Diet and Large Intestinal Disease in Dogs and Cats

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ABSTRACT Large intestinal disease, and more especially colitis, is a commonly seen problem in small animal practice. Although colitis is most frequently diagnosed in dogs, it is becoming increasing common in cats. The etiology of colitis is not known, but there is general agreement that an immune-mediated response to luminal antigen is involved. In particular, parasites, bacteria and dietary factors may be involved. In ~10% of dogs presented with typical signs of colitis, no pathologic lesion will be found on investigation. These dogs have a functional diarrhea associated with some stress factor and are thought to have irritable bowel syndrome (IBS). This condition is most frequently observed in working dogs, although highly nervous and excitable dogs may also exhibit similar clinical signs. Until the underlying etiology of colitis is determined, treatment regimens will remain symptomatic. Recent studies have placed considerable importance on the value of diet in the prevention, immediate and long-term therapy of colitis in dogs and cats. In particular the value of "novel" protein diets, fermentable fiber and polyunsaturated fatty acids is receiving the most attention. It is now possible to maintain patients in long-term remission and to modify the severity and chronicity of colitis by using diet alone. This paper will review the subject of dietary management of colitis and IBS and present results from the author's clinical research program. J. Nutr. 128: 2717S–2722S, 1998.

KEY WORDS: • colitis • fermentable fiber • polyunsaturated fatty acids • novel proteins • hypoallergenic diets • irritable bowel syndrome

FUNCTIONS OF THE LARGE INTESTINE

The large intestine of dogs and cats comprises the cecum, colon, rectum and anus. The cecum has no known function, whereas the colon is responsible for the resorption of water and salt from ileal chyme (Murdoch 1996). Resorption occurs primarily in the ascending and transverse colon associated with segmented contractions and retrograde peristalsis. After adequate resorption, mass peristalsis moves fecal residues to the rectum for storage before defecation.

The colon maintains a large population of bacteria numbering between $10^{10}$ and $10^{11}$ colony forming units per milliliter (CFU/mL), which are predominantly anaerobes, whereas the small intestine harbors an aerobic population of between $10^5$ and $10^6$ CFU/mL (Batt and Rutgers 1997). Colonic bacteria have an important role to play in preventing colonization by pathogenic bacteria and in the fermentation of dietary fiber, resulting in the production of short-chain fatty acids (SCFA) including acetate, butyrate and propionate (Reinhart 1993). SCFA and particularly butyrate are considered to be important sources of energy for colonocytes (Guilford 1994, Hillier et al. 1991, Reinhart 1993, Simpson 1997, Vilaseca et al. 1990). Absorption of the SCFA stimulates sodium and water resorption from the colonic lumen (Eastwood 1992, Herschel et al. 1994, Johnson 1992, Simpson 1997), various possible causes of which are shown in Table 1. Although many of these conditions are common in dogs and cats, colitis is the most frequently diagnosed in the author's experience. Up to 45% of cases of suspect intestinal disease referred to the Royal (Dick) School of Veterinary Studies are associated with colitis (Table 2). A smaller but important group of patients present with irritable bowel syndrome (IBS), which can be readily confused with pathologic colonic disease.

DISEASES OF THE LARGE INTESTINE

The diseases most frequently associated with the large intestine are shown in Table 1. Although many of these conditions are common in dogs and cats, colitis is the most frequently diagnosed in the author's experience. Up to 45% of cases of suspect intestinal disease referred to the Royal (Dick) School of Veterinary Studies are associated with colitis (Table 2). A smaller but important group of patients present with irritable bowel syndrome (IBS), which can be readily confused with pathologic colonic disease.

Both canine and feline colitis is classified into lymphocytic-plasmacytic, eosinophilic, histiocytic and granulomatous forms (Bright et al. 1994, Hall et al. 1994, Johnson 1992, Simpson 1995). This is a pathologic classification, representing the predominant cell type observed in the inflammatory response and provides no information regarding the underlying etiology. Although the etiology of colitis is not known (Burrows 1992, Richter 1992, Simpson 1997), various possible causes have been suggested (Table 3). Diet is considered to play an important role in the etiology.
of colitis. When the patient has small intestinal disease, deconjugated bile acids and hydroxy fatty acids may reach the colon where they induce secretory diarrhea (Murdoch 1996). Unabsorbed substrate may also reach the colon, resulting in bacterial fermentation and osmotic diarrhea. Most interest regarding diet is currently centered around dietary hypersensitivity. This is based on the observation that colitis frequently responds to hypoallergenic diets (Leib et al. 1989a, Nelson et al. 1984, Simpson et al. 1994). Although the choice of diet remains controversial (Guilford 1994), protection against colitis may be achieved by ensuring that adequate levels of fermentable fiber and consequently SCFA are included in the diet (Clemens 1996, Simpson 1995). In addition, recent work has suggested that the ratio of (n-6):(n-3) polyunsaturated fatty acids will modify the composition of lipid membrane fatty acids and consequently the levels of arachidonic acid and eicosanoids produced (Reinhart and Sunvold 1996, Vaughan et al. 1994).

