**BETAINE**

A. Betaine and trimethylglycine are synonyms. Betaine, as its alternative name trimethylglycine makes clear, is a trimethylated molecule of the amino acid glycine. With three bioactive methyl groups, betaine is probably the best methyl donor there is. You will find betaine in several NUTRI-SPEC supplements. --- It is found in the highest concentration in Diphasic A.M.

B. Betaine, folate, B12, and methionine all function as methyl donors throughout the body, particularly in the liver. The methyl donors have a direct effect at decreasing fatty liver. But, fatty liver is a multifaceted problem. There is both a Dysaerobic and an Anaerobic component to fatty liver.

The Dysaerobic component includes deficiency of betaine and glutathione, and an excess of HOHUM PUFAs. The HOHUM PUFAs (probably more so in the presence of betaine, glutathione and lipoic acid deficiency) so derange liver mitochondrial function as to contribute to build up of triglycerides in the liver.

The Anaerobic (and Ketogenic) component of fatty liver involves a deficiency of sulphhydryl groups, along with a deficiency of taurine and lipoic acid, and insufficient bile salts. Bile salts have a direct effect in minimizing fatty liver, and a secondary effect by activating thyroid function in the liver.

1) Many of these liver pathologies appear to have a Sympathetic component. Betaine has a very nice anti-Sympathetic effect in that it protects against Sympathetic-induced fatty acid oxidation. (In that regard, it opposes carnitine.)

2) In dealing with either hepatitis or cirrhosis patients, take all the above-mentioned Anaerobic and Dysaerobic (and Ketogenic/insulin resistant) effects into consideration as you evaluate the NUTRI-SPEC Metabolic Imbalances. You will give the QRG-indicated NUTRI-SPEC supplements plus an additional booster of betaine and lipoic acid (using Diphasic A.M.), plus an extra shot of Oxy D+ if the patient is leaning toward the Dysaerobic side, or, if Anaerobic, you will pump up the Taurine, bile salts, and Oxy A+, along with the Diphasic A.M.

3) Supplement with extra betaine in any liver pathology --- a large amount of betaine if the patient is Dysaerobic, a moderate amount if not Dysaerobic, and a small amount if Anaerobic.
C. No presentation on betaine would be complete without discussing another critical nutrient, SAMe. SAMe has astonishing biological activity. It has been shown to inhibit lipid peroxidation in the brain (which is crucial in preventing pathological brain aging). SAMe also enhances the endogenous glutathione anti-oxidant system throughout the body. SAMe is also a particularly crucial part of liver detoxification functions.

With all the benefits research shows can be derived from SAMe, this nutrient became a darling of the health food industry a few years ago. In fact, every pill peddler worth his salt came out with a SAMe product, and hyped it to the moon.

Where do you find SAMe in your NUTRI-SPEC products? You don’t. You see, SAMe is yet another case of blatant health food industry charlatanism. The dirty little secret that this pathologically dishonest industry is keeping from you is that SAMe is so unstable it cannot be put into a tablet or capsule. Even if the pill maker puts SAMe into the product, it is totally decomposed into components with no biological activity long before the pill reaches you or your patients. Even though the cat is out of the bag, the industry is still doing everything it can to keep the truth from being known. A few companies are quietly removing SAMe products from their catalogs, yet others will keep pushing the stuff as long as there are unsuspecting buyers they can separate from their money.

Why are we telling you the nasty story of the SAMe fraud? We are always quite happy to expose the irresponsible and dishonest behavior that typifies the health food industry – but in this case our motives go beyond that. You see, there is a way to supplement the body with SAMe, but it is not by taking preformed SAMe.

What you must do is take the SAMe precursor. And what might that be and where can you find it? It is the methyl donor betaine (also known as trimethylglycine) and is found in your Diphasic A.M. How close is the association between betaine and SAMe? Supplementation with betaine has been shown to double the SAMe level in the liver. Furthermore, in alcohol-fed laboratory animals (whose SAMe levels have thus been depleted) betaine supplementation raises the SAMe level by 500%, and, protects against fatty infiltration.

