



Research Summary: SIBO #1

As featured in Dr. Kenny Mittelstadt's video:
"What Causes SIBO to Keep Coming Back? (Root Cause Blueprint)"
Date of Publication: 11/10/2025

Research Context:

This week's topic explores how SIBO isn't really about bacterial overgrowth. It's about three interconnected systems that break down and invite that overgrowth to occur in the first place. Below are the key studies that help connect these dots and show why treating bacteria alone, whether with antibiotics or herbal protocols, might be missing the bigger picture of barrier integrity, motility dysfunction, and immune resilience.

Key Findings from the Research:

Study 1 (PMID 37051344):

This comprehensive review looked at how gut barrier breakdown, what is colloquially called "leaky gut," connects to both digestive and non-digestive diseases. The researchers found that when the tight junctions between intestinal cells loosen up, food particles and bacterial toxins can cross into places they shouldn't be, triggering immune reactions and inflammation. What's especially interesting is that this barrier dysfunction doesn't just happen in isolation. It's foundational to conditions like dysbiosis. The barrier actually breaks down first, creating the environment where bacterial imbalances can take hold. The research also showed that specific foods and nutrients can either promote gut barrier health or make it worse, which means dietary intervention could become a personalized tool for disease prevention. This study helps explain why you can't just antimicrobial your way out of SIBO. You have to seal the barrier first.

Study 2 (PMID 30134201):

This study dug into how your gut's immune system uses IgA antibodies. Think of them as little protective flags waving on the surface of your intestinal lining. These antibodies are your gut's way of identifying what's friend versus foe. The researchers found that a healthy microbiome produces signals that enhance this IgA response, giving you stronger immune resilience in your digestive tract. When your microbiome shifts into dysbiosis, these protective signals get dampened, and your immune interface weakens. This is where bacteria that aren't supposed to be in your small intestine can start to thrive. What's fascinating is that there are two types of IgA responses happening at once... one that broadly manages your normal microbiome residents, and another that responds specifically to pathogens. This research shows us that the gut-brain-immune connection isn't just metaphorical. It's a measurable, active communication network that either supports or sabotages your ability to keep SIBO at bay.

Study 3 (PMID 35145413):

This paper reviewed treatment options for functional gastrointestinal disorders and motility problems, and the findings are eye-opening. The researchers found that these disorders are heterogeneous and interconnected with multiple mechanisms: altered gut motility, intestinal barrier dysfunction, gut immune dysfunction, visceral hypersensitivity, changes in digestive secretions, microbial dysbiosis, and changes to the gut-brain axis. Sound familiar? These are the exact three systems we're talking about with SIBO. The challenge they identified is that most current treatments only target symptoms rather than the underlying mechanisms, which is why people experience temporary relief but then relapse. The study emphasized that cause and effect get tangled up when we only treat symptoms. If your motility is slow, bacteria accumulate, but bacteria can also slow down motility. This creates a self-perpetuating cycle. The research points to the need for treatments that address the cellular and molecular mechanisms driving these patterns, not just the surface-level symptoms.



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Functional Medicine Connections:

Here's how these pieces fit together: Your small intestine sits at the intersection of three major communication networks in your body... the barrier system (what gets in and what stays out), the motility system (how things move through), and the immune-digestive capacity system (how well you break down and respond to what you eat). When stress, inflammation, food poisoning, medications, or chronic digestive burden hits your system, these networks start breaking down in a specific order. The barrier loosens first, allowing things through that trigger immune responses. Your motility slows down, so bacteria and partially digested food stick around longer than they should. Your digestive enzymes, bile, and stomach acid production drops, leaving more undigested carbohydrates for bacteria to feast on.

These three systems talk to each other constantly... barrier inflammation affects motility, poor motility damages the barrier indirectly, and low digestive capacity means you can't even absorb the nutrients needed to repair the barrier in the first place. This is why SIBO isn't a bacterial problem. It's a systems communication breakdown. The bacteria are just the messengers showing you where the real problem lives.

Practical Reflections & Takeaways:

Think about your own SIBO pattern: Did it start after food poisoning, a stressful life event, or a round of antibiotics? Did you develop new food sensitivities around the same time your digestive symptoms began? Do you notice that your symptoms get worse when you're stressed or not sleeping well?

These aren't random coincidences. They're your body showing you which of the three systems went offline first.

If SIBO comes back after treatment, ask yourself: Did I stop having regular bowel movements? Did a major stressor hit? Did I stop supporting my barrier or digestive capacity? Your lived experience is meaningful data. The symptoms you notice, the timing of flares, and what makes things better or worse are all clues pointing you toward which system needs the most attention right now.

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