

“HEALTH FOOD INDUSTRY MYTHOLOGY: You Need the Truth About Folic Acid.”

We receive many questions regarding the various forms of folic acid/folate/folacin. Most of those questions quote some “research” purporting to demonstrate that one form is superior to another. Almost always, that “research” is nothing more than a sales pitch from a pill peddler. It is easy to be confused by the ubiquitous and ridiculous health food industry nonsense regarding folic acid.

The terminology used to describe folic acid is confusing. The terms folic acid and folate are routinely used interchangeably. The real name of the vitamin, and the name for which folic acid and folate are accepted as synonyms, is folacin. Another name, also used synonymously, is pteroylglutamic acid (PGA).

The many different terms that are now accepted as synonymous derive from the many research pathways by which folic acid was originally isolated and identified. In other words, many researchers discovered the same nutrient at about the same time by different research methods, and each researcher had his own name for it. So now, folacin (the official term), folic acid, folate, and PGA are all used to describe pteroylmonoglutamate, the mother substance from which many different related coenzymes are derived.

There are many, many substances in both plant and animal foods that serve as sources for pteroylmonoglutamate and all its subsequently derived coenzymes. Furthermore, micro organisms in the human gut (the major source of the nutrient) also produce many forms of the vitamin from which folacin can be formed. In summary: We have many food and intestinal sources of PGA, which then become folacin, which then becomes many active coenzymes with a huge diversity of biological functions.

Many of the sales pitches regarding folacin are not for products that supply a different form or superior form of folacin. All they do is supply one or more of the countless natural sources from which folacin can be derived.

The most common question we get regarding folic acid is about the “superiority” of methylfolate. Methylfolate is not a superior form of folic acid, it is merely one form. Out of the zillion functions of folic acid, methylfolate performs only one --- methylation in the homocysteine methionine pathway.

There is so much hype from the methylfolate peddlers regarding the role of methylfolate in lowering elevated homocysteine that we must look closely at that metabolic pathway. First, understand that homocysteine is an essential intermediary metabolite. Without homocysteine, your body cannot produce its ubiquitous and most important antioxidant, glutathione. Methionine becomes homocysteine, which then becomes cystathionine, which becomes cysteine,

which then becomes glutathione. The only problem with the essential metabolite homocysteine is when it accumulates in excess. The most common reason excess homocysteine accumulates is a lack of methyl donors. Methylfolate can serve as one such methyl donor, but it is actually inferior in that role to many other one carbon donors such as betaine, methionine, vitamin B12, and dimethylglycine. From the hype regarding methylfolate, you would assume that this form of the B vitamin is the one essential way to lower homocysteine, yet it really is not a major player at all.

Furthermore, folacin that has been converted to methylfolate is incapable of performing the many other functions of the vitamin. Methylfolate actually performs exactly the opposite biochemical function as all other forms of the vitamin. In many of the essential physiological activities of folacin, the vitamin acts as a methyl acceptor. It is clear to see, therefore, that methylfolate, as a methyl donor, is incapable of performing all the zillion functions of the vitamin except for one.

Not only is methylfolate not effective in all but one of the zillion functions of folacin, supplementation with methylfolate can cause a deficiency of folacin in certain people. Anyone who is low in methionine for any reason will slip into what is called the “folate trap.” The essential amino acid methionine is so critical that, when necessary, folate will be diverted into the methionine synthesis pathway to the exclusion of other uses of folate. That folate trap can create a folate deficiency in any case, but in those who obtain a substantial percentage of their folate intake in the form of methylfolate, a folic acid deficiency, as well as an exacerbation of the methionine deficiency, is almost a certainty.

[Additional note: Folacin (folic acid, folate, PGA, pteroylmonoglutamate) does not cause cancer. There is some (but limited) evidence that consumption of folic acid in amounts 100 times the recommended daily amount may promote the growth of certain cancers. Also, since folacin is Anti-Dysaerobic in its metabolic effect it will stimulate the growth of advanced Anaerobic cancers.]