Nutrition Research Reviews (2014), **27**, 295–307
doi:10.1017/S0954422414000213

Dietary fibre and the importance of the gut microbiota in feline nutrition: a review

Kristel Rochus*, Geert P. J. Janssens and Myriam Hesta

Laboratory of Animal Nutrition, Department of Nutrition, Genetics and Ethology, Faculty of Veterinary Medicine, Ghent University, Heidestraat 19, 9820 Merelbeke, Belgium

Abstract

Domestic cats are obligate carnivores and in this light hindgut fermentation has been considered unimportant in this species. However, a diverse microbiota has been found in the small and large intestines of domestic cats. Additionally, *in vitro* and *in vivo* studies support the hypothesis that microbial fermentation is significant in felines with potential benefits to the host. Results on microbiota composition and microbial counts in different regions of the feline gastrointestinal tract are compiled, including a description of modulating host and technical factors. Additionally, the effects of dietary fibre supplementation on the microbiota composition are described. In a second section, *in vitro* studies, using inocula from fresh feline faeces and focusing on the fermentation characteristics of diverse plant substrates, are described. *In vivo* studies have investigated the effects of dietary fibre on a broad range of physiological outcomes. Results of this research, together with studies on effects of plant fibre on colonic morphology and function, protein and carbohydrate metabolism, and the effects of plant fibre on disease conditions that require a decrease in dietary protein intake, are shown in a third section of the present review. Conclusively, for fructans and beet pulp, for example, diverse beneficial effects have been demonstrated in the domestic cat. Both dietary fibre sources are regularly used in the pet food industry. More research is warranted to reveal the potential benefits of other fibre sources that can be used on a large scale in feline diets for healthy and diseased cats.

Key words: **Dietary fibre: Domestic cats: *In vitro* studies: *In vivo* studies: Oligosaccharides**

Introduction

Despite a short colon and the lack of a functional caecum as evolutionary adaptations to a strict carnivorous diet, considerable microbial fermentation occurs in the hindgut of domestic cats[1]. Microbiota of domestic cats are capable of fermenting a broad range of dietary plant fibres[1–4]. An *in vitro* study on the fermentation of various plant fibre sources using faecal inoculum of different species and ruminal fluid of cattle showed that the feline faecal inoculum produced the highest concentrations of total SCFA and acetate, supporting the hypothesis of substantial fermentation activity of the feline colonic microbiota[5]. In addition, *in vivo* research demonstrated that concentrations of volatile fatty acids in the colon of healthy cats were comparable with those measured in the forestomach of ruminants and large intestines of other single-stomached mammals, while concentrations in the small intestine were higher than those reported in other species[1]. The study, therefore, provides another indirect proof of the fermentation potential of the feline gut microbiota.

As in other species, the supplementation of plant fibre to feline diets has been associated with beneficial effects on several disease conditions (for example, Elliott[6], Rutgers & Biourge[7], Freiche *et al.*[8] and Fischer *et al.*[9]) and on the general health of the gastrointestinal tract[10–12]. The majority of the information on the benefit of dietary plant fibre is, however, extrapolated from human nutrition as described by Sunvold[13]. Dietary and metabolic interspecies differences should be taken into account, and specified *in vitro* and *in vivo* studies have been undertaken in dogs and to a lesser extent in cats. A first overview of *in vitro* and *in vivo* studies on the effects of dietary plant fibre supplementation to canine and feline diets was done by Sunvold[13] and Buddington and Sunvold[10]. Additionally, more recent studies in felines are described and discussed in the present review. Besides plant fibres, the importance of animal fibre for carnivorous species has been discussed by Depauw *et al.*[14] in cheetahs. Animal fibre is defined by the latter authors as low- to non-digestible (glyco)protein-rich substances that are potential substrates for large-intestinal fermentation[14]. Plantinga *et al.*[15] hypothesised that the consumption of whole prey, which is

Abbreviations: CKD, chronic kidney disease; FOS, fructo-oligosaccharide; GOS, galacto-oligosaccharide; MOS, mannanoligosaccharide; sc-FOS, short-chain fructo-oligosaccharide.

* **Corresponding author:** K. Rochus, fax +32 92647848, email rochuskristel@gmail.com

a major source of animal fibre, by feral cats might enhance gut health and affect the microbiota differently as compared with foods of plant origin. In domestic cats, however, little research has been done on animal fibre, despite the fact that animal meal, which is a source of animal fibre, is the main protein source in the majority of the commercially available extruded and wet diets[16,17] and despite the growing popularity of feeding raw meat-based diets containing animal fibre to pets[18]. The results of the scarce *in vivo* research in domestic cats on the effects of animal fibre on nutrient digestibility and faecal fermentation endproduct concentrations are also described in the present review.

Bacterial populations in the gastrointestinal tract

Apparent quantitative and qualitative differences have been shown among bacterial populations present in the different anatomic regions of the gastrointestinal tract of cats[10,19]. Qualitatively, the microbiota in different intestinal compartments tend to be more similar within the individual, than between corresponding compartments of different cats, suggesting a large inter-individual variation[19]. Quantitative data not only depend on host characteristics, such as age, gastrointestinal disease and environment, but also on the sample collection method, and the enumeration technique (culture plating *v.* molecular techniques). An overview of studies investigating the effects of these factors is given in Tables 1 and 2. For a review of different enumeration techniques, their advantages and disadvantages, see Suchodolski[20].

Microbiota in the different regions of the feline gastrointestinal tract

An overview of the microbiota in different regions of the gastrointestinal tract is given in Fig. 1 and has recently been reviewed by Minamoto *et al.*[21].

Stomach. Microbiota in the stomach contents of suckling kittens and adult cats were studied by Osbaldiston & Stowe[22]. The samples were collected after euthanasia, laparotomy and incision of the stomach wall, and culture-plating techniques were used[22]. In this part of the gastrointestinal tract, the microbiota mainly constituted of enterococci ($\log_{10}$ 5·64 (SD 0·97)). Likewise, all kittens and six out of nine adult cats showed similar counts of lactobacilli ($\log_{10}$ 5·55 (SD 0·67)). Comparable counts of five other genera of anaerobic micro-organisms were isolated from the stomach contents of some cats (*Streptococcus* spp., *Staphylococcus* spp., *Proteus* spp., *Bacillus* spp., *Pasteurella* spp., *Mima* spp., *Escherichia* spp., *Clostridium* spp., *Catenabacterium* spp., *Eubacterium* spp., *Bacteroides* spp. and *Veillonella* spp.). No effects of diet

Table 1. Overview of effects of the host factors age, gastrointestinal (GI) disease and environment on quantitative data of the gastrointestinal microbiota in cats

Host factor	Comparison	Main results	Reference
Age	Suckling *v.* weaned kittens	Diet change most important influence on changes in microbiota	95
	Suckling (4 weeks) *v.* weaned (9 months) kittens	Before weaning more diverse and variable faecal microbiota compared with post-weaning	43
	Suckling kittens *v.* adult cats	Predominant GI microbiota similar (qualitative)	22
	Weaned kittens *v.* adult cats	Faecal *Clostridium perfringens* conc.: 4·7 *v.* 11·6 $\log_{10}$ CFU/g faeces*	95,96,97
		Faecal *Escherichia coli* conc.: 2·7 *v.* 9·0 $\log_{10}$ CFU/g faeces*	95,97
		Faecal *Lactobacillus* spp. conc.: 6·0 *v.* 12·4 $\log_{10}$ CFU/g faeces*	95,97
		Faecal *Bifidobacterium* spp. conc.: 7·9 *v.* 7·5 $\log_{10}$ CFU/g faeces*	95,97
	Elderly *v.* young and adult cats	Lower faecal *Bifidobacterium* spp. conc.	96
	Geriatric cats *v.* weaned kittens	Predominant gastrointestinal microbiota similar (qualitative)	42,43
GI disease	IBD-inflicted *v.* healthy cats	Total bacteria: 10·0 *v.* 10·3 $\log_{10}$ CFU/g faeces	98
		Bifidobacterium spp.: 7·6 *v.* 9·3 $\log_{10}$ CFU/g faeces	98
		Bacteroides spp.: 8·3 *v.* 9·1 $\log_{10}$ CFU/g faeces	98
		Desulfovibrio spp.: 7·8 *v.* 7·3 $\log_{10}$ CFU/g faeces	98
		Similar faecal microbiota (qualitative)	99
		Enterobacteriaceae associated with duodenal mucosa: 17 *v.* 0 bacteria/mm^2†	100
	GI tract disease-inflicted *v.* healthy cats	↓ *Pasteurella*, *Bacteroides*, *Lactobacillus* spp. in duodenum (data not shown in 27)	27
Environment	Outdoor predatory *v.* indoor	Faecal *Bacteroidetes*: 2 *v.* 16 % of clones	36
		Faecal *Proteobacteria* absent *v.* present	36
		Lactobacillus spp. *v. Bifidobacterium* spp. most prevalent faecal micro-organism	36

conc., Concentrations; CFU, colony-forming unit; IBD, inflammatory bowel disease; ↓, decrease.
* Mean over both diets (34 and 53 % DM crude protein) and different ages of kittens (8, 12 and 16 weeks)
† Median of seventeen IBD-inflicted and ten healthy cats, ranges are 0–4219 bacteria/mm^2 for IBD-inflicted and 0–3 for healthy cats; values determined by fluorescence *in situ* hybridisation.

