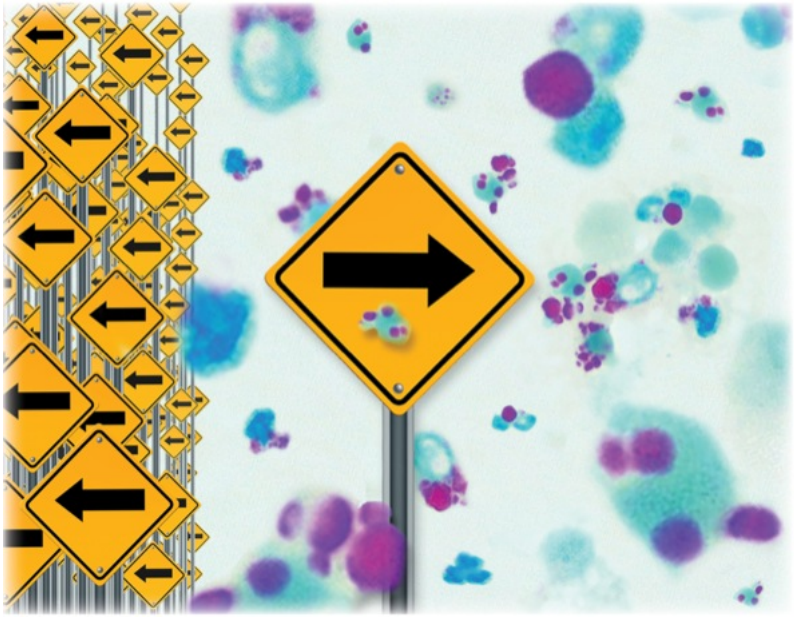
**In the News (/Section/In-the-News/187)**

AUGUST 10, 2017

# A MAP for Crohn's Disease: Old Theory Gains Momentum



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Julie Doyle first began experiencing symptoms of Crohn's disease on a family trip to the Statue of Liberty when she was 14 years old. Twenty-five years, four surgeries and innumerable failed treatments later, she was—as she puts it—functionally cured.

Ms. Doyle and her doctors initially believed she had some kind of flu. "My mom thought I got sick from the Statue of Liberty, because the bathrooms there were really bad," she said. She didn't receive a definite diagnosis of Crohn's until she was in college.

Ms. Doyle tried almost every medication on the market, which initially limited her to prednisone, other steroids and sulfasalazine. None was effective for long. (She avoided taking 6-mercaptopurine drugs when they became available after discussing their risks and benefits with her physician.) She thought that if she ignored the problem, it would go away on its own.

It didn't.



After having her first child, Ms. Doyle went in for a routine screening, and although she didn't feel particularly sick, her doctors decided to image her mucosa using capsule endoscopy. The capsule became lodged in her intestines—this was before dissolvable patency capsules were available—and surgery would be required to remove it.

“I went to the hospital in the middle of the night in excruciating pain; I knew I had a blockage,” Ms. Doyle recalled. “I always joke that when you start vomiting bile, it’s time to get yourself to the hospital.”

Ms. Doyle went into surgery with the expectation of undergoing a simple laparoscopic procedure. Instead, she woke up attached to an ileostomy bag, having undergone an ileostomy bypass. “I asked the surgeon, ‘Did you get the capsule?’ and he said, ‘Oh yeah, I got the capsule in about five minutes. There were also 18 inches of terribly diseased intestine that were going to make you go septic or give you cancer in a year, so I took all of that out, too.’”

She was 32.



Ms. Doyle was prescribed adalimumab (Humira, AbbVie), but it was ineffective. Meanwhile, her Crohn’s became metastatic, resulting in painful skin lesions. Ms. Doyle switched to infliximab, which worked for six months before her body built up antibodies against the drug.

“After that, my doctor said, ‘We don’t really have much else for you,’” Ms. Doyle said. “And I said, ‘What do you mean? I have two little kids. I can’t not have anything left.’ And she said, ‘Well, I don’t think biologics are gonna work for you.’ So I got pretty nervous. Up until that point, I’d just listened to whatever the doctors said, but I started researching anything I could.”

