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REVIEW ARTICLE

Gut Health: The Foundation of Resilience in Food Producing Animals

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ABSTRACT

Optimal gut health is of vital importance to the performance of animals. Gut is responsible for regulating physiological homeostasis that provides the host the ability to with stand infectious and non-infectious stressors. The gut microbiota confers health benefits to the host, including aiding in the digestion and absorption of nutrients, contributing to the construction of the intestinal epithelial barrier, development and function of the host immune system and competing with pathogenic microbes to prevent their harmful propagation. Modulating gut health in animals involves manipulating the gut microbiome to improve overall animal health and productivity. This can be achieved through various strategies like dietary adjustments, prebiotics, probiotics, postbiotics and even faecal microbiota transplantation. These methods aim to shift the balance of gut microbes toward a more beneficial composition, thereby enhancing nutrient utilization, boosting immunity and reducing the risk of disease.

Keywords: Faecal microbiota transplantation, gut health, intestinal epithelial barrier, postbiotics, prebiotics, probiotics

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INTRODUCTION

According to Hippocrates 460-370 BC, "all disease begins in the gut and health is determined by the microbiota in the gut!". The intestine represents one of the largest interfaces of the animal body with the external environment. The gastrointestinal tract is responsible for regulating physiological homeostasis that provides the host the ability to with stand infectious and non-infectious stressors[1]. Most of the studies addressing health and animal production have been focused on gut microbiota, which is justified by the crucial role of these microorganisms in nutrition, fitness and performance traits[2]. Public concerns about the use of growth-promoting antibiotics (AGPs) in animal agriculture have led to significant policy changes. The European Union has banned AGPs, while the United States is reassessing their use. These actions stem from growing evidence that AGP use contributes to antibiotic resistance, posing a threat to both animal and human health[3]. In India, several antibiotics are banned and some are restricted for use in livestock and poultry, primarily to combat antimicrobial resistance. Removal of AGPs from animal feeds results in an increase in enteric disorders, infections as described[4,5]. The ban on AGP has triggered a renewed scientific interest in the intestinal health of animals. While in the past, the focus of gut health research was almost exclusively on the veterinary aspects of pathogenic organisms invading the intestine and/or intestinal tissues, causing severe damage to the host mucosa and resulting in clinical symptoms of disease[6]. The current focus is on the fundamental aspects of the numerous complex and subtle interactions between the host mucosa, the intestinal content and all organisms residing in the intestinal tract.

Gut health is defined as "a steady state where the microbiome and the intestinal tract exist in symbiotic equilibrium and where the welfare and performance of the animal is not constrained by intestinal dysfunction"[7]. In animals raised for food, gut health is closely related to animal health and is directly related to the animals' growth and performance. A damaged gut can have a negative impact on feed conversion ratio, digestion and nutrient absorption, which can result in financial loss and increased susceptibility to disease[8,9]. However, a healthy gut is essential for the well-being of companion animals, and alterations in gut microbiota have already been linked to a number of illnesses, including allergies, cardiovascular disease and inflammatory bowel disease (IBD)[10,11].

Recently, it has also been shown that there is extensive communication between the brain and the microbiota *via* the brain-gut-microbiome axis. Through this bidirectional communication, signals from the brain can influence the motor, sensory and secretory functions of the gut and visceral messages from the gut can influence brain function[12]. Similarly, the gut-kidney axis involves the interplay between the gut microbiome, intestinal barrier, microbial metabolite production and renal physiology[13].

The intestine is the site of the highest antigen load caused by microbial and feed antigens in the gut lumen. The intestine is made up of an epithelium, a robust and diverse immunological system that contains most of the body's immune cells and commensal bacteria, which outnumber the host cells overall. Understanding of the interaction between all these interrelated components of the gut is what cumulatively makes the gut the basis for the health of animals[9].

Intestinal barrier

The intestinal homeostasis is determined by the intestinal epithelium, gut microbiome and host immune system. This functional unit is entirely dependent on the integrity of the gut epithelium, which is maintained by junctional proteins such as adherent junctions, tight junctions and desmosomes that join neighbouring epithelial cells and provide a physical barrier together with the lamina propria[14]. The glycocalyx on the surface of intestinal epithelial cells contributes to barrier function by preventing exogenous molecules and live bacteria from gaining access to the epithelial brush border membrane[15]. Key cell types in the physical barrier are absorptive enterocytes, Paneth cells and goblet cells. The epithelial cells in the small intestine form a continuous layer and the space between the cells is sealed by tight junctions. These tight junctions are a critical element of the gut barrier. Although Paneth cells produce antimicrobial peptide AMPs, goblet cells have a key role in barrier function by producing gel-forming (MUC2 and MUC6) or transmembrane (MUC3, MUC12, MUC13, MUC15 and MUC17 in the small intestine; MUC4, MUC20 and MUC21 in the large intestine) mucins. The mucus layer which prevents bacterial adhesion. Lysozyme and secretory IgA are key factors of the chemical barrier[16].

