

748

Erythrocyte Membrane Fatty Acid Composition in Cancer Patients

NINA MIKIROVA, Ph D; HUGH D. RIORDAN, MD; JAMES A. JACKSON, MT (ASCP) CLS, Ph D;
KELLY WONG; JORGE R. MIRANDA-MASSARI, PharmD*; MICHAEL J GONZALEZ, DSc, Ph D, FACN*.

Essential fatty acids (EFA) have an important role in complex metabolic reactions. The metabolism of essential polyunsaturated fatty acids (PUFA) appears to be one of the critical targets in the complex metabolic stages that lead to, or are associated with cancer. The goal of our research was to analyze the erythrocyte specific types of membrane fatty acid content, level and distribution in cancer patients as compared to non-cancer patients. Changes in fatty acid composition may affect different aspects of cell structure and function, including proliferation. Analyses of RBCs membrane fatty acids were performed for 255 patients with different types of cancer (breast, prostate, liver,

pancreas, colon, and lung), 2,800 non-cancer patients and 34 healthy volunteers. Our research study demonstrated a lower level of stearic acid and an increased content of oleic acid in RBC of cancer patients in comparison with control and non-cancer patients. According to the results of this investigation, the ratio of Eicosa pentaenoic acid (EPA) and Decosa hexaenoic acid (DHA) to Alpha-linolenic acid (ALA) may be useful to estimate PUFA imbalances in cancer patients. EPA and DHA acid may be recommended as supplementation and in addition to current therapy during cancer treatment.

Key words: Erythrocytes, Fatty acids, Cancer

Many studies have demonstrated that dietary fats may affect the incidence of cancer and markedly influence tumor development. Dietary fatty acids (FA) may modulate various important membrane parameters such as: membrane associated receptors, tumor antigens, prostaglandin synthesis membrane potential and membrane fluidity. Essential fatty acids (EFA) have an important role in complex metabolic reactions. The metabolism of essential polyunsaturated fatty acids (PUFA) appears to be one of the critical targets in the complex metabolic stages that lead to, or are associated with cancer. A decrease in PUFA level is one of the biochemical abnormalities that strongly correlate with cell transformation (1, 2). The loss of specific PUFA is either due to changes in enzyme activity or decreased concentration of substrates that may be accompanied by an array of changes that may promote the development of malignancies.

Neoplastic tissues exhibit differences in the lipid metabolism as compared to normal cells. An elevation in

distinct lipids (3) and a decrease in PUFA levels (4) have been strongly correlated with cell transformation.

Tumor cells also tend to have a decreased activity of EFA desaturase enzymes (5). The activities of the desaturase enzymes are under hormonal and nutritional control, but the molecular mechanisms of this control have not been totally elucidated (6). Especially, the delta -6-desaturase enzyme appears important, since its activity determines the tissue level of polyunsaturated acids, such as gamma-linolenic acid (GLA), dihomogamma-linolenic acid (DGLA) and the series 1 eicosanoids.

Many types of tumor cells exhibit a decreased rate of lipid peroxidation compared to that seen in normal cells, as a result of lower content of PUFA. A high rate of cell division correlates inversely with the rate of lipid peroxidation in both normal and tumor cells (7). The extreme importance of lipid peroxidation products (LPP) in cell proliferation is now acknowledged (8, 9). LPP were previously considered as components that should be prevented by antioxidants, LPP are now recognized as metabolites involved in the control of tumor growth and determinants of tumor sensitivity to anticancer agents. Incorporation of omega-3 PUFA in tissues provides excellent substrates for lipid peroxidation.

Animal studies indicate that omega-6 fatty acids promote tumor growth while omega-3 fatty acids inhibit tumor development. For example, a fish oil-derived concentrate enriched in eicosapentaenoic (EPA) and

From the Center for the Improvement of Human Functioning International, Inc., 3100 N. Hillside Avenue, Wichita, Kansas, 67219 and the *RECNAAC II Project, University of Puerto Rico, Medical Sciences Campus, PO Box 365067, San Juan, PR 00936.

Address correspondence to: Dr. Michael J. González, Graduate School of Public Health, Nutrition Program, PO Box 365067, San Juan PR 00936-5067, Email: mgonzalez@rcm.upr.edu

MIKIROVA, NINA