

New insights into how omega-3 fatty acids reduce inflammation also hints at novel disease treatments

Science Daily

There is strong evidence that eating foods rich in omega-3 fatty acids, such as some fish, plant-derived oils and nuts, or taking omega-3s as a dietary supplement reduces inflammation and lowers the risk of illness and death from cardiovascular and other inflammatory diseases, said Bruce A. Freeman, Ph.D., professor and chair of the Department of Pharmacology and Chemical Biology, Pitt School of Medicine, and one of the study's senior authors.

"What has been a provocative question for people familiar with these impressive clinical actions is how omega-3 fatty acids actually induce such beneficial pharmacological effects," he said. "This study has given us fresh and revealing perspective into that process."

In this study, also led by Pitt assistant professor Francisco J. Schopfer, Ph.D., the researchers examined metabolic byproducts of omega-3 fatty acids that are produced by activated macrophages, a type of immune cell that is always present in inflamed tissue, and discovered previously unknown biochemical mediators of inflammation.

Using a small molecule called beta-mercaptoethanol (BME) as a reactive bait, Chiara Cipollina, Ph.D., one of the study's lead authors and a post-doctoral student from Palermo, Italy's Ri.MED Foundation, "hooked" several derivatives of omega-3 fatty acids that were produced by immune cells. These derivatives were chemically modified to become electrophilic fatty acid oxidation products (EFOX), meaning they are attracted to electrons and therefore react with critical molecular targets in many different cell types.

By interacting with certain protein residues that have electrons available for chemical binding, these derivatives stimulate changes in cellular protein function and the genetic expression patterns of cells, resulting in a broad range of antioxidant and anti-inflammatory responses.

The research team found that an enzyme called cyclooxygenase-2 (COX-2), which is the molecular target of common drugs such as aspirin, ibuprofen and acetaminophen, mediates the transformation of omega-3 fatty acids into EFOX. Notably, cellular EFOX concentrations were significantly increased in the presence of aspirin, suggesting another mechanism for that drug's beneficial effects.

"There is a lot of evidence that supports minimizing inflammation as a fundamental therapy for many diseases," Dr. Freeman said. "Our new insights help explain in part the multitude of beneficial actions observed for both omega-3 fatty acids and

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Published on Chem.Info (<http://www.chem.info>)

aspirin, and the discovery of this new class of omega-3 fatty acid-derived anti-inflammatory mediators could point drug development activities in new and fruitful directions."

For example, drugs that, like aspirin, enhance the production of EFOX could be of value, or new agents might be synthesized that are able to induce anti-inflammatory signals that are similar to those induced by EFOX, he explained. Drs. Freeman and Schopfer and their drug discovery team now are working on some of these approaches.

The research team also included co-lead author Alison L. Groeger, Ph.D., Marsha P. Cole, Ph.D., Steven R. Woodcock, Ph.D., and Gustavo Bonacci, Ph.D., all of the Department of Pharmacology and Chemical Biology, University of Pittsburgh School of Medicine. Co-authors Tanja K. Rudolph, M.D., and Volker Rudolph, M.D., have since returned to their positions at the University Heart Center, Hamburg, Germany.

The study was funded by start-up support from the University of Pittsburgh School of Medicine and grants from the National Institutes of Health, the American Diabetes Association, and the Ri.MED Foundation.

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Source URL (retrieved on 09/16/2014 - 11:12pm):

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