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Effect of Wheat Dextrin Fiber on the Fecal Microbiome and Short-Chain Fatty Acid Concentrations in Dogs: Randomized, Single-Blinded, Parallel-Group Clinical Trial †

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† This article is a revised and expanded version of an abstract entitled "Effect of Wheat Dextrin on Fecal Short-Chain Fatty Acids in Dogs with Chronic Diarrhea", which was presented at the American College of Veterinary Internal Medicine Forum, Austin, TX, USA in 2023.

Abstract: The purpose of this study was to assess the practical implications of supplementing soluble fiber in the diet of dogs. Dogs with a history of managed or active chronic enteropathy were randomized to receive either wheat dextrin (fiber group) or maltodextrin (placebo group) mixed with food once daily for 28 days. Owners recorded a daily fecal score one week prior to and during the supplementation period. Shallow shotgun sequencing, quantitative PCR abundances of core bacterial taxa, and short-chain fatty acid (SCFA) concentrations via gas chromatography/mass spectrometry were performed on fecal samples collected before and after supplementation. Seventeen dogs completed the study (fiber group: nine dogs; placebo group: eight dogs). The change in fecal score differed between groups, with the fiber group developing softer stools ($p = 0.03$). Alpha diversity, quantified PCR abundances of the SCFA-producing taxa, and fecal SCFA concentrations were not different after supplementation in either group. Fecal microbial communities differed between baseline and day 28 for fiber and placebo groups ($p = 0.02$, respectively); however, the size effect (ANOSIM R = 0.18 and R = 0.26, respectively) was minimal. In this small group of dogs fed variable commercial diets, the additional intake of wheat dextrin powder supplement was well accepted, but had minimal discernable clinical benefit, and could soften stools.

Keywords: microbiome; wheat dextrin; dysbiosis index; fiber; metagenomics

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1. Introduction

A subset of dogs with chronic enteropathy have functional dysbiosis and alterations in microbiome functions [1]. While the etiology and pathogenesis of chronic enteropathy remain poorly understood, dysbiosis and altered microbial metabolites are thought to play a role in the development of disease [2–4]. A subset of dogs with chronic enteropathy have decreased gut microbial diversity, reduced abundances of major bacterial groups that are primary producers of beneficial short-chain fatty acids (SCFAs), and subsequently lower fecal SCFA concentrations compared to healthy dogs [2,5,6]. As such, therapeutic interventions that manipulate gut microbiota and the production of SCFAs have been of interest as a treatment option in dogs with chronic enteropathy.

Soluble dietary fibers are indigestible plant-based carbohydrates that are used in the management of diarrhea in dogs. Depending on the properties of the soluble fiber, some fibers can help to normalize stool form (i.e., viscous gel-forming fibers), and some have no effect at physiologic doses (i.e., readily fermented fibers) [7]. Soluble fibers are fermented by gut bacteria in the distal intestine to a variable degree. They can improve host health by promoting the growth of beneficial saccharolytic bacteria, such as *Bifidobacterium* and *Lactobacillus*, and by increasing the microbial production of SCFAs in the gut [8]. SCFAs, particularly butyrate, are an energy source for colonocytes and have anti-inflammatory properties by inhibiting the production of pro-inflammatory cytokines, such as interleukin-6 and tumor necrosis factor-alpha [2,9]. SCFAs can also strengthen the gut barrier by promoting the expression of tight junction proteins [10]. Lastly, the production of SCFAs results in an acidic luminal environment that prevents the overgrowth and colonization of some enteropathogens [11].

Previous studies in healthy dogs have evaluated the benefit of various fibers formulated into a diet [12–17]. In some cases, caregivers are unable or unwilling to feed their dog a veterinary therapeutic diet enhanced with fiber, and veterinarians may provide guidance to caregivers to use a fiber supplement added to the dog's food to infer a potential clinical benefit, such as to help in forming stool with viscous fibers or for a prebiotic effect with fermentable fibers. Limited studies are published evaluating the potential benefit to clinical parameters, such as stool form, and the gut microbiome by adding a fiber supplement to the food [18–21]. More information is needed to support the clinical practice of adding a fiber supplement to pet food.

