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Navigating Microbiota Dynamics applicable to Pet Nutrition

PROCEEDINGS

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The gastrointestinal microbiota has emerged as a central player in pet health, influencing digestion, immune function, metabolic balance, and disease progression. As our scientific understanding deepens, it is becoming increasingly clear that nutrition is one of the most powerful tools available to modulate microbial ecosystems in dogs and cats. As we come together for this symposium on **Navigating microbiota dynamics applicable to pet nutrition**, we will explore the evolving science behind microbiota dynamics and cutting-edge translate cutting edge research into practical, evidence based nutritional strategies for companion animals.

Throughout the day, leading experts shared insights into biomarkers, feeding practices, and dietary interventions that help decode the complex interactions between diet, the gut microbiome, and host physiology. From early stage research to clinical applications, the sessions highlighted how new advances on the area are reshaping the way we assess gut health and manage gastrointestinal disorders in veterinary practice.

A particular focus will be placed on chronic enteropathy, an area where nutrition driven microbiota targeted approaches are transforming clinical management. By examining both established and emerging strategies—ranging from tailored nutritional solutions to fecal microbiota transplantation—this program fostered a deeper understanding of what is currently known, where uncertainties remain, and how science can continue to guide innovation.

Through expert discussions and collaborative round tables, participants will connect evidence with real world application, reinforcing a shared commitment to advancing pet nutrition through robust science, open dialogue, and continuous learning with the final aim of ensuring that our beloved pets enjoy long, healthy, and happy lives. Together, we hold the power to transform their well-being and enrich their journeys with us. I hope you enjoy the PURINA® PRO PLAN Symposium on **Navigating microbiota dynamics applicable to pet nutrition.**

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Connecting the Dots: The Use of Biomarkers for Better Understanding of Gut Health and Disease

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CONNECTING THE DOTS: THE USE OF BIOMARKERS FOR BETTER UNDERSTANDING OF GUT HEALTH AND DISEASE

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Intestinal physiology and pathology – interconnection between host and microbiome

The normal gastrointestinal tract (GIT) requires the interaction between host function, immune system, and the intestinal microbiome. The function of the GIT is digestion of nutrients, which are ultimately absorbed in the brush border of the small intestine. In animals with chronic gastrointestinal signs, many underlying pathologies can co-exist. These include inflammation, which over time leads to chronic mucosal remodeling. Furthermore, epithelial damage and increased cell shedding can be a feature in CE. Intestinal epithelial cells contain lipid rafts, cholesterol- and sphingolipid-rich microdomains incorporating long-chain fatty acids such as nervonic, arachidonic, and palmitic acids, that regulate signaling and immune responses.[1] Consequently, mucosal injury can lead to the shedding of these cellular components, increasing their fecal concentrations in both dogs and cats.[1-3] Long-term inflammation changes epithelial architecture and function, with loss of transporters.[4] Therefore, increased amounts of primary bile acids, amino acids, fatty acids and carbohydrates have been reported in fecal samples of dogs and cats with chronic enteropathies (CE), pointing towards malabsorption as an important component of the disease process.[5] The intestinal microbiota reacts to this increased luminal substrate, and some bacteria will preferentially utilize substrates and outcompete others. When extreme, this leads to dysbiosis, defined as significant microbial shifts with loss of microbial function (ie, abnormal bile acid conversion, decreases in short-chain fatty acids, and increases in intestinal lactate).[6]

Of importance is that these pathologies occur on a gradient, with only subsets of animals having more severe changes.[3,7] For example, an increased dysbiosis index has been observed in up to 70% of dogs and cats with CE, and those animals with dysbiosis had more functional changes in the intestine (ie, increased primary bile acids, increased fecal carbohydrates and lipids) compared to animals with CE but without dysbiosis.[5] This would explain, at least in part, why animals with CE have a heterogeneous response to treatments.

Another important new concept is that these mucosal and functional changes in CE, once established, are likely present for months to years and maybe even life-long in some animals.[2,8,9] For example, dogs with increased Dysbiosis Index at presentation have been shown to typically have persistence of dysbiosis over time, even with clinical remission, indicating that the underlying pathology remains, requiring long-term management rather than expecting full resolution of the disease.[8,10] Based on these studies, an increased DI can serve potentially as a staging marker for CE, as these dogs are less likely to achieve full resolution, and have an increased likelihood of the need for repeated and lifelong treatment, as was demonstrated in these studies.

In line with this, several studies have shown that dogs with more severe dysbiosis typically respond less favorable to fecal microbiota transplantation (FMT), with often only short term response, requiring repeated FMTs to manage clinical signs, compared to dogs with mild dysbiosis.[10-13]

Microbiota assessment – Dysbiosis Index

The Dysbiosis Index (DI) is an analytically validated panel of quantitative PCR assays designed to evaluate the severity of dysbiosis in clinical settings. The assays independently quantify fecal abundances of key core bacterial taxa commonly present in

healthy dogs and cats. These include functionally important bacteria such as Faecalibacterium and P. hiranonis, which typically decrease in dysbiosis. Conversely, pathobionts such as E. coli and lactic acid–producing Streptococcus species often increase.

The DI integrates these individual bacterial abundances into a single numerical value: reductions in beneficial taxa combined with increases in pathobionts result in a higher DI, reflecting greater dysbiosis severity.[7,14] Once the DI is elevated, additional core taxa are altered as well. Severe dysbiosis is associated with reduced microbial enzymatic capacity and altered metabolic function, including decreased SCFA production, impaired bile acid conversion, and increased fecal lactate concentrations.[15,16] The magnitude of the DI is clinically relevant and can inform therapeutic decision-making.

