Petfood Applications of Inulin and Oligofructose

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ABSTRACT Published data on intestinal microbiota of dogs and cats are limited but suggest the presence of a complex and diverse colonic bacterial population (34 genera including 129 species) which majority of which are anaerobes. During the colonic fermentation of endogenous and undigested amino acids, several putrefactive compounds (i.e., ammonia, aliphatic amines, indoles, phenols and volatile sulfur-containing compounds) are produced and are responsible for the malodor of dog and cat feces. These fecal odor components also have been implicated as causes of colorectal cancer; therefore, dietary manipulation of gut microbiota towards a potentially more remedial community (Bifidobacterium and Lactobacillus) is gaining more attention. The health benefits derived from dietary supplementation of prebiotics (e.g., oligofructose and inulin) have been documented in human. However, little is known of a potentially similar role in companion animals. Feeding another prebiotic (i.e., lactosucrose) to dogs or cats is reported to increase the numbers of bifidobacteria and decrease the numbers of pathogens and the concentration of fecal odor components. In our laboratory, oligofructose supplementation numerically decreased the concentrations of ammonia and amines and increased the numbers of bifidobacteria in dog feces. J. Nutr. 129: 1454S–1456S, 1999.

KEY WORDS: • oligofructose • inulin • dogs • cats • petfood

Unlike the case for other monogastric animals (e.g., rats, mice, and pigs) or humans, published data on intestinal microbiota of dogs or cats are limited (Balish et al. 1977, Clapper 1970, Davis et al. 1977) but suggest the presence of a complex colonic bacterial population. This microbial population was found to be diverse (34 genera including 129 species; Balish et al. 1977), to contain large numbers of anaerobic bacteria (i.e., $10^{10}$ organisms/g dry feces; Davis et al. 1977) and to promote significant colonic fermentation (Banta et al. 1979). These colonic bacterial species not only contribute to normal physiologic function but also participate significantly in the causation or prevention of various diseases by biotransforming a variety of ingested or endogenous compounds to beneficial or harmful derivatives. This biotransformation was suggested to influence drug efficacy, toxicity, carcinogenesis and aging (Mitsuoka 1990). There is evidence that the composition of colonic microbiota can be altered by dietary ingredients. For example, source and level of dietary protein were found to influence the occurrence of pathogens (e.g., Clostridium perfringens) in dog feces (Amitberg et al. 1980). Therefore, the potential exists for dietary manipulation of colonic fermentation by changing the composition of microbiota in the colon toward a potentially more remedial community.

Origin and health hazards of fecal odor components

During the colonic fermentation of endogenous and undigested amino acids, several putrefactive compounds are produced (Bakke 1969a and 1969b, Tabor and Tabor 1985, Williams 1959) and are responsible for the malodor of the feces. These compounds include ammonia, aliphatic amines (alpha-, cadaverine, phenylethylamine, putrescine and tyramine), branched-chain fatty acids (isobutyrate and isovalerate), indoles (indole, 3-methylindole [skatole], 2-methylindole, 2,3-methylindole and 2,5-methylindole), phenols (phenol, p-cresol and 4-ethylphenol), and volatile sulfur-containing compounds (dimethyl disulfide, diethyl disulfide, di-n-propyl disulfide and di-n-butyl disulfide) (MacFarlane and Cummings 1991). These compounds are produced from amino acids by deamination (e.g., ammonia), deamination-decarboxylation (e.g., short-chain fatty acids) or decarboxylation (e.g., aliphatic amines) processes.

Many of these putrefactive compounds have adverse effects on colonic health. Some of these fecal odor components have been implicated as causes of colorectal cancer (Johnson 1977, Silverman and Andrews 1977). Ammonia may promote tumorigenesis (Lin and Visek 1991, Visek 1978). Phenol and p-cresol have been implicated in colonic cancer (Bingham 1988) and may exacerbate diseases such as ulcerative colitis (Ramakrishna et al. 1991). Despite the importance of the problem, no significant control measures have been taken because of the lack of knowledge of the relationship between diet and fecal odor components.

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Dietary manipulation of colonic fermentation

Because the gut microbiota can play a major role in host health, there is currently some interest in manipulation of the composition of the gut microbiota towards a potentially more remedial community. Prebiotics are nondigestible food ingredients that beneficially affect the host by selectively stimulating the growth and/or activity of one or a limited number of bacterial species already resident in the colon, the desired outcome is to improve host health (Gibson and Roberfroid 1995). Intake of prebiotics can significantly modulate the colonic microbiota by increasing the number of specific bacteria and thus changing the composition of the microbiota. In the early 1990s, Terada et al. (1992 and 1993) reported in the dog and cat, respectively, that feeding lactosucrose, a prebiotic, increased bifidobacteria while decreasing pathogenic organisms (i.e., C. perfringens).