Dextrin is a plant-based soluble fiber made from starchy foods (e.g., wheat, potato, corn) that has been shown in people and animal models to have a prebiotic effect and be readily fermented by gut microbiota into SCFAs [22–25]. Wheat dextrin is an affordable and readily accessible over-the-counter supplement. This supplement is flavorless and readily mixes with water so presumably would be well accepted by dogs when mixed with food. To date, no studies have evaluated the clinical application of supplementing wheat dextrin to dogs, including those with chronic enteropathy. Therefore, the primary objective of this study was to assess the practical implications of supplementing the diet of dogs with wheat dextrin soluble fiber and examining its impact on fecal consistency. To determine the effect of wheat dextrin on stool form, baseline fecal scores were obtained and compared to fecal scores obtained during supplementation in dogs that received wheat dextrin and dogs that received a placebo supplement. Adverse effects and the impact of supplementation on food intake were also recorded. The second objective was to determine the effect of wheat dextrin fiber supplementation on fecal microbial community composition and fecal concentrations of short-chain fatty acids (SCFAs). We hypothesized that wheat dextrin supplemented into the diet of dogs with a history of chronic enteropathy would be well accepted and tolerated, would not impact fecal form, would shift the fecal microbial diversity and community composition toward normal, and would increase fecal concentrations of SCFAs.

2. Materials and Methods

This study protocol was reviewed and approved by the Institutional Animal Care and Use Committee at Oregon State University (IACUC protocol 2020-0113). The study was designed as a randomized (allocation ratio 1:1), single-blinded, placebo-controlled parallel-arm trial. Client-owned dogs with a history of managed or active chronic large bowel, small bowel, or mixed diarrhea (>3 weeks duration) were recruited from the patient population at the Carlson College of Veterinary Medicine Veterinary Teaching Hospital between June 2021 and June 2022. Dogs that had a favorable (complete or partial) response to a therapeutic veterinary diet and/or immunosuppression could be enrolled. Owners were

given written consent prior to the enrollment of their dog in the study. Prior to enrollment, health status was determined by review of medical history, physical examination, CBC, serum biochemistry panel (including liver enzymes, kidney values, electrolytes, cholesterol, albumin, and globulin), urinalysis, and fecal helminth parasite analysis by the Modified Wisconsin Sugar Flotation Technique. Exclusion criteria included concurrent disease (such as intestinal parasitism, endocrinopathy, neoplasia) and the current use or historical use of an antibiotic within the past 2 weeks.

Dogs were randomized by weight (<20 kg or ≥20 kg) using an online randomization tool (https://ctrandomization.cancer.gov/tool/; accessed on 1 May 2021) to receive either a placebo powder (placebo group; maltodextrin, Spectrum Clinical MFG Corp) or commercial wheat dextrin powder (fiber group; Benefiber Original, Haleon Consumer Healthcare) mixed in their food once daily for 28 days. Maltodextrin is a manufactured digestible polysaccharide food additive commonly used as a filler and thickening agent in the food industry. Study powders were indistinguishable (both white, tasteless, and soluble in water), and owners were blinded to which powder their dog received.

Figure 1 is a schematic overview of the study timeline. Prior to supplementation with the study powder, dogs were observed for 7 days by pet owners and baseline fecal and appetite scores were obtained (observation period). To allow for gradual introduction of fiber supplementation and to avoid adverse effects associated with a rapid change in fiber intake, the amount of study powder (wheat dextrin or placebo) was gradually increased to the maximum amount (<20 kg: 3 teaspoons, equal to 4.5 g of soluble fiber; ≥20 kg: 6 teaspoons, equal to 9 g of soluble fiber) over the first 2 weeks of powder supplementation (adaptation period). For dogs in the placebo group, this equates to 9 g and 18 g of maltodextrin, respectively. The dogs received the maximum amount for the final 2 weeks of the study (treatment period). Owners were instructed to use a level teaspoon when measuring the study powder. A physical examination and CBC and serum biochemistry panel were performed at the end of the 28-day treatment period to monitor health status. A body condition score (1–9; World Small Animal Veterinary Association Global Nutrition Committee) was obtained during exams. A freshly voided fecal sample was collected at enrollment prior to initiating the study supplement (baseline) and on the 28th day of the supplementation period (day 28). Owners recorded a fecal score (1–7; 1 = very hard and dry, expelled as pellets; 7 = watery with no texture; Purina Fecal Scoring Chart, Nestle Purina Healthcare) and appetite score (0 = no food consumed; 1 = 25% consumed; 2 = 50% consumed; 3 = 75% consumed; 4 = all food [100%] consumed) daily during the 7-day observation period and during the 28-day supplementation period. Owners completed a questionnaire for the Canine Inflammatory Bowel Disease Activity Index (CIBDAI) score at enrollment and at the end of the 28-day supplementation period (insignificant disease, 0–3; mild, 4–5; moderate, 6–8; severe, 9–18) [26].

Feces were collected and stored at 4 °C for up to 12 h prior to storage in −80 °C. Fecal samples were shipped frozen on dry ice to Texas A&M Gastrointestinal Laboratory for DNA extraction using the MoBio Power soil DNA isolation kit [1], for quantitative PCR of core bacterial taxa [27], and for measurement of fecal SCFA concentrations [2], as previously described.