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press

Table 2. Overview of the effects of sampling and enumeration techniques on quantitative data of the gastrointestinal microbiota in cats

Techniques	Main results	Reference
Sampling techniques		
Direct needle aspiration *v.* endoscopy	Similar counts in duodenal fluids	25,26
Diluted *v.* undiluted endoscopic samples	Diluted endoscopic samples: underestimation of duodenal populations	25
Enumeration techniques		
Culture-plating *v.* molecular techniques	Culture plating: underestimation of the bacterial diversity, overestimation of *Bifidobacterium* spp.	26,39,44
16S rRNA gene analysis *v.* other molecular techniques (for example, FISH, analysis of the 60 kDa chaperonin gene)	Uncommon detection of *Bifidobacterium* spp.	36,39,44

FISH, fluorescence *in situ* hybridisation.

were observed between adult cats on a standard feline diet (Hill's Prescription Diet Feline c/d; Hill's Pet Nutrition Inc.) and adult cats on a chemically defined, liquid elemental diet (amino acids, carbohydrates, fats, vitamins and minerals), both fed for 6 weeks[22]. In humans, the microbiota of individuals on elemental diets decrease both in number and diversity, due to a lack of continuous supply of nutrients to the large intestine, since these diets are highly digestible[23]. In cats, the reason for the lack of diet effect was postulated to be the different passage rates through the gastrointestinal tract compared with humans.

The elemental diet thus provided a sufficient and continuous supply of nutrients necessary for bacterial survival and replication throughout the whole feline gastrointestinal tract on both diets[22]. No information on the number of meals was available in the above-mentioned study. However, this factor could influence the study outcome significantly. No raw meat or 'natural' diets were fed to the cats in this study.

Small intestine microbiota: duodenum. The microbiota, determined by culture-plate techniques, in the proximal part of the small intestine have been found to show quantitative and qualitative changes over time, and a considerable variation among individual cats at the same time point, regardless of the sampling technique (direct needle aspiration after laparotomy *v.* endoscopy; diluted *v.* undiluted endoscopic samples)[24,25]. Despite this variation, both studies found large numbers of bacteria in the proximal part of the small intestine in healthy cats, which is in accordance with the results of Johnston *et al.*[26,27] and Smith[28]. Due to the use of diluted endoscopic samples (Table 2), lower numbers of bacteria were observed by Muir *et al.*[29].

Controversy has arisen about the reliability and the clinical significance of the absolute numbers of bacteria counted from small-intestinal fluid samples[30]. In cats, considerably higher numbers of bacteria were found in the proximal small intestine in comparison with humans[31] and dogs[32], possibly as an adaptation to an obligate carnivorous diet[27]. The causative factors in the carnivorous diet responsible for this high bacterial number in the proximal small intestine are yet to be unravelled. Furthermore, it is suggested that feline host defences to indigenous microbiota may be particularly well developed, and small-intestinal bacterial overgrowth is not a common clinical syndrome in cats with chronic non-obstructive gastrointestinal disease (Table 1)[27].

Qualitatively, the most abundant bacterial phylum in the feline duodenal microbiota was Firmicutes, consisting primarily of Clostridiales, detected with both culture-plating

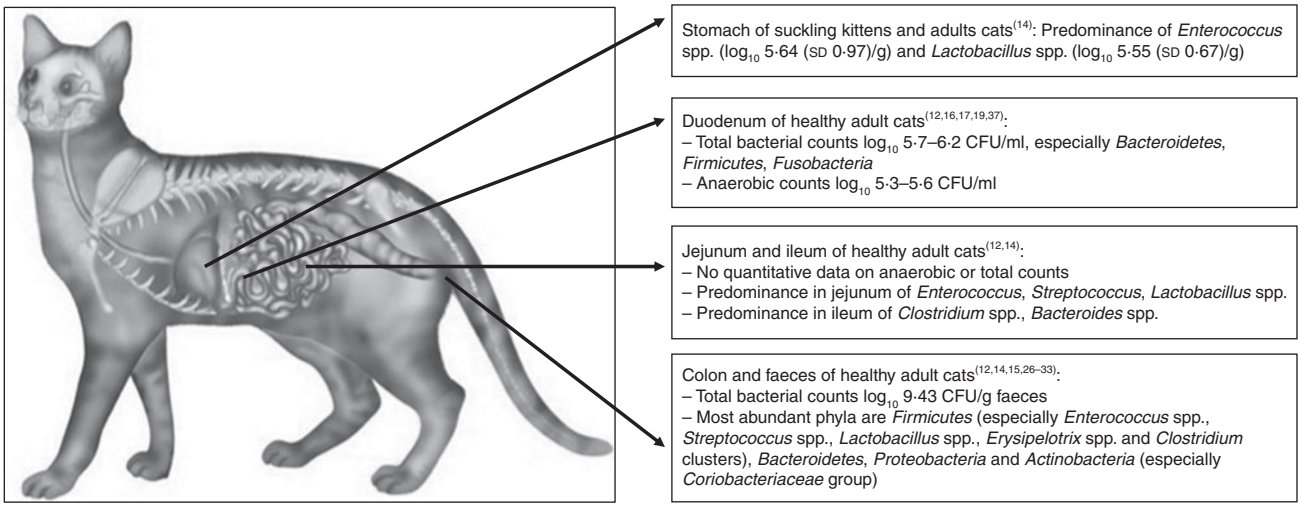

Fig. 1. Overview of the feline microbiota in different regions of the gastrointestinal tract. CFU, colony-forming unit.

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press

as well as molecular techniques[19,25,27]. The majority of the identified Clostridiales belonged to *Clostridium* clusters I and XIVa[19], the latter of which is known to encompass beneficial butyric acid-producing species[33]. *Clostridium* spp. were found in more than 50 % of the duodenal aspirates together with *Enterococcus faecalis*, *Bacteroides* spp., *Pasteurella* spp., *Streptococcus* spp., and unidentified Gram-negative aerobic rods[24]. In contrast, Johnston *et al.*[26,27] found *Bacteroides* spp. to be the most abundant anaerobic bacteria accompanied by *Eubacterium* spp. and *Fusobacterium* spp. Again, *Pasteurella* spp. were found to be the most abundant aerobic bacteria in duodenal fluid[26]. Ritchie *et al.*[19] did not find *Bacteroides* spp. in the duodenum, which could be explained by the low number of cats in which duodenal samples were obtained. The differences in the recovery of *Bacteroides* among the above-mentioned studies is most probably not affected by diet or age differences, since in all studies commercial wet or dry diets were fed and only young cats with an age between 1 and 2 years were included. Effects of cat sex could be excluded, as Sparkes *et al.*[24] used both female and male cats, whereas Johnston *et al.*[26] used female cats only, and both studies recovered *Bacteroides* spp. as (one of) the major bacterial species. Body-weight differences between studies are assumed to be small, since in all studies healthy cats were included, so an effect of body weight on the recovery of *Bacteroides* spp. seems unlikely.

Small intestine microbiota: jejunum and ileum. In the feline jejunum, the predominant species were *Enterococcus* spp., *Streptococcus* spp. and *Lactobacillus* spp., detected with both culture-plating as well as molecular techniques[19,22]. In addition, Osbaldiston & Stowe[22] observed higher frequencies of the latter two species and coliforms in the jejunum as compared with the stomach. In comparison with the microbiota of the stomach, jejunal microbiota showed the presence of three other bacterial species (*Micrococcus* spp., *Klebsiella* spp., *Enterobacter* spp.) and, in one cat, yeasts were cultured from jejunal contents. Again, no differences due to diet were found[22]. For more details on the diet types, see the 'Stomach' section.

The ileal microbiota consisted mainly of *Clostridium* spp. and *Bacteroides* spp.[19]. For the Clostridiales class, the predominant clusters were the same as in the duodenum[19]. Papasouliotis *et al.*[34] observed significantly higher numbers of bacteria, particularly aerobes, in the ileum compared with the duodenum after culturing intestinal juice, simultaneously aspirated at the two sites. As in the study of Johnston *et al.*[26], *Pasteurella* spp. were the predominating aerobe species in the duodenum, whereas in the ileum, enterococci and *Escherichia coli* were the most common aerobes[34,35]. As in Ritchie *et al.*[19], the predominating anaerobes in both segments were *Clostridium* spp., especially clusters I and XIVa[34,35].

Large intestine and faeces. In the colon and faeces of domestic cats, the most abundant phylum was Firmicutes, which was dominated by *Enterococcus* spp.[19,22,36], *Streptococcus* spp.[22], *Lactobacillus* spp.[19,22,36,37], *Erysipelotrix* spp.[37] or *Clostridium* clusters[37–39]. In two studies, the phyla Bacteroidetes and Proteobacteria were the second and third most abundant, respectively[39,40], whereas in the study of Tun *et al.*[41] the Bacteroidetes/Chlorobi group was the predominant phylum. In contrast, two studies[37,38] observed that the phylum Actinobacteria was the second most abundant in cats, while in the study of Desai *et al.*[36] even higher numbers of Actinobacteria were found. Likewise, in kittens and geriatric cats, the Coriobacteriaceae group, belonging to this phylum was highly present in the faecal microbiota[42,43] (see Table 1).