It is therefore likely that the etiology of colitis is multifactorial, requiring the presence of luminal antigens, poor colonic flora and inflammation and a breakdown of the natural protective mechanisms (Table 4) to induce disease (Simpson 1997). In particular, failure to secrete immunoglobulin A (IgA), loss of immune tolerance or increased mucosal permeability may be involved (Burrows 1992, Magne 1992, Richter 1992). After initiation of an immune response, oxygen free radicals, tissue ischemia and eicosanoid production perpetuate inflammation, resulting in further damage (Simpson 1997). Drug and dietary management is designed to break this vicious cycle of inflammation and in some cases remove the initiating agent. Diet may also be useful in disease prevention.

Patients with IBS present with clinical signs similar to those with pathologic colitis; thus it is not possible to make a diagnosis based on the clinical examination alone. Equally, there is no definitive diagnostic test for IBS; the diagnosis can be made only by exclusion of other causes of chronic diarrhea (Simpson 1995). Stress is an important initiating cause of IBS, and the success of treatment depends on identifying this factor, which is usually easier to determine in working dogs than in those of a nervous or excitable nature. IBS has not been diagnosed in cats. The condition is associated with disordered colonic motility, with failure of segmentation and increased frequency and urgency of defecation.

### TABLE 1

**Conditions involving the large intestine of dogs and cats**

<table>
<thead>
<tr>
<th>Inflammation</th>
<th>Neoplasia</th>
<th>Motility disorders</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphocytic plasmacytic colitis</td>
<td>Adenocarcinoma</td>
<td>Irritable bowel syndrome</td>
<td>Stricture formation</td>
</tr>
<tr>
<td>Eosinophilic colitis</td>
<td>Lymphosarcoma</td>
<td>Megacolon</td>
<td>Foreign bodies</td>
</tr>
<tr>
<td>Histiocytic colitis</td>
<td>Rectal polyps</td>
<td>Feline dysautonomia</td>
<td>Diverticulum</td>
</tr>
<tr>
<td>Granulomatous colitis</td>
<td>Anal tumors</td>
<td></td>
<td>Perineal hernia</td>
</tr>
</tbody>
</table>

### TABLE 2

**Annual distribution of clinical cases referred with suspect intestinal disease to The Royal (Dick) School of Veterinary Studies**

<table>
<thead>
<tr>
<th>Disease</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exocrine pancreatic insufficiency</td>
<td>14</td>
</tr>
<tr>
<td>Small intestinal disease</td>
<td>27</td>
</tr>
<tr>
<td>Colitis</td>
<td>45</td>
</tr>
<tr>
<td>Intestinal neoplasia</td>
<td>7</td>
</tr>
<tr>
<td>Megacolon</td>
<td>4</td>
</tr>
<tr>
<td>Irritable bowel syndrome (IBS)</td>
<td>3</td>
</tr>
</tbody>
</table>

### TABLE 3

**Possible etiologic agents that may result in colitis in dogs and cats**

<table>
<thead>
<tr>
<th>Dietary factors</th>
<th>Parasites</th>
<th>Infection</th>
<th>Genetic</th>
<th>Stress</th>
<th>Autoimmune</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein hypersensitivity</td>
<td>Trichuris vulpis</td>
<td>Clostridial infections</td>
<td>Boxers, French bulldogs</td>
<td>Irritable bowel syndrome</td>
<td>60% of human inflammatory</td>
</tr>
<tr>
<td>Unabsorbed fats, hydroxy fatty acids</td>
<td>Giardiasis</td>
<td>Salmonella spp.</td>
<td></td>
<td>Immune mediated</td>
<td>bowel disease patients have</td>
</tr>
<tr>
<td>Deconjugated bile acids</td>
<td>Hookworms</td>
<td>Campylobacter spp.</td>
<td></td>
<td>Increased permeability</td>
<td>antibodies to colonocytes</td>
</tr>
<tr>
<td>Excess or deficient dietary fiber</td>
<td>Coccidia spp.</td>
<td></td>
<td></td>
<td>Reduced immune tolerance</td>
<td></td>
</tr>
</tbody>
</table>