Twenty-one banked fecal samples obtained from healthy dogs that were age- and sex-matched to study dogs were included in the shallow shotgun sequencing analysis. These dogs were used as a control population to compare microbial composition between fecal samples (beta diversity) to determine if there was a shift toward normal after supplementation. These fecal samples were obtained from previous studies collected at different institutions in the United States [1]. These dogs were clinically healthy for at least 1 year without

any gastrointestinal signs and did not receive antibiotics, antacids, anti-inflammatory medications, or corticosteroids within the 6 months prior to sample collection.

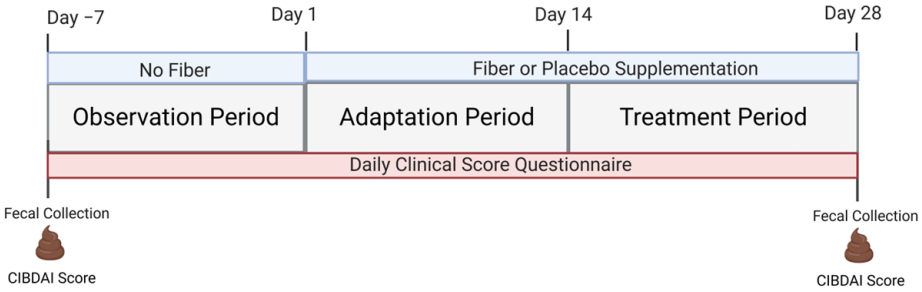


Figure 1. Schematic timeline. Dogs with chronic enteropathy were randomized to receive either wheat dextrin or placebo powder mixed with their food daily for 28 days. Dogs were monitored for 7 days prior to starting the study powder supplement (observation period). Dogs received the study powder supplement once daily mixed with their food for total of 28 days, including a 14-day period of gradually increasing amount of daily powder supplement to the maximum dose (adaptation period). Voided fecal samples were collected before and on the 28th day of powder supplementation.

2.1. Quantitative PCR Analysis and Calculation of Dysbiosis Index

A quantitative PCR for *Faecalibacterium*, *Turicibacter*, *Streptococcus*, *Blautia*, *Fusobacterium*, *Escherichia coli*, and *Clostridium hiranonis* was performed and a quantitative PCR-based dysbiosis index (DI) was calculated as previously described [27]. DI values were classified as normal (DI < 0 with all evaluated bacterial taxa within reference interval, RI), minor changes (DI < 0 with any bacterial taxa out of RI), mild to moderate changes (DI between 0 and 2), and significant dysbiosis (DI > 2). This classification has recently been shown to correlate with overall microbiome shifts as assessed by metagenomic sequencing [1].

2.2. Fecal Short-Chain Fatty Acid Measurement

Fecal concentrations of acetate, propionate, and butyrate were measured using a stable isotope dilution gas chromatography/mass spectrometry assay as previously described [2]. To account for differences in water content among fecal samples, final concentrations of fecal SCFAs were adjusted by fecal dry matter.

2.3. Fecal Metagenomics

Shotgun metagenomic sequencing was performed at an outside laboratory (BoosterShot, Diversigen, New Brighton, MN, USA). Library prep and downstream analysis using QIIME 2 2021.11 was performed as previously described [1]. Within-sample diversity was assessed via calculation of alpha diversity metrics (number of Operational Taxonomic Units [OTUs], Shannon, Chao1) [28–30] and between-sample differences were assessed via beta diversity measurement as Bray–Curtis dissimilarities (distances) [31] using the OTU tables rarefied to the sample with the lowest sequencing depth (67,300). Species richness can be defined as the number of unique OTUs and can be used as a proxy for bacterial species within the fecal sample. Chao1 estimates the minimal number of species (i.e., richness) present in a fecal sample. Shannon estimates the evenness of bacterial species distribution within the fecal sample. Metagenomic sequences are available via BioProject ID PRJNA975215.

2.4. Statistical Analysis

A sample size calculation was performed using an estimated effect size of 1.4 (Cohen's d) using fecal score data collected from the first 6 dogs that completed the study and assuming beta of 0.2 and alpha of 0.05 [32]. The calculation suggested enrolling at least 8 dogs per group.