Interpretation of the DI should always include evaluation of individual taxa, particularly P. hiranonis, as its reduction is a major contributor to microbial imbalance and is closely associated with gastrointestinal pathology.

Mild microbial shifts are characterized by a DI below 0, with at least one bacterial taxon outside the reference interval. These changes are often transient and of uncertain clinical significance. However, even when the DI remains below 0, a reduction in P. hiranonis is considered meaningful, given its essential role in bile acid metabolism and microbial homeostasis.

Moderate dysbiosis is defined by a DI between 0 and 2 in dogs and between 0 and 1 in cats. Importantly, a DI above 0 indicates dysbiosis even if all measured taxa fall within their respective reference ranges. In such cases, beneficial bacteria often cluster near the lower limits of normal, while pathobionts approach the upper limits, reflecting a subtle but measurable shift.

Significant dysbiosis is diagnosed when the DI exceeds 2 in dogs or 1 in cats. Higher DI values correspond to greater disruption of the intestinal microbiota.

In both uncomplicated and hemorrhagic acute diarrhea, microbiome alterations are typically mild. The DI is often normal or only slightly elevated (<2), and P. hiranonis usually remains within normal limits. These changes are primarily driven by increases in E. coli and especially C. perfringens.

Acute hemorrhagic diarrhea syndrome (AHDS) is strongly associated with C. perfringens strains harboring the netF toxin gene, detected in a substantial proportion of affected dogs and rarely found in those with uncomplicated or chronic diarrhea. Importantly, C. perfringens abundance declines rapidly within a few days in most cases of acute diarrhea without targeted therapy.[17] Therefore, antibiotic treatment is generally unnecessary in acute diarrhea, which is typically self-limiting.

In contrast, more pronounced increases in the DI occur in a subset of dogs with chronic inflammatory enteropathy (CIE). Chronic inflammation can result in mucosal remodeling and loss of transporters responsible for absorbing bile acids, fatty acids, and carbohydrates. Consequently, malabsorption contributes to increased luminal substrates, which promote microbial imbalances. Elevated concentrations of fatty acids and carbohydrates have been documented in dogs with CIE, particularly in those with high DI values.

Bacterial communities respond selectively to these altered substrates, leading to expansion of certain taxa and depletion of others, thereby increasing the DI. Notably, CIE is a heterogeneous condition, and not all affected animals exhibit dysbiosis, suggesting distinct pathophysiological mechanisms among subgroups.

A markedly elevated DI, especially when accompanied by depletion of P. hiranonis, indicates more severe functional intestinal disturbances. Persistently high DI values may serve as a marker of disease severity and are associated with lower likelihood of sustained clinical remission and increased need for long-term therapy. Dogs with severe dysbiosis often show only temporary improvement after FMT and typically require repeated treatments.

Importantly, dysbiosis characterized by reduced P. hiranonis does not automatically imply bile acid diarrhea. Many dysbiotic dogs and cats respond to dietary modification, immunomodulation, and FMT. Bile acid sequestrants should be considered only in the small subset of patients with persistent clinical signs and dysbiosis despite standard therapy.

A minority of clinically healthy animals (up to 15%) may demonstrate subclinical increases in the DI. In such cases, repeat testing after one to two months is advisable. Persistent abnormalities may indicate early functional changes. Screening of FMT donors should exclude subclinical dysbiosis

Therapeutic Approaches to Dysbiosis

Based on these new concepts, differentiating mild microbial shifts from severe dysbiosis and identifying the underlying cause are useful, as long-term outcomes vary accordingly. Severe dysbiosis often reflects chronic mucosal alterations and requires a multimodal treatment strategy targeting both the primary intestinal disorder and the associated microbial imbalance.

Dietary modification should always be implemented first, with adjunctive therapies—including fiber supplementation, probiotics, FMT, immunomodulatory treatment, and, in selected cases, antibiotics—added as appropriate. Each modality addresses distinct pathophysiological mechanisms.[18]

In dogs with CIE, increased DI values and decreased P. hiranonis frequently persist for months to years despite apparent clinical remission, likely reflecting ongoing mucosal dysfunction. Nutritional interventions can improve clinical signs by modifying luminal substrates but may not fully restore microbial balance.

Anti-inflammatory therapy with corticosteroids has been shown in one study to gradually reduce the DI and restore P. hiranonis over several months.[19] Among available treatments, FMT is the only intervention that consistently produces rapid normalization of the microbiome, often reducing the DI within days, particularly in antibiotic-induced dysbiosis.[20]

However, in animals with persistent mucosal dysain to consider is that of physical health. Both the behavioural responses and the physiological changes associated with anxiety have the potential to impact on the physical health of the dog. This may happen within the context of justified anxiety and acute stress and impact on physiological parameters which are traditionally used in a veterinary context to assist in diagnosis of disease. For example, altered heart rate, respiratory rate or biochemical markers can make interpretation of clinical examination and blood tests more challenging. In cases where anxiety is unjustified or ongoing the resulting chronic physiological impact and allostatic load can have significant impact on the physical health of the individual. This impact can be related to a range of physical health presentations. These include but are not limited to gastrointestinal, dermatological, cardiovascular, endocrine and metabolic conditions as well as sleep disturbance, reproductive impact and limitations to recovery from illness and surgery.

