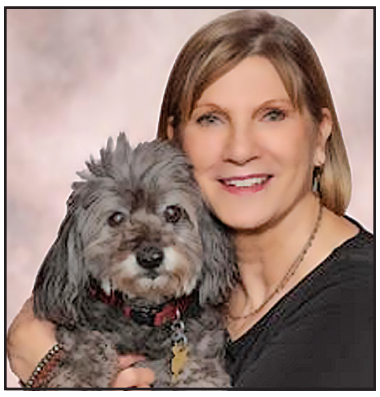
C Diff—from Animal to Soil to You!!



Dr. Kate Thomsen and Silky

Clostridium difficile is a bacteria whose name has recently been changed to Clostridioides difficile, but everybody calls it C diff. It is currently the most common cause of hospital acquired infectious diarrhea. Having this condition in the hospital can cause you to be in isolation, take more antibiotics, add 2 weeks to your hospital stay (and at least another $10,000), and can sometimes become severe enough to cause pseudomembranous colitis (inflammation of the inner lining of the large intestine/colon) which may lead to dehydration, kidney failure, toxic megacolon, colon perforation, sepsis and death. Lovely. Sounds like we shouldn't mess with this bug. But that may not be an option….

To a microbiologist, C diff is a "gram positive, spore-forming, toxin-producing anaerobic bacteria." This means it can only survive in oxygen free environments. It can form a thick protein shell (its spore form) to protect itself when in an unfriendly environment. And it can secrete toxins that can damage human or animal cells. It is also quite antibiotic resistant.

Clostridium bacteria live in soil, in water and in the intestinal tracts of humans and other animals. They were here on the planet long before we were, so how (and why) did they find a second home in the human intestinal tract? Why did we co-evolve with them as part of us? The Clostridia (and other bacterial species) found a perfect home in our intestines where there is an abundance of: 1) dark crevices or folds in our intestinal wall where there is no oxygen, and 2) a steady flow of our undigested dietary roughage which they ferment as an energy source. We let them stay because they have become essential for the development and function of the immune system. They provide a "barrier" function to overgrowth of harmful bacteria and are tolerant of other beneficial bacteria. They produce B vitamins, Vitamin K and short chain fatty acids such as butyrate which is food for their hosts, the cells of the intestinal wall. We need them as much as they need us. It's a good arrangement. In fact most Clostridia are classified as commensal or "friendly" bacteria – and are key players in maintaining homeostasis in the intestinal tract. C Diff is not a commensal. It is a pathogen that causes infection – but not always. Like some viruses, C Diff can find us, live in us, and not cause any health problems.

So why would some of these Clostridia, like C diff, suddenly decide to produce toxins that harm their home and hosts? It's when the terrain has changed, for example after their host has taken an antibiotic. This reduces the number and composition of normal bacteria in the colon. C diff (not affected by the antibiotic) finds itself in an environment free of the bacterial products that usually suppress it's growth. Suddenly it is not competing for limited nutrients!! So C Diff proliferates. Some C diff bacteria will not contain the genes for toxin production. The ones that do will produce toxins A and B and damage the cells of the colon wall. Diarrhea usually begins 4 – 9 days after an antibiotic is started but can also develop up to 8 weeks after an antibiotic is discontinued. C diff is resistant to penicillin, cephalosporins and clindamycin and these antibiotic classes are more likely to precipitate C diff antibiotic associated diarrhea.

Some people with CDI (C Diff infection) will have mild, frequent, smelly diarrhea, and others will have liquid diarrhea, dehydration, fever, appetite loss, abdominal pain, tenderness, and progression to colitis or pseudomembranous colitis. But C diff does not cause disease if no toxin is produced. Even when a toxin is produced, some people become "carriers" with no or very mild symptoms. Other people will have multiple relapses usually occurring within 1 – 8 weeks after a successfully treated episode of CDI with each episode increasing the likelihood of a further recurrent episode. What's different about people who get milder disease or no recurrences? Again it's the terrain: they have a brisker antibody response to the toxins. Symptom-free carriers of infection have been shown to have more antibodies to C Diff toxins A and B. Recurrent C diff infection appears to be associated with a lack of protective immunity to C Diff toxins.

C Diff in it's vegetative (growing) state and spore state are found in the feces of infected people who can spread the infection to surfaces, foods, and other objects, which are touched by other people. This is called fecal-oral transmission. C diff spores can survive on surfaces for months and are resistant to conventional disinfectants. Hospital studies have shown that more than 50% of the hands of health care workers, hands of patients with diarrhea, sites like blood pressure cuffs and call buttons are contaminated with C Diff. One study of 400 hospitalized patients negative for C Diff on admission showed that 21% tested positive for C Diff and 8% developed symptomatic CDI during their hospitalization.

