ESSENTIAL FATTY ACIDS AND B COMPLEX FACTORS

How often do we see patients who require B vitamin supplementation for the correction of the following problems: neuralgias, paresthesias, allergies, muscle cramping, changes in vision, depression, anxiety, and other neuroses and psychoses, acne, eczema, dermatitis, adrenal problems, menstrual difficulties, cardiovascular abnormalities, fatigue, poor sleep, glossitis, stomatitis, and vaginitis? And how frequently do we encounter osteoarthritis, rheumatoid arthritis, bursitis, multiple sclerosis, systemic lupus erythematosus, irritable bowel syndrome, epilepsy, obesity, gall bladder problems, easy bruising, hot flashes, infertility, and diabetes mellitus? All of the above conditions and symptoms, and many more, are related to improper fatty acid ingestion and/or metabolism. Laboratory and clinical investigations during the last thirty years have opened the door to our understanding of the body's fat metabolism in relationship to the other factors in natural health care. The use of EFA gives us powerful tools with which we may dramatically affect our patients' health problems, from both therapeutic and preventive perspectives.

Much of the original biochemical and clinical information in this section originated from a ground-breaking conference entitled "Essential Fatty Acids and Prostaglandins in Human Health and Disease," which took place in Lenox, Massachusetts, in December, 1982. Prior
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to 1981, laboratory investigation methods had not developed far enough to allow for a true understanding of essential fatty acids, prostaglandins and related substances. At this 1982 conference, Donald Rudin, M.D. presented his findings on the relationships of various B complex factors and certain essential fatty acids (EFA). He elaborated on two of his published papers which discussed the relationships of niacin and thiamine to the omega-3 essential fatty acids (ω-3 EFA). These papers were entitled, "The Major Psychoses and Neuroses as Omega-3 Essential Fatty Acid Deficiency Syndrome: Substrate Pellagra," 15 and "The Dominant Diseases of Modernized Societies as Omega-3 Essential Fatty Acid Deficiency Syndrome: Substrate Beriberi." 16 This was pioneering information which opened the door for many of the clinical advances that followed. Rudin stated that vitamins, particularly niacin and thiamine, are only functional in the body when the appropriate EFA are present as substrates on which the B vitamin-dependent enzymes are able to act. Rudin's particular area of interest is the ω-3 EFA which are converted by B vitamin-dependent enzymes to the prostaglandin 3 (PG3) series tissue hormones. Similar patterns exist in the relationships of omega-6 essential fatty acids (ω-6 EFA) and the B complex factors. The ω-6 EFA are converted into the prostaglandin 1 (PG1) and prostaglandin 2 (PG2) series tissue hormones, as will be elaborated on later in this section.

B vitamins are active in the body in the form of various coenzymes which have their effects on the metabolism of the macronutrient factors in the body, protein, carbohydrate, and fat. These vitamin B coenzymes convert the macronutrients into various other forms for the release of energy, regeneration of tissues, or production of metabolic controlling factors (hormones and enzymes). Rudin stated that the major deficiency diseases pellagra (classic niacin deficiency) and beriberi (classic thiamine deficiency) may, in some cases, be as


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much EFA deficiency diseases as B vitamin deficiency diseases. That is, even if adequate B factors are present, a deficiency of EFA substrates on which these B factors work will result in many of the same symptoms as the niacin or thiamine deficiencies.

Rudin suggests that if all of the symptoms of a niacin or thiamine deficiency are compared to those symptoms of major protein (amino acid) deficiency (kwashiorkor), and the kwashiorkor symptoms are subtracted from the niacin or thiamine deficiency lists, the resulting list of symptoms are those of EFA deficiency. Further, a certain (small) percentage of pellagra patients will not respond to niacin but will respond to the appropriate (i.e., ω-3) EFA. In other words, there must be a matching of the essential B complex factors with the essential fatty acid factors (and one would assume, the essential amino acid factors) for optimal health to be present.