Summary

In summary, CE appears to be a gradual disease leading in some dogs and cats to mucosal remodeling with loss of transporters and therefore malabsorption of carbohydrates, lipids, and amino acids. These increased luminal substrates are available for bacteria leading to dysbiosis. A more severely increased DI, especially with depletion of the key bacterium P. hiranonis, can be a marker for more severe functional intestinal changes. These findings further explain and emphasize that diet needs to be the cornerstone therapy of canine CE, as highly digestible diets together with fiber and changes in other nutrients will have the largest impact in modulating the abnormal luminal environment due to malabsorption in CE.

These new studies clearly demonstrate that CE is a heterogeneous condition and cannot be treated with a one-size-fits-all approach; successful management of CE requires a multimodal approach focused on addressing the underlying pathophysiologic processes as well as host and microbiome function.

Key messages

1. Distinguishing between mild microbial shifts and severe dysbiosis is essential, as severity of dysbiosis influences long-term outcomes

2. A markedly elevated Dysbiosis Index (DI) is more suggestive of advanced disease—the greater the dysbiosis, the more profound the functional intestinal alterations

3. Diet, in combination with fiber, should always be the first-line therapy for acute diarrhea and chronic inflammatory enteropathies

4. FMT can be highly beneficial as adjunct therapy in CIE; however, patients with severe dysbiosis often require repeated administration

5. Clinical remission does not necessarily equate to functional restoration of the microbiome or complete resolution of underlying intestinal pathology

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Influence of Feeding Practices on Gut Health in Dogs and Cats

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INFLUENCE OF FEEDING PRACTICES ON GUT HEALTH IN DOGS AND CATS

Dr. Jan S. Suchodolski and Dr. Kelly S. Swanson

Gut Microbiome

A normal gastrointestinal tract (GIT) requires the presence of normal multiple functional pathways, both from the host and the microbiome. Dietary nutrients are broken down into smaller molecules mostly by mechanic and enzymatic host processes (e.g., gastric, pancreatic and duodenal enzymes) which will then be absorbed by the intestinal brush border. Some of these dietary substrates (e.g., carbohydrates, proteins and lipids) will be metabolized by bacteria, resulting in small molecules which provide energy, modulate immune responses and motility, and enhance gut barrier. Therefore, a normal microbiome is beneficial to the host, and changes in the microbiome will occur in response to the luminal environment. The microbiome is influenced by differing concentrations of macronutrients, luminal pH, and intestinal transit time.

Dietary Macronutrients

In healthy animals, amounts of dietary macronutrients affect microbiota and metabolites, although these changes are typically very minor (especially when compared to antibiotics and chronic intestinal disease) and remain mostly within reference intervals for the Dysbiosis Index (DI) (Oba et al., 2025a). Higher protein and fat are often associated with increases in E. coli, Fusobacteria and C. perfringens. On the other side, higher dietary fiber is associated with increases in saccharolytic bacteria such as Faecalibacterium, Megamonas, and Prevotella. Therefore, within established reference intervals, these groups (and others) change predictably in response to the amount of macronutrients. Especially digestibility of proteins is important. For example, in one study, dogs fed a homemade raw food diet which was very high in undigestible protein and high in fat but low in fiber had a mildly increased DI (approx. 0-2), driven by increased E. coli and C. perfringens, low Faecalibacterium and normal P. hiranonis (Schmidt et al., 2018). When raw food diets with more balanced macronutrient profiles were fed to dogs, all core bacteria and the DI remained in normal ranges (unpublished data). Therefore, it appears that nutrient profiles and digestibility seem more important compared to the actual format of the diet (kibble vs. wet vs. raw). This was also speculated as reason why some owners report frequent intestinal signs in dogs fed homemade raw diets, as they or often unbalanced and often contain low digestible components (Baum et al., 2024).

In healthy animals, the microbiome is stable when targeted assays are used (Sung et al., 2024). It is notable that some animals are highly stable for core taxa and the DI, while other animals can show more severe fluctuation over time, although again mostly within normal reference intervals. The bacteria that fluctuate more are typically E. coli, C. perfringens, and lactic acid bacteria like Streptococcus. These bacteria tend to overgrow when undigestible food components are present with the gut. One explanation in healthy animals is that the intestinal transit time is accelerated (e.g., through stress), therefore more undigested food enters the colon leading to blooms of these bacteria.

Fiber and Prebiotics

The microbiota is influenced by fiber and prebiotics included in diets. Fiber and prebiotics are indigestible carbohydrates that promote growth of beneficial microorganisms and can be divided into soluble/non-soluble and fermentable/non-fermentable fibers. The effect on the microbiota is variable, depending on the type and amount. Fermentable fibers and prebiotics are converted by colonic bacteria to short-chain fatty acids.

Diet Type

Different diet types (e.g., highly digestible, hydrolyzed protein, fiber-enriched, and novel protein diets) have been shown to induce clinical remission in chronic inflammatory enteropathy (CIE). While the exact mechanism why so many animals are food responsive remains unknown, a highly digestible diet reduces undigested substrates in the lumen, reducing bacterial overgrowth. This is important, as malabsorption of nutrients is an important part in CIE pathophysiology. Commonly used hydrolyzed or novel protein sold as gastrointestinal diets have typically high digestibility. Also, in fiber-enriched gastrointestinal diets, the macronutrients such as protein are typically also highly digestible, and together with the fiber component this can modulate the intestinal microbiota. Some studies have associated clinical remission in food-responsive enteropathies with partial, but not complete, normalization of the microbiome.

