

So even if there were not any changes in the AA/EPA ratio between groups, a depression of DGLA levels in the aggressive prostate cancer group would easily explain the clinical observation. Unfortunately, that interpretation requires an extensive background in understanding eicosanoid biochemistry, which is not easily found in academic clinical-research centers.

This is not the first time that the potential benefits of DHA are in question. In the largest cardiovascular intervention study ever done, it was demonstrated that adding high levels of EPA to the diet of Japanese patients with high cholesterol levels (who already with a very low AA/EPA ratio of 1.6), dramatically decreased their likelihood of future

cardiovascular events (6). This reduction was only correlated with increases in EPA levels as well as with a decrease in the AA/EPA ratio from an already low 1.6 to an even lower 0.8 (7). The levels of DHA in these patients had no significance for predicting future cardiovascular events.

Likewise other studies using DHA alone to treatment post-partum depression, improve neurological functioning of children or treating Alzheimer's have also been found to be negative (8,9).

It's not that DHA is bad, it just doesn't do much to reduce cellular inflammation. DHA does a lot of other useful things, but reducing cellular inflammation is not one of them.

References

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