D. If betaine did nothing more than double SAMe levels, it would be a remarkable supplement and an indispensable addition to your Diphasic A.M. supplement. But, betaine’s benefits go far beyond its influence on SAMe. For instance, betaine is a powerful cholangogue. This means it facilitates the conversion of cholesterol into biliary acids in the liver for excretion through the gall bladder. The two obvious benefits are improved gall bladder function and a lowering of elevated serum cholesterol.
It has long been known that betaine so favorably influences the liver that it decreases the toxic effects of carbon tetrachloride and other powerful chemical poisons. Now it is known that the mechanism by which it does this is by actually increasing the number of mitochondria in liver cells.

Liver insufficiency = need methyl donors = choline, methionine, B15, and betaine. [Also benefit from iodine, calcium orotate, vitamin A, and bioflavonoids for cholesterol metabolism, and from lecithins to emulsify cholesterol.]

Betaine has another protective function – it protects the brain against the toxic effects of ammonia produced in the liver (but it does not do this as well as carnitine, another ingredient in your Diphasic A.M.).

E. The benefits of betaine extend from the liver to the cardiovascular system. Betaine will actually decrease the incidence of thrombo embolisms. Add this to its cholesterol-lowering effect, and you will see that it is invaluable in the protection against cardiovascular disease.

But wait – there is more. We have saved the best news on betaine for last. Consider this: There are only a few truly independent risk factors for cardiovascular disease. (Cholesterol, by the way, is not one of them.) One of the best independent risk factors for cardiovascular disease (as you have often heard us discuss) is serum triglycerides. There are only a couple of others, and the one that is the most significant by far (in other words has the greatest predictive value of a person’s risk for cardiovascular disease) is the serum level of homocysteine.

Not long ago, we had an Electrolyte Stress patient with a history of cardiovascular disease (including myocardial infarct), when reporting to his cardiologist for his semi-annual blood work, who requested, in addition to cholesterol and triglycerides, a homocysteine test.

[This, by the way, was a patient who after only three months under NUTRI-SPEC care was able to totally eliminate three out of the four medications he was taking after having had his heart attack, and the fourth medication, a beta blocker, was down to just two days each week. His blood pressures are maintained at perfectly normal. His clinostatic pulse response and orthostatic blood pressure response are perfectly normal. His cholesterol and triglycerides have come down to normal. And, he has more energy than he has had in years. All this is attributable to his NUTRI-SPEC regimen for an Electrolyte Stress Imbalance.]

Insisting on a homocysteine test at the request of his chiropractor/nutritionist did not win any points for this man from his cardiologist. In fact, the cardiologist took what could best be described as a
tantrum – ranting and raving that the homocysteine test was a total waste of time and money. When my patient pointed out that it was the best of the few independent risk factors for cardiovascular disease the cardiologist blurted out, “So what, what’s this guy think he is going to do about it anyway if it is high?”

Good question. And you, as a NUTRI-SPEC practitioner, have a good answer. Research has shown that betaine (when combined with just very small amounts of vitamin B6 and folic acid, as found in your Activator) will lower elevated homocysteine levels. Nothing else will. [See Addendum on homocysteine below.]

F. Betaine (trimethylglycine) is a quaternary ammonium compound which is biologically active as an osmolite, stabilizing osmotic pressure in cells.

- urea and betaine counteract each other/must be in balance

G. Betaine hydrochloride is a betaine molecule that is converted to a salt by adding hydrochloric acid. Betaine hydrochloride, as found in your NUTRI-SPEC supplement Proton Plus, is a powerful acidifier, supplier of hydrochloric acid as a digestive aid, and, it also contributes betaine, with its methyl donor activity, to the mix.

H. When phosphoethanolamine is elevated in plasma, but extremely low in urine:

1) Possible inhibition of choline and acetylcholine synthesis due to impaired methionine metabolism involving methylation by SAMe.
   --- Need B12, folate, and betaine

I. Betaine, and particularly its relationship to the whole methionine and glutathione series of metabolic pathways, is fascinating. Here are a few critical points:

Betaine, as you have just read, is by far the best way to lower elevated homocysteine. B6 and B12 offer nothing of lasting benefit, except in the existence of actual B6 or B12 deficiency. Folate does nothing unless there is a folate deficiency, but there can be a folate deficiency associated with a methionine deficiency.