Abrupt Diet Change

Over the life of a pet, the diet may be changed multiple times. Most experts recommend a gradual diet transition (e.g., 7-10 day period) so that the gastrointestinal tract and microbiota can adjust. If the change is too abrupt, stool quality may suffer. In addition to changes in nutrient content, the moisture content and format of the diet is also important, as it can influence gut fill, transit time, and risk of gastrointestinal upset. Similar to what has been demonstrated in humans (David et al., 2014), a study conducted in dogs demonstrated that fecal microbiota and metabolite profiles rapidly adapt to dietary change (Lin et al., 2022). Whether dogs were shifted from a control kibble diet to the same diet supplemented with fiber or to a protein-rich canned diet, most fecal characteristics (i.e., pH, scores, dry matter content) and metabolites were stabilized within only 2 days. For fecal microbiota, the stabilization process took 6-10 days. More recent canine studies have shown similar results (Oba et al., 2025b; Wilson et al., 2025; Wren et al., 2025). To our knowledge, such studies have not been conducted in cats.

Altered Transit Time

Gastrointestinal microbiota populations and gut health outcomes may also be impacted by factors that influence gastrointestinal transit time. As summarized by Falony et al. (2018), increases in gastrointestinal transit time increase water absorption, stool firmness, and microbial population density. Longer transit time also increases microbial richness and causes a shift from saccharolytic to proteolytic metabolism. These shifts would be expected to increase fecal pH and shift fecal metabolite profiles. Of the nutrients, dietary fibers and other non-digestible carbohydrates (e.g., resistant starches, oligosaccharides) have the greatest impact on transit time. In general, soluble, viscous fibers have a tendency to delay gastric emptying and slow small intestinal transit rate, increasing transit time. The opposite is usually observed with insoluble fibers that provide bulk, increase transit rate, and reduce transit time (Fahey et al., 2004). Greater food consumption has also been shown to affect gastrointestinal transit time in dogs (Liang et al., 2026) and cats (Opetz et al., 2023). In both cases, greater food intake resulted in a reduction in transit time.

Physical Activity

While transit time and stool quality is not always impacted, studies in animal models and humans have demonstrated that physical activity can impact the composition and functional capacity of gut microbiota (Coenen et al., 1992; Mailing et al., 2019; Charlesson et al., 2025). Various potential mechanisms exist, including ischemia, heat stress, metabolic flux, and changes to gut barrier, mucus layer, hormone and bile acid production. Similarly, physical activity is typically expected to reduce transit time, affect stool quality, and alter gut microbiota populations in dogs and cats. The most extreme example is that of sled dogs, who have a high prevalence of diarrhea and other morbidity signs during long-distance racing events. In fact, diarrhea is a leading cause for discontinued racing during distance athletic events (Long, 1993; McKenzie et al., 2010). More moderate levels of exercise may not cause diarrhea and morbidity, but it may increase transit rate and loosen stools, consequently affecting fecal microbiota and metabolite profiles (Templeman et al., 2020; Oba et al., 2023).

Environmental Stressors

Environmental stressors may also impact gastrointestinal transit time, stool quality, and/or microbiota populations. It is common for animals to experience stress-induced defecation, including dogs and cats. While increased defecation frequency and loose stools may be inconvenient for pet owners, gastrointestinal stability is of great importance for dogs working in the military, police force, or search-and-rescue missions. A few studies have been conducted in working dogs to investigate the impact of transport stress. A pilot study conducted by Venable et al. (2016) demonstrated that commercial airplane travel increased fecal scores (looser stools) and modified fecal microbiota populations in Federal Emergency Management Agency (FEMA) search-and-rescue dogs without impacting search performance. A similar pilot study by Perry et al. (2017) reported increased salivary cortisol and rectal temperature of FEMA search-and-rescue dogs following helicopter travel, but without changes in search performance and fecal microbiota. More research is needed, but recent studies in working dogs have identified microbiome markers of behavioral traits and suggest an important role of gut microbiota in work performance (Craddock et al., 2022; Lin et al., 2024).

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Microbiota Modulation Through the Diet: How Much Do We Know?

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MICROBIOTA MODULATION THROUGH THE DIET: HOW MUCH DO WE KNOW?

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As in humans, there is great interest in supporting the gut health of dogs and cats. Recently, an expert consensus panel assembled by the International Scientific Association for Probiotics and Prebiotics (ISAPP) defined gut health as "a state of normal gastrointestinal function without active gastrointestinal disease and gut-related symptoms that affect quality of life" (Marco et al., 2026). Because gut functions are complex and operate as an integrated system, this gut health definition and concept include several functional domains: digestive physiology, gut microbiome, gut barrier, immune function, metabolism, and gut-brain axis. Even though the importance of the gut microbiome has been well accepted for many years, characterizing its composition and functional capacity was limited until the development of molecular tools in the early 2000's. The microbiome is composed of a complex and dense population of bacteria, archaea, protozoa, fungi, and viruses. Of these groups, bacteria are the most abundant and well-studied population. Gastrointestinal microbiome changes contributing to or resulting from digestive diseases have been documented in dogs and cats (Redfern et al., 2017; Ziese and Suchodolski, 2021). Animals under high stress or undergoing antibiotic therapy are also known to have poor stool quality and an altered gut microbiota (i.e., dysbiosis) (Pilla et al., 2020; Martini et al., 2025).