The discrepancy in predominant phyla is probably due to the underestimation of *Bifidobacterium* spp. when using the 16S rRNA target gene[19,39,44] (see Table 2). Desai *et al.*[36] stated that, in general, the overall taxonomic profile is similar in domestic cats to that of most of the studies in mammals. In mammalian faecal microbiota a predominance of bacteria belonging to the phylum Firmicutes has been shown with variation in constituent bacterial species due to animal species[45]. This finding was confirmed in a recent study using the newer massive parallel 16S rRNA gene pyrosequencing technique on faecal samples of pet dogs and cats[38].

On the species level, Sparkes *et al.*[46] observed *Bacteroides* spp. and *Clostridium perfringens* as predominant anaerobes, and *E. coli*, *Lactobacillus* spp. and *Plesiomonas shigelloides* as predominant aerobes in the faecal microbiota of cats. Again, Itoh *et al.*[47] found Bacteroidaceae and Eubacteriaceae (phylum Firmicutes) as the most predominant families in the faecal microbiota of conventionally raised cats, followed by Clostridiaceae, Streptococcaceae and Lactobacillaceae families.

A potential problem with the use of faecal samples to estimate the microbiota in the large intestine is a possible underestimation of *Lactobacillus* spp., especially when enumerated with general bacterial primers. The use of group-specific primers might circumvent this problem[39]. Additionally, the lumen and the mucosa of the gastrointestinal tract harbour bacteria, which are hypothesised to be both represented in the faecal microbiota[48]; hence, faecal samples appear to represent the 'total' gut microbiota, at least qualitatively. Quantitatively, the faecal excretion of mucosal bacteria might be an underestimation of the actual number of bacteria attached to the mucosa. The fraction of mucosal bacteria that is excreted in the faeces remains to be investigated. In addition, using faecal samples has advantages of non-invasive sampling techniques[36] and a larger availability compared with intestinal fluids[38].

Effects of dietary fibre on the gastrointestinal microbiota

The results of the studies on the effects of supplementing fructo-oligosaccharides (FOS), short-chain FOS

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press

(sc-FOS), galacto-oligosaccharides (GOS), sc-FOS+GOS and lactosucrose[24,40,46,49–53] on the faecal microbiota are depicted in Table 3. In most of the studies, an increase in faecal *Bifidobacterium* spp. was observed. Barry *et al.* concluded that FOS might be a useful fibre source for promotion of feline gastrointestinal health based on changes in the faecal microbiota[51]. The lack of a significant treatment effect in the studies of Sparkes *et al.*[24,46] and a rather low response (increase of $\log_{10}$ 0·5) in the study of Kanakupt *et al.*[52] might have been due to the low inclusion level of (sc-)FOS. When GOS or a sc-FOS + GOS combination was supplemented, the increase in *Bifidobacterium* spp. counts was higher (increase of $\log_{10}$ 0·7 and 0·9, respectively)[52]. In contrast, the decreased numbers of bacteria in cats fed diets supplemented with sc-FOS in the study of Johnston *et al.*[49] might also have been due to other differences in diet composition (different level of protein, fat and other carbohydrates). Additionally, the number of animals used in this experiment was very low (three for the control diet and four for the 'FOS diet')[49].

In two feline studies, the effects of a synbiotic formulation on the faecal microbiota of healthy cats were studied[37,53]. The first study used a mixture of seven probiotic strains and a blend of FOS and arabinogalactans[37], whereas in the second study a combination of GOS and a specific *Bifidobacterium* strain was used[53]. No changes in major bacterial phyla were discovered between faecal samples before, during or after administration of the synbiotic supplement[37]. During product administration, probiotic species were detected in eleven out of twelve cats, and abundances of *Enterococcus* spp. and *Streptococcus* spp. were increased in at least one time point during administration and decreased back to baseline values after discontinuation of the supplementation[37]. Likewise, in another study, cats were supplemented with a probiotic for 4·5 weeks, and the supplemented species (*Lactobacillus acidophilus*) was recovered from faeces of cats during the supplementation period, but not before or after the supplementation was ceased[54]. In contrast, 10 d after cessation of daily synbiotic administration, a significant increase in faecal bifidobacteria content was observed compared with counts before supplementation[53].

In vitro and *in vivo* fermentation studies

As stated previously, intestinal fermentation might be an important process in healthy domestic cats. A literature overview of the fermentation studies done *in vitro* and in domestic cats is given in the next sections. Furthermore, for several disease conditions, dietary fibre can exert beneficial effects. An overview of the effects of dietary fibre on a broad variety of diseases was beyond the scope of the present review. Therefore, the focus is on

Table 3. Overview of the effects of dietary fibre on the feline gastrointestinal microbiota

Fibre source	Control	Dosage	Main results (fibre source *v.* control)	Unit	Reference
FOS	No added fibre	0·75 % DM	No changes in duodenal microbiota	$\log_{10}$ CFU/ml	24
FOS	No added fibre	0·75 % DM	Trend towards ↑ total anaerobes (+0·5) and total bacteria (+0·4) Significant ↑ *Lactobacilli* (+2·2), *Bacteroides* spp. (+1·5) Significant ↓ *Escherichia coli* (−1·2), *Clostridium perfringens* (−1·7)	$\log_{10}$ CFU/g faeces	46
FOS	Cellulose	4·0 % as-is	↑ *Bifidobacterium* spp.: 11·6 *v.* 10·4 ↓ *E. coli*: 8·4 *v.* 9·3	$\log_{10}$ CFU/g faecal DM	51
sc-FOS	Cellulose	0·5 % as-is	↑ Faecal *Bifidobacterium* spp.: 9·9 *v.* 9·4	$\log_{10}$ CFU/g faecal DM	52
sc-FOS	No added fibre	1 g/d	Trend towards lower numbers of bacteria in duodenum (no data specified in 49)	$\log_{10}$ CFU/ml	49
GOS	Cellulose	0·5 % as-is	↑ Faecal *Bifidobacterium* spp.: 10·1 *v.* 9·4	$\log_{10}$ CFU/g faecal DM	52
sc-FOS + GOS	Cellulose	1·0 % as -is	↑ Faecal *Bifidobacterium* spp.: 10·3 *v.* 9·4	$\log_{10}$ CFU/g faecal DM	52
FOS	Cellulose	4·0 % as-is	↑ Faecal Actinobacteria: 11 *v.* 7	%	40
Pectin	Cellulose	4·0 % as-is	↑ *Clostridium perfringens*: 11·5 *v.* 10·3 ↑ *Lactobacillus* spp.: 11·2 *v.* 10·9		51 51
Pectin	Cellulose	4·0 % as-is	↑ Faecal Firmicutes: 43 *v.* 34	%	40
Lactosucrose		175 mg/d	↑ *Lactobacilli* spp. (day 7, +0·9; day 14, +0·8)†, *Bacteroides* spp. (day 14, +0·2)†, bifidobacteria‡ (day 7, +9·0; day 14, +8·7)†, Spirochaetaceae (day 14, +0·9; only frequency of occurrence significantly different) ↓ *Clostridiae** (day 7, −1·0; day 14, −0·2)†, *Enterobacteriaceae* (day 7, −3·5; day 14, −1·6)†, Fusobacteria (day 7, −1·2)†, Staphylococci (day 7, −1·9)†	$\log_{10}$ CFU/g faeces %	50

FOS, fructo-oligosaccharides; CFU, colony-forming unit; ↑, increase; ↓, decrease; sc, short-chain; GOS, galacto-oligosaccharide.
*Mean of lecithinase-positive and -negative Clostridia at all time points.
† Differences between specified time point and day before administration are calculated.
‡ Absent before lactosucrose administration.

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press

diseases in which dietary protein restriction is a treatment cornerstone, such as chronic kidney disease (CKD).

In vitro *fermentation studies*

All *in vitro* fermentation studies done in cats used fresh faecal samples as a source of inocula[3,4,55,56]. Samples were immediately placed in a sterile sample bag[55] or within 15 min post-voiding in a container pre-filled with carbon dioxide and kept on crushed ice before incubation (maximum 4 h post-voiding of first sample)[56]. A recent study with canine faeces revealed that chilling the faecal samples for 24 h in crushed ice maintained the fermentation characteristics of substrates compared with incubation with fresh faecal inoculum[57]. Furthermore, in another *in vitro* study from the same group, faeces appeared to be applicable for inoculum preparation for *in vitro* screening purposes, although the fermentation in the proximal and transverse colon might be overestimated[58]. These inocula were used to ferment different fibre sources. In the studies of Sunvold *et al.*[3,4], fermentation was described based on organic matter disappearance from the culture media and the concentrations and ratios of SCFA. Only one study in cats investigated the interdependency of fermentation characteristics of select fermentable fibres[56]. The main conclusions of the *in vitro* studies are compiled in Table 4.

