

# Diarrhoea and digestive upsets – management and treatment

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**KIT STURGESS** MA, VetMB, PhD, CertVR, DSAM, CertVC, MRCVS discusses the different presentations, clinical signs and approaches to therapy in relation to gastrointestinal conditions in small animal patients

**DIARRHOEA is the most common non-routine cause for presentation of a pet to a primary care practice on The Small Animal Veterinary Surveillance Network database (25,346 – 28.5 per cent – of 89,277 consultations).**

Diarrhoea can present both as an acute or chronic condition, with primary features suggesting small or large intestinal or mixed diarrhoea. Although usually associated with gastrointestinal tract disease, diarrhoea can occur secondary to systemic disease, such as fever or hypoadrenocorticism.

The most common accompanying features of diarrhoea include vomiting, inappetence and weight loss ([Figure 1](#)). This article aims to provide a logical overview of the management and treatment of cats and dogs with diarrhoea and digestive upset. The most common causes of acute and chronic diarrhoea in cats and dogs are listed in [and](#) .

The majority of cases of diarrhoea and digestive upset are acute and probably self-limiting, or chronic and low-grade, making treatment on an outpatient basis appropriate.

In acute, probably self-limiting or intermittent, low-grade chronic diarrhoea, suitable non-specific treatments include:

- ensuring adequate hydration
- appropriate worming
- diet
- probiotics
- mucosal protectants
- prebiotics
- motility modifiers

Unless there are specific indicators, antibacterial agents, corticosteroids and starvation are inappropriate first line treatments. In chronic disease, however, while such treatments may provide symptomatic relief they are unlikely to lead to long-term resolution of the problem and the client should be made aware of this.

In some situations symptomatic relief is all that is required as signs are very intermittent and the patient is otherwise well, with a normal appetite and stable weight. In cases in which signs are progressive, frequent or associated with significant hyporexia, vomiting or weight loss then further investigation is recommended so targeted treatment can be given.

Key indicators that suggest more aggressive management, such as haematology, biochemistry, imaging ([Figure 2](#)) hospitalisation and/or surgery or further investigation, are required include:

- signs of severe systemic disease, such as collapse, hypotension or shock
- significant other signs, such as:
  - dehydration
  - vomiting
  - pain
  - fever
  - abdominal swelling
  - marked melaena or haematochezia
  - jaundice
- repeated bouts of diarrhoea – especially if they become worse or there is significant weight loss and/or hyporexia

## **Non-specific treatments**

### **Oral rehydration fluids**

In dehydrated patients or patients with high fluid demands, hydration is important, with solutions containing salts, dextrose, amino acids and small quantities of cereals being most effective. However, acceptance can be relatively low so it is essential patients are carefully observed to ensure they meet their fluid requirements.

## **Diet**

In acute diarrhoea, standard advice is a period of starvation of 12 to 48 hours followed by a bland diet for three to seven days (Guilford, 1994), but this is not evidentially based.

It should be remembered any change on to a new diet will perturb the intestinal flora and excite an immune response to the new protein, which may delay recovery and can potentially lead to food hypersensitivity to the new protein as it has been introduced into an inflammatory environment. In patients with repeat episodes of acute diarrhoea, starvation can lead to protein-calorie malnutrition.

There is mounting evidence feeding through diarrhoea has benefits in maintaining calorie intake and the integrity of the mucosal barrier. The author would therefore advise continuing to feed patients with acute diarrhoea (assuming they will eat and are not vomiting) on a highly digestible, low fat, preferably wet food that contains a protein they have previously encountered.

In patients with chronic diarrhoea, where dietary hypersensitivity may well play an important part, feeding a novel or hydrolysed protein diet is of value. It is essential – at least in the initial phase – to feed the new diet exclusively to everything but water.

It is likely if the patient is going to respond to dietary management alone there will be a measurable, though not necessarily complete, response within two weeks – a feeding duration with which most owners can cope.

## **Motility modifiers**

The inflammation in enteritis can cause significant dysmotility in a variety of ways, leading to hypermotility and hypomotility, segmental dilation and reducing the rate of gastric emptying by affecting rhythmic segmentation and peristalsis. This can result in flatulence, eructation, pain, vomiting and diarrhoea. Restoring “normal” bowel motility should help to reduce the severity of clinical signs.