Many factors, including genetics, age, medications, disease status, may affect the composition and/or functionality of the gut microbiota, but diet is known to be one of the most important determinants (Barko et al., 2018; Alessandri et al., 2020; Wernimont et al., 2020). The gut microbiome and metabolome have been shown to quickly adapt and restabilize following a dietary change (Lin et al., 2022). The ingredient profile, nutrient composition, and processing conditions of the diet may all determine what is digested by the host and what reaches the colon where most of the microbial fermentation occurs. Carbohydrate-based substrates support saccharolytic microbial metabolism, primarily leading to the production of lactate, short-chain fatty acids (SCFA; acetate, propionate, butyrate), and gases (e.g., hydrogen, carbon dioxide, methane). SCFA serve as an energy source for colonocytes, increasing colon weight, mucosal surface area, and mucosal hypertrophy (Reinhart et al., 1994) and increase the production of gut peptides such as glucagon-like peptide (GLP)-1 that aid in curbing appetite, improving glycemic response and promoting weight loss (Massimino et al., 1998). Collectively, the increased epithelial cell growth, increased intestinal surface area, reduced intestinal pH coming from greater SCFA production increase the solubility and absorption of certain minerals such as calcium and magnesium (Whisner and Castillo, 2018). Microbial fermentation of protein-based substrates contributes to SCFA production, but to a lesser degree. The primary byproducts of protein fermentation include branched-chain fatty acids (BCFA), phenols, indoles, ammonia, biogenic amines, and sulfur-containing compounds. While some of these compounds may play a role in intestinal homeostasis, they are often associated with poor stool quality and intestinal disease (Davis et al., 2026).

Many dietary components may modify the abundance and/or activity of the microbiota, but dietary fibers and prebiotics are some of the most influential (Wilson and Swanson, 2024; Bhosle et al., 2025; Swanson et al., 2025). According to the United States Food and Drug Administration (FDA), fibers are "nondigestible soluble and insoluble carbohydrates with ≥3 monomeric units and lignin that are either intrinsic and intact in plants or isolated

and synthetic and demonstrate a physiologic health benefit in humans" (FDA, 2016). Chemically speaking, dietary fibers include cellulose, hemicelluloses, lignin (complex polyphenolic linked with cellulose and hemicelluloses), pectins, beta-glucans, gums, and mucilages (Livingston et al., 2016; Fahey et al., 2019). Non-digestible oligosaccharides and resistant starches are distinct categories, but function similarly in the gut. Common fiber sources in pet foods include beet pulp, wood cellulose, peanut hulls, grain co-products, fruit and vegetable pomaces and pulps, miscanthus grass, and gums.

Fibrous substances differ regarding their particle size, chemical linkages, molecular weights, and physiochemical properties, consequently impacting the microbial groups that contribute to their breakdown, rate and extent of fermentation, and amount and type of byproducts produced. In general, fibers that are soluble generally have a higher fermentability. The inclusion of "functional fibers", which provide nutritional and health benefits, increase the abundance and activity of beneficial bacterial groups that contribute to saccharolytic fermentation and SCFA production. Many of the primary SCFA-producing bacteria that break down resistant starches (Bacteroides, Ruminococcus), cellulose (Bacteroides, Ruminococcus), hemicelluloses (Bacteroides, Prevotella, Roseburia), pectin (Bacteroides, Eubacterium, Faecalibacterium), beta-glucans (Atopobium, Clostridium cluster XIVa, Enterococcus, Eubacterium, Lactobacillus, Prevotella), gums (Bifidobacterium, Lactobacillus, Ruminococcus), and non-digestible oligosaccharides (Bifidobacterium, Lactobacillus) have been identified (Dalile et al. 2019). Dietary fibers may be used to beneficially modify the gut microbiota and improve or maintain stool quality, but it depends on the fiber concentration and the extent to which they are fermented. Dietary formulas that contain too much fermentable fiber may result in flatulence, loose stools, and greater defecation frequency. Cats and large breed dogs are more responsive to the effects of microbial fermentation, so inclusion levels of soluble, fermentable fibers in their foods should be monitored more closely. Formulating diets to include a moderate fermentable fiber concentration and balance of insoluble and soluble fibers is typically recommended.

Prebiotics, defined as "substrates that are selectively utilized by host microorganisms conferring a health benefit" (Gibson et al., 2017), are another common functional ingredient category used in pet foods to support gut health. As reviewed by Wilson and Swanson (2024) and Swanson et al. (2025), prebiotics have been shown to beneficially modulate the gut microbiota, improve gut barrier and immune function, reduce the incidence of diarrhea, reduce insulin resistance, and support the metabolism of healthy dogs and cats. There is little evidence, however, to support prebiotic use in dogs and cats with existing gastrointestinal disease. To date, most accepted prebiotics used in pet foods fall into the category of non-digestible oligosaccharides. Fructans are the most common class of prebiotic used in pet foods and may include highly pure sources of short-chain fructooligosaccharides or long-chain inulins, or ingredients that naturally contain them in lower concentrations (e.g., chicory root). Galactooligosaccharides and lactulose are other common prebiotics added to pet foods intentionally or as components of other ingredients (e.g., legumes). Bacteroides, Bifidobacterium, Faecalibacterium, and Lactobacillus are some of the microbial taxa known to metabolize these substances and/or be increased in animals consuming them (Dalile et al., 2019; Wilson and Swanson, 2024). Because prebiotic concentrations can vary widely depending on source, prebiotic purity must be considered carefully when deciding on ingredient inclusion levels for dietary or supplement formulations.