Changes in the expression level and functioning of tight junctions cause gut leakage, characterized by body fluids leaking into the intestinal lumen, which may ultimately result in diarrhoea[17]. In this context, the organised intestinal barrier prevents uncontrolled microbial induced immune reactions in the gut. Disruptions of the intestinal barrier result in substantial alterations to the delicate equilibrium between luminal antigens and the local immune system. Consequently, a leaky gut permits the translocation of bacteria, other microorganisms and luminal antigens into the bowel wall, thereby inducing an overwhelming proinflammatory mucosal immune response[18,19].

Intestinal mucosa maintains immune tolerance to a wide array of antigens while also inducing appropriate immune responses to external pathogens[20]. To maintain the health of intestine, its mucosa contains variety of innate and adaptive immune cells, including innate lymphoid cells, granulocytes, dendritic cells, macrophages, B cells and both α-β and γ-δ T cells. These cells can support barrier function through direct killing of invading pathogens, production of soluble mediators, such as cytokines (IL-10, IL-17 and IL-22), neutrophil extracellular trap formation or the local induction of protective immune responses against antigens, which form an immune barrier towards invading antigens and pathogens[21]. Innate and adaptive immune responses in the intestine are constrained by the local production of anti-inflammatory cytokines (e.g. IL-10 and TGFβ), which suppress effector functions of multiple immune cell lineages and promote the population expansion of regulatory T cell responses[22]. This homoeostatic cytokine balance is crucial for preventing excessive inflammatory responses in the intestine.

Intestinal barrier dysfunction

Dysfunction of intestinal barrier and alterations in intestinal permeability is also known as "leaky gut." The effects of pathogenic organisms on host intestinal epithelial cells are complex. These primary pathogen-host interactions may result in disturbances in the normal intestinal barrier, activation of the inflammatory cascade and alterations of normal fluid and electrolyte secretion. Enteric pathogens can bind to the cell surface and induce changes in the expression of tight junction proteins[23]. In addition, the production of toxins by pathogens can promote cellular damage through disruption of intracellular protein interactions, leading to increased cellular permeability and ultimately triggering cell death[24].

IBD affect both human and animal patients and are associated with gastrointestinal dysfunction due to infiltration of the mucosa, submucosa or lamina propria with abnormal populations of immune cells. In dogs with IBD compared with normal controls, the expression of the protein E-cadherin was lower in the villus epithelium, suggesting the role of this protein in the pathogenesis of IBD in dogs. In horses with large intestinal disease, a significant difference in TNF-α expression was found in diseased mucosa, suggesting a possible role for this cytokine in the pathogenesis of equine IBD. TNF-α increases myosin light chain kinase phosphorylation, which may alter paracellular permeability through its association with actin and myosin. Myosin light chain kinase expression and enzymatic activity are increased in cases of IBD and correlated with disease activity[25].

Gut microbiota

The gut microbiome of domestic animals is a complex community of microorganisms; viruses, bacteria, fungi, protozoa and other microbes residing in their digestive tracts, with each region harbouring distinct microbial populations. The intestinal microbiota contributes to several physiological, protective (pathogen displacement, nutrient competition, receptor competition, production of antimicrobial factors), structural (GIT barrier fortifi-

cations, induction of IgA, apical tightening of tight junctions, immune system development) and metabolic functions (ferment non-digestible dietary residue and endogenous epithelial-derived mucus, synthesize vitamins, control intestinal epithelial cell differentiation and proliferation, ion absorption)[26-29]. Several of the metabolites produced by the microbiota also stimulate the neuroendocrine cell in the GIT and therefore, the microbiota plays an important role in the endocrine regulation of gastrointestinal functionality[7,30].

The microbiome is dynamic and changes depending on things including nutrition, age and environment. The makeup of this microbiome can affect many facets of an animal's health, such as immunity, digestion and even behaviour[31]. The gut microbiome of cattle and sheep is dominated by Bacteroidetes and Firmicutes[32]. The dominant bacterial phyla in the poultry gut microbiome are Firmicutes, Bacteroidetes, Proteobacteria and Actinobacteria. Lactobacilli are predominant in the upper and middle GIT of poultry[33]. The canine gut microbiome is primarily composed of three dominant bacterial phyla; Firmicutes, Bacteroidetes and Fusobacteria. These phyla, along with others like Actinobacteria and Proteobacteria, contribute to a diverse and dynamic gut ecosystem[34].

