The Omega-3 Index and Inflammation: Results from the Framingham Study

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Omega-3 fatty acids are often referred to as "anti-inflammatory" fatty acids. The studies in animals are convincing but measuring inflammation in humans in notoriously difficult. Here we review a paper (Fontes JD, et al. 2015, Atherosclerosis) that was recently published by Dr. Bill Harris, the President and CEO of OmegaQuant, and colleagues from Boston University. It compares the components of the Omega-3 Index, omega-3 fatty acids EPA and DHA in red blood cells, to the blood levels of 10 inflammatory markers in over 2500 people. Below are some of the important points of the study:

- **Using the iconic Framingham Cohort**: This study uses samples from the Framingham cohort. This is an iconic study population that began in 1948 in the city of Framingham, Massachusetts (a suburb of Boston). It was one of the first studies to follow thousands of people for years and measure both blood and lifestyle factors to try to figure out what caused heart attacks. Now the children of the original cohort are participating in a second study called the Framingham Offspring study. Also, the researchers have since recruited a more diverse group of people to participate called the Framingham Omni cohort. For this study, samples from 2724 participants from both cohorts (mean age 66 $\pm$ 9 years; 54% women and 8% minorities) that were collected between 2005-2008 were analyzed for fatty acids and inflammatory markers.

- **Interpreting inflammatory markers is complicated**: Inflammatory markers measured in humans are hard to interpret. It's not correct to say that inflammation is "good" or "bad" – it depends! Inflammation is the tool the immune system uses to kill unwanted organisms in the body, like viruses and certain bacteria. When we aren't infected, levels of inflammation

should be low (there's nothing to fight off), but when we need to fight off a disease, they should be high. Measuring multiple inflammatory markers is necessary because levels are highly dynamic, and it's likely that at least one will be elevated by chance or a different physiological process. Now, we are finding that inflammation is even a factor in "non-infectious" diseases like heart disease, which was the focus of this study. Here, 10 inflammatory markers were measured from different pathways and statistical measures were taken to ensure that the correlations observed were not due to chance. The 10 inflammatory markers measured were as follows (they sound like gibberish, but they are real!): urinary 8-epi-PGF_2a isoprostanes (normalized to creatinine); blood C-reactive protein (CRP), interleukin-6, intercellular adhesion molecule-1 (ICAM-1), lipoprotein-associated phospholipase-A2 (LpPLA2) activity and mass, monocyte chemoattractant protein-1 (MCP-1), osteoprotegerin, P-selectin, and tumor-necrosis factor receptor 2 (TNFR2).

- **The higher the Omega-3 Index, the lower the inflammatory markers**: So, what did the researchers find? There were significant inverse correlations between <u>all</u> inflammatory markers and the Omega-3 Index when adjusting for age and sex (Ps<0.0001). However, these relationships were small-to-moderate effect sizes (Rs between -0.07 and -0.18). What these effect sizes mean is difficult to say but they are most likely due to the small range of values for both inflammatory marker levels and the Omega-3 Index. Also, the "true" anti-inflammatory effect of having a higher Omega-3 Index is most likely subtle, but we cannot infer this from this study as we'll discuss below. When further adjusting for 15 other blood and lifestyle variables including fish oil intake, 8 of 10 inflammatory markers were still inversely associated with the Omega-3 Index. For the relationship between the Omega-3 Index and inflammatory markers to remain significant after adjusted for many other variables that presumably affect inflammation means that there is probably something there.

- **Correlation does not equal causation!**: In this blog we have harped on the fact that positive or negative associations between omega-3 (whether intakes or blood levels) and some disease do not mean that the omega-3 fatty acids *caused* (or *prevented*) the disease; one simply cannot tell what caused what with a cross-sectional study design (i.e., where all data are collected at one point in time). So, in the spirit of fairness and transparency, we must make that same point here and say that we can't tell whether increasing the Omega-3 Index causes a reduction in inflammatory markers or not; this simply cannot be answered in an observational study.

Bottom line: This study in the Framingham population using gold-standard analyses of variables, i.e. the Omega-3 Index and multiple inflammatory markers, helps further demonstrate a link between omega-3s and lower inflammation. However, because it's an observational study, it cannot prove that omega-3s lower inflammation.

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