Ms. Doyle began a deep dive into the literature on Crohn’s disease, looking for anything that might help her. “I came across all sorts of things that I hadn’t been aware of, things like helminthic therapy and fecal matter transplant. And then I came across the MAP theory. And it just made sense to me.”



**‘Functionally Cured’**

The etiology of Crohn’s disease is unknown. Certain genetic mutations, such as *NOD2* and *IL23R*, are known to confer risk, but not all patients exhibit these mutations, nor do all people with these mutations develop the disease. The prevailing theory is that Crohn’s disease results from a complex interaction between genetics and environmental factors, but precisely which environmental factors are responsible remains a mystery.

The MAP theory centers on a bacterium called MAP, short for *Mycobacterium avium* subspecies *paratuberculosis*. MAP causes a Crohn’s-like disease called Johne’s (pronounced “YO-nes”) disease in cattle—and, proponents of the theory believe, is responsible for Crohn’s in at least a subset of patients.

The theory is not new. In fact, it’s older than the name “Crohn’s disease” itself, having first been proposed in 1913 by Dr. Thomas Dalziel of Glasgow, Scotland, almost two decades before Dr. Burrill Crohn would unwillingly have his name given to the disease. But it is far from mainstream. As a result, few gastroenterologists offer treatment based on the MAP infection paradigm of Crohn’s disease, known as atypical mycobacterial antibiotic therapy (AMAT).

But for Ms. Doyle, something clicked. “When you’re sick for 25 years, you learn how your body works. And I always knew that Flagyl [metronidazole, Pfizer] had worked well for me as a short-term solution. So it made sense to me that other antibiotics, if this were a pathogen, might work well.”

Ms. Doyle dug deeper into the MAP theory and AMAT. In her research, she had repeatedly come across the name of a particular physician, with blog posts from Crohn’s patients attesting that they had been cured. She managed to contact that physician, who spoke with her at length about her situation and offered to do a small trial of AMAT therapy if her physician would be open to it.

“So they talked, and they put me on antibiotics,” particularly 600 mg of rifampin, 1,000 mg of clarithromycin and 500 mg of levofloxacin. “I was sick as a dog for about a week, and then I got better. And then I got better, and better and better. After 12 months, I got a repeat colonoscopy, and I was healed. Not just in remission—I’d gotten a colonoscopy before starting the antibiotics, and at 12 months after treatment, there was not a trace of Crohn’s left.”

To generate a before-and-after record, Ms. Doyle had her blood drawn and tested for inflammatory markers two weeks before starting AMAT. Her blood showed moderate Crohn’s disease, which was entirely consistent with her experience.

Six weeks after starting the therapy, Ms. Doyle was feeling much better and had gained 10 pounds. She eventually dropped the levofloxacin due to joint pain, and began taking low-dose naltrexone instead, which relieved the pain. She had her blood tested for inflammatory markers every three months by her gastroenterologist, who agreed to follow her progress but was not supportive of AMAT. Blood tests a year after starting treatment showed only dormant bacteria where active MAP organisms once proliferated.

Despite his initial skepticism, Ms. Doyle said her gastroenterologist was eventually impressed by her complete mucosal healing. She continues on AMAT to this day since beginning in 2014, and has no intention of stopping. She considers herself “functionally cured” of Crohn’s disease, and her lab tests would seem to bear that out.

Apart from the initial flu-like phase, which is generally thought by AMAT proponents to be the result of the MAP bacteria dying off en masse, she has experienced no adverse effects. In 2016, she helped found the Human Paratuberculosis Foundation, which promotes education and research into Crohn’s disease and other potentially MAP-related autoimmune diseases.

**Of Prednisone and Paradigms**

The physician who put Ms. Doyle on the path to recovery was William Chamberlin, MD, a gastroenterologist at San Antonio Military Medical Center. In his early years, Dr. Chamberlin worked in a number of far-flung locations, including a tiny provincial hospital in Papua New Guinea, where he routinely treated a number of mycobacterial illnesses, including tuberculosis (*Mycobacterium tuberculosis*), leprosy (*Mycobacterium leprae*) and skin lesions (*Mycobacterium ulcerans*). "Maybe that altered my perceptions of the disease types that mycobacteria could cause, so that when I was exposed to the idea that MAP could cause Crohn's disease, it really fell on fertile ground for me," he said.