The microbiome has a direct effect on the development and function of the mucosal immune system. The gut microbial alterations in animal gastrointestinal system or the differences in gut microbiome composition and function have been associated with a variety of diseases, ranging from metabolic conditions and gastrointestinal inflammation leading to colitis and respiratory illnesses[35,36]. Age, gender and species are important internal factors that influence the composition and structure of the gut microbiota[37]. Additionally, external factors such as heavy metals, antibiotics and pesticides can markedly disrupt the gut microbiota composition, leading to dysbiosis[38]. Moreover, the effects of the gut microbial community extend beyond the gastrointestinal system and can cause other systemic diseases[36].

From eubiosis to dysbiosis

Eubiosisis the balance of the intestinal microbial environment, which has positive impacts on the animal as a whole. Overall, healthy gut microbial communities are characterized by high taxa diversity, high microbial gene richness and a stable functional core of microbiome[39]. Gut dysbiosis is defined as an imbalance in the composition of the gut microbiota that may result in modifications to the transcriptome, metabolome or proteome of micro-organisms[40].

Neonatal calf diarrhoea is the leading cause of neonatal morbidity and mortality globally. The bacterial pathogens associated with calf diarrhoea include *E. coli*, *Salmonella* spp., *Clostridium perfringens* and *Clostridium difficile*. The two main viruses implicated in calf diarrhoea are bovine coronavirus and bovine rotavirus (BRoV). Calves with rotaviral diarrhoea had a lower relative abundance of Firmicutes and Bacteroidetes and a high abundance of Proteobacteria compared to their healthy counterparts[41]. At the genus level, the genera *Escherichia*, *Clostridium* and *Streptococcus* increased in BRoV-infected calves, while *Blautia*, *Bacteroides*, *Lactobacillus* and *Coprococcus* decrease[42]. Irrespective of the causative agent responsible for the onset of calf diarrhea, there are significant changes in bacterial communities of the gut microbiota[43]. During diarrhoea there is a shift from obligate anaerobes to facultative anaerobes in the GIT, resulting in dysbiosis[44]. The abundance of *Faecalibacterium prausnitzii*, *Lachnospiraceaesp.* and *Ruminococcacea sp.* bacteria associated with gastrointestinal health decreases significantly during calf diarrhoea[45]. Concurrently, an increase in *Lactobacillus*, *Streptococcus* and Enterobacteriaceae, especially *E. coli* is observed[46]. It is frequently noted that diarrheal calves have higher levels of *Enterobacteriaceae* bacteria[47]. Dysbiosis associated with inflammation results in alterations in the metabolites available to and originating from bacteria in the GIT of calves, resulting in an environment that favours the growth of *Enterobacteriaceae*. *Salmonella* spp. and *E. coli* benefit from the production of ethanolamine, lactate, glucarate/galactarate 1, 2, propanediol, succinate and L-serine during dysbiosis[48]. Infection with *Cryptosporidium parvum* in calves results in a reduction in the microbial diversity, and this reduction is proportional to the number of oocytes detected in the feces. Furthermore, an increase in the fecal abundance of *Fusobacterium* is reported in diarrheic calves infected with *C. parvum* compared to uninfected calves[49,50].

Rumen acidosis is one of the most prevalent gastrointestinal diseases affecting cattle, significantly threatening their health and growth performance. Rumen acidosis can induce alterations in the composition and diversity of the gut microbiota in calves. Notably, the levels of certain beneficial bacteria, such as Prevotella, Succinivibrio and Succinivibrionaceae decreased significantly. These substantial changes in intestinal composition and abundance may serve as critical driving factors for the development of rumen acidosis[51].

In pigs, *Enterotoxigenic Escherichia coli* (ETEC) induced diarrhoea is associated with a decrease in the Bacteroidetes/Firmicutes ratio. ETEC-induced diarrhoea in piglets decreases the microbial diversity in the jejunum and lowers the abundance of *Prevotella* compared to healthy counterparts. ETEC in piglets is also associated with an increased abundance of *Lactococcus* in the jejunum and *Escherichia Shigella* in the feces[52].