Statistical analysis was performed using GraphPad Prism 10.1.1 (GraphPad Software, Boston, MA, USA). Normality was determined based on a combination of a Shapiro–Wilk test and an evaluation of QQ plots. Descriptive data are presented as median and range. Data were log-transformed to meet the assumption of normality. If normality was not met after log-transformation, a nonparametric test was performed. Either a paired Student *t*-test or Wilcoxon matched-pair signed-rank test was used to compare CIBDAI, fecal SCFA concentrations, qPCR bacterial taxa data, DI, and alpha diversity parameters between baseline and day 28 for each treatment group. For beta diversity, an analysis of similarity tests (ANOSIM) using the PRIMER 7 (Plymouth Routines in Multivariate Ecological Research Statistical Software, v7.0.13) software package on the Bray–Curtis dissimilarity index was performed to evaluate global differences in fecal microbial composition between treatment and healthy control groups. The R value generated by ANOSIM is scaled between −1.0 and 1.0. An ANOSIM R value close to 1.0 suggests dissimilarity between groups, and an R value close to 0 suggests similarity between groups.

For the relative abundance of microbial taxa, a Wilcoxon matched-pair signed-rank test was used for within-group analysis on the main microbial phyla, classes, orders, families, and genera (median relative abundance $\geq$ 0.1%) present in all fecal samples combined. Analysis of the species and strain levels was performed only if a significant difference was identified at the genus level.

For the fecal score data, an average daily score was calculated if more than 1 fecal score was recorded in 1 day. The fecal scores obtained during the 1-week observation period, 2-week adaptation period, and 2-week treatment period were averaged for each dog, and average fecal scores were compared between the 3 study periods (observation, adaptation, treatment) for each group using a Friedman test with Dunn's multiple comparison test. Additionally, the change in the average fecal score between the observation period and 28-day treatment period (28-day treatment period minus observational period) and between treatment groups (placebo and fiber) was compared using a Mann–Whitney U test. *p* values were adjusted for multiple comparisons using the Benjamini, Krieger, and Yekutieli method. A *p* or q value < 0.05 was considered statistically significant.

3. Results

3.1. Dogs

Twenty dogs were enrolled in this study. Two dogs randomized to the placebo group were removed after developing a health issue unrelated to the intestinal tract that required a change in their treatment regimen. In the fiber group, an owner opted to remove their dog from the study after the dog developed acute large bowel diarrhea 5 days after starting wheat dextrin. Of the 17 dogs that completed the study, 9 dogs were randomized to receive wheat dextrin, and 8 dogs received the placebo. The wheat dextrin group consisted of five large-breed dogs with a mean body weight of 30.4 kg (SD 6.7 kg; Labrador retriever [2], Australian shepherd, Great Pyrenees, Malamute [one each]) and four small-breed dogs with a mean body weight of 10.1 kg (SD 6.7 kg; Boston Terrier, Chihuahua, Shetland sheepdog, undefined mixed breed [one each]). Of the nine dogs, five were spayed females and four were neutered males, and the median body condition score was 4.5 (range, 3–7). The placebo group consisted of four large-breed dogs with a mean body weight of 33.1 kg (SD 16.5 kg; Gordon setter, Great Dane, Rottweiler, golden retriever mixed breed [one each]) and four small-breed dogs with a mean body weight of 12.3 kg (SD 4.7 kg; beagle [2], French bulldog, Chinese crested [one each]). Of the eight dogs, five were spayed females and three were neutered males, and the median body condition score was 5 (range, 4–7). There was no difference in body weight between treatment groups (p = 0.9). Dogs in the fiber group were supplemented with a median wheat dextrin soluble fiber dose of 0.4 g/kg/day (range,

0.2–0.9 g/kg/day), and dogs in the placebo group were supplemented with a median maltodextrin dose of 0.7 g/kg/day (range, 0.3–1.4 g/kg/day).

All dogs were primarily fed an adult maintenance commercial diet, of which most dogs (9/17) were fed a veterinary therapeutic diet (gastrointestinal diet: four dogs [two placebo group; two fiber group]; hydrolyzed protein diet: five dogs [four placebo group; one fiber group]). The total dietary fiber content of the commercial foods fed to the dogs at enrollment could be determined based on manufacturer product guides for 14/17 dogs (placebo group: 6/8; fiber group: 7/9). The total dietary fiber was not different between the groups (placebo group: median, 2.0 [range, 1.1–6.3 g/100 kcal]; fiber group: median, 2.0 [range, 0.8–5.8 g/100 kcal]; $p = 0.8$). Dogs were on their diet for at least 2 months prior to study enrollment. One dog in the placebo group was receiving budesonide. Three dogs (placebo group: two of eight; fiber group: one of nine) had a history of an upper gastrointestinal endoscopy that documented intestinal lymphoplasmacytic inflammation. One dog in the placebo group and two dogs in the fiber group were receiving a commercial probiotic. Baseline laboratory evaluation was largely unremarkable with the exception of mild hypercholesterolemia in six dogs and mild eosinophilia in three dogs. Fecal sugar centrifugation was negative for helminth ova in all dogs. Two dogs had a low-grade heart murmur on examination.