Dr. Chamberlin first encountered the idea during his fellowship, in a 1984 paper by Roderick Chiodini and colleagues, who had isolated a then-unclassified mycobacterial species from three Crohn's patients (*Antimicrob Agents Chemother* 1984;26:930-932). Then, while working in Asia in 1989, he diagnosed a young woman with Crohn's disease, and the patient asked him whether she should go on antimycobacterial antibiotics. "And I said, wow, they're already doing it."

A few years later, Dr. Chamberlin returned to working for the U.S. Army as a gastroenterologist. "Patients came to me who were being treated with prednisone, and from everything I knew, prednisone was the last thing you wanted to treat a mycobacterial infection with. At that point, I went back to my basic medicine textbooks, and I started reading about *M. leprae*, *M. tuberculosis*—what was known about them, what the immune responses were—and it just became more and more obvious to me that Crohn's disease was a mycobacterial infection. And that idea was, by this time, very, very controversial."

Dr. Chiodini had been able to culture MAP in vitro, as had researchers in the veterinary world. But prominent researchers in the inflammatory bowel disease community had tried and failed to identify MAP in Crohn's patients, Dr. Chamberlin said. "And they failed for very basic reasons: They did not know how to handle this atypical, non-tuberculosis *Mycobacterium*. It's a different species, a different organism."

Dr. Chamberlin began treating Crohn's patients with a triple antibiotic regimen around 1994. "I was lucky in that my first few patients had very good responses."

Dr. Chamberlin used AMAT on roughly a dozen more patients with a high success rate, although not everyone responded to the treatment. Yet, apart from his patients, not many people were enthused by his success. "As the years passed, no one believed me. People said, 'Oh, we've already looked at this,' and my results were just dismissed."

Then, at a conference in the late 1990s, Dr. Chamberlin saw a poster by an Australian physician who had treated a series of 12 Crohn's patients with antibiotics, reporting virtually the same results. "So I got in touch with him, and by then I thought, 'We are really onto something.'"

That physician was Thomas Borody, MD, PhD, director of the Centre for Digestive Diseases (CDD), in Sydney. Like Dr. Chamberlin, Dr. Borody had field experience with mycobacterial infections, from treating tuberculosis and leprosy in the Solomon Islands. His research led him to the lab of Dr. John Hermon-Taylor, a retired surgeon turned molecular biologist who, in 1987, wrote a paper with Dr. Chiodini demonstrating that mycobacteria isolated from Crohn's patients were identical to MAP, as determined by DNA probes that differentiate between mycobacterial species (*J Clin Microbiol* 1987;25:796-801).

Dr. Hermon-Taylor has studied MAP since. He operates one of the few labs in the world that can consistently culture MAP, and is raising funds for a clinical trial of a MAP vaccine to treat Crohn's disease, which he is developing with researchers in England.

"I noticed that he was treating patients with double therapy, and I came from the land of tuberculosis, where we would start treating with a minimum of three drugs together," Dr. Borody said. "But because we had people running off to the bush over there in the Solomons, in Malaita, we sometimes had to use up to seven different drugs to get control of what had developed into quite resistant strains of tuberculosis on an island of about 80,000 people."

Like Dr. Chamberlin, Dr. Borody's experiences with mycobacterial infections left him open to the idea that a pathogen might be causing Crohn's disease. In his clinic, which has a yearlong waiting list consisting of patients from around the world, he claims to have an 85% remission rate for Crohn's patients from a combination of quadruple antibiotics and adalimumab.

In 2011, Drs. Borody and Chamberlin, together with Dr. Jordana Campbell at CDD, published a meta-analysis of studies examining primary treatment of Crohn's disease with antibiotics, finding mostly positive results (*Expert Rev Clin Immunol* 2011;7:751-760). One trial (*Gastroenterology* 2007;132:2313-2319) showed that antibiotics were not effective against Crohn's disease; however, that study later was found to be underpowered and flawed, and a more straightforward intent-to-treat analysis of the raw data by Marcel Behr, MD, MSc, a tuberculosis expert at McGill University, in Montreal, found that even a suboptimal dose of antibiotics compared favorably to adalimumab and infliximab.

