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What Is Akkermansia — A Keystone Microbe in Your Gut (An Interview with Dr. Colleen Cutcliffe)

Analysis by Dr. Joseph Mercola | July 26, 2026

STORY AT-A-GLANCE

- The composition of your gut microbiome may have an encompassing influence on your health, including metabolic, neurological, and digestive function
- The diversity of your microbiome varies depending on your diet, age, location, and other factors. Age-related loss of diversity may be a meaningful contributor to age-related changes in function and the onset of chronic conditions
- In conventional antibiotic treatment, roughly 20% to 30% of Clostridium difficile cases recur after initial therapy, and severe or refractory cases carry meaningful mortality risk. Fecal microbiota transplant (FMT) has been reported in some trials to have cure rates around 80% to 90%
- Akkermansia muciniphila is among the gut bacteria most studied as a key contributor to microbiome health
- Reduced integrity of the mucin layer has been associated with what researchers describe as leaky gut syndrome. Akkermansia muciniphila is among the strains most closely studied for its interaction with the mucin layer

The video above features an interview with Dr. Colleen Cutcliffe, a molecular biology scientist and the CEO and cofounder of Pendulum, a company creating microbiome products. This interview was done as part of my early explorations into the obligate anaerobe microbiome field. I was particularly drawn to Dr. Cutcliffe's work with Akkermansia.

How Your Gut Microbiome May Influence Health

Yogurt and probiotics have been popular for decades, but what really catalyzed interest in the human microbiome was the advent of DNA sequencing. This allowed scientists to begin identifying and differentiating the various microorganisms found in the human body — along with the introduction of fecal transplants. As explained by Cutcliffe:

> *"One of the interesting things that people realized was that the gut microbiome has the ability to change symptoms and change diseases. This discovery largely came about as a result of research into fecal microbiome transplants (FMT), where you take feces from a healthy person and place it into a sick one.*
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> *That may sound really disgusting until you know that it actually has incredible medical value. Some of the first places where these fecal microbiome transplants were used were for treating Clostridium difficile infections.*
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> *[When] you take an antibiotic, it kills off most of the bacteria in your gut. This strain called Clostridium difficile now has no competitors, so it can start to divide and replicate. When it gets to high levels, it becomes extremely toxic and ultimately fatal.*
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> *What they found was that if you transplanted stool into these people who were suffering from this infection, and you basically inundated the microbiome with all these new competitors and were able to swamp out [C. difficile], you could save a person's life better than the therapies that were being used."*

In conventional antibiotic treatment of C. difficile, recurrence has been reported in roughly 20% to 30% of patients, with materially higher rates in severe or refractory cases. FMT cure rates reported in published trials vary widely, but are generally in the range of 80% to 90%. Despite these positive outcomes, FMT has still not become standard of care for C. difficile infections.

Small, early-stage studies have explored FMT in other contexts as well, including metabolic, gastrointestinal, and neurological conditions, though the evidence base is preliminary and quality varies considerably. That said, a key, open question is: which of the many microorganisms transferred are responsible for the observed effects?

Potential Safety Concerns of FMT

Important safety questions remain, as there's much we do not yet know about the microbiome. For example, a pathogenic microorganism that has not yet been fully characterized could be inadvertently transferred via FMT.

Your microbiome is made up not just of bacteria, but also viruses, bacteriophages, fungi, and yeast, and we don't yet understand exactly how all these organisms affect human health, let alone their interactions.

What's more, your microbiome changes depending on location, so the microbiome of someone living in the U.S. will not be the same as that of someone living in India or Norway. The reason for this is because your microbiome is heavily influenced by diet, medical interventions, toxic exposures, and the natural environment, all of which vary from one person and location to the next.

To address some of these issues, companies are now working on developing manufacturing processes to survey and clean up the stool, and then deliver the final product in pill form.

> *"There are several companies out there that are doing this kind of an intervention, which I think is a little bit safer than the pure stool,"* Cutcliffe says.

Surveying the Human Gut Microbiome

Many scientists are now working on surveying the gut microbiome. It started with the American Gut Project, where academics from around the U.S. surveyed all the different parts of the human microbiome, not just the gut microbiome but also the microbiome in

your nasal passages and ears, and on your skin.

"They're trying to understand, what does the population look like? One of the really interesting high-level themes that's come out is that the more so-called advanced or industrialized a country is, the less diverse their microbiome.