Interesting relationship between folate and methionine: There is a phenomenon called the “Folate Trap.” When there is even a marginal deficiency of methionine, the body will convert folate to methionine. This folate trap can cause a functional folate deficiency at the cellular level even in the presence of normal blood levels of folate. Causes of the methionine
deficiency diverting folate into the methionine production pathway are many.

1) Methionine is a heat labile amino acid destroyed in cooking. Many people have a primary deficiency of methionine. (Methionine and the other heat labile amino acids are, as you know, found in your Activator -- just one of many reasons it is superior to all other multiple supplements.)

2) Methionine is converted to SAMe, which can be diverted into the catecholamine metabolic pathway. SAMe is consumed as norepinephrine is converted into epinephrine. So, patients with a Sympathetic Imbalance, or a Sympathetic compensation for a Glucogenic Imbalance can divert all their SAMe into the catecholamine pathway, thus depleting methionine. As NUTRI-SPEC practitioners, we must be very careful not to over-supplement with tyrosine or even phenylalanine, for fear of potentiating the catecholamine pathway and depleting methionine.

3) Paradoxically, it seems that homocysteine can be elevated even in this methionine deficiency state. The elevated homocysteine that sometimes (though not often) occurs may be due to the low functional folate.

4) The deficiency of methionine, and sulfur-containing amino acids in general, directly decreases the production of glutathione. Anything that depletes methionine (or sulfur amino acids in general) will also deplete glutathione.

5) An interesting fact about a vitamin B12 deficiency is that the body mistakenly interprets the vitamin B12 deficiency as a methionine deficiency. In a desperate attempt to increase the perceived low methionine, the several pathways of folate conversion to methionine are activated inappropriately.

J. Now that you have seen the story on the wonders of betaine, you appreciate even more how nothing can do more to protect your patients against both pathological hyperplasia and pathological disintegration – the two phases of pathological aging – than your Diphasic Nutrition Plan can.

K. [Addendum --- Homocysteine]

“How does homocysteine fit into the picture of age-associated inflammation?” --- Thyroid insufficiency causes elevated homocysteine (and elevated prolactin) that decrease to normal after correction of hypothyroid. Elevated homocysteine is associated with inflammation. There seem to be many mechanisms involved with homocysteine’s association with inflammation, but here are some choice fragments of truth:
1) Homocysteine blocks the metabolism of vitamin A. (This failure of vitamin A metabolism explains a connection between homocysteine and birth defects. --- Ironic, isn’t it, that there is such an irrational fear of vitamin A supplementation in pregnancy, when it is actually vitamin A deficiency, not vitamin A excess that is associated with birth defects?)

Low vitamin A, just like low vitamin D, is associated with excessive Th1 immune activation. Excesses of the Th1 inflammatory cytokines such as TNF-α, IFN-γ, IL-1 and IL-2, are associated with many autoimmune diseases, including particularly Hashimoto’s autoimmune thyroiditis and rheumatoid arthritis and Type I diabetes.

2) The association between homocysteine and immune activation appears to involve a positive feedback loop. Elevated homocysteine activates the immune system, but immune system activation also increases homocysteine production.

3) Some autoimmune conditions or pathologies involving ImmunoNeuro-Endocrine stress such as idiopathic Parkinson’s are associated with enteric-nervous system inflammation/damage. Serum homocysteine is elevated in 43% of idiopathic Parkinson subjects, and the elevated homocysteine is not entirely explained by low serum vitamin B12. But, we recall that 70-75% of the immune system is in the gut mucosa, and 60% of idiopathic Parkinson subjects are hydrogen breath test positive for small intestine bacterial overgrowth. It seems that the increased bacterial utilization of vitamin B12 (as opposed to a deficiency of B12 production) is responsible for a functional vitamin B12 deficiency and subsequent elevated homocysteine.