Prokinetics affecting the stomach would include metoclopramide, ranitidine and erythromycin. Prokinetics affecting the small bowel would include metoclopramide and cisapride.

Opiates such as loperamide will act to slow gastric emptying, increase tone and segmentation, decrease propulsion and increase the tone of the ileocolic and anal sphincter.

Anticholinergic drugs such as atropine or propantheline bromide, which reduce peristalsis, are rarely indicated as they can worsen ileus.

## **Antacids**

Antacids will not directly affect diarrhoea, but may be helpful if there is hyperacidity associated with delayed gastric emptying, with many clinicians viewing ranitidine as an appropriate first choice as it has some antacid and prokinetic activity.

Proton pump inhibitors such as omeprazole are rapidly absorbed and excreted in dogs and will cause profound acid suppression within 48 hours (Tolbert et al, 2011) and probably sooner.

Some evidence suggests that for maximum effect twice daily dosing is required (Bersenas et al, 2005). There is minimal clinical data in cats, but omeprazole at 0.75mg/ kg to 1mg/kg orally every 24 hours appears safe and its antacid properties have been demonstrated in experimental models. When acid suppression is ceased there is likely to be rebound hyperacidity. For this reason it is not appropriate to reduce dose frequency prior to stopping therapy.

## **Mucosal protectants and adsorbents**

A variety of products are available that are designed to bind toxins and bacteria. Rarely is sufficient given to actually effectively “coat” the mucosal surface.

Experimental evidence of their benefit is limited. They can also improve stool consistency through their adsorbent properties.

Commonly used in veterinary medicine are kaolin, pectins and bismuth subsalicylate. Barium will also have a similar action and diarrhoea will often temporarily improve after a barium x-ray series.

## **Probiotics**

Probiotics are becoming the most widely used non-specific treatment for diarrhoea in cats and dogs. They are relatively easy to administer and palatable veterinary-specific products are available alone or in combination with prebiotics and/or mucosal adsorbents.

Probiotics act to improve the epithelial barrier, modulate the mucosal immune system and alter the intestinal flora. They have been shown to reduce the duration of diarrhoea compared to controls (Herstad et al, 2010), canine inflammatory bowel disease activity score and diarrhoea in a rescue centre (Bybee et al, 2011) and improve faecal score in cats with chronic diarrhoea (Hart et al, 2012).

Changes in cytokine expression have been demonstrated, although these changes have not been consistent. A variety of probiotic bacteria are used including *Enterococcus faecium* EC1707 and SF68.

## **Prebiotics**

Prebiotics are designed to alter luminal microflora to promote growth of endogenous enteric protective bacteria and include lactulose, inulin, psyllium, fructooligosaccharide and mannan-oligosaccharides. They are frequently combined with probiotics and termed synbiotics.

Demonstration of their efficacy is limited due to the difficulties in accurately defining intestinal bacterial populations at a given time point, but they have been shown to reduce the duration of diarrhoea in experimentally infected dogs (Gouveia et al, 2013).

## **Approach to diarrhoea treatment**

There is limited published evidence of the efficacy of non-specific treatments of cats with diarrhoea and no published studies looking at management of acute diarrhoea.

Pragmatically, feeding a highly digestible, moderate fat diet and administering probiotics/synbiotic compounds would be appropriate first choice medication in a patient that is otherwise clinically well.

Motility modifiers have a role where there is urgency and the cat is defecating in the house/outside the litter tray. There is more evidence in dogs to support the use of probiotics in management of acute diarrhoea, making a similar strategy appropriate.

Chronic diarrhoea is more likely to be successfully treated by dietary intervention with probiotic/synbiotic in the absence of a specific diagnosis. A number of studies have demonstrated a relatively high incidence of faecal parasites in diarrhoea cases, particularly in cats – some of which will have multiple enteropathogens (Paris et al, 2014) making a full faecal examination an important early investigation in such cases.

Antacids, prokinetics, mucosal protectants and motility modifiers may all have a role to reduce the severity of clinical signs and will be effective if the underlying disease process is self limiting. In non-self limiting disease they will result in temporary improvement followed by relapse.