Until recently, CDI was considered Hospital Acquired (HA-CDI) most of the time. While HA-CDI is still a major health care problem, the emergence and increasing prevalence of Community Acquired C Diff infection (CA-CDI) has researchers busy looking for community reservoirs. CA-CDI now accounts for almost 48% of annual cases in the United States. The antibiotic classes clindamycin, fluoroquinolones, carbapenums, and macrolides were most likely to be associated with CA-CDI.

Researchers have published data to explain the rise of CA-CDI including: asymptomatic carriers disseminating the bacteria in the community, contact with children under 2yo (70% of whom are colonized), having health care exposure other than hospitalization, living near nursing homes, proximity to livestock farms or facilities that handle raw farming materials.

In 1950 farmers began adding antibiotics to animal feeds for faster weight gain. This practice has been banned in the US since 2017. But animal contact is suggested as a potential risk factor for the development of CA-CDI because of the high prevalence of C Diff existing in pigs, cattle and poultry on large scale intensive farms. Animal manure and human biosolids are composted for agricultural purposes and an Australian study found C Diff in 22.5% of composted products, 29.7% of soil conditioners, 16.7% of mulches and 13.6% of garden mixes. A 2022 study of spinach fields detected C Diff in 3.3 % of spinach samples and in 10% of soil samples. A worldwide study of environmental samples from healthcare and non-healthcare sites performed from 2014 to 2017 found 26% of samples tested positive for C Diff strains. Shoe soles had the highest positivity rates, with 45% of samples testing positive for the bacteria. No where to run, no where to hide.

Guidelines recommend that a patient with 3 or more unformed bowel movements in 24 hours be tested for C Diff. Blood tests look for antibodies to C Diff and the toxins A and B. PCR tests can also be done for the toxins. Testing positive, one would eliminate the precipitant (current antibiotic) if possible. Antibiotics (ironically enough) are used to eliminate "out of control" C Diff. These include vancomycin or fidaxomicin. Vancomycin will suppress both normal bacteria and growing C Diff but not the spores. So when Vanco is finished, the spores may open and the infection may start again. In our office, we always add another herbal antimicrobial hoping to prevent this. We also always add Saccharomyces boulardii, a probiotic non-colonizing yeast that neutralizes C Diff and it's toxins A and B. Dietary advice includes drinking plenty of water with electrolytes to prevent too much fluid loss and avoiding spicy, fatty, or fried foods and any other foods that make symptoms worse (gluten? dairy?).

When the diarrhea is under control we start rebuilding the intestinal microflora with a Weeding, Seeding and Feeding protocol. This rotation of supplements will continue to remove undesirable microbes while supporting the keystone species of bacteria which have such a huge effect on both the health of the intestines and the status of the microbial communities within the intestines. Slow and steady addition of dietary fibers gradually help to restore the terrain—and this is the best prevention against future infection.

Newer ideas in the literature suggest to protect the gut lining during active infection with compounds that block reactive oxygen species (free radical damage) and the use of misoprostol which was used in the past to prevent ulcers in people taking aspirin and ibuprofen. Fecal microbial transplantation (FMT) recolonizes an infected gut with donor fecal material. It is FDA approved for recurrent C Diff infection. Healthy cleaned stool can be administered directly into the colon with a scope, through a nasogastric tube or in a crapsule.

Prevention strategies include judicious use of antibiotics individually and worldwide. In high-risk environments there should be stringent infection control with deep environmental cleaning and appropriate hand hygiene. The Vancouver hospital system has trained dogs to identify C Diff contamination with 97% accuracy allowing for more targeted decontamination protocols.

The FDA has approved bezlotoxumab (Zinplava), a monoclonal antibody, indicated for reducing the risk of CDI recurrence. Animal studies have shown that a high zinc diet alters the gut microbiota, making them more susceptible to C Diff infection. Lowering dietary zinc in humans and animals has been suggested as a strategy to prevent infection. Banning antimicrobial use in livestock worldwide and finding better treatments for human biosolids and animal manure are goals of One Health, the integrative effort to provide optimal health for people, animals and the environment. They and others are also working on, you guessed it, a vaccine – always the answer these days.

I favor lots of hand washing and building up the gut terrain!!!! And of course, take off your shoes when you come inside!!!

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Risk factors that increase the chance of contracting C Diff are also terrain-based

- The health of one's immune system
  - being older than 65
  - having a severe underlying illness
  - being immunocompromised
  - receiving chemotherapy
  - having had a previous episode of CDI
- Disruptions of homeostasis
  - taking antibiotics, especially over a long period of time
  - taking proton pump inhibitors to lower stomach acid (adequate stomach acid would kill active C Diff – but not spores)
- High exposure to C Diff
  - prolonged hospital stay or nursing home residence

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