Undigestible oligosaccharides in general, and oligofructose in particular, are prebiotics. They have been shown to stimulate the growth of endogenous bifidobacteria, which, after a short feeding period, become predominant in human feces (Gibson and Roberfroid 1995). Oligofructose, for example, stimulated the growth of bifidobacteria at the expense of others that are pathogens or less desirable (i.e., bacteroides, clostridia and coliforms); these were reduced in number to very low levels (Wang and Gibson 1993). Bifidobacteria (a major group of saccharolytic bacteria) constitute up to 25% of the total bacterial population in the gut of adult humans (Kawase et al. 1981), and their positive biological activities have received much attention (Tamura 1983).

Evidence from canine and feline species

Few published reports exist concerning the effects of oligofructose and inulin on canine or feline colonic bacterial populations. Willard et al. (1994) reported on supplementation of dog diets with 1% oligofructose. This research dealt with small intestinal bacterial overgrowth, and the results indicated positive health responses to such supplementation. After an adaptation period (46–51 d), oligofructose decreased ($P < 0.05$) both aerobic and facultative anaerobic bacterial numbers in fluid from both the duodenum and proximal part of the jejunum, as well as in the duodenal mucus.

In a study reviewed by Buddington and Sunvold (1998), adult beagles were fed diets containing cellulose or beet pulp plus oligofructose as the fiber source. Both groups had similar fecal concentrations of total anaerobes and total aerobes, but dogs fed oligofructose tended to have fewer Enterobacteriaceae and clostridia and greater numbers of lactobacilli. In addition, dogs fed oligofructose had longer and heavier small intestines, with more mucosa and greater absorptive surface area.

In a study reviewed by Gruffydd et al. (1998), cats fed diets containing 0 or 0.75% oligofructose had increased ($P < 0.05$) fecal concentrations of lactobacilli ($7.9 \times 10^3$ vs. $5.0 \times 10^3$). The cats fed oligofructose also had decreased concentrations of C. perfringens ($7.9 \times 10^3$ vs. $4.0 \times 10^3$) ($P < 0.10$) and Escherichia coli ($3.2 \times 10^9$ vs. $2.0 \times 10^9$) ($P < 0.05$) compared with the controls. This may indicate that oligofructose supplementation elicits a more remedial colonic microbial population.

In a recent study from our laboratory (E. A. Flickinger, A. R. Patil, H. S. Hussein and G. C. Fahey, Jr., unpublished data), 16 adult male beagles were fed a corn-based diet without or with 0.3, 0.6 or 0.9% supplemental oligofructose for 18 d. Dietary supplementation increased ($P < 0.05$) fecal short-chain fatty acid concentrations. Fecal ammonia concentrations were numerically lower ($P = 0.30$) in the oligofructose-fed groups than in the control group (2.20 vs. 4.07 mg/g dry feces). Fecal concentrations of putrescine and cadaverine were numerically lower ($P = 0.28$) in all of the oligofructose-supplemented groups (putrescine = $0.54, 0.29, 0.30$ and 0.20 and cadaverine = $0.20, 0.16, 0.06$ and 0 mg/g dry feces for dogs fed 0, 0.3, 0.6 or 0.9% oligofructose, respectively). The dogs that were fed the highest level of oligofructose tended to have lower ($P = 0.07$) concentrations of total phenols (0.61 vs. 0.80 mg/g dry feces) and higher ($P = 0.06$) numbers of bifidobacteria ($6.3 \times 10^5$ vs. $2.5 \times 10^3$ colony-forming units) in their feces compared with the control group.

Oligofructose in petfood ingredients

The practice of dietary supplementation of oligofructose, inulin or other oligosaccharides will depend on the contribution of natural prebiotics in petfood ingredients. Because of the lack of a database on concentrations of oligofructose as well as other oligosaccharides in petfood ingredients, 25 common petfood ingredients were selected and analyzed for oligofructose concentration (Hussein et al. 1998). In this study, the concentrations of three major subcomponents of oligofructose (i.e., 1-kestose [GF3], nystose [GF4] and 1F-β-fructofuranosyl-nystose [GF3F4]) in these ingredients were assayed. No oligofructose was detected in corn, corn distiller’s solubles, hominy, milo, brown rice, white rice, brewer’s rice, rice hulls, seaweed or soybean meal. On a dry matter basis, wheat co-products (bran, germ and middlings) contained the highest concentrations (0.40, 0.47 and 0.51%, respectively) of total oligofructose, followed by peanut hulls (0.24%), alfalfa meal (0.22%), barley (0.19%) and wheat (0.14%). The remaining ingredients (e.g., corn gluten meal, oats, rice bran, beet pulp, soybean hulls and canola meal) contained very low concentrations (<0.04%). This database should be expanded to include other petfood ingredients; in addition, the analyses should be expanded to include other oligosaccharide groups.

SUMMARY

It is important to establish accurate relationships between dietary ingredients (especially protein) and the microbial ecology of the dog and cat colon. Such relationships should provide guidelines for formulation of diets that contain the optimal levels and sources of proteins to meet amino acid requirements and contain prebiotics that support dog and cat health. These guidelines would only result from future in vivo experiments that focus on examining dietary supplementation of oligofructose, inulin and other oligosaccharides under different dietary and physiologic conditions.

LITERATURE CITED