Probiotics are defined as "live microorganisms that when administered in adequate amounts confer a health benefit" (Hill et al., 2014). Probiotics are thought to exert their effects within the gastrointestinal tract and gut-associated lymphoid tissue by displacing pathogens and restricting their growth, improving intestinal barrier function and reducing inflammation, producing antimicrobial substances, and regulating innate and/or adaptive immune responses (reviewed by Wilson and Swanson, 2024). Probiotics may not lead to large shifts in the gut microbiota composition, but they have been shown to reduce the incidence or duration of acute diarrhea in a few studies (Kelley et al., 2009; Bybee et al., 2011; Gookin et al., 2022). Their success in treating chronic gastrointestinal disease is less convincing. Probiotics are available in many formats, including powders, capsules, gels, pastes, and liquids. While many are provided in supplement form, probiotics are commonly included as a functional ingredient in dry kibble diets for dogs and cats. Probiotics are most commonly of the Bacillus, Bifidobacterium, Enterococcus, and

Lactobacillus genera. Microorganism viability is a key issue with probiotics, especially for those applied to complete and balanced pet foods.

Many pet food and supplement manufacturers develop blends of biotic substances and other functional ingredients. If a blend is composed of probiotic(s) and prebiotic(s), it may meet the criteria of a synbiotic. Synbiotics are defined as a "mixture comprising live microorganisms and substrate(s) selectively utilized by host microorganisms that confer a health benefit" (Swanson et al., 2020). Synbiotics may be classified as being complementary or synergistic, with potential benefits and mechanisms of action being similar to that of its individual components. Thus far, several synbiotic studies have been conducted in dogs and cats, with mixed results being reported. In addition to carefully establishing the dosage of all components, synbiotics must be strategically designed to match the live microorganisms with fermentable substrates that will enhance their stability, viability, and efficacy.

The final biotic category is that of the postbiotic, which is a "preparation of inanimate microorganisms and/or their components that confer a health benefit" (Salminen et al., 2021). A key benefit of postbiotics is their stability, as the microorganisms involved are intentionally inactivated. Postbiotics may be derived from yeast or bacterial species and inactivated by pasteurization, autoclaving, ultrasonication, high pressure, or other methods. In addition to modulating the gastrointestinal microbiota, postbiotics may enhance epithelial barrier function, modulate local and systemic immune responses, and modulate systemic metabolism. While the characterization of most probiotics and prebiotics should be relatively straightforward, postbiotics may include inactivated bacteria, bacterial lysates, and fermentation products that are highly complex. A few postbiotic studies have been conducted in healthy animals, with some benefits on gut microbiota (i.e., greater Bifidobacterium, Prevotella, Faecalibacterium) and immune response being reported (Lin et al., 2019; Koziol et al., 2023). A lot more research is needed in this emerging ingredient category to fully elucidate their potential benefits on dogs and cats.

Even though many significant advances have been made in this field over the past few decades, practical challenges pertaining to product commercialization and quality assurance exist. From a scientific perspective, the lack of consistency in regard to experimental methods and a high variation in product efficacy are other issues. Continued advancements in microbiome science, laboratory tools and assays, and machine learning and artificial intelligence are expected to improve our understanding of gut microbiota populations and how they interact with and affect host organisms, leading to next-generation strategies that have greater precision and efficacy. Emerging research in humans demonstrates the importance of designing synergistic fiber mixtures to support complementary microbial groups related to health (Cantu-Jungles et al., 2025). Moreover, individual variability in gut microbiota responses affect the health benefits coming from fiber interventions (de Campos Costa et al., 2026). While it requires more research in the target hosts (i.e., dogs, cats), the personalization and systematic design of functional fiber and biotic mixtures based on the canine and feline gut microbiota populations may be used to design pet foods and supplements in the future.

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Rebalancing the Gut: Microbiota-Targeted Strategies for Managing Chronic Enteropathies

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REBALANCING THE GUT: MICROBIOTA-TARGETED STRATEGIES FOR MANAGING CHRONIC ENTEROPATHIES

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Nutrition as a causative factor

Epidemiological studies support the role of diet as a risk factor in the pathogenesis of inflammatory bowel disease (IBD) in humans. For example, many human patients with IBD report diet as a trigger factor in the relapse of their disease [1]. A western diet, which is high in fat and red meat and low in fruits and vegetables is associated with an increased risk of IBD in humans [2]. We have recently identified possible nutritional risk factors in dogs with chronic enteropathy pre-illness [3]. Therefore, in all animals with chronic enteropathy, the diet history should be thoroughly scrutinized to identify any nutritional risk factors, which if avoided may help the animal attain remission.

Nutrition as a therapeutic strategy

Commercial therapeutic hydrolysed diets

Hydrolysed diets have been used successfully in the management of chronic enteropathy in dogs and cats [4-9], with some studies reporting a response in two-thirds of animals [6,8,10]. Commercial therapeutic hydrolysed diets contain proteins that have been hydrolysed to a size that theoretically evades a type $_1$ hypersensitivity response. However, it is currently unknown what type of immunological disturbance is present in canine and feline chronic enteropathy. In addition, some animals that have been sensitised to the intact protein can still react to the hydrolysed protein. However, there is scientific evidence that commercial therapeutic hydrolysed diets help companion animals with chronic enteropathies attain remission [5-8], as well as beneficially modulate the intestinal microbiota [9,11], immune system [12,13], and the intestinal brush border [5]. Due to these beneficial effects, this category of diets is often chosen first to trial in dogs and cats with chronic enteropathy.