### TABLE 4

**Natural protective mechanisms that help to protect against luminal antigens**

<table>
<thead>
<tr>
<th>Mechanical</th>
<th>Immunologic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motility, peristalsis</td>
<td>Peyer’s patches, lymphoid follicles</td>
</tr>
<tr>
<td>Gastric acid secretion</td>
<td>Secretion of immunoglobulin A.</td>
</tr>
<tr>
<td>Pancreatic, intestinal and biliary secretions</td>
<td>Intra-epithelial lymphocytes</td>
</tr>
<tr>
<td>Mucus production</td>
<td>Lymphocytes in the lamina propria</td>
</tr>
<tr>
<td>Colonic flora</td>
<td>Immune tolerance</td>
</tr>
<tr>
<td>Tight junctions between colonocytes</td>
<td>Hepatic RE system</td>
</tr>
</tbody>
</table>

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peristaltic activity (Bush 1985, Tams 1992). The most effective treatment is removal of the stress factor, but in many cases, drug and dietary management may be required to maintain remission.

**MANAGEMENT OF COLITIS**

It is very important to ensure that there is no concurrent systemic or small intestinal disease associated with colitis because such cases will fail to respond to treatment for colitis until the underlying cause is addressed. When a specific cause of colitis can be identified (Table 3), specific treatment will usually effect a complete cure. However, in the majority of cases, the etiology is not known; thus, treatment remains symptomatic. Such therapy normally involves both drug and dietary management and although it may provide a clinical remission, it will rarely effect a cure. Long-term remission is now possible using diet alone in the majority of cases.

**Drug therapy.** Traditionally, drug therapy has played a key role in the management of colitis, with many cases receiving daily drug therapy. Such therapy usually involves the use of anti-inflammatory drugs such as sulfasalazine and prednisolone, which can have significant side effects, particularly in the long term (Barnett and Joseph 1987, Bush 1985, Richter 1992). For this reason, alternative management regimens have been devised to avoid the need for long-term drug therapy (Simpson 1997).

However, to assist in breaking the vicious cycle of inflammation, drug therapy still has an important role to play in the initial stages of colitis management. Sulfasalazine remains the drug of choice in the treatment of colitis, although prednisolone and azathioprine either alone or in combination are also used (Table 5) (Burrows 1992, Simpson 1997). Once remission is achieved, drug therapy is required only if clinical signs recur. In such cases, the immediate use of anti-inflammatory drugs for a period of 3–5 d will usually resolve the clinical signs of colitis and reestablish remission with diet alone.


**Elimination diets.** The use of elimination diets in the treatment of colitis implies that the condition is associated with dietary factors and in particular dietary hypersensitivity. The objective is to provide the patient with a "novel" protein source to which there has been no previous exposure together with an appropriate carbohydrate source, usually boiled rice. This diet should be home prepared with no additives or supplements. Such a diet should be fed to the exclusion of all other foods for at least 4 wk before considering an alternative diet.

Once the patient has responded to the elimination diet, components of the original diet should be reintroduced one at a time and the response observed (Paterson 1995). In this way, it should be possible to identify the dietary hypersensitivity present and design a dietary regimen for the long-term well-being of the patient.

Unfortunately, this technique relies heavily on owner compliance, and in the author’s experience and that of others, it is often difficult to persuade owners to proceed with provocative testing once a satisfactory resolution of chronic diarrhea has occurred often for the first time in many months or years (Leib et al. 1989).

**Hypoallergenic diets.** Hypoallergenic diets with or without added fiber have been advocated by several workers (Guilford 1994, Leib 1990, Simpson et al. 1994, Willard 1988). The aim is to select a "novel" protein source with which the patient has no previous exposure. Veterinary diets are now available with a wide range of protein sources including chicken, fish, rabbit, duck, lamb and venison. This diet should be fed to the exclusion of all other food sources and the response observed. There is evidence to suggest that the introduction of a second "novel" protein diet may be required after ~4 wk due to increased mucosal permeability and reduced immune tolerance, i.e., induced sensitivity to the first protein source (Guilford 1996).

The second protein source can usually be used in the long-term management of the patient.

Recent work compared the value of a hypoallergenic, a low fat and a high fiber (mainly insoluble fiber) veterinary diet in the treatment of colitis (Simpson et al. 1994, Simpson 1995). In the study, patients with clinical signs of colitis were investigated to confirm that there was no evidence of small intestinal disease and that lymphocytic plasmacytic colitis was present. Patients were randomly allocated to one of the three diets and given sulfasalazine at 20–40 mg/(kg · d) as part of the initial therapeutic regimen. Patients were then monitored for signs of clinical improvement over the next 4 mo using an analog scale (Dixon and Bird 1981) to record improvements in fecal consistency and reduction in fecal mucus, blood and tenesmus. The level of drug therapy required to maintain remission during the study period was also recorded.

