Cancer Prevention Research

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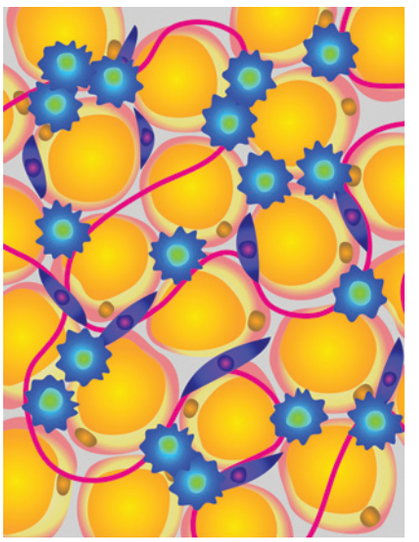

Obesity-related inflammation is implicated in the development of breast cancer. Obesity-induced adipocyte hypertrophy, hypoxia and macrophage infiltration, and adipose stromal cell hyperplasia with increased local and systemic pro-inflammatory adipokines and cytokines fuel the process.

Preclinical and epidemiologic studies suggest that the inflammation resolving marine omega-3 fatty acids eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) reduce risk of breast cancer development. A key mechanism is thought to be replacement of arachidonic acid in cell membranes resulting in the formation of less inflammatory eicosanoids, as well as protectins and resolvins. In addition, EPA and DHA disrupt lipid rafts reducing activation of tyrosine kinase receptors such as EGFR. The dose of DHA or EPA combined or as single agents necessary for inflammation resolution or lipid raft disruption in humans is unclear; however, pre-clinical studies suggest that DHA may be superior to EPA for breast cancer prevention. Beginning on page 203, Gucalp and colleagues present results of a placebo-controlled trial in which 3 months of DHA at a dose of 2g/day did not impact expression of several inflammatory genes or prevalence of crown-like structures in benign breast tissue from overweight and obese high-risk women and breast cancer survivors. Potential explanations for these null results include the type and dose of fatty acid used, cohort heterogeneity, and the biomarkers selected, as discussed in an accompanying editorial by Fabian and Kimler on page 187.

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