Much of our concern about Clostridium is primarily with Clostridium perfringens and either clinical or subclinical necrotic enteritis. Many have theorized that control of C. perfringens may be a major reason for the effectiveness of the growth promoting antibiotics. In a survey in 2000, it was estimated that the cost of subclinical necrotic enteritis was as high as 5 U.S. cents per bird (20). Using these estimates and 1999 estimates on world broiler meat production, the cost to the poultry industry globally of necrotic enteritis (N.E.) is nearly $2 billion (2). Both clinical and subclinical N.E. is common in all poultry growing areas of the world (20). The disease was first described by Parish in 1961 (15) and has subsequently been reported from most areas of the world (7). The causative agent of N.E. is Clostridium perfringens which is a nearly ubiquitous anaerobic bacteria that can be readily found in soil, dust, feces, feed, poultry litter, and in intestinal contents (7).

C. perfringens, the Opportunist. Given the ubiquitous nature of C. perfringens, it is not surprising that we cannot attribute the clinical disease, N.E., to only one cause. However, it does appear that conditions that result in damage to the intestinal mucosa (coccidiosis, mycotoxicosis) or disturbance to the normal intestinal microflora predispose birds to proliferation of Clostridium (1,6,7,8). Smith et al. found that manipulating the diet affected the population of C. perfringens in the intestine suggesting N.E. may be precipitated by the diet (17). High levels of animal byproducts, i.e. fishmeal, wheat, barley, oats, or rye, have been shown to predispose birds to the disease (5,7,10,11). Therefore, in those poultry growing areas of the world that feed wheat or large amounts of fishmeal, the incidence and also the severity of the disease is more extreme than those poultry growing areas feeding a corn/maize based ration.

Subclinical Necrotic Enteritis. The subclinical form of the disease may be the most economically important form since it has been shown to impair the feed conversion ratio in broilers (19). Another form of subclinical N.E. is a hepatitis or cholangiohepatitis found in broilers at processing (13). It has been estimated that broiler flocks in Norway had losses due to liver condemnations as great as 20% (16).

C. perfringens, a Component of Healthy Gut Microflora. Why do we even care about the bacteria flora in the intestines of chickens? One answer is the healthy normal flora is the first line of defense for all animals' bodies to invading pathogens. All birds have a normal flora of bacteria on their skin, respiratory tract and the intestines. It has been estimated that the normal flora of the chicken’s gastrointestinal tract is approximately 1 x 10^{11} colony forming units/gram of intestinal content (18). The predominant bacteria present in the chicken ceca are primarily obligate anaerobes (3). There have been at least 38 different types of anaerobic bacteria isolated from the chicken ceca (4) with more than 200 total bacterial strains isolated (14). However, we are limited in our knowledge of all of the bacteria in a bird’s ceca because many of the bacteria cannot be grown using cultural techniques (14).
A second reason to care about the healthy normal flora may be the economic benefit of improved bird growth and feed efficiency. We know that the use of antibiotics continuously in the feed of birds will improve their growth, is this due to the antibiotics effect on specific pathogenic bacteria, such as *C. perfringens*? It has been shown that antibiotic growth promotants (AGP) inhibit the negative effects of *Clostridium perfringens* to cause necrotic enteritis (9). However, the actual mode of action of the AGP’s has not been determined.

If we go back to our original question of “Why do we care about the bacterial flora in the intestines of chickens?” We can see that a complete understanding of all the bacteria present may allow us to understand how the AGP’s work and may allow us to determine methods to achieve present day growth rate and feed efficiency without using antibiotics to manipulate the intestinal flora. We have used molecular techniques, 16SrDNA clone libraries, to examine various areas in the intestines of healthy broilers on a corn-soy diet with and without AGP’s. The results of this work have shown that the AGP’s activity, as expected, is primarily against the gram-positive bacteria of the intestines. The major effects appear to be against the *Lactobacillus* spp. and the *Clostridium* spp. This effect appears to be primarily in the ratio of the Clostridium to Lactobacillus in the ileum. As more of the data becomes available, it may be possible to determine which Clostridium species are beneficial and how we can best maintain the intestinal environment to favor those species to prevent both the clinical and subclinical effects of *C. perfringens*.

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References