After the 4-mo trial, 82% of dogs in the low fat group required both drug and dietary therapy. Only 18% were maintained on diet alone. In the high fiber group, 50% required drug and dietary management over the first 3 mo, but this improved to 25% in the last month. Half of the dogs were subsequently changed to the hypoallergenic diet because of poor owner acceptance, with a dramatic improvement in clinical response. In contrast, only 15% of dogs receiving the hypoallergenic diet required drug therapy by mo 4 and remained drug free for a further 8 mo of follow-up. All of the dogs in the low fat and hypoallergenic groups showed a marked improvement in clinical signs with a reduction in fecal tenesmus, mucus and blood and improvement in fecal consistency. A similar improvement was not observed in the high fiber group. It was concluded that the hypoallergenic diet provide the best dietary management for dogs with colitis although it

**TABLE 5**

Drugs that are commonly used in the treatment of colitis in dogs and cats

<table>
<thead>
<tr>
<th>Drug name</th>
<th>Trade name</th>
<th>Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sulfasalazine</td>
<td>Salazopyrin</td>
<td>Dog: 20–40 mg/(kg · d)</td>
</tr>
<tr>
<td>Olsalazine</td>
<td>Dipentum</td>
<td>Cat: &lt;25 mg/(kg · d)</td>
</tr>
<tr>
<td>Mesalazine</td>
<td>Asacol</td>
<td>Dog: 10–20 mg/(kg · d)</td>
</tr>
<tr>
<td>Prednisolone</td>
<td></td>
<td>1 mg/(kg · d)</td>
</tr>
<tr>
<td>Azathioprine</td>
<td>Imuran</td>
<td>Dog: 1–2 mg/(kg · d)</td>
</tr>
<tr>
<td>Metronidazole</td>
<td>Flagyl</td>
<td>10 mg/kg, twice a day</td>
</tr>
<tr>
<td>Tylosin</td>
<td>Tylocare</td>
<td>20–40 mg/(kg · d)</td>
</tr>
</tbody>
</table>
was not possible to conclude what factor(s) in the hypoallergenic diet was responsible for the improvement. However, it does suggest that diet plays an important role in the pathogenesis of colitis, which is in agreement with previous studies (Nelson et al. 1988).

It is interesting to note that these results compare favorably with other studies in which hypoa llergenic diets or restricted diets have been fed to dogs and cats with colitis (Leib et al. 1989b, Nelson et al. 1984 and 1988). Although provocative testing was not possible in the study by Simpson et al. (1994), other studies have suggested that it results in relapse of diarrhea (Nelson et al. 1984 and 1988). Other have suggested that hypoallergenic diets should be supplemented with various fiber sources to obtain the best response (Guilford 1996, Willard 1988).

**Dietary fiber.** The high fiber group gave the poorest results in the dietary trial described above. This may reflect the type of fiber used in the diet rather than a failure of “fiber” as a therapeutic agent. Previously, all types of dietary fiber were assumed to have a similar effect in the gastrointestinal tract, but the classification of fiber according to solubility and fermentability has resulted in a change of opinion (Table 6). Soluble fiber retains large volumes of water and readily forms gels, which increase luminal viscosity, alter gastrointestinal transit, tend to be fermentable and alter nutrient absorption. Insoluble fiber retains little water and does not form gels; it speeds intestinal transit, promotes colonic peristalsis and increases fecal bulk (Dimski 1992). Thus, different types of fiber have different effects on the intestine (Burrows and Merritt 1983).

There is now considerable evidence to suggest that the fermentability of dietary fiber is of importance in maintaining colonic health (Clemens 1996, Murdock 1996) and improving colonic water and salt absorption (Herschel et al. 1981). These effects are due to the anaerobic fermentation of dietary fiber, resulting in the production of SCFA such as acetate, butyrate and propionate (Reinhart 1993, Roediger 1980). SCFA provide >70% of the animals colonocyte energy requirements (Hague et al. 1997) and have many beneficial effects on the colon (Guilford 1994, Hague et al. 1997, McIntryre et al. 1993, Finnie et al. 1995) (Table 7). Several studies have shown that butyrate inhibits growth of colorectal tumors and induces colonocyte differentiation (Hague 1997). Poorly fermentable fiber yields low levels of SCFA, which are inadequate to meet colonocyte requirements, whereas highly fermentable fibers yield excess SCFA and gases that may induce diarrhea and flatulence while seriously reducing digestibility of the diet. The use of moderately fermentable fiber such as beet pulp appears to produce an appropriate amount of SCFA to meet the animal’s requirements (Reinhart 1993).

