Liposaccharides (LPS), Metabolic Endotoxemia, and the Megaspore Study

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So here's a bunch of other things. Leptin resistance. When this LPS is allowed to get into your enteric nervous system, that enteric nervous system is a nervous system that covers your entire digestive tract. It has some of the most dense nerve endings, even compared to the spinal cord itself. And so it's a really, really elaborate and significant neuronal system. And when LPS enters that neuronal system and disrupts the neuronal signaling, you end up with a gut brain communication disruption, which can lead to things like leptin resistance. Leptin resistance means that you end up gaining weight more easily because one of the things that leptin is supposed to do, once you eat food, leptin increases in concentration because your microbiota produces signals, like short chain fatty acids, to get your body to produce more leptin.

As leptin increases than what you start to see is your satiety starts to increase and hunger goes way down. So it forces you to stop eating. But when leptin is not functioning when we are immune to the response of leptin because of continuous overeating, continuous leaky gut, continuous inflammation, and the disruption between the gut brain connection, we tend to chronically overeat and end up with metabolic syndrome and obesity. Chronic constipation works the same way. That enteric nervous system also controls your bowel movements. And so if the enteric nervous system is shot and not allowed to function the way it's supposed to, your bowels aren't going to move the way they're supposed to. Mood and appetite disorders. It can also disrupt ghrelin, which is the hunger hormone. Also dopamine in the case of depression. So this LPS can actually get into parts of your brain where it interferes with dopamine receptors.

So you don't have that neurological response or dopamine, other cognitive declines because they can cross a blood brain barrier and cause inflammation in that region itself, which causes cognitive decline. You can cause loss of memory by entering places like the hippocampus and the amygdala and causing inflammation in those deep recesses of the brain. Those deep areas control things like memory. And so when inflammation sets in, in those areas of the brain, you're going to have issues with memory recall and ability to understand things, analytical thinking and so on. Depression, it can be triggered through disruption of serotonin turnover. Anorexia nervosa also through serotonin turnover in the synapsis of the CNS. Anxiety, which it can do through messing up the HPA axis, the Hypothalamic Adrenal Pituitary axis, increasing the expression of corticosteroid releasing hormones. So you end up with more stress hormones in response to everyday things and that can create severe anxiety, chronic pain.

It can actually get into neurons, into these things called nociceptors and trigger pain signals, which a lot of people who suffer from severe inflammation and illness, they also end up with chronic non-specific pain. Parkinson's disease. I mentioned Alzheimer's. This is also the major inducer of Parkinson's disease. Intracranial LPS. So migrating from the lumen of the gut, past the barriers, into the circulatory system, and entering the cranium and can cause microglia activation, so immune cell activation, that ends up causing neuronal loss and that leads to the onset of Parkinson's. Hypogonadism, low testosterone in men, high levels of serum LPS associated with low testosterone, and of course autoimmune disease like we talked about as well. So at the end of the day, this LPS moving through, being allowed to move through from the lumen because you have a dysbiotic microbiota, because you have a dysfunctional mucosa, and you have a disrupted intestinal barrier, on a regular basis especially when you eat food, this LPS is allowed to migrate through and enter the circulatory system and it causes all kinds of havoc.

So when we were looking at total gut restoration, our first focus was can we stop this LPS from migrating through? And that's where megaspore came in. So we designed this product to be able to stop that LPS from migrating through and it does it through several mechanisms, but at the end of the day what we're seeing is an alleviation of the leaky gut. So let me show you a little bit of data. We recruited somewhere around close to 100 college students, screened lots of them for this leaky gut response to

eating a meal so they would be responders versus non-responders. Non-Responders means they did not have a leaky gut response to the meal, responders mean that they did have a leaky gut response to the meal, and those that did have the leaky gut response were entered into the study.

Now it's interesting to note that around 50 to 55% of healthy young individuals with no frank disease, no illnesses had this severe leaky gut response and this is what you see. At baseline when we measure their serum LPS levels, this endotoxin from the gut, you tend to have very low circulating levels that don't really cause that much of an issue. But once we feed them a meal and in the next five hours we see this five to six X increase in serum LPS. So now what we've had is a five to six fold increase in the amount of LPS that's now in their circulatory system. This now causes a huge amount of inflammatory response in the body and that same kind of CD14 inflammation that we talked about earlier, that's exactly the kind of inflammatory response that it sets off.

Now we took the people that had the severe inflammatory response and we gave them probiotic for 30 days and after 30 days, we had almost a complete blunting of the inflammatory and the LPS response. So what this is showing us at the spores is somehow preventing the LPS from migrating from the lumen, past the mucosal, past the intestinal barrier, and into the circulatory system.