To Drs. Borody and Chamberlin, a key piece of the Crohn's puzzle that has gone largely ignored is a 1986 publication by Van Kruiningen et al, in which the *Mycobacterium* that would later be known as MAP was harvested from the diseased ileum of a child and fed in cream to four infant goats, while four others received only cream (*Dig Dis Sci* 1986;31:1351-1360). The four goats that ate the *Mycobacterium* developed granulomatous lesions virtually identical to those seen in Crohn's disease. The original *Mycobacterium*, identified as Linda, was then recovered from all four infected goats, and not found in the control group.

"If you're working from the paradigm that Crohn's is not an infectious disease, you say, 'OK, so you had a Crohn's patient, and you identified a goat pathogen that just happens to be there. You gave it to a goat, and the goat got Johne's disease. That doesn't prove anything,'" Dr. Chamberlin said. "Which leads me to believe that David Hume was correct—that you can never prove causality. You can only show an association between A and B, and tell your story, and if people believe you, then it becomes true."

To Dr. Chamberlin, calling Crohn's disease a "disease of immune dysregulation" is like saying "water is wet, or that World War II was caused by people failing to get along. It doesn't say anything about the etiology of the disease, only the pathogenesis. I've come to understand the following. In order to understand the paradigm that a nontuberculous mycobacteria [NTM] can cause Crohn's disease, you have to have working knowledge in three areas: Number one, what is Crohn's disease—and a lot of physicians have that, that's easy; number two, knowledge about NTM, as separate from tuberculosis mycobacteria—how they function, what they are—and there's virtually nothing in the gastroenterologist's background to give them any understanding at all. Anyone can go and get this understanding, especially with the internet, but we just don't.

"And the third area is a really good working knowledge of the immune system. And there is nothing in the training of the gastroenterologist about the immune system. There's a really superficial introduction, but no real depth of understanding. So of that triad, two of the legs are lacking in physicians who are supposed to be expert in Crohn's disease."

**A Case of Mistaken Identity— And Accidental Assassination**

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John Aitken, Otakaro Pathways Laboratory

When Ms. Doyle needed her blood tested for MAP, she sent it, at Dr. Chamberlin's recommendation, to John Aitken, a microbiologist and senior director of Otakaro Pathways, Ltd., in Christchurch, New Zealand. In 1973, the same year that Mr. Aitken began his specialist training in microbiology in the pathology department of Christchurch Hospital, he was misdiagnosed with Crohn's disease. "For three years, I suffered under the misapprehension that I had Crohn's disease, and at the same time I was training in the hospital as a medical laboratory scientist," Mr. Aitken said. "So that had created a small space in my head that whenever I saw something on Crohn's disease, I would read it."

Mr. Aitken was subsequently put in charge of a satellite hospital laboratory that worked with respiratory infections, including tuberculosis, at which point he began working with mycobacteria. His interest in Crohn's disease led him to the MAP theory early on, and he made attempts to replicate the work of other researchers. He began working more frequently in Crohn's in 2004, and in 2011 co-founded his current lab, which focuses predominantly on Crohn's disease and MAP (Figures 1-4).

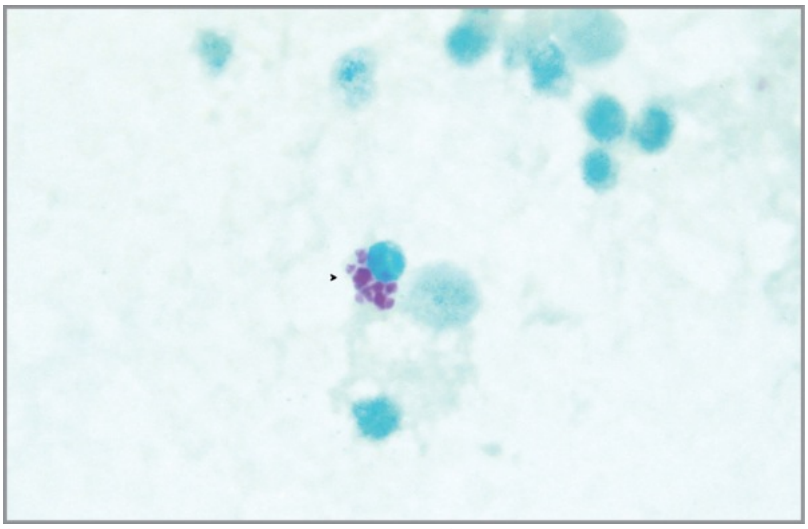

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**Figure 1. Typical large forms of Mycobacterium avium subspecies paratuberculosis lack rigid cell walls. The blue-green bodies are macrophages.**