Besides the source (intestinal fluid *v.* faeces) of the inocula, another important factor that influences the outcome of *in vitro* fermentation studies is the diet of the inoculum donors[4,55]. This statement contrasts with the above-mentioned findings of Osbaldiston & Stowe, where no effect of diet on the microbiota composition was found (see the 'Stomach' and 'Jejunum and ileum' sections)[22]. However, the latter study used culture techniques, which underestimate the diversity of the microbiota[19], whereas in the *in vitro* system, the end-product and metabolite concentrations and the volumes of gas produced upon fibre fermentation are measured, hence the metabolism of the microbiota as a whole is evaluated. In the study of Barry *et al.*[55], cats were adapted to different dietary fibre sources (cellulose, FOS, pectin) *in vivo* before fermenting these fibres *in vitro*. Overall, *in vivo* adaptation to FOS or pectin resulted in higher *in vitro* SCFA concentrations and more gas produced as compared with adaptation to cellulose[55]. The differences found in the present study were of a great magnitude and most probably biologically relevant considering careful extrapolation to *in vivo* situations (for example, total *in vitro* gas produced in ml/g DM averaged over the three adaptation diets: 0·0 for cellulose *v.* 90·5 for FOS *v.* 61·1 for pectin; total SCFA in mmol/g DM averaged over the three adaptation diets: 0·1 for cellulose *v.* 5·0 for FOS *v.* 2·8 for pectin)[55]. Sunvold *et al.*[4] compared *in vitro* organic matter disappearance and SCFA production from fermentation of different fibre sources, using inocula from cats fed a diet without supplemental fibre or supplemented with beet pulp. *In vitro* fermentation of fibrous substrates by faecal microbiota from cats increased when fermentable fibre was included in the donor diet[4].

In vivo studies

The *in vivo* effects of dietary fibre, including oligosaccharides and animal fibre, on nutrient intake and digestibility, faecal characteristics, the morphology and function of the colon, N and energy metabolism, and some disease conditions are compiled in this section.

Effects of dietary fibre on nutrient intake, nutrient digestibility and faecal characteristics: viscous fibres. Sunvold *et al.* performed *in vivo* investigations following *in vitro* fermentation trials[3,4]. These authors concluded that the *in vitro* method appeared to be a good estimator of *in vivo* fermentation with the exception of the most fermentable fibres (pectin and gums with high viscosity). However, the *in vivo* fermentation calculation, based on the comparison of organic matter in food and faeces, was lower than could be predicted *in vitro* because of a decrease in digestibility of the other nutrients in the diet[4].

Besides difficulties of extrapolating *in vitro* data, another disadvantage of supplementing viscous fibres *in vivo* was an increased defecation frequency and a poor stool quality with a supplementation level of 9·5 % total dietary fibre[3].

Table 4. Overview of *in vitro* fermentation studies of plant fibres using feline faecal inoculum

Study	Main results	Reference
In vitro fermentation of selected fibre sources by cat faecal inoculum	Citrus pectin, guar gum and locust bean gum highly fermentable, cellulose poorly fermentable	3
In vitro fermentation of selected fibre sources by cat faecal inoculum – influence of diet composition on fermentation parameters	Fermentable fibre included in donor diet resulted in ↑ *in vitro* fermentation of fibrous substrates	4
Adaptation of healthy adult cats to select dietary fibres *in vivo* affects fibre fermentation *in vitro*	*In vivo* adaptation to FOS or pectin resulted in ↑ *in vitro* SCFA concentrations and gas production	55
Incubation of select fermentable fibres with feline faecal inoculum – correlations between *in vitro* fermentation characteristics	Differences in fermentation rate resulted in typical changes in bacterial fermentation products Fibre supplementation, stimulating bacterial propionic or butyric acid production, cause of beneficial effects might also be a decrease in the large-intestinal production of putrefactive compounds	56

↑, Increase; FOS, fructo-oligosaccharides.

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press

Loose stools with a strong odour were also confirmed by Bueno *et al.*[11,12] when a pectin–gum arabic blend was included in the diets (total dietary fibre 8·6 %). It has to be noted that the doses used in the above-mentioned experiments are very high. Barry *et al.*[51] supplemented a lower dose of pectin (4 % as fed) to domestic cats and observed softer faeces as compared with the control group, cellulose. The decrease was, however, small and the authors concluded that pectin, like FOS, might be a useful fibre source for the promotion of feline gastrointestinal health[51]. Sunvold *et al.*[3], on the contrary, advised the use of a moderately fermentable fibre source, such as beet pulp, in feline diets (see the 'Effects of dietary fibre on nutrient intake, nutrient digestibility and faecal characteristics: moderate (beet pulp) and less fermentable fibre sources' section). In a recent feline study, another viscous fibre source, guar gum, was used as the sole source of fermentable soluble fibre, supplemented to a moderate-protein diet[59]. The apparent protein digestibility coefficient tended to be lower in guar gum- (71·8 (SEM 3·6) %) than in cellulose (79·7 (SEM 1·0) %)-supplemented cats, and the faecal and plasma metabolites from protein fermentation (for example, faecal ammonia in mg/l: 390 for guar gum *v.* 227 for cellulose; plasma isovaleryl- +2-methylbutyrylcarnitine in μmol/l: 0·32 (SEM 0·06) for guar gum *v.* 0·21 (SEM 0·05) for cellulose) were higher in the former cats, which confirmed highly viscous fibres to be less suitable soluble fibre sources for *in vivo* use in domestic cats[59].

Effects of dietary fibre on nutrient intake, nutrient digestibility and faecal characteristics: fructans, mannano-ligosaccharides, galacto-oligosaccharides and lactosucrose.

Studies on the effect of fructans (FOS, oligofructose, inulin), mannano-oligosaccharides (MOS) and GOS on faecal characteristics (consistency score and pH) are shown in Table 5[50–53,60–62]. Differences in results might be explained by differences in the levels of supplementation among studies: for example, up to 9·0 % as fed oligofructose in Hesta *et al.*[60]; maximum 0·6 % DM MOS in Aquino *et al.*[62]; 1 % synbiotic in Biagi *et al.*[53].

Decreased faecal consistency (looser stools) can have implications for the host animal, for the use of fibres in the pet food industry and for the appreciation of the foods by pet owners. On the contrary, the effects on stool consistency can be advantageous in the treatment of cats with constipation[8]. A decrease in faecal pH is caused by an increased production of bacterial endproducts, such as lactic acid and SCFA, in the hindgut. This decrease can exert several effects both on the microbiota and the host animal, such as stimulation of the growth of beneficial bacteria like *Lactobacillus* spp.[63] or an increased mineral absorption from the hindgut[64,65]. Likewise, the absorption of ammonia from the hindgut can be reduced by decreasing pH. Lactic acid molecules produced can be converted to weaker acids, such as acetic, propionic and butyric acids, by cross-feeding bacteria[66], which prevents a severe decrease in pH and the development of lactic acidosis. The latter disease condition can be observed in cats with gastrointestinal disease[67].

The above-mentioned SCFA have been associated with different beneficial effects on the hosts' general health[68] and the function of the gastrointestinal tract[11,12]. Besides faecal characteristics, the *in vivo* studies listed in Table 5

Table 5. Overview of the effects of dietary fructans, mannanoligosaccharides (MOS), galacto-oligosaccharides (GOS) and lactosucrose on faecal characteristics in cats

Fibre source	Dosage	Main results	Reference
OF	6, 9 % as-is	More, wetter faeces resulted in lower consistency scores (−0·5 and −0·7, respectively)*	60
FOS	4·0 % as-is	More, wetter faeces resulted in higher consistency scores (+0·8)†	51
Pectin	4·0 % as-is	More, wetter faeces resulted in higher consistency scores (+0·7)†	51
FOS	3·11 % as-is	Trend towards ↑ faecal output, due to ↑ moisture content + faecal DM production No difference in faecal consistency score	61
MOS	0·2, 0·4, 0·6 % DM	No difference in faecal consistency score	52
Probiotic + GOS	1·0 % as-is	No difference in faecal consistency score	53
OF	3, 6, 9 % as-is	Faecal pH: 6 % < 0 % (−0·38); 6 % < 3 % (−0·22); 9 % < 0 % (−0·64); 9 % < 3 % (−0·48); 9 % < 6 % (−0·26) Highest faecal total SCFA concentrations in highest supplementation groups	60
Inulin	3, 6 % as-is	Faecal pH = 0 % group: 6·86 (0 %) *v.* 6·81 (3 %) *v.* 6·94 (6 %)	60
sc-FOS + GOS	1·0 % as -is	↓ Faecal pH (−0·7), trend towards ↑ total SCFA and branched-chain fatty acids	52
GOS	0·5 % as-is	Faecal pH = 0 % group	52
Probiotic + GOS	1·0 % as-is	Faecal pH = 0 % group	53

OF, oligofructose; FOS, fructo-oligosaccharides; ↑, increase; sc, short-chain.
* Consistency score: 1 represented watery diarrhoea; 3, normal consistency; and 5, constipation[60].
† Consistency score: 1 represented dry, hard pellets; 2, hard, dry, formed stool; 3, soft, formed, moist stool; 4, soft, unformed stool that assumes form of container; 5, watery liquid that can poured[51].

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press

also investigated the effects of various oligosaccharides on nutrient digestibility. In the study of Hesta *et al.*[60], the apparent digestibility of protein decreased significantly as the level of fructan inclusion increased (from 87·0 % with 0 % inulin to 82·8 and 77·3 % with 3 and 6 % inulin inclusion, respectively; 83·1 % with 3 % oligofructose inclusion in the diet), which was confirmed with the supplementation of sc-FOS + GOS to feline diets[52] (from 84·2 % in control to 81·9 % in sc-FOS + GOS). The latter decrease[52] was lower than compared with the study of Hesta *et al.*[60], most probably caused by the lower inclusion levels of the fibres. The decreased protein digestibility was probably due to a higher faecal excretion of bacterial protein with a higher level of fructan in the diet[60] (from 25·4 % with 0 % inulin to 31·1 and 35·2 % with 3 and 6 % inulin inclusion, respectively; 32·8 % with 3 % oligofructose inclusion in the diet). Low-level MOS supplementation did not affect nutrient digestibility when supplemented to a wet diet, but slightly improved the DM digestibility if supplemented to a dry commercial diet[62] (from 73 (SD 4) % in control to 76 (SD 4) % in a 0·6 % MOS diet). In addition, a beneficial effect on palatability was only seen with MOS supplementation to the dry diet (for example, 11 % increase in DM intake between control and the 0·6 % dry diet)[62]. According to these authors, MOS is thus preferably supplemented to dry diets[62].