Based on the fact the most common relapsing cause of diarrhoea in cats is idiopathic inflammatory bowel disease (chronic inflammatory enteropathy; Norsworthy et al, 2013), there is a rationale to using prednisolone in such cases in the absence of histologic support. In those cats and dogs where biopsy is not an option this is a reasonable approach, but wherever possible, should be accompanied by having measured vitamin B<sub>12</sub>, pancreatic lipase and trypsin-like immunoreactivity first.

The problem with prospective prednisolone use – especially as some side effects are likely to occur – is the dose and duration of the prednisolone treatment before success or failure has been demonstrated. If the patient appears to fail to respond, does this represent a different underlying cause, inadequate time, inadequate dose or inappropriate choice of anti-inflammatory/immunosuppressive agent? The situation in dogs is further complicated as there are

a greater number of individuals with other causes of their diarrhoea, such as antimicrobial responsive diarrhoea or motility issues.

## Use of antimicrobials in diarrhoea and digestive upsets

Antimicrobials remain widely prescribed for the management of diarrhoea in dogs and cats. The rationale for their use is questionable unless:

1. there is significant immunosuppression, making translocation of bacteria from the gut a significant risk – for example, parvovirus
2. it is likely the intestinal mucosal barrier is breached, increasing bacterial translocation across the wall – for example, haemorrhagic enteritis
3. a pathogen that is likely to respond to and requires antimicrobial treatment has been identified (for example, enteroinvasive *Escherichia coli*), in which case the narrowest spectrum antimicrobial should be used.
  - Whether *Campylobacter* and *Salmonella* should be treated is controversial and in many circumstances allowing an appropriate immune response to mature may be a better long-term option depending on the zoonotic risk that exists for that individual case.
4. antimicrobial-responsive diarrhoea (ARD) is likely

For many cases in 2) and 4) metronidazole represents an appropriate choice, although some cases of ARD seem to respond more favourably to tetracyclines.

Regardless of the approach adopted, a logical, sequential, well-recorded approach to the patient with diarrhoea and digestive upset is much more likely to lead to appropriate treatment and investigation only where necessary.

• Please note some drugs in this article are not licensed for use in dogs and cats, and are prescribed under the cascade.

## References and further reading

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**Figure 1.** Ptyalism associated with nausea and diarrhoea.





**Figure 2.** Fluid-filled intestinal loops in a collapsed 16-year-old cat with severe diarrhoea.

Parasitic	Helminths, nematodes – <i>Ancylostoma</i> (dog), <i>Trichuris</i> (dog), ascarids Protozoans – <i>Cystoisospora</i> , <i>Cryptosporidium</i> , <i>Giardia</i> , <i>Tritrichomonas</i> (cats)
Infection	Viruses – parvovirus, coronavirus, <i>Rotavirus</i> , distemper (dogs) Bacteria – <i>Campylobacter</i> , <i>Salmonella</i> , enteropathogenic <i>Escherichia coli</i> , toxigenic <i>Clostridium perfringens</i>
Diet	Food intolerance – for example, lactose Food poisoning – consumption of foods containing preformed toxins or toxigenic bacteria Sudden dietary change
Inflammation	Idiopathic haemorrhagic gastroenteritis Acute phase or flare-up of inflammatory bowel disease
Abdominal obstruction	Vomiting more commonly – small intestinal foreign body or intussusception
Pancreas	Acute pancreatitis
Fever (>40°C)	Various causes

**Table 1a. Common causes of acute diarrhoea in dogs and cats**

Parasitic	Helminths, nematodes – <i>Ancylostoma</i> (dog), <i>Trichuris</i> (dog), ascarids Protozoans – <i>Cystoisospora</i> , <i>Cryptosporidium</i> , <i>Giardia</i> , <i>Tritrichomonas</i> (cats)
Infection	Bacteria – <i>Campylobacter</i> , <i>Salmonella</i> , enteropathogenic <i>Escherichia coli</i> , toxigenic <i>Clostridium perfringens</i> Antimicrobial responsive diarrhoea (dogs)
Diet	Food intolerance – for example, lactose Food hypersensitivity
Inflammation	Idiopathic inflammatory bowel disease
Neoplasia	Lymphoma
Dysmotility	Intestinal hurry
Pancreas	Chronic pancreatitis Pancreatic insufficiency
Endocrinopathies	Hyperthyroidism (cats) Hypoadrenocorticism (dogs)
Renal	IRIS stage IV chronic kidney disease

**Table 1b. Common causes of chronic diarrhoea in dogs and cats**