You can go into populations in the rainforest, where they've been relatively untouched by Western civilization, and they have incredibly diverse microbiomes, meaning they have lots and lots of different types of strains that are doing lots and lots of different activities.

As you get to more industrialized countries, like the United States, we have a less diverse population of strains. One of the other interesting things is that if you look at an individual as they age, younger people have a more diverse microbiome compared to people who are older.

So, one of the overriding theories is that this loss of diversity represents a loss in functionality that is tied to reduced health, and potentially even perpetuating disease. And so, can you give these [microorganisms] back and help people improve disease?

For example, as people age, many experience new food sensitivities that they didn't used to have, or they experience more GI distress more frequently than they used to. Unfortunately, many feel like there's nothing they can do about that; that it's just part of aging. But that's not true. It's [due to] this depletion in the microbiome, and there is something that you can do about it.

Ultimately, all of these guys are housed inside your body, so you are in complete control over who's living, who's dying, who you're feeding through the foods that you're eating. You can get to a point where you're sensitive to these foods because you've lost [certain] strains in your gut, but you can replenish them and get back to eating those foods."

The traditional strategy has been to improve your diet, eat more real food. This will work over time, but it's a slow process that can take many months. A more efficient strategy would be to repopulate your gut with essential strains in addition to the appropriate dietary changes. The question is which strains matter most.

What Is Akkermansia and What Does It Do?

Since it was first described 20 years ago, considerable research has been published on Akkermansia and its possible roles in health. Much of Cutcliffe's work has focused on this bacterium, given the breadth of research interest in it. "Akkermansia is quickly becoming known as a keystone strain in your microbiome," she states.

While the microbiome is an ecosystem of many strains, certain strains like Akkermansia appear to play disproportionate roles. Akkermansia was first described in 2004 by Derrien and colleagues in a study published in the International Journal of Systematic and Evolutionary Microbiology.[1] Subsequent research has examined associations between Akkermansia levels and metabolic markers, including body weight. Here are examples:

- **Associations with glycemic status —** Observational research has linked low or absent Akkermansia levels to populations with prediabetes and Type 2 diabetes.
- **Interacts with the gut mucin layer —** Researchers have found through both human and animal studies that Akkermansia is a key strain associated with maintenance of the mucin layer.[2] Cutcliffe describes it as "the 'glue' that keeps your gut lining strong." She further explains:

> *"You have these epithelial cells and the junctions between them are held together by glue, which is called mucin. When the mucin layer gets too thin, you lose those tight junctions, and that's where you can start to get things moving across that boundary that are not supposed to move across it.*

So, it's important to have a strong gut lining and Akkermansia is one of the strains we know of that is there at the mucin layer, both interacting with and helping regulate that layer. That's why it's of interest in research on a range of conditions."

- **Akkermansia and the gut-immune axis —** Researchers have hypothesized that reduced mucin-layer integrity may contribute to immune dysregulation in some contexts. This remains an active area of investigation. Akkermansia helps support mucin-layer integrity, which in turn may influence what crosses the intestinal barrier.
- **Akkermansia and gut-barrier function —** When the mucin layer is thin, larger molecules may cross more readily. Some research suggests that supporting Akkermansia levels may help support mucin-layer function and, by extension, gut-barrier integrity.

How Akkermansia May Influence GLP-1 Signaling

Drug companies are now promoting injectable glucagon-like peptide 1 (GLP-1) agonists for weight loss, and these drugs can have troublesome side effects. Researchers have noted overlap between GLP-1 agonist activity and pathways involving Akkermansia, though the mechanisms and contexts differ substantially. Cutcliffe explains how Akkermansia is thought to influence GLP-1:

"When it was observed that people with Type 2 diabetes or prediabetes were low in Akkermansia, it was believed that it was because of this mucin deficiency. But as people started to study Akkermansia more, and the microbiome in general, what's become clear is that it's a lot more direct than just the mucin layer.

What happens in your body naturally, if you've got all the right microbes, is that you eat a meal, your microbiome metabolizes that food and generates postbiotics [excretions from beneficial bacteria] like butyrate [and] a protein called P9. Some of these postbiotics then signal your body to produce GLP-1.