ESSENTIAL FATTY ACIDS AND PROSTAGLANDINS

The major importance of EFAs in the body is their relationship to the formation of tissue hormones called eicosanoids. “Eicosa” is the prefix for twenty. All of the eicosanoids are derived from 20-carbon chain EFAs. EFAs become active when they are converted to eicosanoids. EFAs can be converted to four different groups of eicosanoid compounds that include prostaglandins, leukotrienes, thromboxanes and prostacyclins. For our purposes, we will focus the discussion on the prostaglandin (PG) group and briefly include the others in passing.

There are three families of prostaglandins which are termed the PG1, PG2, PG3 series. The lists of functions of the various prostaglandins are seemingly endless, and excesses or deficiencies of any PG family can create problems. The PG hormones are very short-lived (for the most part, less than five minutes, some as short as milliseconds) and are derived from fatty acids that are present in cell membranes or that circulate in the body as free fatty acids, phospholipids, and cholesterol esters (combinations of cholesterol and fatty acids). Despite their short lives, PGs are constantly being produced and their effects on the
tissues they contact are of major importance.

PGs act as the intermediates between hormones, neurotransmitters, and other metabolic stimulants and the actual tissue effects. Many of these effects occur in cell membranes, where the lipids have a dual role as primary building blocks of these membranes as well as having major functional impact. Other effects occur in the other compartments of the body. PG hormones mediate the control of local tissue effects such as inflammation, platelet aggregation, response to other hormones such as the catecholamines, tumor growth, neurotransmitter response, and in general, the homeostatic response to various stimuli and stressors which the tissues encounter.

In reality, there are no such things as “good prostaglandins” or “bad prostaglandins.” However, the PGs of the PG2 series are usually those which are present in excess in our society and therefore, these are often called the “bad prostaglandins.” These negative actions include such common problems as platelet aggregation (hence, increased blood clotting), increased blood pressure, asthma, menstrual cramps, increased tumor growth, and increased inflammatory response with all of its attendant tissue damage and pain.

To summarize, the PG 2 family causes:

- ↑ INFLAMMATION
- ↑ BLOOD PRESSURE
- ↑ CHOLESTEROL
- ↑ BLOOD CLOTTING
- ↓ NATURAL KILLER CELL ACTIVITY (INCREASED TUMOR GROWTH)
- MENSTRUAL CRAMPS
- ASTHMA

The PG 1 & PG 3 families do the opposite from the PG 2 family.

Two other groups of substances called leukotrienes (Lts) and thromboxanes (TXs) are also derived from EFA. Leukotrienes are produced by white blood cells (i.e., leukocytes, hence
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the name, leukotrienes). Thromboxanes are produced by platelets (i.e., thrombocytes, hence the name, thromboxanes). Those LTs and TXs which are derived from arachidonic acid cause many of the same "bad" problems that are caused by the PG2s. We will pay little attention to LTs and TXs in the present discussion, but be aware that when we are discussing the production of PG2 substances from arachidonic acid, the same principles usually hold true for the LT products (LT4 series) and the TX products (TX2 series) of arachidonic acid. LTs and TXs will receive more attention below.

Most of the bad effects of PG2 can be balanced by the presence of adequate amounts of PG1 and PG3 and therefore, PG1 and PG3 are often referred to as “good prostaglandins.” Because of the far-reaching effects of the PGs, and the fact that they are derived from EFA, the dietary intake of EFA takes on heightened importance in our understanding and manipulation of our patients' nutritional intakes. To put EFA in perspective, they are as fundamentally important as the B vitamins and other vitamins, possibly no more important, but certainly no less important. EFA metabolism should be one of the first, if not the first, nutritional factors assessed on a patient. This is made very clear when using Indicator Testing for EFA as will be discussed below. EFA assessment is so important that it should be addressed very early in the treatment process. In my practice I usually consider EFA supplementation during the first or second treatment session.

The simplified EFA summary shown in Figure 16-3 is amplified by the relationships of EFA and PG which are shown in Tables 17-1A and 17-1B. This information is compiled by this author from presentations and notes from many sources.