Dysbacteriosis, its causes and its impact

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Poultry and animal intestines are the niche of complex and dynamic ecosystems formed by all microbes at luminal and mucosal levels within each section of the gut. Microbes in the gut can be grouped into microbial communities (MC) composed of groups of bacteria, protozoa, fungi, yeasts, bacteriophages and other virus that constitute the microbiome of each animal. Members of each microbiome can be categorized as symbionts, commensals, and pathobionts. These MC influence host’s responses to nutrient digestion, absorption, mucosa metabolism, general physiology, and local or systemic immunological reactions. When these ecosystems are in equilibrium, the net balance of the relationship between the microbiome and the animal host is usually positive for the host. However, when drastic qualitative and quantitative changes occur in the gut microbiome composition and their metabolic products, the animal host can face a dysbiosis, dysbacteriosis, and, alternately, intestinal bacterial overgrowth. These events generally cause clinical signs, such as flushing, diuresis or diarrhea. The causes of dysbacteriosis, its impact on health and performance, and options for prevention will be reviewed in the following paragraphs.

The Gut Ecosystem in Equilibrium

As the animal age, intestines are colonized by countless diverse MC that are affected by the flow of nutrients from the diet, secretions and systemic responses of the host (animal) that are dictated by its immune, endocrine and nervous systems. Most of the gut microbiota competes with the host for nutrients by various means. Nevertheless, as the animal grows a natural equilibrium between MC and the host is established. This equilibrium implies that symbiont and commensal MC are in the highest concentrations and are dominant in the gut ecosystem. This results in some positive impacts on the animal. Commensal or symbiotic microbiota benefits the host by 1) promotion of gut maturation, 2) enhancement of gut integrity, 3) antagonisms against pathogens (competitive exclusion), and 4) immune modulation. The symbiotic microflora also plays a significant role in maintaining intestinal immune homeostasis by preventing inflammation. As a result, the animal expends less energy and nutrients to maintain this tissue.

Pathobiont MC are always present, but in very low concentrations that are sometimes not detectable by culture methods. Some pathobionts and most of the commensal MC can have some positive effects under stable gut conditions, but the same pathobiont MC can contribute hazardous metabolites to the host when intestinal conditions change in a dysbacteriosis. This double-sided role means that the same MC could be positive and negative depending on circumstances. Consequently, maintaining the gut ecosystem in equilibrium is extremely important to sustain this balance between all three types of MC.

The host animal has several physiological mechanisms to control bacterial proliferation within its intestines, keep the microbiome ecosystem in equilibrium, and avoid the possible translocation of bacteria from the intestinal lumen to the blood stream. The main intestinal barriers to pathogen infection that also modify the microbial...
profile for each animal include: 1) peristalsis (flow rate, transit time), 2) secretions (water, electrolytes, HCl, enzymes, bile salts, and immunoglobulin A), 3) mucus (physical properties), 4) associated microflora, 5) mucosal integrity, 6) efficient nutrient digestibility and absorption, and 7) the gut-associated lymphatic tissue (GALT).

Nutrient absorption by the host animal is highly competitive with enteric microflora by limiting substrates. Improving nutrient digestibility and nutrient absorption in the foregut is the main way to minimize bacterial proliferation and possible dysbiosis. In poultry, it is important to maintain an average particle size of grains of 800 to 1000 μm after grinding or guarantee a minimum of 2.5% total dietary fiber to stimulate gizzard function.

The gizzard sets the digesta passage rate and appropriate functioning can contribute to better peptic digestion.

### Causes of Dysbiosis

Changes in intestinal motility, modifications of gastric acidity, reduction in the production of bacteriostatic peptides in the pancreas, alterations in the amounts of mucus produced or in its composition, reduced IgA secretion, and focal ulcerations of mucosa result in failure of nutrient absorption, tissue necrosis, and shifts in gut MC numbers and metabolism. Any perturbation in gastroenteric physiology or immunity of the bird caused by temperature stress (heat or cold) or other environmental discomfort can cause dysbiosis and/or enteritis associated with lower absorption of nutrients by the host. Exposure to the stress hormones norepinephrine and epinephrine, significantly increases the proliferation of several enteropathogenic bacteria, such as Escherichia coli, Yersinia enterocolitica, Pseudomonas aeruginosa, Salmonella enteritidis, Salmonella cholerasuis, and Salmonella typhimurium. Even under the best nutritional conditions, environmental stresses in commercial production conditions can increase intestinal bacteria proliferation and make animals more prone to dysbiosis. This situation is more frequent in young animals with unstable and immature microflora or immunosuppressed animals.