A decreased ileal protein digestibility might result in an increased large-intestinal protein and amino acid fermentation[59,69]. Besides SCFA, microbial degradation of amino acids can result in putrefactive endproducts, such as branched-chain fatty acids, valeric acid, ammonia (NH_3) and phenolic compounds (for example, indole, *p*-cresol, phenol)[70]. Some putrefactive endproducts, such as polyamines, appear to be required for normal development and repair of the gastrointestinal tract[71,72]. However, many of these compounds are suggested to be related to colorectal disease in humans and rats[73–75]. Different sources of dietary fibres have, therefore, been applied in domestic cats in an attempt to reduce the production and excretion of these potentially harmful substances and to attempt the reduction of faecal odour[50–53,61,76]. Faecal concentrations of ammonia (339 (SD 210) μg/g wet faeces before supplementation), indole (48 (SD 19) μg/g wet faeces before supplementation), ethylphenol (20 (SD 6) μg/g wet faeces before supplementation) and urinary ammonia (17 (SD 8) mg/ml urine before supplementation) were reduced significantly on day 14 of lactosucrose administration[50] (to 162 (SD 34), 30 (SD 12), 8 (SD 3) μg/g wet faeces and 10 (SD 4) mg/ml urine, respectively). This decrease might be explained by the concomitant decrease in counts of Clostridia (−0·25 log numbers per g faeces for the average of lecithinase-positive and -negative Clostridia, difference between before and after 14 d of lactosucrose administration) and Enterobacteriaceae (−1·6 log numbers per g faeces, difference between before and after 14 d of lactosucrose administration) due to the lactosucrose supplementation, as both groups are known to produce these putrefactive substances. In addition, the environmental ammonia (ammonia in room from 22 (SD 2) parts per million (ppm) before administration to 16 (SD 1) ppm after 14 d of administration) and the faecal odour (no quantitative data available) decreased remarkably during administration[50]. Likewise, supplementation of oligofructose led to decreased faecal concentrations of histamine, spermidine and indole[76] (quantitative data not available), and a synbiotic combination of GOS and a *Bifidobacterium* strain decreased faecal ammonia concentrations (in μmol/g faecal DM) even 10 d post-supplementation[53] (from 353 before to 288 at 1 d post- administration and 281 at 10 d post-administration). In contrast, Hesta *et al.*[61] found no effects of FOS supplementation to cats on twenty-seven different odour components, and Kanakupt *et al.*[52] observed no differences in faecal protein catabolites among control, sc-FOS-, GOS- and sc-FOS + GOS-supplemented cats. In the latter study, no differences in protein catabolite-producing bacteria were observed[52]. Increased faecal concentrations of ammonia (small differences), 4-methyl phenol and indole were observed when feline diets were supplemented with FOS (differences compared with cellulose in mmol or μmol/g of faecal DM: 0·1, 2·1 and 1·0, respectively) and pectin (differences compared with cellulose in mmol or μmol/g of faecal DM: 0·1, 1·9 and 0·7, respectively), possibly due to the fast fermentation of both supplements[51]. Different outcomes in the studies might again be explained by different sources and inclusion levels of fibres.

Effects of dietary fibre on nutrient intake, nutrient digestibility and faecal characteristics: moderate (beet pulp) and less fermentable fibre sources. The effects of moderately and less-fermentable fibre sources on nutrient intake have been studied by different groups. Fekete *et al.*[77] observed slightly different DM intake among diets supplemented with high levels (10 % DM) of beet pulp (263 (SD 42) g/4 d), peanut hulls (274 (SD 31) g/4 d) and alfalfa meal (257 (SD 37) g/4 d). In contrast, Sunvold *et al.*[3] did not observe differences in DM, organic matter or N intake between beet pulp- and cellulose-supplemented cats and cats on a control diet without supplemented fibre. Both studies used a similar inclusion level of the dietary fibre sources. Likewise, the inclusion of cellulose at a high level (17 % DM) did not alter food intake in cats[78,79] and the addition of psyllium husks and seeds did not decrease diet acceptance in cats with constipation[8].

In all the above-mentioned studies nutrient digestibility was studied as well. In peanut hull- and alfalfa meal-supplemented cats, a decreased DM digestibility (−22·4 and −7·2 % compared with the control diet) was seen[77], which was confirmed in healthy beet pulp-[3] and cellulose[78]-supplemented cats (−7·6 and −15 % (average of the three types of cellulose used) compared with control). Likewise, another study confirmed a decreased DM

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press

digestibility when diets of overweight cats were supplemented with beet pulp (−11 % compared with control), wheat bran (−13 % compared with control) or sugarcane fibre (−21 % compared with control)[9]. Only peanut hull and alfalfa meal supplementation decreased protein digestibility[77] (−10 and −2 % compared with control, respectively). Likewise, Fischer *et al.*[9] observed different effects on protein and fat digestibility depending on the chemical composition of the supplemented fibre source. No other studies in which cats were supplemented with peanut hulls, alfalfa meal, wheat bran or sugarcane fibre were found. Conclusively, moderately fermentable fibre sources, such as beet pulp, appear to be beneficial for normal-weight cats. In contrast, low-fermentable fibres, such as sugarcane fibre, might have adequate properties in low-energy weight-loss diets[9]. However, the high water-binding activity of the latter fibre source, comparable with that of long-fibre cellulose[79], can lead to extremely dry faeces, limiting the inclusion level in the diet[9]. This problem might be overcome by using short-fibre or microcrystalline cellulose[79]. An important factor in determining the outcome of the supplementation of fibres is the inclusion level, which was very high in the above-mentioned studies. Therefore, more research using lower levels is warranted.

Effects of dietary fibre on nutrient intake, nutrient digestibility and faecal characteristics: animal fibre. Differences in nutrient digestibility and fermentation end-products between a high-protein, extruded feline diet and a raw and cooked beef-based diet, which is a source of animal fibre, were studied by Kerr *et al.*[18]. The extruded diet had a much higher protein content (57 % crude protein) compared with a conventional extruded feline diet (for adult cats between 32 and 35 % crude protein) to mimic the composition of the beef-based diets. The effect of processing of the diets was investigated. However, due to a different ingredient composition between the extruded and beef-based diets, this comparison was biased. The extruded diet contained, for example, chicken meal as the major protein source. The latter might have contained a considerable amount of animal fibre from cartilage, for example, and might be considerably less digestible than beef meat. Two other studies that investigated the effects of feeding raw-meat diets on nutrient digestibility and faecal characteristics in domestic cats were found[80,81]. Comparisons of results between both studies are again biased by the use of different ingredient sources, such as different sources of plant fibre. Further studies using diets with an equal ingredient composition of all diets are necessary to study the effects of animal fibre fermentation on nutrient digestibility, the host's metabolism and the interaction with plant fibres in domestic cats.

Effects of dietary fibre on colonic morphology and function. The influence of fibre fermentation and concomitant SCFA production on colonic morphology was studied in adult healthy cats by Bueno *et al.*[11,12], and in overweight cats by Fischer *et al.*[9]. Beneficial effects on morphology (colonic weight, mucosal cell density, crypt structure), function (mucosal tissue energetics, transport of SCFA, etc.) and microbiota of the colon (less pathogenic bacteria) were observed[11,12]. According to these authors, the supplementation of a moderately fermentable fibre source to feline diets generated the best combination of beneficial effects on morphology, function and microbiota. In contrast, in overweight cats, the supplementation of beet pulp, wheat bran or sugarcane fibre did not affect the histological image of the colon biopsies[9]. Nevertheless, these fibres demonstrated other beneficial properties upon *in vivo* fermentation, as stated in other parts of the present review.

