Polyunsaturated Fatty Acids for Membrane

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The role of omega-3 polyunsaturated fatty acids (PUFAs) in controlling tumor cell membrane fluidity, drug resistance, and altered membrane biophysics are still under question.

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1. Introduction

From a chemical point of view, just as matter is formed by the combination of atoms, our body organ system is developed according to this parasitic trend from cells to tissues, from tissues to organs, and from organs to body systems [1]. After a certain period, each cell dies. When the cell divides at a much higher rate than the normal division rate, it is called a tumor [2]. If the cell spreads in the body system and then forms a new tumor, then the division of these cells is uncontrolled, and the cells gain immortality; then, it is termed cancer.

In cancer, a large amount of fatty acid biosynthesis occurs in the cell membrane, resulting in new membranes in tandem with uncontrolled cell division [3]. In the case of breast cancer, not only are fatty acids and phospholipids converted into a cell membrane, but the membrane's biophysical atmosphere is also affected. In this type of cancer, the estrogen receptor driven by the estrogen hormone in the cell membrane plays a crucial role. The role of omega-3 polyunsaturated fatty acids (PUFAs) in controlling tumor cell membrane fluidity, drug resistance, and altered membrane biophysics are still under question.

On the other hand, it has been found that saturated fatty acids increase the risk of breast cancer [4][5]. Omega-3 PUFAs are not directly synthesized in our body, and they must be taken from outside. Why have polyunsaturated fatty acids been chosen instead of saturated fatty acids? How do these types of fatty acids prevent breast cancer by altering membrane biophysics?

2. Introductory Concept of Omega-3 PUFAs

Omega-3 PUFAs gather fundamental polyunsaturated unsaturated fats that assume significant roles in biological cell construction and cell flagging. The longer chain omega-3 PUFAs, eicosapentanoic acid (EPA), and docosahexaenoic acid (DHA), usually referred to as marine unsaturated fats, are most proficiently obtained from fatty cold water fish such as salmon. Whether ingested or synthesized, PUFAs are either oxidized for fuel, stored in triacylglycerol, taken up in phospholipid films for inevitable use as substrates by cyclooxygenase (COX) and lipoxygenase (LOX) compounds, or utilized as ligands for G receptors [6][7][8]. The omega-3 unsaturated fats EPA and DHA and their subordinates are significant for retinal and mental health, intellectual capacity, and the creation of negligibly reactive eicosanoids, proresolving mediators named resolvins, and different tissue protectins [9].

However, most of the bioactive lipid mediators of interest are a consequence of COX and LOX compound movement in the long-chain PUFAs. Cancer is likely known to increase mammary tumor expansion. EPA and DHA contend with AA as substrates for COX and LOX proteins, even though EPA is a less suitable substrate than AA, especially for COX. Upon inflammatory stimulus, the catalyst phospholipase A2 discharges AA from phospholipid layers of monocytes, and overwhelmingly proinflammatory subsidiaries are created.

3. Intermediate Cross-Talk of ALA and Gamma-Linolenic Acid (GLA)

A decrease in oncogenic protein is monitored through disturbance of plasma film lipid rafts, a decrease in cytokine production, and an expansion in apoptosis following the enactment of the plasma layer GRP120 protein [10][11][12][13]. ALA and GLA disturb lipid rafts, sphingolipid/cholesterol-improved miniature spaces of plasma layers that streamline motioning

by concentrating proteins. A lessening in epidermal development factor receptor and human epidermal development factor-2 flagging would be relied upon to diminish multiplication [14]. A reduction in Ki-67 has been observed in favorable and dangerous mammary tissue after ALA and GLA supplementation in most preclinical models.

4. Translational Impact of PUFAs

steroidal anti-inflammatory drug (sulindac sulfide) induced apoptosis, leading to enhanced growth suppression of human colon cancer xenografts. PUFAs have also enhanced the susceptibility of human colorectal cancer cells combined with 5-fluorouracil for mammary carcinoma in addition with celecoxib, tamoxifen in breast cancer, cisplatin in lung cancer, gemcitabine in breast and liver cancers, doxorubicin, vincristine, and fludarabine in leukemia. Various investigation results indicated that EPA at 2 g/d for 3 mo also reduces colonic crypt cell proliferation and increases apoptosis in normal colonic mucosa [15]. In patients given 3 g of omega-3 PUFA daily for 7 days before surgery for colorectal cancer, EPA was rapidly absorbed into the colonic mucosa and the colonic muscle layer, supporting assertions about the therapeutic benefits of omega-3 PUFA.

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