3.2. Clinical Scores and Adverse Effects

No clinically significant hematological or biochemical changes were observed between baseline and day 28. No adverse effects were reported by the owners of dogs that completed the study. All dogs maintained a normal appetite (score 4) during the 5-week study and accepted the study powder when mixed with their food. The placebo group had a lower CIBDAI score on day 28 when compared to baseline ($p = 0.02$). For the fiber group, the CIBDAI score was not different between baseline and day 28 ($p = 0.6$; Table 1). The average fecal score during the observation period (placebo: median, 2.5 [range, 2–3.5]; fiber: median, 3 [range, 1–5]) was not different from the average fecal score for the adaptation period (placebo: median, 2.75 [range, 2–3]; fiber: median, 3 [range, 2–4.75]; $p = 1.0$, respectively) and treatment period (placebo: median 2 [range, 2–3]; fiber: median, 4 [range, 2–5]; $p = 0.6$ and $p = 0.3$, respectively) for either groups. The change in the fecal score over the 28-day supplementation period differed between groups (placebo: median, 0 [range, −1.0 to 0]; fiber: median, 0.7 [range, −0.25–2.0]; $p = 0.03$) with a change to softer stools (i.e., higher fecal score) in the fiber group and minimal change in the placebo group.

Table 1. Canine Inflammatory Bowel Disease Activity Index, alpha diversity indices, fecal short-chain fatty acid concentrations, fecal qPCR abundances of select bacterial taxa, and dysbiosis index before and after 28-day supplementation with placebo powder or the fiber wheat dextrin. Data presented as median and range.

	Placebo Group (n = 8)		**Wheat Dextrin Group (n = 9)**	
	Baseline	**Day 28**	**Baseline**	**Day 28**
CIBDAI	3.5 (0–5) [a]	2 (0–3) [b]	3.0 (0–4)	3.0 (0–4)
Alpha Diversity				
OTU	269 (201–412)	344 (273–419)	352 (183–433)	316 (193–382)
Chao1	341 (258–494)	420 (304–574)	439 (354–567)	383 (214–478)
Shannon	3.7 (2.8–4.6)	3.7 (2.6–4.5)	4.3 (3.4–4.8)	4.2 (3.6–4.9)
Fecal Short-Chain Fatty Acids (μmol/g of fecal dry matter)				

Table 1. *Cont.*

	Placebo Group (n = 8)		Wheat Dextrin Group (n = 9)	
	Baseline	**Day 28**	**Baseline**	**Day 28**
Total SCFAs	213 (11–541)	180 (33–470)	358 (106–402)	283 (198–467)
Acetate	130 (7.2–373)	121 (21–267)	172 (56–278)	148 (118–252)
Propionate	59 (0–242)	38 (0–131)	95 (42–177)	128 (50–141)
Butyrate	23 (1.2–71)	18 (7.1–103)	37 (8.0–75)	23 (12–74)
Fecal qPCR Abundance (Log DNA)				
Faecalibacterium (RI: 3.4–8.0)	5.3 (2.5–7.1)	4.3 (2.4–6.9)	6.4 (3.4–7.4)	6.1 (3.2–7.0)
Turicibacter (RI: 4.6–8.1)	5.3 (4.0–6.6)	5.2 (4.3–7.2)	5.6 (4.5–6.9)	5.4 (4.6–6.5)
Blautia (RI: 9.5–11.0)	9.9 (7.4–10.3)	8.1 (6.9–9.3)	10 (9.1–10.2)	9.8 (9.3–10.1)
Fusobacterium (RI: 7.0–10.3)	8.1 (6.9–9.3)	8.2 (7.1–9.9)	8.7 (7.8–9.7)	9.0 (7.5–9.2)
Escherichia coli (RI: 0.9–8.0)	5.3 (1.1–7.9)	5.6 (1.0–7.2)	4.0 (1.1–6.6)	3.2 (0.9–6.3)
Streptococcus (RI:1.9–8.0)	4.2 (2.9–8.0)	3.4 (1.3–7.6)	4.5 (2.8–7.2)	4.9 (2.9–6.9)
Clostridium hiranonis (RI:5.1–7.1)	3.4 (0.1–6.0)	2.0 (0.1–6.1)	5.9 (4.7–6.3)	5.3 (3.9–6.1)
Dysbiosis Index (normal < 0)	1.0 (−4.9–8.0)	0.4 (−6.0–7.5)	−3.6 (−6.0–2.0)	−2.5 (−5.9–1.0)

For each row, columns within patient groups bearing a different superscript were significantly different ($p < 0.05$). CIBDAI, Canine Inflammatory Bowel Disease Activity Index; qPCR, quantitative PCR; RI, reference interval.