Effects of dietary fibre on nitrogen and energy metabolism. The principle of the N trap is that blood urea concentrations decrease when fermentable fibre is supplemented to the diet. Fermentable fibre stimulates the growth of and provides energy to the anaerobic microorganisms in the large intestine[82]. For bacterial protein anabolism, not only an energy source, such as fermentable fibre, but also a source of N should be available[83]. N sources include undigested dietary protein entering the large intestine, endogenous protein and blood urea[82]. Bacterial protein is not absorbed in the large intestine, but is excreted in the faeces[7]. When blood urea is the major N source to the intestinal microbiota, blood urea concentrations decrease and a decreased N excretion by the kidneys is observed, while N excretion in the faeces increases. Furthermore, fermentable fibre increased caecal blood flow in rats[82], which might enhance the passive diffusion of urea from the blood to the intestine. The N trap hypothesis has been proven in the rat[82,84] and dog[85], whereas in the cat tendencies towards an N shift from urine to faeces were found using diets supplemented with oligofructose[61,76]. Verbrugghe *et al.*[86–88] studied the effects of oligofructose and inulin on glucose and amino acid metabolism in domestic cats. In the first study[86], a control diet was tested against a prebiotic diet with 2·5 % of a blend of oligofructose and inulin. Diets did not affect fasting plasma glucose and insulin concentrations, blood glucose and insulin responses to glucose administration, or area under the glucose and insulin curves. In contrast, a decreased mean blood glucose concentration and area under the curve were achieved by supplementing sugarcane fibre to diets of overweight cats[9]. Despite an apparent absence of effects on carbohydrate metabolism in the study of Verbrugghe *et al.*[86], analysis of plasma acylcarnitine profiles revealed higher propionylcarnitine concentrations when the prebiotic diet was fed, suggesting colonic fermentation and propionate absorption. Prebiotic supplementation reduced methylmalonylcarnitine and aspartate aminotransferase concentrations, both indicating reduced gluconeogenesis from amino acids[86]. Further studies confirmed the amino acid-sparing potential of propionic acid from dietary

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press

fructan fermentation[87] and signs of sparing of the amino acid valine with propionylated high-amylose maize starch supplementation provided that protein intake was sufficient[89]. The quantification of the amino acid-sparing potential of propionic acid in healthy and disease-inflicted cats remains to be done. In contrast, this mechanism was not confirmed with guar gum supplementation, due to its high viscosity[59]. The guar gum's high viscosity impaired the small-intestinal protein digestion, causing a large load of undigested protein being fermented in the large intestine, which biased the proper assessment of the amino acid sparing by the produced propionic acid[59]. Further research using guar gum with lower viscosity might be warranted. The applicability of low-viscous guar gum might be restricted to dry extruded feline diets or homemade diets, since, for the production of canned diets, the gelling properties of highly viscous guar gum are advantageous.

Effects of dietary fibre on diseases with dietary protein restriction as a treatment cornerstone, such as chronic kidney and liver disease. CKD is a very common disease in middle-aged to elderly cats[90]. The cornerstones of the dietary management of CKD are a modification of the quantity (decrease) and quality (increase) of protein and a restriction of the dietary P intake[91]. Liver disease encompasses a range of different aetiologies, such as hepatic lipidosis and portosystemic shunts. The symptomatic treatment of liver disease, specifically in case of hyperammonaemia and hepatic encephalopathy, is based on dietary protein restriction[92,93]. Additionally, the supplementation of dietary fibre can exert beneficial effects on these disease conditions and the mechanisms behind these effects are explained in the next paragraphs.

The N trap principle, explained in the previous section, might be advantageous in animals suffering from hyperammonaemia (liver disease) or azotaemia (CKD), since an increase in the faecal N excretion and decreases in blood urea and ammonia concentrations can be achieved[82]. Until now, no studies investigating the N trap principle have been performed in cats with CKD. As mentioned above ('Effects of dietary fibre on nutrient intake, nutrient digestibility and faecal characteristics: fructans, mannanoligosaccharides and galacto-oligosaccharides' section), a decrease in large-intestinal pH occurs when SCFA and lactate are produced upon fermentation of dietary fibre. Another consequence of this decrease, besides the ones mentioned above, is that the overload of protons (H^+), that are present in a more acidic environment, leads to the ionisation of the ammonia (NH_3) molecules present to ammonium (NH_4^+) ions[82]. The absorption of NH_4^+ ions from the intestine to the bloodstream is far less effective, and the majority of these ions are excreted in the faeces[94]. That way, the ammonia concentration in the blood decreases, which benefits patients with hyperammonaemia[7]. Furthermore, less urea will be produced in the liver, resulting in lower blood urea N concentrations, beneficial to azotaemic patients. Additionally, the recently suggested amino acid-sparing potential of propionic acid[86–89] (see above) might be advantageous to patients whose diseases urge a dietary protein restriction, such as kidney[91] or liver[92,93] disease patients with evidence of hyperammonaemia or hepatic encephalopathy.

Conclusions

Despite the growing interest in dietary fibre supplementation to feline nutrition, research on this topic remains scarcer in cats compared with dogs and humans. The newest molecular techniques for qualitative and quantitative assessment of the microbiota have been applied to feline samples of different parts of the feline gastrointestinal tract. Therefore, a detailed assessment of the complex and diverse microbiota is available in the literature. Furthermore, *in vitro* batch culture systems are assumed to be suitable for screening several indigestible substrates for fermentation kinetics and endproduct concentrations and this technique has been used in several animal species. However, the use of this technique with feline inoculum remains scarce, despite the fact that valuable estimations of the fermentation potential and kinetics of various fibre sources for felines can be gained non-invasively. Research on *in vivo* fermentation is rather scarce in cats, despite the fact that fermentation endproducts might appear to exert different beneficial effects on the host animal. More research is warranted to reveal potential (plant or animal) fibre sources that can be used on a large scale in feline nutrition for healthy and diseased cats.

Acknowledgements

The present review was written as a part of the postgraduate study of the first author (K. R.), which was funded by the Institute for Promotion of Innovation through Science and Technology in Flanders (IWT; grant no. 091050 to K. R.). The IWT had no role in the design, analysis or writing of the article.

K. R. was responsible for the manuscript drafting. M. H. and G. P. J. J., promoter and co-promoter of K. R., respectively, contributed to the manuscript drafting.

There are no conflicts of interest.

References

1. Brosey BP, Hill RC & Scott KC (2000) Gastrointestinal volatile fatty acid concentrations and pH in cats. *Am J Vet Res* **61**, 359–361.
2. de Godoy MRC, Kerr KR & Fahey GC Jr (2013) Alternative dietary fibre sources in companion animal nutrition. *Nutrients* **5**, 3099–3117.
3. Sunvold GD, Fahey GC, Merchen NR, *et al.* (1995) Dietary fibre for cats: *in vitro* fermentation of selected fibre sources by cat faecal inoculum and *in vivo* utilization of diets containing selected fibre sources and their blends. *J Anim Sci* **73**, 2329–2339.