Commercial therapeutic limited-ingredient novel protein diets

Studies have shown that nearly 50% of cats and 60% of dogs with chronic gastrointestinal signs responded to a novel protein diet [14,15]. One study assessing a salmon and rice based diet fed to 29 dogs with chronic enteropathy demonstrated that 19 dogs responded favourably to the diet despite there being no change in the mean histologic grade of intestinal biopsy specimens following dietary treatment.16 However, another study assessing a limited-ingredient salmon and rice based diet at the 10 day point showed a positive response in 10/26 (38%) dogs with chronic enteropathy with the number of macrophages and epithelial cells staining positive for activated NF-▌B decreasing significantly 4 weeks after treatment.[17] Typically the author trials this category of diet third, after either two different hydrolysed diets or one hydrolysed diet and a second trial of either fibre-enriched (dog and cat) or low-fat (dog).

Commercial therapeutic gastrointestinal diets

Studies have proven the efficacy of this category of diets in cats with idiopathic chronic gastrointestinal signs [18,19]. However, one study demonstrated that although this category of diet was able to induce remission in dogs with chronic enteropathy, the dogs were less likely to remain asymptomatic at subsequent rechecks when compared to dogs managed with a hydrolysed diet [20]. Therefore, these diets are often used when a dog will not eat a hydrolysed diet and novel protein is not available or unsuitable (i.e., higher fat content, not formulated for growth). The option of low-fat formulas in this category of diets makes them particularly desirable for the management of

intestinal lymphangiectasia or chronic enteropathy in dogs where low fat would also be beneficial, such as nausea, vomiting or osmotic/secretory diarrhoea. Interestingly, dietary fat did not seem to affect the outcome of cats with chronic diarrhoea [21], therefore, dietary fat may be less of a concern in cats with chronic enteropathy. In addition, the option of fibre-enriched in this category of diet also makes them desirable for those dogs and cats with large intestinal disease [22-26]

Stress management

Given the impact of stress on the gastrointestinal microbiome [27], reduction of this particularly around mealtimes is important to ensure the animal is appropriately transitioned to the therapeutic diet and that gastrointestinal signs are controlled and not exacerbated. Recommendations are typically made on an individual case by case basis utilizing the diet history form.

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Chronic Enteropathy II: Fecal Microbiota Transplantation in Dogs with Chronic Enteropathy

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CHRONIC ENTEROPATHY II: FECAL MICROBIOTA TRANSPLANTATION IN DOGS WITH CHRONIC ENTEROPATHY

Dr. Linda Toresson, DVM, PhD, Swedish Specialist in small animal internal medicine

Introduction

Fecal microbiota transplantation (FMT) is used to transfer feces from a healthy donor to a recipient with a disease to restore the intestinal microbiota and metabolome and decrease disease activity. In dogs, FMT has proven to be a safe, useful and microbiota friendly treatment for both acute or chronic gastrointestinal (GI) disorders, including non-responsive enteropathy.[1–8] Studies on FMT in cats are scarce and more studies are needed.[9–11]

FMT in people with gastrointestinal disorders

In people with recurrent Clostridiodes difficile infection (rCDI), FMT has greater efficacy than antibiotic treatments. Furthermore, numerous randomized controlled trials (RCTs) proves that FMT is linked to reduced disease activity and induction of remission in people with inflammatory bowel disease (IBD).[12] The microbial composition of the donor's stool and that of the recipient are critical factors influencing the success of the procedure.[13] In this study, the intestinal core bacteria were less depleted and bile acid conversion was preserved in FMT-responders both before and after FMT, in contrast to non-responders.

FMT can be administered via rectal retention enema, orally as lyophilized feces in capsules, or through endoscopic delivery in the duodenum. Neither the route of administration, nor using fresh or thawed frozen feces appeared to significantly influence the therapeutic outcome in patients with rCDI.[14]

FMT in dogs with chronic enteropathy

Studies and case reports in CE dogs suggest that most dogs treated with FMT have a good clinical response. In two prospective studies of CE dogs, 20 of 27 dogs showed reduced disease activity 15 days after 30 days of treatment with oral lyophilized FMT capsules, while rectal FMT in 17 out of 20 dogs with mild disease resulted in a significant decrease in Canine Inflammatory Bowel Disease Activity Index (CIBDAI) over three months.[4,6] Ten dogs remained stable for one year following FMT in one of these studies.[6]

In a large retrospective case series, 41 dogs with CE were treated with a median of 3 rectal FMTs as adjunct therapy, with a follow-up period of 3-41 months.[5] CIBDAI decreased significantly from 2-17 (median 6) at baseline to 1–9 (median 2) after FMT. Treatment response was noted in 31/41 dogs, of which 26 had a long-lasting response and 5 a short-lasting response. A similar response rate was seen in a recent prospective study using the same FMT protocol. FMT was administered to 39 dogs with refractory or partially refractory CE.[7] Combining data from these 2 studies, 43/59 responders (73 %) showed further improvement of clinical signs after FMT 2 or 3.[5,7] Treatment with a single rectal FMT may still be effective, especially in some younger dogs with less advanced disease,[6] but for CE dogs with long-lasting disease that have been refractory to multiple dietary and medical trials, current knowledge supports the use of repeated FMT.[5,7]

Clinical improvement following FMT was noticed as improved fecal scores and increased activity level.[5,7] Additionally, fewer flare-ups, decreased defecation frequency, increased appetite in hyporectic dogs and weight gain in underweight dogs was noted. FMT treatment enabled tapering of corticosteroids in a subset of dogs with CE. The corticosteroid doses were successfully tapered or withdrawn in 32% of the dogs after 2-3 FMTs.[7]

After FMT, most dogs appeared more playful, active

and interested in social interactions. Decreased fatigue and improved self-assessed quality of life has been reported in people with irritable bowel syndrome and IBD after FMT.[15,16] Potentially, FMT can result in altered levels of neurotransmitters, as several intestinal microbes directly or indirectly can synthesize neurotransmitters including GABA, glutamate, noradrenaline, dopamine and serotonin.