All that signaling is happening from the microbiome directly to the L cells. And so you eat a meal, your microbiome digests them, these postbiotics get created and tell your L cells, 'Hey, go produce GLP-1,' and then you get a spike in GLP-1 in your body.

GLP-1 stimulates your body, too. It says, 'We've got to metabolize the sugar in the bloodstream, release insulin.' It also signals to your brain, 'We just ate, we're full, we don't need to eat again.' After a period of time, GLP-1 goes down — until the next time you eat a meal. Then it spikes again.

So, that's the natural way of things. There are only two strains that have been published, to date, that have been shown to be able to stimulate L cells to produce GLP-1, and one of them is Akkermansia. It actually secretes three different [postbiotics] that stimulate L cells to produce GLP-1.

So, what's been found is that if you are low or missing Akkermansia, your body is not naturally producing as much GLP-1 as it's supposed to be. By giving people back Akkermansia, you can now have these physiological benefits of reducing A1C and lowering blood glucose spikes.

To be clear, the natural GLP-1 you produce is different from the drug. The drug is a mimic. It's an analog. It looks like GLP-1. It gets injected into the bloodstream directly, which means that rather than the natural spike after you eat [followed by a decline], the [drug] is keeping those levels really high all the time.

So, this signaling of 'we got to metabolize sugar in the blood and we're full, we just ate' is going on constantly. That's why people experience these incredible, amazing overnight effects because that's how those drugs are working. But if you actually have the right microbes, you can generate your body's natural GLP-1 and get back into this natural cycle."

Akkermansia and Food Cravings — Emerging Research

Early research has examined whether Akkermansia supplementation may influence food cravings, with some studies reporting effects on sugar-related cravings.

> *"What that tells us is that your microbiome has the ability to change your cravings, and that can really guide you to better eating habits and then, in turn, replenish the good microbes,"* Cutcliffe says. *"So, you end up being on a good cycle and getting off of that bad one.*
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> *To reiterate, the GLP-1 drugs are not what we're talking about with the microbiome. They're two different things. One is your body's natural way to increase GLP-1. The other is a synthetic drug, and we are in no way suggesting that [Akkermansia] is a drug.*
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> *When you do it naturally, you're not going to see that immediate overnight result because it's not going to be hammering your body with high levels of GLP-1 signaling all the time. It's going to do it the way your body naturally does it, which is that you eat a meal and then your body tells you we're full. Over a period of time you will start to see the benefits. So, it's not going to be an overnight change, but it will be a sustainable way."*

Probiotic Potency Explained: CFU, AFU, and TFU

When evaluating the potency of probiotics, there are three units of measurement you need to be aware of: colony forming units (CFU), active fluorescent units (AFU), and total fluorescent units (TFU).

- **Colony forming units (CFU) —** This is the most widely recognized and utilized metric for quantifying the number of viable bacteria or fungal cells in a probiotic product. One CFU represents a single microorganism capable of dividing and forming a colony under specific laboratory conditions. This measure is important because the activity of probiotics is associated with the number of live microorganisms that reach your gut.

Probiotic manufacturers typically list CFU counts on product labels, indicating the number of live organisms per serving. Higher CFU counts are often marketed as more potent, though the optimal CFU level can vary depending on the specific strains and the health context.

Consumers are also advised to check that the CFU amount listed on the label is specified as the CFU level at the end of shelf life (its expiration date).[3] As noted by The Probiotics Institute,[4] "The amount of probiotic (CFU) present on the 'manufacturing date' is not as important as the amount present at the 'end of shelf life.'"

- **Active fluorescent units (AFU) —** This unit is a less conventional and not widely standardized measure in the context of probiotics, with the exception of Akkermansia. While CFU shows the number of bacteria that are alive, AFU refers to the total number of bacteria present, both dead and alive. It is primarily a unit used to measure enzymatic activity.

  For instance, AFU could be used to evaluate the activity levels of specific enzymes produced by probiotics, which contribute to their function, such as breaking down lactose or producing vitamins. In some specialized applications, AFU is also used to assess the metabolic activity or functional potency of probiotic strains beyond mere viability.

  Most companies that sell Akkermansia probiotics use AFU instead of CFU, and there's a scientific reason for that. Akkermansia is a strict anaerobe and as such it plate-counts poorly under standard probiotic quality control conditions. Many viable-but-non-culturable (VBNC) cells aren't captured by CFU even though they're metabolically active.