In the nutrient side, the most crucial factor for dysbiosis is the lack of digesta, since MC can change in periods of hours when nutrients are not present. Water quality is crucial to maintain normal intestinal function and digesta pH, and to avoid bacterial proliferation in the intestines. Sulfates, magnesium and other salts can increase passage rate or enhance water consumption. The gut MC are also affected by enteropathogen infections, such as those caused by Eimeria spp. Coccidiosis is a common parasitism and one of the more common causes of enteric problems. Infection with Eimeria spp. changes MC and the patterns of fermentation in the ileum and ceca initiating dysbiosis.

### Consequences of Dysbiosis

Dysbiosis can have profound effects on the host. Acute dysbiosis events can result in the proliferation of pathobiont MC that become enteropathogenic. Pathogenic bacteria can produce toxins and metabolites that increase gut motility, increase fermentation with gas production, change gut pH, irritate the mucosas, cause inflammation, and increase secretion of mucus. This process reduces digestibility and absorption of nutrients and water increasing even more the dysbiosis. This results in wet droppings, enteritis and diarrhea that could sometimes be lethal. However, the most costly condition for animal production is the chronic inflammatory responses of the animal to constant minor dysbiosis. These chronic responses can reduce weight gain and cause poor feed conversion efficiency. Wet litter is a general outcome of dysbiosis that may affect the air quality of the house with higher production of ammonia accompanied by a higher incidence of respiratory problems. Additionally, food borne pathogens such as Salmonella spp. and E. coli proliferate more in dysbiotic intestines and can become persistent residents of the hindgut. Coccidiosis infection and any other enteric disease can be aggravated when dysbiosis is prevalent. Generally animals with dysbiosis have high concentrations of Clostridium that generate more toxins leading to necrotic enteritis.

### How to prevent dysbiosis

The most important factors to prevent dysbiosis are minimizing environmental stress, maintaining good water quality, improving feed digestibility, and avoiding antinutritional factors, mycotoxins and rancidity. Since, this is not an easy task in animal production, some feed additives could be used to modulate MC and avoid dysbiosis. Growth promotant antibiotics are well known for the inhibition of undesired MC and the negative effects of their metabolites and selection for beneficial bacteria, but somehow they diminish natural diversity of gut MC. This negative aspect of antibiotics make them more prompt to generate bacterial resistance.

Other products have been proposed as alternatives to growth promotant antibiotic utilization taking into consideration the increasing bacterial resistance to some antibiotic categories, ban of their use in some countries, and poultry consumer rejection. Alternative new feed additives have been classified as probiotics, prebiotics, enzymes, organic acids, and herb extracts. Probiotics introduce desirable live microorganisms into the gut. Prebiotics promote the growth of desirable bacteria in the gastrointestinal tract. Enzymes help to eliminate the anti-nutritional effects of water-soluble polysaccharides and/or change the substrates to improve proliferation of some beneficial MC while organic acids cause the inhibition of bacterial growth. Finally, phytobiotics have very variable working mechanisms that depend on their composition. Phytobiotics can be bacteriostatic, bacteria modulators, immune-stimulating, or anti-inflammatory.

### Conclusions

Maintaining the equilibrium of the gut ecosystem is key to avoiding dysbiosis. The predominant gut MC are relatively stable over time in healthy animals. However, drastic shifts in MC occur, especially in young and immunosuppressed animals. Dysbiosis can be caused by periods of starvation or intestinal insults caused by antinutritional factors, mycotoxins, rancid fats and enteropathogens such as coccidiosis infection. It is noteworthy that unstable MC are frequently correlated with intestinal disorders and reduced growth or worsening of feed conversion. Improving feed digestibility and using feed additives that modulate gut microflora help to maintain more stable gut ecosystems even during periods of intestinal stress. Preventing dysbiosis may help to improve poultry performance, health, and welfare, and reduce foodborne pathogens and the environmental impact of poultry production.