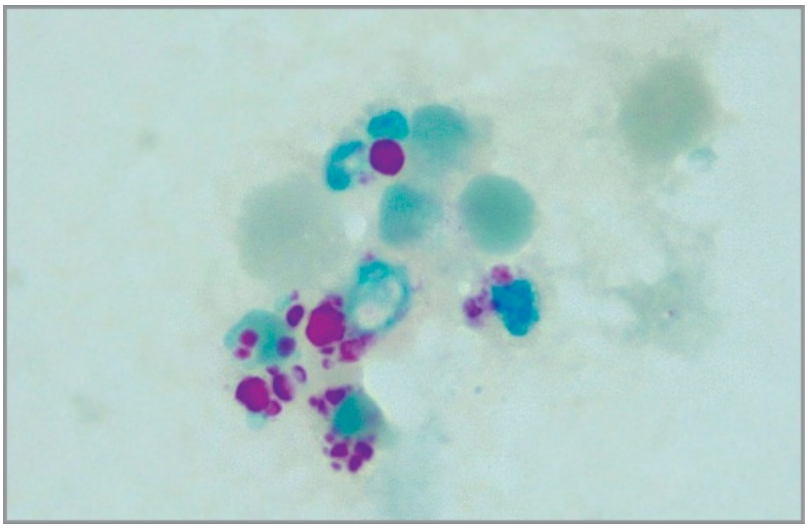

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**Figure 2. Mother and daughter large-form cells actively replicating, amid a mix of "scout cells" produced to test the environment for replication and colony formation.**

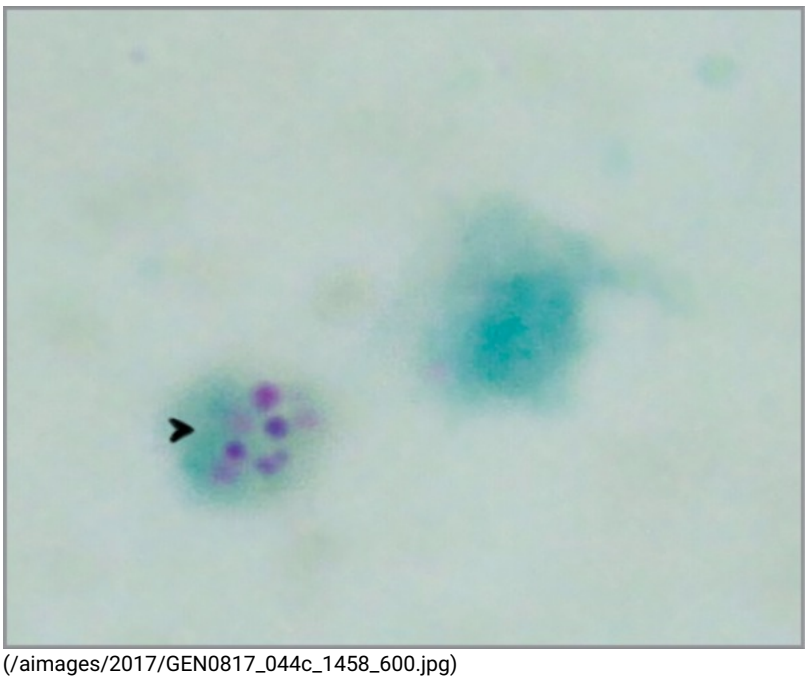

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**Figure 3. Large–form cells.**

