

Position Statement: Crohn's Disease and Mycobacterium avium paratuberculosis (MAP)

Crohn's disease likely arises in people who are genetically predisposed to the disease after one or more environmental triggers initiate abnormal immune responses that lead to uncontrolled intestinal inflammation. The behavior and distribution of the resultant inflammation is potentially dictated by a complex interplay between the involved genes, triggers, and altered responses. Researchers have long sought potential triggers that serve as causative forces behind the disease in some if not all affected patients.

A particular bacteria, Mycobacterium avium paratuberculosis (MAP), has generated interest as a potential trigger for Crohn's disease because it causes an intestinal illness called Johne's disease, in many species of animals including cattle, goats, sheep, and primates. Although Johne's disease in animals is not identical to Crohn's disease in humans, the two illnesses do share similarities including the following:

- Tend to begin in childhood or early adulthood;
- Largely affect the ileum (the lower part of the small intestine);
- Demonstrate comparable injuries to intestinal tissue, and;
- Cause persistent diarrhea, malabsorption, and weight loss.

Moreover, MAP is more frequently recovered from the intestines of patients with Crohn's disease compared to people with ulcerative colitis and individuals without either disease.

However, several findings have caused many researchers to discount a causative role for MAP in Crohn's disease. First, MAP cannot be detected in many patients with Crohn's disease and has been frequently found growing in people without the disease. Second, medical therapy specifically targeted against MAP has not been found to consistently alleviate the symptoms or eradicate the inflammation associated with Crohn's disease. Third, other medical therapies that suppress the immune system (e.g., immunosuppressants) or target specific inflammatory proteins (e.g., biologic agents) are effective in Crohn's disease, but would likely be associated with no improvement or worsening of disease if caused by MAP. Most clinicians accordingly believe that MAP may be a part of the normal intestinal bacterial flora of many people exposed to this organism through common food sources but is present in greater quantities in patients with Crohn's disease because of the underlying immune dysfunction. Clinical trials studying MAP and Crohn's disease are ongoing.

In summary, Mycobacterium avium paratuberculosis may play a role in the development of Crohn's disease as one of many different microbes that might act as a trigger for an abnormal inflammatory response in genetically susceptible individuals. But until more convincing scientific proof emerges, it cannot be described as a primary or the sole cause of Crohn's disease. The Crohn's & Colitis Foundation looks forward to peer-reviewed published results of this study and encourages continued research to help us better understand this disease. IBD researchers are still seeking to understand other environmental, genetic, and immune system factors that can play a role in the development of Crohn's disease.

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