



Enhancing Your Body's Ability to
Fight the COVID-19 Virus with EFAs
Including Appendix Detailing How Essential EFAs are Related to
Killing Viral-Relating Diseases / Conditions
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Enhancing Your Body's Ability to Fight the COVID-19 Virus with EFAs Including Appendix Detailing How Essential EFAs are Related to Killing Viral-Relating Diseases / Conditions

Most of us are attempting to avoid contracting the COVID-19 Virus by using masks, gloves, disinfectants, and antiseptics. Shouldn't we also focus on strengthening our body's immune system? There is more than one strain of corona virus. However, the following analysis generally applies. "[Inactivation of Enveloped Viruses and Killing of Cells by Fatty Acids](#)," and 2019's "[Characterization of the Lipidomic Profile of Human Coronavirus-Infected Cells: Implications for Lipid Metabolism Remodeling upon Coronavirus Replication](#)," specify that the long chain fatty acids; in particular, Parent omega-6 and its long-chain metabolite AA can destroy the virus at the cellular level.

I thank my colleague, EFA/eicosanoid specialist Paul Beatty, from Toronto, Canada for providing these articles presented here and many of the references in the Appendices.

Today, due to food processing — unless you are consuming organic Parent omega-6 — it will be adulterated and inactive to a large extent. For optimal protection, an EFA formulation should optimize cell membrane structure (viruses must bind and join the cell's membrane) and maximize blood flow. EFAs must be consumed on a daily basis.

We all understand that a strong immune system is key to staying healthy. Parent omega-6 also optimizes cellular health by flooding each of your cells with oxygen. Please understand this review of two important journal articles assumes you understand the basis of my work for the last twenty years. Specifically, that our daily diet unfortunately consists of a variety of *adulterated* oils. You must offset this damage by supplementing with *unadulterated* oils, specifically Parent Omega-6 and Parent Omega-3. Therefore, when I reference Omega-6 and Omega-3, unless otherwise noted, it is fully functional and unadulterated.

In an effort to assist you, I have summarized these two articles:

The 1st article analyzed nine fatty acids found in human breast milk for their anti-viral effect.

Novel lipids-based pharmacognosy solutions

Enveloped viruses like the Corona Virus are susceptible to inactivation and destruction by certain fatty acids. This journal article study makes clear that all fats are not equal in anti-viral effectiveness — only (unprocessed / fully functional) medium-chain saturated, and long-chain unsaturated fatty acids were antiviral (but at varying concentrations):

- “The polyunsaturated long-chain fatty acids [like PEOs] were the most active and effective antiviral in killing enveloped viruses.
- “Antiviral fatty acids were found to affect the viral envelope, causing leakage and at higher concentrations, a complete disintegration of the envelope and the viral particles. They also caused disintegration of the plasma membranes of tissue culture cells resulting in cell lysis and death...”

The 2nd article analyzed 24 lipids. There are several pathogenic corona virus types similar to the current 2019-nCoV and the researchers looked at the highly pathogenic MERS coronavirus — which had an enormous 34% mortality rate. LA [Parent omega-6] is the metabolic precursor of AA — both of which are key components of the cell membrane and biological signaling precursors. Researchers found that optimal coronavirus replication required a specific composition of cellular lipids and any disruption could decrease the efficiency of coronavirus replication:

- Supplementation of LA [Parent omega-6] / AA [omega-6 series long-chain derivative] significantly suppressed both HCoV-229E (which can impair the respiratory system), and also significantly suppressed the highly virulent MERS-CoV described above. They can be metabolized to important eicosanoids and metabolites, which play multiple roles in the host immune response and the pathogenesis of viral infections.
- “LA metabolism and AA metabolism pathways presented higher impact than the other pathways. Our data demonstrated that LA and AA potently suppressed MERS-CoV replication in a similar manner as HCoV-229E. Overall, our results demonstrated that exogenously supplied [from a supplement] LA and AA could interfere with the optimal replication of human-pathogenic coronaviruses....”

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“There are millions or billions of these viruses out there. The immune system fights back and attacks the virus; this is what causes inflammation and fever. But in extreme cases, the immune system goes berserk; particularly in the “Coronavirus” (SARS-CoV-2); [causing a cytokine explosion], causing more damage than the actual virus.” (“Why the Coronavirus Has Been So Successful” Ed Yong, Science Section — *The Atlantic*, March 20, 2020).