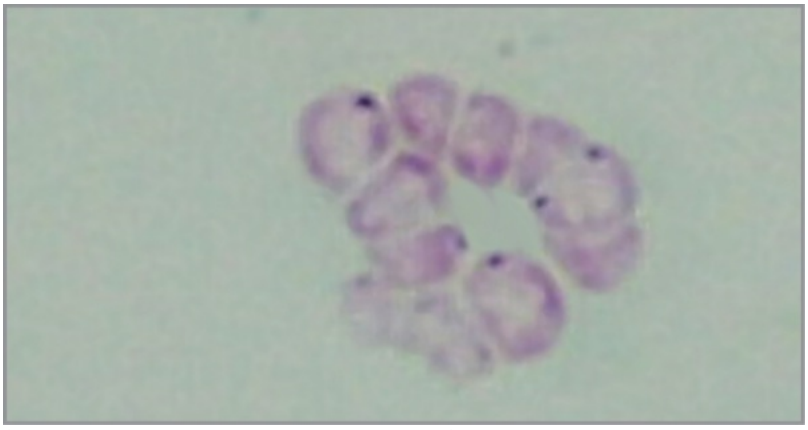

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**Figure 4. Lipid bodies showing the organisms, free of white cells, attempting to build cell walls. Dark spots adherent to the inside of the cell wall are lipid deposits in the first stage of peptidoglycan production, which ends with the construction of a cell wall.**

“One of the strange things is, people publish papers where they report that they have grown MAP, but when you read the paper very carefully, what they have found was the DNA sequences that indicate that they had MAP in the culture,” Mr. Aitken said. “Occasionally, at 18 months incubation, they will find that they have an organism growing on a culture that usually has a cell wall, whereas the strain we work with doesn’t have a cell wall at all, it just has a semipermeable membrane. So my question always was, ‘If it takes 18 months to grow, and if it’s got a cell wall, how come a pathologist can’t see it from a biopsy?’”

The papers he was reading were reporting that MAP had been grown and identified with relative ease, whereas laboratory pathologists were having considerably more difficulty identifying MAP in tissue and blood samples from Crohn’s patients. Those researchers were reporting their findings, but their methods were not uniformly reproducible in other labs. That made no sense to Mr. Aitken.

“The organism we’re working with does not have a cell wall, which makes it very difficult to stain and perceive,” Mr. Aitken said. “You have to apply a lot of work to get them, but eventually you make the breakthrough. It’s easy enough once you figure it out.”

Mr. Aitken estimated that by autumn he would have a patented supplement that could be added to basic culture broth to enhance the detection of MAP.

“We are establishing beta-testing sites in medical laboratories now, so that scientists on the bench can judge for themselves,” he said. “What we believe is happening is that the organism loses its cell wall, which makes it less open to detection by the body, like a soldier who ditches his uniform. Then, when it gets into the body, it’s taken up by a white blood cell.”

Once that happens, Mr. Aitken said, MAP lives and multiplies inside the white blood cell until the cell dies. At that point, MAP bacteria are released into the blood, triggering inflammation through production of biofilm and peptidogylcans—which, Mr. Aitken said, signals other white blood cells to pick them up and repeat the process.

Mycobacteria live only inside white blood cells. *M. tuberculosis* uses the same strategy to lay dormant in the blood, sometimes for decades. Moreover, some Crohn’s patients, as well as some with TB, have the *NOD2* genetic mutation, which interferes with the ability of white blood cells to do their job of consuming the pathogen.

Echoing Dr. Chamberlin, Mr. Aitken said a lack of basic microbiological knowledge prevents most gastroenterologists from understanding the MAP theory of Crohn’s disease. “If you look at any paper looking at the detection of MAP in the blood, they will specify using EDTA [ethylenediaminetetraacetic acid] as the anticoagulant. Most people on the bench in the lab know that EDTA is bactericidal. So you’ve got the vast majority of researchers out there who are looking at Crohn’s using a bactericidal compound to look for a rather delicate bacteria. They’re killing the thing they’re looking for.”

Mr. Aitken said the paradigm shift in Crohn’s disease, should it occur, will come not from academics but from patients.

“Having worked in laboratories for a number of years, I’ve only been in contact with clinicians, and I saw very little of the patient side of things,” he said. “But this last 10 years or so, I’ve been confronted with the patients that I work with, and it’s been a humbling experience to see these people working away, trying to get tests and therapies for their child, or for themselves. They are just unstoppable. Give me a platoon of Crohn’s mothers and I will conquer the world!”