3.3. Fecal Short-Chain Fatty Acid Concentrations

No significant difference in fecal concentrations of total SCFAs, acetate, butyrate, and propionate were found between baseline and day 28 for both the placebo group and fiber group (Table 1).

3.4. Fecal qPCR and Dysbiosis Index

The abundance of *Faecalibacterium*, *Turicibacter*, *Blautia*, *Fusobacterium*, *Escherichia coli*, *Streptococcus*, and *Clostridium hiranonis* and the DI were not significantly different at day 28 compared to baseline for both groups (Table 1).

At baseline in the placebo group, three dogs had normal DI, two dogs had mild to moderate changes, and three dogs had significant dysbiosis. After placebo supplementation for 28 days, most dogs had similar DI values to baseline except that two dogs that had a normal DI at baseline experienced minor changes. At baseline in the fiber group, all dogs except one dog (eight of nine) had a normal DI. This dog had mild to moderate dysbiosis based on a DI value of 2.0. On day 28 of fiber supplementation, DI values were similar in most dogs except one dog had experienced minor changes, and this dog continued to have mild to moderate dysbiosis (Figure 2).

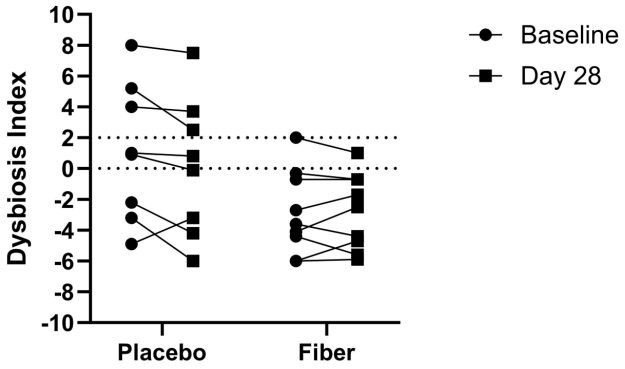


Figure 2. Scatter plot of dysbiosis index in dogs with chronic enteropathy at baseline (circles) and after 28 days (squares) of supplementation with either the placebo maltodextrin (n = 8 dogs) or the fiber wheat dextrin (n = 9 dogs). Dotted lines represent the normal ranges for the DI. A DI less than 0 indicates no shifts in the overall diversity of intestinal microbiota. DI between 0 and 2 suggests a mild to moderate shift in the overall diversity. DI above 2 is consistent with a shift in the overall diversity.

3.5. Fecal Shallow-Sequence Metagenomic Analysis

3.5.1. Evaluation of Fecal Microbiota

The sequence analysis yielded a total of 4,621,116 quality sequences (mean, 1,429,077) for all analyzed fecal samples (55 total; 34 from dogs with chronic enteropathy [baseline and day 28] and 21 from control dogs). Relative abundances of the main bacterial taxa were analyzed at the phylum, class, order, and genus taxonomic levels. Within-group analysis identified differences between time points in the relative abundance of the genera *Butyricicoccus* (p = 0.03) and *Blautia* (p = 0.02) for the placebo group and the genus *Coprococcus* (p = 0.02) for the fiber group. However, these findings were not significant after correcting for multiple comparisons.

3.5.2. Alpha Diversity

The number of OTUs and the Chao1 and Shannon diversity indexes were used to assess microbial richness and evenness within fecal samples. For both groups, the number of OTUs and the Chao1 and Shannon diversity indexes were not significantly different on day 28 compared to baseline (Table 1).

3.5.3. Beta Diversity

The Bray–Curtis dissimilarity index was calculated to assess microbial compositional differences between fecal samples and was visualized using principal coordinate analysis plots (Figure 3). When Bray–Curtis index was compared within treatment groups over the 28-day supplementation period (baseline vs. day 28), a difference was found for the placebo group (p = 0.02; ANOSIM R = 0.26) and fiber group (p = 0.02; ANOSIM R = 0.18), but the R values suggest significant overlap in microbial composition between time points. When the placebo group at baseline and day 28 was compared to the healthy controls, samples were different from the healthy control samples with some overlap (p = 0.01; ANOSIM R = 0.3) at baseline, and no evidence of a difference was appreciated at day 28 (p = 0.8; ANOSIM R = −0.10). For the fiber group, no evidence of a difference was found when compared to the healthy controls at baseline (p = 0.3; ANOSIM R = 0.05) with a difference noted at day 28, albeit minimal (p = 0.03; ANOSIM R = 0.19).