Essential EFAs are known to limit this excessive overreactive cytokine reaction:

“Essential fatty acids are natural anti-inflammatory agents and therefore **decrease the production of cytokines and histamine, which can contribute to neurotransmitter imbalance** (from the “ESSENTIAL FATTY ACIDS chapter of the book, *Integrative Medicine for Children*, 2009).”

Because EFAs work so well to kill viruses, it is worthwhile to review the medical literature regarding other viral-related disorders in the Appendices below. **Engineering often makes great use of past discoveries. Tragically, at least in the lipids area, modern medicine often fails to make use of significant undisputable past discoveries.** To forward the Essential EFA/anti-viral connection, we will remedy that deficiency in the following appendices:

APPENDICES:

Dr. Horrobin, noted authority on EFAs and eicosanoids has written a significant medical journal article [DF. Horrobin (1990) Essential Fatty Acids, Immunity and Viral Infections, *Journal of Nutritional Medicine*, 1:2, 145-151]:

“There is evidence of a close interrelationship between essential fatty acid (EFA) metabolism and the ability to respond to viral infections. EFAs are required to **enable interferon** to exert its **antiviral effects.** **Viral infections** both in vivo and in vitro **induce abnormalities of EFA metabolism.** In a placebo-controlled study in post-viral fatigue syndrome EFA supplementation with n-3 and n-6 EFAs has been found effective in reversing the plasma EFA abnormalities and treating the clinical state. **These observations suggest that normal EFA metabolism is of major importance in regulating the response to viral infections.”**

PGE₁ [made from Essential omega-6 —or more directly from GLA], activates (Cytotoxic) T Lymphocytes—your body’s powerful “killer cells” of intruders/infections/bacteria/viruses, etc. (British Society for Immunology).

From “thepharmaletter” [20-04-1992]:

“EFAs COULD REFORM ANTIVIRAL THERAPY [INCLUDING HIV]”

A new approach to treating viral infections, based on supplying the patient with supplemental essential fatty acids (EFAs), could represent a major pharmaceutical advance combatting virus-induced illness, including AIDS.

In this study, 12 patients with AIDS, diagnosed on the basis of evidence of HIV infection from a blood test, severe weight loss and at least two other symptoms of the disease, were selected for the study. **Within 12 weeks of treatment with EFAs, the patients had put on weight and experienced a considerable improvement in symptoms with a reduction in fatigue, diarrhea and an improvement in the severity of skin rashes.**

20 months after the study began, five of the 12 patients remain alive and relatively well. There was also a **significant improvement in the CD4 lymphocyte count** of treated patients, from 59 to 261/mm³. No patients experienced any adverse effect as a result of the treatment with EFAs.

While **EFAs** are known to have direct antiviral activity, they are also **necessary for the antiviral actions of interferon**. In this regard, research work conducted in the USA in the 1970s demonstrated that **interferon requires EFAs before it can function as an antiviral**. And **without this** vital interaction, whereby the EFAs are converted into prostaglandins, **the antiviral actions of interferon are either lost or greatly diminished**.

Physicians at the Istituto Dermatologico Santa Maria e San Gallicano in Rome have reported that blood from Italian AIDS patients shows the same pattern as have researchers at the Royal Free Hospital, London, and the University of Miami, Florida. As for other virus infections, a team at Ohio State University, Columbus,

reported in 1988 that students infected with the **Epstein Barr virus** (the cause of infectious mononucleosis) also **had reduced blood EFA concentrations**. Furthermore, the **efficacy of EFAs against post-viral fatigue syndrome has been demonstrated** in a double-blind, placebo-controlled trial conducted at the University of Glasgow with 63 patients.

Post-viral Fatigue Syndrome: 85% of patients improved with Essential EFA treatment

Sixty-three (63) diagnosed post-viral fatigued patients of prior 1-3 years of illness suffered severe fatigue, myalgia, and a variety of psychiatric symptoms. (Behan, et al., “Effect of high doses of essential fatty acids on the post-viral fatigue syndrome,” (Acta Neurol Scand. 1990 Sep;82(3):209-16). **At just 3 months, the improvements were 85% with the EFAs and 17% placebo (p<0.0001)**. The **Essential EFA levels** were abnormal at baseline and **corrected by active treatment**. There were no adverse effects.

