

Overview: CM PLEX

Speculation on Mechanism:

I. Improves Membrane Fluidity

Improves fluidity of all cell membranes especially those of white blood cells (WBCs) and endothelial cells which line all blood vessels in the body

Endothelial cells act as a semi-permeable barrier between the blood and the tissues. Arguably the largest organ in the body, endothelial cells determine what nutrients, hormones, etc. are allowed to pass from blood to tissues. The endothelium, which collectively refers to all endothelial cells is the largest gland in the body and secretes a number of substances that play a role in regulating blood pressure, immunity, and inflammation.

Inflammation can occur when injury, dietary conditions (trans fats, free radicals), or other insults stimulate or stiffen cell membranes. WBCs, which normally bounce randomly off the surface of endothelial cells, now begin to roll and stick to the surface of the endothelial cells. The “activated” endothelial cells secrete a number of adhesion molecules that attract and cause the white cells to accumulate. As other factors come into play an inflammation cascade occurs as WBCs and the entire “inflammatory” process spreads from the endothelium to the inflamed area. When a joint becomes inflamed, we have arthritis.

Myristoleate and oleate are cis monounsaturated fatty acids (MUFA). Cis-MUFAs have been shown to offer protection against endothelial cell activation, the production of adhesion molecules, and stimulation of the inflammatory cascade. Of interest, omega 3s have been found to have a greater beneficial effect than MUFA on preventing endothelial activation. It is possible that some of the cetyl fatty acid esters in our CM Complex increase membrane fluidity in both the endothelium and white blood cells.

II. Inhibits Inflammation

Indirectly and directly inhibits the production of inflammatory products like leukotrienes and those from the COX 2 pathway

Perhaps by improving membrane fluidity and reducing the entire inflammatory cascade, the CM Complex may prevent the stimulation and production of other inflammatory substances like leukotrienes and products of the COX 2 pathway. Omega 3s, specifically DHA more than EPA decrease endothelial activation at the cell membrane level. EPA, another omega 3, acts to competitively inhibit the conversion of arachidonic acid that would otherwise travel down the COX 2 and lipoxygenase pathways leading to the production of leukotrienes and COX 2 inflammatory products. Perhaps components of our CM Complex may play a similar role.

III. Uniquely Stabilizes Protein Receptors and Signaling

Proteins in cell membranes play critical roles in acting as receptors for a number of substances and in signaling gene regulated functions. They are what allow for communication between cells and between cells and hormones, eicosanoids, etc. In order to function optimally it is critical that these proteins become anchored securely to the inner face of the cell membrane. This anchoring of proteins is accomplished by bonding with long chain fatty acid or “sticky fingers” which appear to be specific for the binding of these proteins. Myristate, a saturated fatty acid, appears to be especially important in this function.

The enzyme n-myristoyltransferase places myristic acid on the amino-terminal sequence of specialized proteins where it participates in signal transduction. Without placing the myristate on these proteins (myristoylation) they do not function during signal transduction. It has been proposed that myristoylation of proteins may be involved in apoptosis—the process of programmed cell death. The majority of human cells have 50 to 60 lifetime cell divisions before they die off completely. This programmed cell death is referred to as apoptosis. Apoptosis may protect us from having old, sickly, malfunctioning cells from dividing uncontrollably. On the other hand tumor or cancer cells have inhibited apoptosis and divide unchecked.

Modification of proteins by myristoylation has been recognized as important in the function of various viral, and tumor enhancing signal transduction proteins. In fact myristoyltransferase (NMT) activity in rat colonic tumors was found to be higher than NMT activity in normal or normal-appearing colonic mucosa. Inhibiting myristoylation is now a target for possibly forming the basis of new approach to cancer treatment.

It is unclear whether our CM Complex affects this process. It could enhance it by preventing apoptosis and preventing the aging of immune and defense mechanisms. Or it could inhibit the process and encourage the apoptosis or destruction of harmful cells.

The cetyl group is a large 16-carbon alcohol. It is unclear how adding a cetyl group to myristate and myristoleate affects the above functions. Perhaps by adding length to the fatty acid chains it allows them to increase fluidity even more, which in turn decreases the ultimate production of inflammatory products.