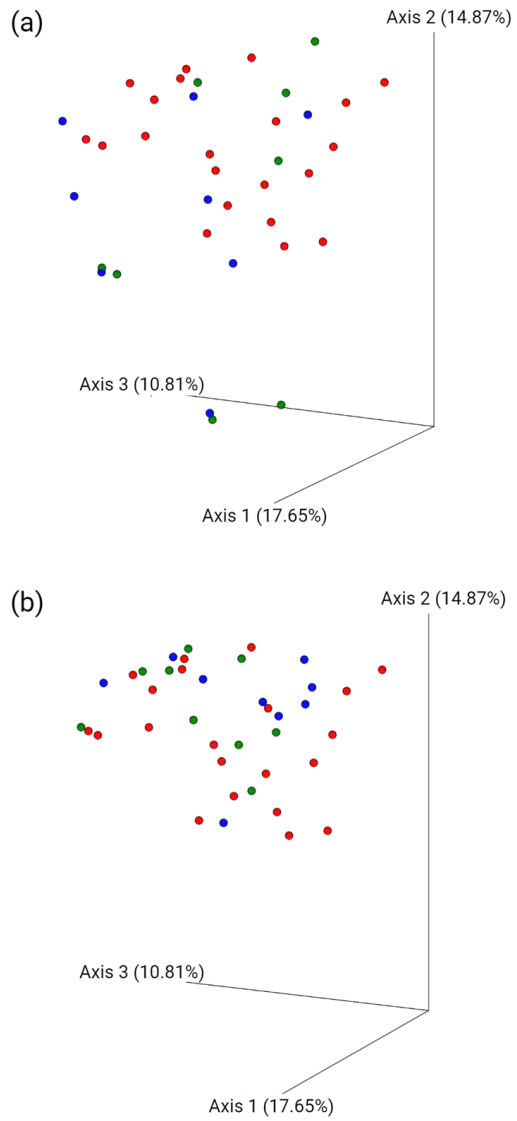


Figure 3. Three-dimensional representation of principal coordinate analysis (PCoA) plots of Bray–Curtis dissimilarity index for healthy dogs and dogs that received (**a**) placebo powder or (**b**) wheat dextrin powder mixed with their food for 28 days. Each dot represents a fecal sample (green: baseline; blue: day 28; red: healthy dogs).

4. Discussion

This was an initial study evaluating the clinical application of adding fiber supplement to the food of dogs, a common practice in veterinary medicine for dogs with diarrhea. The study evaluated a wheat dextrin fiber supplement that is affordable and readily accessible to pet owners. This study found that dogs readily accepted the wheat dextrin powder when mixed with the food, and this was attributed to the powder being tasteless and soluble in water. Based on owner-reported fecal scores, softer stools were reported with wheat dextrin supplementation in some dogs. This finding was mainly driven by fecal scores recorded for dogs <20 kg that received, on average, a higher amount of soluble fiber per day (1.5 g/kg/day) compared to dogs ≥20 kg (0.4 g/kg/day). This finding was contrary to our hypothesis, as readily fermentable fibers, like wheat dextrin, have no effect on stool output or stool water content and have no laxative effect in people [33]. Wheat dextrin is classified as a non-viscous fiber in people and thus lacks the stool-normalizing benefit of vicious fibers that form a gel when mixed with water, such as psyllium husk. Previous

studies showed that psyllium husk supplementation improved fecal form in dogs with diarrhea [18,19].

Contrary to our hypothesis, supplementing wheat dextrin fiber to dogs with chronic enteropathy did not result in a major shift in the fecal microbial community based on qPCR analysis of core bacterial taxa and shallow shotgun sequencing. Included in the qPCR analysis were four bacterial taxa (*Faecalibacterium*, *Turicibacter*, *Blautia*, *Fusobacterium*) known to produce SCFAs via fiber fermentation in the colon. Although in vitro studies using human fecal microbiota show that wheat dextrin increases the production of SCFAs within 24 h after supplementation, fecal SCFA concentrations did not change over the 4-week supplementation period for the dogs that received wheat dextrin in this study, contrary to our hypothesis [22–24]. This study did not evaluate the global metabolic capacity of microbiota before and after fiber supplementation. It is plausible that a benefit of prebiotic fibers is attributable to alterations in the metabolic activity of microbiota despite no accompanying alteration in microbial composition [25]. Characterization of the fecal metabolome in future studies could help us to better understand the impact of fiber on gut health.