Chronic Fatigue Syndrome (CFS) / Myalgic Encephalomyelitis (ME)

A superb analysis posted in 2005 (Clinical Improvements in CFS/ME: The Role of Fatty Acids, Prof. Basant Puri, 2005) shows the following:

In well-conducted studies of the immune system show changes in particular types of white blood cells have been noted [Puri BK. Chronic fatigue syndrome: A natural way to treat ME. Hammersmith Press. London. ISBN 1-905140-00-2. 2005].

In summary, these are:

- **Reduced** NK cell (killer cell) activity;
- **Reduced** Th1 cell (helper cell type 1) activity;
- **Increased** Th2 cell (helper cell type 2) activity;
- **Increased** Tc cell (cytotoxic cell) activity.

The changes described above are consistent with the immune system reacting to a pre-existing long-term viral infection.

By carrying out a specialized brain examination known as proton neurospectroscopy, using a magnetic resonance imaging scanner, we were able

directly to examine the chemistry of the living brain in patients with CFS (Puri BK et al. Relative increase in choline in the occipital cortex in chronic fatigue syndrome. Acta Psychiatr Scand. 106: 224-226. 2002). We found strong evidence of a chemical signature in the patients which was consistent with an *inability of the body to create the all-important phospholipid molecules in cell membranes* in the brain. This could easily result from a **lack of long-chain polyunsaturated fatty acids in the brain**. In turn, this could be caused by a **viral infection stopping the body from being able to manufacture these fatty acids**. Our Hammersmith Hospital **results were replicated** by another group, working in Glasgow, under Professor (then Dr) Abhijit Chaudhuri, who also found evidence of the same chemical signature [Chaudhuri A et al. Proton magnetic resonance spectroscopy of basal ganglia in chronic fatigue syndrome. Neuroreport. 14: 225-228. 2003].

The Essential “Parents” omega-6 (LA) and omega-3 (ALA) fatty acids have very important roles in maintaining the correct structure of cell membranes. Your cell membranes also need to have sufficient levels of the omega-6 long-chain polyunsaturated fatty acid arachidonic acid (AA).

Another important role is that of the synthesis of *eicosanoids* in the body. These functions fall to two of the omega-6 long-chain polyunsaturated fatty acids, dihomo-gamma-linolenic acid (DGLA) contained in Evening Primrose oil and arachidonic acid (AA), and one of the omega-3 long-chain polyunsaturated fatty acids, namely eicosapentaenoic acid (EPA); which is made from the Essential EFA, Parent omega-3. Starting from dihomo-gamma-linolenic acid (DGLA), arachidonic acid (AA) and EPA, **the body can make all the families of prostaglandins, thromboxanes and leukotrienes (which collectively are eicosanoids)**. They are intimately involved in processes that are of the **utmost importance in maintaining the health and well-being of the body and in fighting disease**.

Essential omega-6 will kill the virus in the bloodstream on contact with it, although, for maximum resistance, your body relies on the long chain metabolites to further assist in viral deactivation. Many viral species block the delta-6-desaturase enzyme. This means that the body is no longer able to produce the long-chain metabolites of Essential omega-6 and Essential omega-3 fatty acids. **Most importantly PGE₁ — the body’s most powerful anti-inflammatory — is made either easily from GLA-containing seed oils or metabolized from Essential LA, IF there are sufficient unadulterated / unprocessed amounts.**

EPA (made from Essential omega-3 (ALA)) is also important in helping your body to combat viral infections. EPA kills viruses in the body, without

harming us in the process. It does this in at least two ways. First, EPA is itself a viricidal. That is, if you add small amounts of EPA solution to harmful viruses (such as the Epstein Barr Virus that causes glandular fever), then the EPA kills the virus. Second, EPA is also indirectly a viricidal. After being acted on by two sets of enzymes, EPA is converted into families of interferons, which, in turn, are also powerfully antiviral.

The lack of DGLA (because of **insufficient fully functional Essential omega-6**), arachidonic acid, and EPA also means that your **body cannot produce enough eicosanoids — the general health and well-being of the body suffers**. Your body cannot mount a proper immune response.

Additionally, blocking that first enzyme (delta-6-desaturase) also means that **cell membranes** cannot get enough arachidonic acid and docosahexaenoic acid so that they **become more rigid and lose their normal flexibility**. The effects on the protein receptor molecules that lie in the cell membranes are profound; the size and shape of these receptors change so that they no longer accept and pass on signals in the right way. **Communication between cells is impaired**. The results of this in the human brain have **cognitive defects, such as problems with short-term memory and with concentration**.

