

# Propionate, A Common Food Preservative, Alters Our Metabolism. Does That Make It A Disruptor?



By *Chuck Dinerstein* — April 24, 2019



Our these croissants full of an endocrine disruptor? Image by skeeze from Pixabay [1]

The rise in obesity and diabetes has many causes, genetic, lifestyle, and of course, our dietary choices especially a perennial villain, sugar. A new study in mice with a pilot study in humans suggests that a naturally occurring compound, found in cheese, and used as a food additive may play a role in altering our metabolism. The additive is propionate or propionic acid; a compound used predominantly in bakery products as a mold inhibitor. Science aside, it raises some interesting questions about generally recognized as safe or GRAS food additives. But first, let's look at what they are reporting.

Propionate is a short chain fatty acid (SCFA) converted by our metabolism into glucose. Some SCFAs are felt to be helpful, especially those that result from fermentation of undigested carbohydrates by our microbiome; but other studies suggest that they are associated with weight changes. We have studied this chemical for over 100 years, and initial studies dating back to 1912 found that the amount of glucose created by ingesting propionate is about 20 times more than expected based on the chemical reactions. In this current study, scientists, using a series of well-conceived experiments were able to tease out the source of that additional glucose at least in our fellow mammal, mice. The found that

- Propionate elevated blood glucose in a dose-dependent fashion when taken orally.
- Much of the “additional” glucose came from glucose production in the liver; gluconeogenesis is the biological term.
- Gluconeogenesis was aided by a rise in glucagon, a hormone that stimulates glucose

production; and by FABP4, a protein that facilitates the movement of propionate, among other fatty acids, in our fat tissue.

- Propionate's action was indirect. For reasons that are not well understood, propionate stimulated our sympathetic nervous system, at least as measured by the release of norepinephrine; which in turn increased glucagon and FABP4.

Quite a bit of metabolic science in those bullet points. To summarize, propionate increases glucose by stimulating our sympathetic nervous system to produce more glucagon and FABP4. So much for our mammalian cousin, but how does it affect humans?

Using a placebo-controlled, double-blinded crossover study in 14 lean humans, they found that a “very low dose of propionate” liberated norepinephrine, resulting in increased glucagon, and FABP4. There was also a late rise in blood glucose, consistent with the time necessary for all these metabolic actions to take place. They concluded that propionate was a metabolic dysregulator – I suppose that is similar but not quite the same as an endocrine disrupter.

The first limitation is, of course, the small sample size of humans. I am not sure that 14 individuals give us anything more than a metabolic impression. The second limitation is related to whether the amount of propionate ingested was it, in fact, a “very low dose?” The reference they cite is a study on propionate in sourdough bread, so I am not sure of its applicability. The FDA’s determination of how much propionate we might ingest daily is 10 – 20 fold less than the 1 gram they gave to their human subjects. [1] So while propionate has metabolic effects mediated through our neurohumoral axis (the sympathetic nervous system), I am not sure these metabolic changes reflect dysregulation and certainly not in the amounts we usually eat. You might see the same effect eating swiss cheese where propionate is a natural fermentation product, not an additive.

## GRAS

A more interesting consequence of the paper is how our increasing knowledge of metabolism as a dynamic process, often referred to as metabolomics, could alter our understanding of generally recognized as safe. Propionate was reviewed by the FDA in 1979 and based upon the expert panel’s review

*“There is no evidence in the available information on propionate that demonstrates, or suggests reasonable grounds to suspect, a hazard to the public when they are used at levels that are now current or might reasonably be expected in the future.”*

[2]

More specifically, there was no significant oral toxicity, mutagenesis, teratogenicity, short-term or long-term effects. Metabolomics and metabolic studies like this one, were not possible when propionate was determined to be GRAS. Should we reconsider the designation based on these findings? Should we begin to point the causal finger at propionate as the source of obesity and diabetes. Despite the murmuring of press releases, this is an interesting study, not a smoking gun or a call to arms. Before we throw out all the baked goods containing propionate or require new labeling, we should do a study involving more than 14 humans and two meals. For the moment we

will have to content ourselves that unless it is inert, everything in our food interacts with our metabolism and changes in metabolic products concentrations or the timing of their action is not dysregulation or disruption.

[1] The FDA determination was made in 1979 and was based on a market survey suggesting a daily intake of 45mg as well as calculations based on the amount of propionate purchased by bakeries, 30mg, and an estimate based on the amount of propionate sold by manufacturers, 83mg.

[2] [Report 79](#) [2]by the FDA Here is a much fuller statement

*“Propionic acid occurs naturally in various foods including butter and cheese. ... it is a normal intermediary metabolite. ...demonstrated low acute toxicity after oral administration to mice or rats...Microbial assays for mutagenicity of propionic acid and calcium and sodium propionate were negative. Investigations of the teratogenicity of calcium propionate in four mammalian systems also were negative. Short-term feeding tests show ...adverse effects on weight gain [weight decreases, not increases] only when propionate intakes are many orders of magnitude greater than the estimate of human dietary intake of propionate used as a food ingredient, about 1 mg per kg per day. Longterm feeding studies of propionic acid and calcium propionate have not been reported. However, a long-term feeding study of sodium propionate showed no adverse effects in rats.”*

Source: The short-chain fatty acid propionate increases glucagon and FABP4 production, impairing insulin action in mice and humans Science Translational Medicine DOI: 10.1126/scitranslmed.aav0120

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[1] <https://pixabay.com/photos/croissants-breakfast-baked-goods-1916425/>

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