  Flow cytometry (AFU) was developed in part to address this conundrum. It labels cells with fluorescent dyes that distinguish intact membranes (live) from compromised ones, and counts each cell as it passes through a laser. In short, flow cytometry captures VBNC cells that plate counts miss.

- **Total fluorescent units (TFU) —** This unit measures the total bacterial mass including both live and dead cells through fluorescent labeling, and is typically used only for pasteurized products. Like AFU, TFU values are higher than CFU counts for the same sample since they include both viable and non-viable cells.

The primary difference between CFU, AFU, and TFU lies in what they measure: CFU quantifies the number of live microorganisms; AFU assesses the functional activity of those microorganisms; and TFU measures the total bacterial mass, regardless of their functional activity. While CFU is an indicator of the potential for colonization and survival of probiotics in the gut, AFU could offer additional insights into the functional capabilities of the probiotic strains.

Current Akkermansia Clinical Trials: Dosages and Applications

As research advances, numerous clinical trials are underway to evaluate the efficacy and safety of Akkermansia-based interventions.[5] Published data in 2024 investigating Akkermansia have reported preliminary results,[6] highlighting its potential across a range of health conditions, including infectious disease,[7] immune-related disease,[8] liver fibrosis,[9] stress management,[10] intestinal-related diseases,[11] metabolic health,[12] and brain function.[13]

These studies, which include both animal and human trials, have used therapeutic doses ranging from 100 million to 10 billion CFU per day.

In studies of obesity, Type 2 diabetes, and metabolic syndrome populations, doses around 10 billion CFU per day have been used. Researchers have examined effects on markers such as insulin sensitivity and glucose metabolism, with study sizes and durations varying.

Conversely, lower doses around 1 billion CFU per day have been examined for more gut-specific conditions and liver health,[14] and even at these reduced levels, some studies report reductions in markers of intestinal inflammation.

**These findings are from research conducted in clinical settings, often in small or proof-of-concept trials. Results may not apply to all individuals.*

Akkermansia Delivery and Form — What the Research Suggests

When choosing an Akkermansia probiotic, bacterial counts in the billions are typically used in published trials, though dose alone is not the whole story — the delivery method appears equally important.

For live formulations, delayed-release capsules and microencapsulation have been designed to help bacteria reach the colon intact. Without such protection, much of the live bacterial load may not survive the journey through the digestive tract.

Akkermansia is highly sensitive to oxygen, and these microbes thrive in oxygen-free environments, which makes the journey through the digestive tract challenging. Even a brief exposure to oxygen can be fatal for live cells, which is why formulations with delayed-release capsules or microencapsulation have been designed to protect the bacteria until they reach the colon.

Theoretically, a lower-dose probiotic that successfully reaches the colon may be more effective than a higher-dose product that does not, which is why the delivery method may be a far more important consideration than the labeled bacterial count.

If you are considering an Akkermansia supplement, formulations with timed-release capsules or microencapsulation have been designed to keep Akkermansia protected until it reaches the colon, typically within two to four hours.

Frequently Asked Questions About Akkermansia

Q: Does Akkermansia help with weight loss?

A: Research has examined and found associations between Akkermansia levels and metabolic markers, including body weight, appetite-related signaling, and gut health.

Q: Is Akkermansia safe?

A: Akkermansia is naturally present in a healthy microbiome. Available studies on Akkermansia supplementation, which remain limited in size and duration, have not identified serious adverse effects,[15] though longer trials are needed to confirm long-term safety.

Q: Does Akkermansia cause diarrhea?

A: Akkermansia has not been linked to diarrhea in the published trial data to date. One study reported that its presence in the gut was associated with reduced occurrence of diarrhea in children.[16] That said, as with any change to gut bacteria, sudden increases in any beneficial bacteria, including Akkermansia, may cause temporary digestive discomfort; gradual introduction is generally preferable.

Q: What causes low Akkermansia levels?

A: Low Akkermansia levels can be influenced by diet quality — high intake of processed foods, added sugars, and harmful fats like seed oils is associated with lower Akkermansia levels. Aging, antibiotic use, chronic stress, sedentary lifestyle,[17] and metabolic conditions can also affect the composition of the gut microbiota,[18] including Akkermansia levels.

This article is for informational purposes only and does not constitute medical advice. Consult a qualified health care provider before making changes to your health regimen.

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