The intestinal microbiota before FMT, assessed with the dysbiosis index (DI), was compared between CE dogs with a long-lasting response versus short-lasting and non-responders combined by pooling data from three different studies.[5–7] According to the combined data, 85% of long-lasting responders had no or mild dysbiosis at inclusion, whereas 67 % of short-lasting and non-responders had marked dysbiosis at inclusion. The DI remained unaltered after 2-3 FMTs in short-lasting and non-responders.[7] In long-lasting responders, the DI decreased significantly after FMT and was stable for a minimum of 6 months. The abundance of Peptacetobacter hiranonis, the primary bile acid (BA) converting bacteria in dogs, was higher in long-lasting responders and positively correlated with the percentage of fecal secondary BA. Long-lasting responders had significantly higher percentage of fecal secondary BA, both before and after FMT, than short-lasting and non-responding dogs combined. These findings align with the effects of FMT in people with ulcerative colitis.[13,17]

FMT in cats

Studies on clinical effects of FMT in cats are scarce and consists of one case report and two prospective studies.[9–11] In the case report, one cat with non-responsive ulcerative colitis responded to two FMTs. In a study of 46 cats with chronic GI disorders, oral FMT capsules were given for 50 days.[10] The intestinal microbiota of the responders became more similar to that of healthy cats after treatment, but a positive response was only based on two owner-assessed questions. A recent study in CE cats compared the effects on disease activity and the DI before and after one single FMT to results from a control group of CE cats.[11] No significant improvement in DI or disease activity was noted after one FMT compared to control cats with CE. Cats with CE may, just as dogs, need more than one FMT for disease activity to decrease significantly. Unpublished clinical observations supports the use of FMT in cats with refractory or partially refractory CE, but further studies are needed.

Donor screening, FMT dose and preparation of feces

A condensed summary of the recommended donor screening, based on guidelines on FMT in companion animals, is available in the textbox.[8] It is recommended to re-test fecal donors every 6 months.
In several studies, 5 g of donor feces/kg BW of the recipient for dogs < 25 kg
BW, and 3 g of donor feces/kg for dogs with a BW > 25 kg have been
successfully used to treat NRE or partially refractory CE.[5–7]
In dogs with long-standing CE, 2-3 FMTs with 10-20 days interval is recom-
mended as one set of treatments. If there is no response after two FMTs,
a third treatment appears not to be effective.[5]

Fresh frozen feces, with or without cryopreservatives can be used. For cryo-preservation, 10% glycerol can be added to the fecal slurry before freezing. Donor feces should be stored in -20 °C. Frozen feces without cryopreserve-atives should be used within 1 month, based on the decline of culturable *P. hiranonis* after prolonged storage.[18] If cryopreservatives are used, frozen feces should be used within 3 months. Feces can be thawed overnight in a fridge, or in a 37°C water bath on the day of the procedure. The volume of the fecal slurry shouldn't exceed 20 ml/kg BW of the recipient.[8]

Donor requirements/screening:
12 months – middle age
Body condition score 4-6/9
Healthy, no medications
CIBDAI ≤ 3
No antibiotics for 6 months (minimum)
No raw food diets
Normal hematology and serum biochemistry

Fecal screening:
Normal DI
Exclude:
Salmonella spp
Campylobacter jejuni
Giardia and other intestinal parasites, including protozoa

Adverse effects

FMT is considered generally safe.[8] Rare adverse events are usually mild and
can occur in both responders and non-responders. The most prevalent adverse
events in one study were self-limiting diarrhea or worsening of diarrhea with-
in 48h after FMT.[5]

Lyophilized FMT capsules

Lyophilized FMT capsules have been used in several published studies. It is quick and convenient, as no special preparation of the dog prior to administration is required. It may, however, not provide the same booster effect as rectal FMT, as it is difficult to give the equivalent amount of feces in capsules as one rectal FMT provides. Lyophilized FMT capsules have been tested by the author to extend the interval between rectal FMTs in a few dogs with poorly responsive CE requiring frequent FMTs,. In several dogs, the capsules were not as effective as rectal FMT to control clinical signs in CE, even when feces from the same donor dog was used for both rectal FMT and lyophilized capsules. The difference in efficacy is likely dose dependent.

Less information on the microbial composition of the donor stool is available in commercial lyophilized capsules compared to personally screened donors. For instance, information on the abundance of the important BA converting bacteria P. hiranonis is rarely available for commercial FMT capsules. A high abundance of beneficial microbes, such as P. hiranonis and Faecalibacterium, is desired in donor feces.

In some European countries, it is illegal to produce FMT capsules and sell to pet owners as a veterinarian, as FMT capsules are classified as a drug, requiring a license to produce and distribute it, as well as quality testing of each batch. However, rectal retention FMT is allowed as treatment, under the hospital exemption.

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