In poultry husbandry systems, coccidiosis is an

economically significant protozoan disease caused by an intracellular parasite that significantly impacts production. *Eimeria acervulina*, *Eimeria maxima* and *Eimeria brunetti* can reduce the abundance of Eubacterium, Lactobacillus and Ruminococcus in the cecum. Conversely, Eimeria infection can increase the abundance of bacteria like Bacillus, Enterococcus and Escherichia in the cecum[53]. Changes in the microbiota due to coccidiosis can affect the immune system's ability to respond to the infection. *E. tenella* infection alters the composition and diversity of caecal microbiota, significantly reducing Proteobacteria and Firmicutes *(Enterococcus)*[54]. Alteration induced by *Eimeria tenella* infection in abundance of the bacterial community may contribute to the severity of pathology and variation observed in tissue damage[55].

In canine, short-term changes in the intestinal environment, such as in cases of acute diarrhea, affect the microbial composition. Acute diarrhoea results in decrease in microbial diversity, with lower numbers of *Bacteroidetes* and *Faecablibacterium* and higher numbers of *Clostridium sp*[56]. Intestinal dysbiosis is linked to several chronic GIT illnesses, including IBD and Mucosa-adherent Proteobacteria genera *(E. coli)*[57].

Modulation of gut health

Modulation of gut health can play a key role in reducing the dependence on antimicrobials for protecting animals from diseases and maintaining production. Modulation of barrier function may be a promising path for the treatment of a wide range of intestinal and extraintestinal diseases. Currently, numerous novel therapeutic concepts are being explored to directly or indirectly enhance barrier function. Currently, there are a few methods for modifying the gut microbiome, including dietary modifications, use of prebiotics, probiotics, synbiotics and postbiotics.

Prebiotics are substances that are selectively utilized by host microorganisms, contributing a health benefit[58]. Inclusion of prebiotics in livestock and poultry feed has shown the capability to improve host health and productivity through the selective stimulation of beneficial gut microbiota[58,60].

The potential benefits of probiotic are diverse and may include immune system activation and modulation, enhanced mucosal barrier function, competitive exclusion of pathogens and decreased risk of infection through production of antimicrobial substances including lactic and acetic acids[61]. Probiotics have been used in the treatment and prevention of IBDs, diarrhea, irritable bowel syndrome and gastroenteritis. Although several organisms have been studied, commonly used species include *Bifidobacterium*, *Lactobacillus* and *Saccharomyces*[62,63].

Plant-derived compounds, such as polyphenols, alkaloids, flavonoids and essential oils exhibit various bioactive properties that improve gut microbiota composition, support immune function and improve nutrient absorption by influencing gut morphology and digestive enzyme activity. Their antioxidant, anti-inflammatory and antimicrobial properties help to maintain and improve overall performance and lower the prevalence of diseases related to gut and intestinal integrity[64].

One novel approach to regulate gut microbiota in animals to re-establish the recipient's intestinal microbiome is faecal microbiota transplantation (FMT). Faecal microbiota transplantation refers to an approach whereby faeces are transferred from a healthy donor to the gut of an unhealthy recipient through multiple methods. FMT is helpful in treating a number of different gastrointestinal and non-gastrointestinal disorders that areclosely linked to dysbiosis[65].

Metagenomics for the identification of gut microbiome composition

Metagenomic analysis has the potential to provide information about the detection of microbial composition of the gut and diversity, novel genes, microbial pathways, functional dysbiosis, antibiotic resistance genes and the determination of interactions in the gut[45].

Methods for testing gut permeability and other markers of intestinal barrier disruption

One of the issues with determining dysfunction of the gut barrier is the lack of specific biomarkers. When testing for intestinal permeability, a variety of parameters can be evaluated. Moreover, the fact that permeability varies along the GIT must be considered with being the small intestine being more permeable than the large intestine[66]. Briefly, methods for testing gut permeability *in vivo* involve the administration of a tracer molecule by oral gavage or intestinal instillation. Tracers commonly used are non-digestible sugars such as lactulose or mannitol, PEG, fluorescently labelled dextrans and[51] Cr-EDTA, which can be later quantified in urine or blood. The size of a tracer can indicate the probable route of permeability. To obtain comprehensive information regarding epithelial leakness, it is recommended that *in vivo* and *ex vivo/in vitro* tests of permeability are used in combination with the detection of permeability associated biomarkers[67].

CONCLUSION

Several complex mechanisms are involved in GIT functionality and health. Gut microbial comparison and analysis have the potential to benefit the understanding of the pathogenesis of various animal gut-linked diseases and the development of corresponding strategies to decrease the collateral damage. It is crucial to deepen our understanding of these interactions so that strategies for the modulation of GIT functionality and health, in

the context of improved animal performance can be developed.

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