

Gamma-linolenic acid, or GLA, or 18:3 n-6

There are two families of fatty acids essential to the body that must be obtained from the diet.

- (1) Omega-6, or n-6, derived from linoleic acid (LA; 18:2n-6):
 - 18 indicates that this fat is 18 carbons long.
 - 2 indicates that this fat is polyunsaturated, it has 2 double bonds.
 - n-6 indicates that the first double bond is at the 6th carbon from the end methyl (CH₃) group.

- (2) Omega-3, or n-3, derived from alpha-linolenic acid (18:3n-3):
 - 18 indicates that this fat is 18 carbons long.
 - 3 indicates that this fat is polyunsaturated, it has 3 double bonds.
 - n-3 indicates that the first double bond is at the 3rd carbon from the end methyl (CH₃) group.

The most common dietary source of omega-6, or n-6, derived from linoleic acid (LA; 18:2n-6) is corn oil, sunflower oil, and safflower oil.

These omega-6, or n-6 fats, derived from linoleic acid (LA) are enzymatically converted to arachidonic acid (AA; 20:4n-6) which in turn is converted to pro-inflammatory eicosanoid prostaglandin E₂ (PGE₂).

Omega-6 fats derived from linoleic acid (LA) are also converted to gamma-linolenic acid (GLA) by an enzyme called "delta-6-desaturase."

Gamma-linolenic acid, or GLA, or 18:3 n-6:

- 18 indicates that this fat is 18 carbons long.
- 3 indicates that this fat is polyunsaturated, it has 3 double bonds.
- n-6 indicates that the first double bond is at the 6th carbon from the end methyl (CH₃) group.

This means that GLA is an omega-6 polyunsaturated fatty acid.

GLA is then converted to dihomogammalinolenic acid (DGLA; 20:3n-6).

DGLA is then converted to anti-inflammatory eicosanoid prostaglandin E₁ (PGE₁), and inhibits (by competing for enzymes) pro-inflammatory eicosanoid prostaglandin E₂ (PGE₂).

"PGE₁ suppresses diverse effector systems of inflammation and reduces acute and chronic inflammation in several animal models."

"PGE1 suppresses human neutrophil activation and reduces levels of circulating immune complexes in patients with autoimmune diseases."

"PGE1 also protects the gastric mucosa against NSAID induced injury and may enhance healing of gastric ulcers."

"PGE1 has a range of desirable potential therapeutic effects distinct from other PGs."

It is suggested that PGE1 therapy might be accomplished by providing the PGE1 precursors GLA and DGLA.

GLA and DGLA fatty acids exert their beneficial effects through modification of the eicosanoid profile and generation of less potent inflammatory mediators and by exerting direct effects on the cells of the immune system.

GLA administration has been shown to be particularly beneficial in patients with rheumatoid arthritis, atopic eczema, and nervous system disorders (neuropathy, diabetic neuropathy, schizophrenia, and dyskinesias [abnormal involuntary movements]).

"n-3 EFAs are now known to be of little importance in the skin compared to n-6 EFAs."

"Both n-6 and n-3 EFAs are major components of both central and peripheral nervous systems. They both have important structural and functional roles. It would not be surprising if abnormal EFA metabolism played a role in several nervous system disorders."

Unfortunately, the activity of the enzyme "delta-6-desaturase" which converts LA to GLA is impaired in many individuals and disease states.

Therefore, if cellular proportions of GLA and subsequently (DGLA) are to be increased, GLA must be supplied exogenously, from the diet.

Some plant seed oils, especially oils from evening primrose and from borage, contain large amounts of GLA.

Most studies indicate that arachidonic acid (AA) is not increased by GLA administration in humans.

Quotes are from:

Medicinal Fatty Acids in Inflammation by Joel Kremer, Birkhauser Verlag, 1998.