**Farm to Table**

The incidence of Crohn's disease has increased dramatically since the 1960s, both in the United States and abroad. Proponents of the MAP theory assert that this growth is due primarily to contamination in the dairy and food supply. An analysis led by the Department of Agriculture found that 91% of dairy herds in the United States are infected with MAP as of 2007 (*Prev Vet Med* 2013;108:234-238).

"This is an infection that, once upon a time—say the 1900s—infected very, very few animals in very few herds and very few countries. And slowly, insidiously over time, the infection has continued to spread," said Mike Collins, DVM, PhD, professor of microbiology at the University of Wisconsin–Madison. "This is truly an epidemic, but it's not like Ebola virus. It's spreading more like a glacier. For the last 117 years, it's been spreading—slowly but surely—in cattle, in sheep, in goats, in bison. And all of these species are food-producing animals.

MAP is hardy and adaptable. Studies have found that the microbe can now survive under simulated pasteurization conditions, and one survey of 702 pints of retail whole milk purchased in California, Minnesota and Wisconsin found viable MAP present in 2.8% of the pints tested (*J Food Prot* 2005;68:966-972). The bacterium also has been found in powdered infant formula, although the risk for contamination by formula appears relatively low (*Int J Food Microbiol* 2017;257:1-9).

Dr. Collins and other researchers around the world are convinced that MAP is dangerous and are working to develop more sensitive and cost-effective methods to detect and eradicate the bacterium before it can reach consumers.

"I'm convinced that as the epidemic has continued to emerge in animals, it's now spilling over into humans," Dr. Collins said. "And there's lots of ways humans could be exposed, but I think food represents one of the major ones."

**Skepticism and Hope**

Outside the realm of those who study MAP and Crohn's disease, the theory of causation continues to meet with skepticism.

"Patients in general may have a placebo response to medication even if the medication is ineffective. As a consequence of this, I would like to a prolonged benefit of a minimum of six months to a year in patients who have been treated with the medication," said Gary Lichtenstein, MD, director of the Inflammatory Bowel Disease Center and professor of medicine at the Hospital of the University of Pennsylvania, in Philadelphia. "We'd like to see healing or substantial improvement of the mucosa of the bowel in areas that are inflamed in patients on active therapy that is significantly better than those patients treated with placebo. I would also like to see biomarkers—such as calprotectin and C-reactive protein—go down and remain at a low or normal level.

"The trials that are published are investigator-initiated, not multicenter, randomized controlled trials, so there may be bias in individual centers," added Dr. Lichtenstein, who is a member of the editorial board of *Gastroenterology & Endoscopy News*.

Sunanda Kane, MD, a gastroenterologist at Mayo Clinic in Rochester, Minn., said she believes AMAT may alter the microbiome in a beneficial way, but that she "is still waiting for definitive proof of MAP being the actual cause of Crohn's disease. It is intriguing and warrants further investigation."

David Graham, MD, professor of medicine and gastroenterology at the Michael E. DeBakey VA Medical Center and Baylor College of Medicine, in Houston, who has published extensively on the potential link between MAP and Crohn's disease, said despite the strong associations, the medical and agricultural mainstream will be convinced only when treatment of MAP results in a cure for Crohn's disease.

"There's a famous saying: History is full of examples of lonely thinkers who were belittled by their peers, and time proved that their peers were correct," Dr. Graham said. "So we may all be wrong."

MAP skeptics and proponents may soon have the answers they seek. RHB-104, an ongoing Phase III, randomized controlled trial of AMAT in approximately 300 Crohn's patients with moderate to severe disease, may provide an interim efficacy analysis this summer. The trial is being conducted by RedHill Biopharma, using the AMAT formula developed by Dr. Borody, who is also a member of RedHill's advisory board.

"I won't even go into the apple carts that will be upset when our theories are incontrovertibly proven," Dr. Chamberlin said. "We're talking about massive industries—immunosuppressive therapies, not to mention the dairy industry—hundreds of billions of dollars-a-year industries that are built on the wrong assumptions."

*—Ajai Raj*

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