There are several potential reasons why wheat dextrin did not have a significant impact on the fecal microbiome and SCFA concentrations in this study. The interplay between baseline microbiological profile (influenced by host health and habitual diet) and the ability of the GI microbiota to metabolize dietary fiber varies by individuals [34,35]. Thus, our population size may have been too small to appreciate a prebiotic effect. Second, habitual fiber intake was negatively associated with prebiotic response in people [35], and in our study, two of the nine dogs that received wheat dextrin were already on a commercial gastrointestinal therapeutic diet (total dietary fiber, 4.9 g/100 kcal and 5.8 g/100 kcal). Therefore, the variable dietary fiber intake of this group of dogs at baseline likely impacted the fermentation capacity of the microbial communities. Third, the dose of wheat dextrin in this study was extrapolated from the amount of total fiber provided by some fiber-enhanced therapeutic diets and chosen based on the reasonable amount of powder that can be mixed with food. Certain fiber-based prebiotics can have dose-dependent effects on the microbiome, and it is plausible that a higher dose would result in a prebiotic effect [36–38]. Lastly, while therapies targeting the microbiome can help in disease management, it is plausible that the underlying disease must be addressed to restore homeostasis of the microbiome.

Maltodextrin, a polysaccharide derived from starch hydrolysis, was selected as a placebo due to its similarity in appearance to wheat dextrin, ease of digestion, and lack of flavor [39]. Unlike wheat dextrin, maltodextrin is digestible. Maltodextrin has been accepted as placebo in various human and veterinary studies as a comparator to the effect of prebiotics, probiotics, and dietary supplements [40–42]. However, a recent systematic review in human studies questioned the validity of using maltodextrin as an inert placebo [39]. In that review, maltodextrin induced alteration on the gut microbiome in half of the studies. Microbial metabolites, including SCFAs, were found to be altered by maltodextrin in a smaller percentage (20%) of studies. These results are in line with our study where dogs receiving maltodextrin had a shift in the fecal microbial community, albeit minor, toward the healthy control group. The improvement in CIBDAI score in the placebo group after supplementation was unexpected as there is growing evidence of the effects of maltodextrin on increasing intestinal markers of inflammation in murine models [43–45]. However, it is unknown whether these changes observed in the placebo group are a direct effect of maltodextrin. In our study, a placebo group was needed to eliminate bias when evaluating clinical scores (i.e., fecal score and CIBDAI) and for determining

potential side effects. Until a more inert placebo can be identified and validated, future microbiome studies may consider no supplementation as placebo.

Our study had limitations. First, dogs were not fed a standardized diet. Some dogs had responded favorably to their current diet, and a change in diet for the purpose of the study could have resulted in relapse of clinical signs. Therefore, diet was not standardized. Additionally, dogs remained on their current feeding regimen, including frequency of meals, and the study supplements were not equally distributed in meals but rather given once daily with a meal. While these are added variables to consider, the practice resembles what can be observed in daily clinical practice. Second, wheat dextrin may contain traces of gluten. Wheat dextrin is starch and a byproduct of the processes that extract gluten from wheat; however, traces of gluten can remain in the product. Albeit rare, gluten can be an allergen in dogs and, in such cases, dogs could have an intolerance to wheat dextrin. Third, information regarding flatulence, bloating, and abdominal discomfort was not collected, and owners did not voluntarily comment on these potential side effects of readily fermentable fiber, like wheat dextrin. Fourth, despite efforts to randomize dogs to treatment groups, most dogs in the fiber group had a normal DI, which can occur in some dogs with chronic gastrointestinal signs [1]. While a normal DI suggests there are no major shifts in the microbiome, it does not exclude dysbiosis. Similarly, most dogs had insignificant to mild clinical signs of gastrointestinal disease, primarily chronic intermittent diarrhea, at the time of enrollment based on CIBDAI scores. Evaluation of wheat dextrin supplementation or other prebiotic fibers in dogs with major shifts in the microbiome or significant clinical signs could yield different results and warrant further investigation. Lastly, dogs did not receive a standardized dose of supplements on a g/kg basis, but rather caregivers were instructed to supplement with a standard amount based on the general size of the dog for ease of administration to mimic dosing recommendations (based on metric teaspoon) often used by veterinarians when recommending fiber supplement. This resulted in differences between the g/kg dose of soluble fiber and maltodextrin that each dog received. While the dose of soluble fiber may have influenced stool quality, no individual dog had a significant change in their microbiome based on the targeted analyses. This study could provide guidance for future studies evaluating a specific dose of wheat dextrin fiber in dogs.

5. Conclusions

Dogs that were fed a variety of commercial diets and supplemented with the fiber wheat dextrin had a change toward softer stools, especially at high daily doses. Wheat dextrin supplementation had minimal impact on the fecal microbiome and fecal SCFA concentrations at the doses used in this study.

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Informed Consent Statement: Owners were given written consent prior to the enrollment of their dog in the study.

Data Availability Statement: Metagenomic sequences are available via BioProject ID PRJNA975215.

Conflicts of Interest: Chi-Hsuan Sung, Rachel Pilla, and Jan Suchodolski are affiliated with the Texas A&M Gastrointestinal Laboratory that offers the dysbiosis index as a commercial test.

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