

# Surprising Butyrate Study

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We've discussed the four carbon fatty acid called Butyrate on earlier Tuesday Minutes and explained Butyrate as the primary nutrient that provides energy to colonocytes which allows them to function at peak performance. Some people are aware that Butyrate also has anti-inflammatory, anti-oxidative, anti-neoplastic, and antimicrobial properties. But there is much, much more to the butyrate story.

A study from Nature.com showed how Butyrate induced T-regulatory cells (Tregs) and how they migrated from the Gut Associated Lymphoid Tissue, also called GALT, to the pancreas and restored immunological tolerance during type 1 diabetes. WOW that's a mouthful and we need to unpack that a little bit because the implications are profound.

I like to think of Tregs as the conductor of the immune system as they have been shown to modulate autoimmunity. Dr. Vasquez in many of his books and webinars has shown that a down regulation of Tregs tips



the scales toward TH1, TH2 and TH17 hyperactivity. Increases in TH17 dramatically increase the chances for autoimmunity.

He has shared how deficiencies in Vitamins D and A, Green tea, probiotics and omega 3 fatty acids and GLA hamper the development or population of Tregs. Now we can add Butyrate to this list. But what I found interesting is not only did Butyrate stimulate the production of Tregs via the GALT system but that they migrated to the pancreas to help regulate function for type one diabetic mice.

I know we are talking about mice but please get the big

picture. Here is one more study showing how the gut is affecting a systemic phenomenon like Type 1 diabetes. The gut, the gut, the gut; how many ways, how many times do we have to hear this message. People laughed at VE Irons and Bernard Jenson when they said “Death begins in the Colon”, I don't think any of us had any idea about the depth of that reality.

Let's hold that thought for a moment as we shift gears to a human study; Butyrate: implications for intestinal function. 1800 mg of butyrate was given with meals for 60 days. In this double-blind,

placebo-controlled study, 49 IBD patients were randomized to oral administration of sodium-butyrate or placebo. Of the IBD patients, 19 had Crohn's disease, and 30 had ulcerative colitis. 18 healthy volunteers were recruited and given starch capsules to provide a healthy microbiota model. As you would expect, at baseline, healthy volunteers showed a healthy diversified microbiota composition compared with IBD patients. Butyrate altered the gut microbiota of IBD patients by increasing bacteria able to produce Short Chain Fatty Acids in both ulcerative colitis patients and Crohn's patients although the bacteria in each were different.

This was the first time that it was shown that butyrate administration promoted the growth of bacteria able to increase the production of butyrate. Dr. Vasquez said it a little differently. He felt the mechanism of action in this study included at least two factors:

First that butyrate reduces inflammation and therefore creates an anti-inflammatory climate which is favorable for the growth of beneficial bacteria. Second, by reducing inflammation, butyrate probably creates a less favorable climate for pathogenic bacteria and therefore the benefits could be mediated simply by a reduction in the number of pathogenic competitors.

One more quick study, a 2014 article in Nutrition Metabolism Cardiovascular Disease titled "Butyrate Impairs atherogenesis by reducing plaque inflammation and vulnerability and decreasing NFkB activation." Authors concluded "Oral butyrate is able to slow the progression of atherosclerosis by reducing adhesion and migration of macrophages and increasing plaque stability. These actions are linked to the reduction of CD36 in macrophages and endothelial cells, decreased pro-inflammatory cytokines and lower activation of NFkB.

All of this data supports a possible role for butyrate as an atheroprotective agent." In that same light, Dr. Mark Houston has shared that damage to the endothelium and ultimately

vascular disease of all kinds have an infinite number of insults.

But here is a big key, even though the assaults are many, there are three finite responses; inflammation, oxidative stress and an overactive immune response. And as we have seen, in this Tuesday Minute and others, butyric acid supports gut repair and the health of the microbiome which in turn supports the health and healing of all three vectors; inflammation, oxidative stress and an overactive immune response. SCFA's especially Butyrate is produced by the microbial fermentation of dietary fiber. So we need healthy fiber as well as a healthy microbiome.

Butyric-Cal-Mag from Biotics Research was one of the first butyric acid supplements commercially available. 2 capsules of Butyric-Cal-Mag supply 1033 mg of butyric acid, 975 mcg RAE of vitamin A as natural mixed carotenoids and acetate, 90 mg of calcium and 77 mg of magnesium. Therapeutic dose is 2 capsules three times a day and reducing to 1 tid in 60 days.

I know we have covered a lot of ground with these 3 totally different studies. But I hope you are making the connection that supporting the gut and healthy Treg production is the first step to overcoming many of the chronic illnesses that we see today. Thanks for watching, I look forward to being with you again next Tuesday.