Because of an EFA deficiency in the cell's membrane — mainly a deficiency of unadulterated / fully functional Essential omega-6, which inhibits Delta 6 desaturase — the cell cannot make enough EFA metabolites to deactivate the virus. In this way your cell becomes the host of the virus.

For additional proof that cellular lipids are altered from viral infection, we look to Williams et al.:

Serum fatty acid proportions are altered during the year following acute Epstein-Barr virus infection (Lipids, 1988 Oct;23(10):981-8).

- **“Abnormal serum fatty acid (FA) proportions had been found at **three months after** infectious mononucleosis (**IM**)... profiles of 20 normal college students were measured at monthly intervals for one year following an acute Epstein-Barr virus (EBV) infection.... **Persistence of low linoleic acid [Essential omega-6]** content beyond six months post-infection occurred in all seven students who showed continued clinical symptoms.**

Estimation of FA enzyme activities over the post-IM year suggested that FA elongation function was normal, but that FA **desaturation enzyme activities were lower than normal**, particularly early after EBV infection. **An inability of the host to normalize the serum total linoleic/oleic acid ratio may parallel a delayed recovery from EBV infection** and may offer insight into its pathogenesis.

The impaired desaturation enzyme activity started with the critical delta-6 desaturase activity and its impairment to produce adequate PGE₁ — the body's most powerful anti-inflammatory and killer T-cell potentiator. More evidence of impaired Essential EFA metabolism is presented in **myalgic encephalomyelitis (chronic fatigue syndrome)**:

“Long-chain polyunsaturated fatty acids and the pathophysiology of myalgic encephalomyelitis (chronic fatigue syndrome)”:

- “...[S]uch infections are likely to **impair the ability of the body to biosynthesise n-3 and n-6 long-chain polyunsaturated fatty acids** by **inhibiting the δ-6 desaturation** of the precursor essential fatty acids — namely, α-linolenic acid and linoleic acid. This would, in turn, impair the proper **functioning of cell membranes**, including cell signaling, and have an **adverse effect on the biosynthesis of eicosanoids from the long-chain polyunsaturated fatty acids dihomo-γ-linolenic acid, arachidonic acid and eicosapentaenoic acid.**”

Many clinical features of epidemics of **Myalgic Encephalomyelitis-like illnesses** are consistent with viral infections. Immune system changes in Myalgic Encephalomyelitis tend to point to **reduced natural killer cell activity, reduced Th1 cell activity, increased Th2 cell activity and increased cytotoxic T cell activity.**

- These findings are **consistent with those of a pre-existing long-term viral infection.** **Viral infections can impair the ability of the mammalian body to biosynthesize long-chain polyunsaturated fatty acids from their short-chain precursors.** In the baseline comparison of erythrocyte membrane fatty acid levels among 63 patients (with what was then termed post-viral fatigue syndrome) and 32 normal volunteers, Behan *et al* found **considerably lower levels** of arachidonic acid, adrenic acid and the **total n-6 polyunsaturated fatty acids**. Warren *et al* used the Oxford Criteria for

diagnosis and found a considerably lower level of eicosapentaenoic acid in patients with chronic fatigue syndrome. This **impairs the biosynthesis of membrane phospholipid molecules in the brain, as long-chain polyunsaturated fatty acids are key components** at the Sn2 position of these molecules. This leads to a reduced incorporation of the polar head group choline in these molecules (at the Sn3 position).

- “As a result of viral, or other, **inhibition of δ -6-desaturase**, an **inadequate supply of the long-chain polyunsaturated fatty acids is available for incorporation into the membrane phospholipid molecules**. Thus, *the ratio* of anabolism to catabolism of membrane phospholipids can be expected to change in an adverse direction. In turn, as far as the brain is concerned, this may be expected to have an unfavorable effect on neurotransmission—for example, it has been shown that **minor changes in fatty acid structure in a small proportion of membrane phospholipids can lead to profound changes in the tertiary and quaternary structures of membrane proteins, and in the functioning of such proteins**.
- “Evidence suggests that **myalgic encephalomyelitis or chronic fatigue syndrome** may be associated with a **persistent viral infection**. Such an infection could **adversely affect the biosynthesis of long-chain polyunsaturated fatty acids and therefore the membrane structure, functioning and production of eicosanoids**. **Treatment with long-chain polyunsaturated fatty acids may offer a potential therapeutic route.**”

The evidence is compelling that unadulterated / fully functional Essential EFAs are fundamental to successfully treating any viral-related illness.