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press

4. Sunvold GD, Fahey GC, Merchen NR, *et al.* (1995) *In vitro* fermentation of selected fibrous substrates by dog and cat faecal inoculum – influence of diet composition on substrate organic matter disappearance and short-chain fatty-acid production. *J Anim Sci* **73**, 1110–1122.
5. Sunvold GD, Hussein HS, Fahey GC, *et al.* (1995) *In vitro* fermentation of cellulose, beet pulp, citrus pulp, and citrus pectin using faecal inoculum from cats, dogs, horses, humans and pigs, and ruminal fluid from cattle. *J Anim Sci* **73**, 3639–3648.
6. Elliott DA (2006) Nutritional management of chronic renal disease in dogs and cats. *Vet Clin Small Anim* **36**, 1377–1384.
7. Rutgers C & Biourge V (1998) Nutritional management of hepatobiliary and pancreatic diseases. In *Encyclopedia of Feline Clinical Nutrition*, pp. 154–155 [P Pibot, V Biourge and DA Elliott, editors]. Aimargues: Aniwa SAS.
8. Freiche V, Houston D, Weese H, *et al.* (2011) Uncontrolled study assessing the impact of a psyllium-enriched extruded dry diet on faecal consistency in cats with constipation. *J Fel Int Med Surg* **13**, 903–911.
9. Fischer MM, Kessler AM, de Sá LRM, *et al.* (2012) Fibre fermentability effects on energy and macronutrient digestibility, faecal parameters, postprandial metabolite response, and colon histology of overweight cats. *J Anim Sci* **90**, 2233–2245.
10. Buddington RK & Sunvold GD (1998) Fermentable fibre and the gastrointestinal tract ecosystem. In *Recent Advances in Canine and Feline Nutritional Research: Proceedings of the 1998 IAMS International Nutritional Symposium*, pp. 449–461 [GA Reinhart and DP Carey, editors]. Wilmington, OH: Orange Frazer Press.
11. Bueno AR, Cappel TG, Sunvold GD, *et al.* (2000) Feline colonic morphology and mucosal tissue energetics as influenced via the source of dietary fibre. *Nutr Res* **20**, 985–993.
12. Bueno AR, Cappel TG, Sunvold GD, *et al.* (2000) Feline colonic microbes and fatty acid transport: effects of feeding cellulose, beet pulp and pectin/gum arabic fibres. *Nutr Res* **20**, 1319–1328.
13. Sunvold GD (1996) Dietary fibre for dogs and cats: an historical perspective. In *Recent Advances in Canine and Feline Nutritional Research: Proceedings of the 1996 IAMS International Nutrition Symposium*, pp. 3–14 [DP Carey, SA Norton and SM Bolser, editors]. Wilmington, OH: Orange Frazer Press.
14. Depauw S, Hesta M, Whitehouse-Tedd K, *et al.* (2013) Animal fibre: the forgotten nutrient in strict carnivores? First insights in the cheetah. *J Anim Physiol Anim Nutr* **97**, 146–154.
15. Plantinga EA, Bosch G & Hendriks WH (2011) Estimation of the dietary nutrient profile of free roaming feral cats: possible implications for nutrition of domestic cats. *Br J Nutr* **106**, S35–S48.
16. Yamka RM, Jamikorn U & True AD (2003) Evaluation of low-ash poultry meal as a protein source in canine foods. *J Anim Sci* **81**, 2279–2284.
17. Dozier WA, Dale NM & Dove CR (2003) Nutrient composition of feed-grade and pet-food-grade poultry by-product meal. *J Appl Poult Res* **12**, 526–530.
18. Kerr KR, Vester Boler BM, Morris CL, *et al.* (2012) Apparent total tract energy and macronutrient digestibility and faecal fermentative end-product concentrations of domestic cats fed extruded, raw beef-based, and cooked beef-based diets. *J Anim Sci* **90**, 515–522.
19. Ritchie LE, Steiner JM & Suchodolski JS (2008) Assessment of microbial diversity along the feline intestinal tract using 16S rRNA gene analysis. *FEMS Microbiol Ecol* **66**, 590–598.
20. Suchodolski JS (2011) Intestinal microbiota of dogs and cats: a bigger world than we thought. *Vet Clin Small Anim* **41**, 261–272.
21. Minamoto Y, Hooda S, Swanson K, *et al.* (2012) Feline gastrointestinal microbiota. *Anim Health Res Rev* **13**, 64–77.
22. Osbaldiston GW & Stowe EC (1971) Microflora of alimentary tract of cats. *Am J Vet Res* **32**, 1399–1405.
23. Winitz M, Adams RR, Sudman DA, *et al.* (1970) Studies in metabolic nutrition employing chemically defined diets. II. Effect on gut microflora populations. *Am J Clin Nutr* **23**, 546–559.
24. Sparkes AH, Papasouliotis K, Sunvold G, *et al.* (1998) Bacterial flora in the duodenum of healthy cats, and effect of dietary supplementation with fructooligosaccharides. *Am J Vet Res* **59**, 431–435.
25. Papasouliotis K, Sparkes AH, Werrett G, *et al.* (1998) Assessment of the bacterial flora of the proximal part of the small intestine in healthy cats, and the effect of sample collection method. *Am J Vet Res* **59**, 48–51.
26. Johnston K, Lamport A & Batt RM (1993) An unexpected bacterial flora in the proximal small intestine of normal cats. *Vet Rec* **132**, 362–363.
27. Johnston KL, Swift NC, Forster-Van Hijfte M, *et al.* (2001) Comparison of the bacterial flora of the duodenum in healthy cats and cats with signs of gastrointestinal tract disease. *J Am Vet Med Assoc* **218**, 48–51.
28. Smith HW (1965) Observations on the flora of the alimentary tract of animals and factors affecting this composition. J Pathol Bacteriol **89**, 95–122.
29. Muir P, Gruffydd-Jones TJ, Cripps PJ, *et al.* (1994) Breath hydrogen excretion after oral administration of xylose to cats. *J Small Anim Pract* **35**, 86–92.
30. German AJ, Day MJ, Ruaux CG, *et al.* (2003) Comparison of direct and indirect tests for small intestinal bacterial overgrowth and antibiotic-responsive diarrhoea in dogs. *J Vet Intern Med* **17**, 33–43.
31. Finegold SM, Sutter VL & Mathisen GE (1983) Normal indigenous intestinal flora. In *Human Intestinal Microflora in Health and Disease*, pp. 7–9 [D Hentges, editor]. New York: Academic Press.
32. Mentula S, Harmoinen J, Heikkilä M, *et al.* (2005) Comparison between cultured small intestinal and faecal microbiotas in Beagle dogs. *Appl Environ Microbiol* **71**, 4169–4175.
33. Collins MD, Lawson PA, Willems A, *et al.* (1994) The phylogeny of the genus *Clostridium*: proposal for five new genera and eleven new species combinations. *Int J Syst Bacteriol* **44**, 812–826.
34. Papasouliotis K, Sparkes AH, Gruffydd-Jones TJ, *et al.* (1996) A comparison of the bacterial flora of the duodenum and ileum in healthy cats. In *Proceedings of the BSAVA Congress*, p. 244. Quedgeley, Gloucester: BSAVA.
35. Gruffyd-Jones TJ, Papasouliotis K & Sparkes AH (1998) Characterization of the intestinal flora of the cat and its potential for modification. In *Recent Advances in Canine and Feline Nutritional Research: Proceedings of the 1998 IAMS International Nutritional Symposium*, pp. 473–482 [GA Reinhart and DP Carey, editors]. Wilmington, OH: Orange Frazer Press.
36. Desai AR, Musil KM, Carr AP, *et al.* (2009) Characterization and quantification of feline faecal microbiota using cpn60 sequence-based methods and investigation of animal-to-animal variation in microbial population structure. *Vet Microbiol* **137**, 120–128.
37. Garcia-Mazcorro JF, Lanerie DJ, Dowd SE, *et al.* (2011) Effect of a multi-species symbiotic formulation on faecal bacterial microbiota of healthy cats and dogs as evaluated by pyrosequencing. *FEMS Microbiol Ecol* **78**, 542–554.

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press

38. Handl S, Dowd SE, Garcia-Mazcorro JF, *et al.* (2011) Massive parallel 16S rRNA gene pyrosequencing reveals highly diverse faecal bacterial and fungal communities in healthy dogs and cats. *FEMS Microbiol Ecol* **76**, 301–310.
39. Ritchie LE, Burke KF, Garcia-Mazcorro JF, *et al.* (2010) Characterization of faecal microbiota in cats using universal 16S rRNA gene and group-specific primers for *Lactobacillus* and *Bifidobacterium* spp. *Vet Microbiol* **144**, 140–146.
40. Barry KA, Middelbos IS, Vester Boler BM, *et al.* (2012) Effects of dietary fibre on the feline gastrointestinal metagenome. *J Proteome Res* **11**, 5924–5933.
41. Tun H, Brar MS, Khin N, *et al.* (2012) Gene-centric metagenomics analysis of feline intestinal microbiome using 454 junior pyrosequencing. *J Microbiol Methods* **88**, 369–376.
42. Jia J, Frantz N, Khoo C, *et al.* (2011) Investigation of the faecal microbiota of geriatric cats. *Lett Appl Microbiol* **53**, 288–293.
43. Jia J, Frantz N, Khoo C, *et al.* (2011) Investigation of the faecal microbiota of kittens: monitoring bacterial succession and effect of diet. *FEMS Microbiol Ecol* **78**, 395–404.
44. Suchodolski J, Camancho J & Steiner J (2008) Analysis of bacterial diversity in the canine duodenum, jejunum, ileum, and colon by comparative 16S rRNA gene analysis. *FEMS Microbiol Ecol* **66**, 567–578.
45. Ley RE, Hamady M, Lozupone C, *et al.* (2008) Evolution of mammals and their gut microbes. *Science* **320**, 1647–1651.
46. Sparkes AH, Papasouliotis K, Sunvold G, *et al.* (1998) Effect of dietary supplementation with fructooligosaccharides on faecal flora of healthy cats. *Am J Vet Res* **59**, 436–440.
47. Itoh K, Mitsuoka T, Maejima K, *et al.* (1984) Comparison of faecal flora of cats based on different housing conditions with special reference to *Bifidobacterium*. *Lab Anim* **18**, 280–284.
48. Eckburg PB, Bik EM, Bernstein CN, *et al.* (2005) Diversity of human intestinal microbial flora. *Science* **308**, 1635–1638.
49. Johnston KL, Lamport A, Ballèvre O, *et al.* (1999) A comparison of endoscopic and surgical collection procedures for the analysis of the bacterial flora in duodenal fluid from cats. *Vet J* **157**, 85–89.
50. Terada A, Hara H, Kato S, *et al.* (1993) Effect of lactosucrose (4G-β-d-galactosylsucrose) on faecal flora and faecal putrefactive products of cats. *J Vet Med Sci* **55**, 291–295.
51. Barry KA, Wojcicki BJ, Middelbos IS, *et al.* (2010) Dietary cellulose, fructooligosaccharides, and pectin modify faecal protein catabolites and microbial populations in adult cats. *J Anim Sci* **88**, 2978–2987.
52. Kanakupt K, Vester Boler BM, Dunsford BR, *et al.* (2011) Effects of short-chain fructooligosaccharides and galactooligosaccharides, individually and in combination, on nutrient digestibility, faecal fermentative metabolite concentrations, and large bowel microbial ecology of healthy adult cats. *J Anim Sci* **89**, 1376–1384.
53. Biagi G, Cipollini I, Bonaldo A, *et al.* (2013) Effect of feeding a selected combination of galactooligosaccharides and a strain of *Bifidobacterium pseudocatenulatum* on the intestinal microbiota of cats. *Am J Vet Res* **74**, 90–95.
54. Marshall-Jones ZV, Baillon MA, Croft JM, *et al.* (2006) Effects of *Lactobacillus acidophilus* DSM13241 as a probiotic in healthy adult cats. *Am J Vet Res* **67**, 1005–1012.
55. Barry KA, Wojcicki BJ, Bauer LL, *et al.* (2011) Adaptation of healthy adult cats to select dietary fibres *in vivo* affects gas and short-chain fatty acid production from fibre fermentation *in vitro*. *J Anim Sci* **89**, 3163–3169.
56. Rochus K, Bosch G, Vanhaecke L, *et al.* (2013) Incubation of select fermentable fibres with feline faecal inoculum: correlations between *in vitro* fermentation characteristics and end products. *Arch Anim Nutr* **67**, 416–431.
57. Bosch G, Wrigglesworth DJ, Cone JW, *et al.* (2012) Effects of preservation conditions of canine faeces on *in vitro* gas production kinetics and fermentation end-products. *J Anim Sci* **91**, 259–267.
58. Bosch G, Pellikaan WF, Rutten PGP, *et al.* (2008) Comparative *in vitro* fermentation activity in the canine distal gastrointestinal tract and fermentation kinetics of fibre sources. *J Anim Sci* **86**, 2979–2989.
59. Rochus K, Janssens GPJ, Van de Velde H, *et al.* (2013) Highly viscous guar gum shifts dietary amino acids from metabolic use to fermentation substrate in domestic cats. *Br J Nutr* **109**, 1022–1030.
60. Hesta M, Janssens GPJ, Debraekeleer J, *et al.* (2001) The effect of oligofructose and inulin on faecal characteristics and nutrient digestibility in healthy cats. *J Anim Physiol Anim Nutr* **85**, 135–141.
61. Hesta M, Hoornaert E, Verlinden A, *et al.* (2005) The effect of oligofructose on urea metabolism and faecal odour components in cats. *Anim J Anim Physiol Nutr* **89**, 208–214.
62. Aquino AA, Saad FMOB, Santos JP, *et al.* (2010) Effects of spray-dried yeast cell wall on digestibility, score of faeces, and palatability of diets for cats. *Arq Bras Med Vet Zootec* **62**, 622–630.
63. Bergman EN (1990) Energy contributions of volatile fatty acids from the gastrointestinal tract in various species. *Physiol Rev* **70**, 567–590.
64. Raschka L & Daniel H (2005) Mechanisms underlying the effects of inulin-type fructans on calcium absorption in the large intestine of rats. *Bone* **37**, 728–735.
65. Xiao J, Li X, Min X, *et al.* (2013) Mannitol improves absorption and retention of calcium and magnesium in growing rats. *Nutr* **29**, 325–331.
66. Duncan SH, Louis P & Flint HJ (2004) Lactate-utilizing bacteria, isolated from human faeces, that produce butyrate as a major fermentation product. *Appl Environ Microbiol* **70**, 5810–5817.
67. Packer RA, Moore GE, Chang C-Y, *et al.* (2005) Serum d-lactate concentrations in cats with gastrointestinal disease. *J Vet Int Med* **26**, 905–910.
68. Salminen S, Bouley C, Boutron-Ruault MC, *et al.* (1998) Functional food science and gastrointestinal physiology and function. *Br J Nutr* **80**, Suppl. 1, S147–S171.
69. Hendriks WH, van Baal J & Bosch G (2012) Ileal and faecal protein digestibility measurement in humans and other non-ruminants – a comparative species view. *Br J Nutr* **108**, S247–S257.
70. Mafra D, Barros AF & Fouque D (2013) Dietary protein metabolism by gut microbiota and its consequences for chronic kidney disease patients. *Future Microbiol* **8**, 1317–1323.
71. Wang Y & Johnson LR (1990) Luminal polyamines stimulate repair of gastric mucosal ulcers. *Am J Physiol Gastrointest Liver Physiol* **261**, 504–511.
72. Loser C, Eisel A, Harms D, *et al.* (1999) Dietary polyamines are essential luminal growth factors for small intestinal and colonic mucosal growth and development. *Gut* **44**, 12–16.
73. Corpet DE, Yin Y, Zhang XM, *et al.* (1995) Colonic protein fermentation and promotion of colon carcinogenesis by thermolyzed casein. *Nutr Canc* **23**, 271–281.
74. Matsui T, Matsukawa Y, Sakai T, *et al.* (1995) Effect of ammonia on cell-cycle progression of human gastric-cancer cells. *Eur J Gastroenterol Hepatol* **7**, S79–S81.
75. Pedersen G, Brynskov J & Saermark T (2002) Phenol toxicity and conjugation in human colonic epithelial cells. *Scand J Gastroenterol* **37**, 74–79.

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press

76. Barry KA, Hernot DC, Van Loo J, *et al.* (2008) Fructan supplementation affects nitrogen partitioning in excreta and stool metabolite concentrations in healthy senior cats. *FASEB J* **22**, 4443.
77. Fekete SG, Hullár I, Andrásofszky E, *et al.* (2004) Effect of different fibre types on the digestibility of nutrients in cats. *J Anim Physiol Anim Nutr* **88**, 138–142.
78. Prola L, Dobenecker B, Mussa PP, *et al.* (2010) Influence of cellulose fibre length on faecal quality, mineral excretion and nutrient digestibility in cat. *Anim Physiol Anim Nutr* **94**, 362–367.
79. Prola L, Dobenecker B & Kienzle E (2006) Interaction between dietary cellulose content and food intake in cats. *J Nutr* **136**, 1988S–1990S.
80. Vester BM, Beloshapka AN, Middelbos IS, *et al.* (2010) Evaluation of nutrient digestibility and faecal characteristics of exotic felids fed horse- or beef-based diets: use of the domestic cat as a model for exotic felids. *Zoo Biol* **29**, 432–438.
81. Kerr KR, Beloshapka AN & Morris CL (2013) Evaluation of four raw meat diets using domestic cats, captive exotic felids, and cecectomized roosters. *J Anim Sci* **91**, 225–237.
82. Younes H, Garleb K, Behr S, *et al.* (1995) Fermentable fibres or oligosaccharides reduce urinary nitrogen excretion by increasing urea disposal in the rat cecum. *J Nutr* **125**, 1010–1016.
83. Bliss DZ (2004) Dietary fibre in conservative management of chronic renal failure. *Ped Nephrol* **19**, 1069–1070.
84. Younes H, Rémésy C, Behr S, *et al.* (1997) Fermentable carbohydrate exerts a urea-lowering effect in normal and nephrectomised rats. *Am J Physiol Gastrointest Liver Physiol* **272**, G515–G521.
85. Howard MD, Kerley MS, Sunvold GD, *et al.* (2000) Source of dietary fibre fed to dogs affects nitrogen and energy metabolism, and intestinal microflora populations. *Nutr Res* **20**, 1473–1484.
86. Verbrugghe A, Hesta M, Gommeren K, *et al.* (2009) Oligofructose and inulin modulate glucose and amino acid metabolism through propionate production in normal-weight and obese cats. *Br J Nutr* **102**, 318–325.
87. Verbrugghe A, Janssens GPJ, Meininger E, *et al.* (2010) Intestinal fermentation modulates postprandial acylcarnitine profile and nitrogen metabolism in a true carnivore: the domestic cat (*Felis catus*). *Br J Nutr* **104**, 972–979.
88. Verbrugghe A, Hesta M, Daminet S, *et al.* (2011) Propionate absorbed from the colon acts as gluconeogenic substrate in a strict carnivore, the domestic cat (*Felis catus*). *J Anim Physiol Anim Nutr* **96**, 1054–1064.
89. Rochus K, Janssens GPJ, Cools A, *et al.* (2013) Dietary supplementation of propionylated starch to domestic cats provides propionic acid as gluconeogenic substrate potentially sparing the amino acid valine. *J Nutr Sci* **3**, e16.
90. Ross SJ, Osborne CA, Kirk CA, *et al.* (2006) Clinical evaluation of dietary modification for treatment of spontaneous chronic kidney disease in cats. *J Am Vet Med Assoc* **229**, 949–957.
91. Polzin DJ, Osborne CA, Ross S, *et al.* (2000) Dietary management of feline chronic renal failure: where are we now? In what direction are we headed? *J Feline Med Surg* **2**, 75–82.
92. Bunch SE (2003) BunchSEHepatobiliary diseases in the cat. In *Small Animal Internal Medicine*, 3rd ed., pp. 506–524 [RW Nelson and C Guillermo Couto, editors]. St Louis, MO: Mosby, Inc.
93. Center SA (1998) Nutritional support for dogs and cats with hepatobiliary disease. *J Nutr* **12**, 2733S–2746S.
94. Cummings JH (1975) Absorption and secretion by the colon. *Gut* **16**, 323–329.
95. Vester BM, Dalsing BL, Middelbos IS, *et al.* (2009) Faecal microbial populations of growing kittens fed high- or moderate-protein diets. *Arch Anim Nutr* **63**, 254–265.
96. Patil AR, Zarneck Maulden GC & Dowling KE (2000) Effect of advances in age on faecal microflora of cats. *FASEB J* **14**, A488.
97. Lubbs DC, Vester BM, Fastinger ND, *et al.* (2009) Dietary protein concentration affects intestinal microbiota of adult cats: a study using DGGE and qPCR to evaluate differences in microbial populations in the feline gastrointestinal tract. *J Anim Physiol Anim Nutr* **93**, 113–121.
98. Inness VL, McCartney AL, Khoo C, *et al.* (2007) Molecular characterisation of the gut microbiota of healthy and inflammatory bowel disease cats using fluorescence *in situ* hybridisation with special reference to *Desulfovibrio* spp. *J Anim Physiol Anim Nutr* **91**, 48–53.
99. Abecia L, Hoyles L, Khoo C, *et al.* (2010) Effects of a novel galactooligosaccharide on the faecal microbiota of healthy and inflammatory bowel disease cats during a randomized, double-blind, cross-over feeding study. *Int J Probiotics Prebiotics* **5**, 61–68.
100. Janeczko S, Atwater D, Bogel E, *et al.* (2008) The relationship of mucosal bacteria to duodenal histopathology, cytokine mRNA, and clinical disease activity in cats with inflammatory bowel disease. *Vet Microbiol* **128**, 178–193.

https://doi.org/10.1017/S0954422414000213 Published online by Cambridge University Press