Butyrate, in particular, has been found to be the preferred energy source for the colon (Allan et al. 1996, Roediger 1980); it is oxidized by the colonocytes using the citric acid cycle (Cummings et al. 1978). Rectal infusions of SCFA and butyrate, in particular, have been used to treat rats and humans with ulcerative colitis where drug therapy has failed (Breurer et al. 1991, Harig et al. 1989) and have been used to reduce the severity and chronicity of inflammation in drug-induced colitis in rats (Scheppach et al. 1996). Orally administered butyrate is completely absorbed in the jejunum and does not reach the colon (Marks 1996); thus, therapeutic doses of butyrate must be administered per rectum. Butyrate utilization by colonocytes has been found to be impaired in patients with active (Roediger 1980) and quiescent colitis (Chapman et al. 1994). This effect was not influenced by administration of SCFA (Allan et al. 1996); it was not due to luminal availability of SCFA (Keighley et al. 1978) or the result of inadequacy in absorption of butyrate into the colonocytes (Roediger et al. 1982). Current opinion suggests that the problem may be associated with the intracellular metabolism of butyrate (Allan et al. 1996). Therefore, although the current trend is to employ low residue hypoallergenic diets in the treatment of colitis, there is a considerable volume of evidence to suggest that inclusion of appropriate levels of fermentable fiber would also be beneficial. The diet should contain a balance of fermentable (for SCFA production) and nonfermentable fiber (to promote normal motility) (Reinhart and Sunvold 1996). Recommendations for fiber vary from 3 to 7% of dry matter in the diet (Reinhart 1993) to empirical levels of metamuscul added to a hypoallergenic diet at 1 and 6 teaspoons per meal (Guilford 1996). Further research into the value of fermentable fiber in the treatment of colitis in dogs and cats is warranted.

**Polysaturated fatty acids.** Recent research has suggested that manipulation of the (n-6) to (n-3) polysaturated fatty acid ratio in the diet can have a major influence on the severity of the inflammatory reaction in allergic skin disease and IBD (Carey 1995, Reinhart 1995). Changes in the proportion of (n-6):(n-3) fatty acids in the diet will change the levels of the same fatty acids in the colonocyte lipid membrane. This effect takes ~6–8 wk to occur after the initiation of a dietary change (Reinhart and Sunvold 1996). Liberation of (n-6) fatty acids by phospholipase A during inflammation results in the production of arachidonic acid and the eicosanoids prostaglandin 3 (PG3), thromboxane 3 (TXA3) and leukotriene 4 (LTB4), which are proinflammatory. Liberation of (n-3) fatty acids in the same circumstances results in the production of less inflammatory eicosapentaenoic acid and the eicosanoids prostaglandin 3 (PG3), thromboxane 3 (TXA3) and leukotriene 4 (LTB4), which are proinflammatory. Liberation of (n-3) fatty acids in the same circumstances results in the production of less inflammatory eicosapentaenoic acid and the eicosanoids PG3, TXA3 and LTB3. Because both (n-6) and (n-3) fatty acids compete for the same enzyme systems, it is possible to alter the response to inflammation by feeding a diet higher in (n-3) fatty acids than normal. It has been suggested that a (n-6):(n-3) fatty acid ratio of between 5:1 and 10:1

### Table 6

<table>
<thead>
<tr>
<th>Fiber type</th>
<th>Solubility</th>
<th>Fermentability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cellulose</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>Pectins</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Guar gums</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Beet pulp</td>
<td>Low</td>
<td>Moderate</td>
</tr>
<tr>
<td>Rice bran</td>
<td>Low</td>
<td>Moderate</td>
</tr>
<tr>
<td>Gum arabic</td>
<td>High</td>
<td>Moderate</td>
</tr>
</tbody>
</table>

### Table 7

Some of the beneficial effects of fermentable fiber on the health of the colon as observed in studies in humans, rats and dogs

- Protects against colonization of pathogenic bacteria
- Increases colonic mucosal mass
- Increases colonic mucosal blood flow
- Improved differentiation of colonocytes
- Protection against neoplasia
- Production of mucin, mucus secretion
- Reduces severity and chronicity of colitis
- Improved healing after injury
provides the ideal ratio of polyunsaturated fatty acids required to reduce the severity of colitis (Carey 1995, Reinhart 1995). Several studies have been conducted in rats, dogs and humans to lend support to this response. In one study, the incorporation of fish oil reduced the severity of colitis by 56% with an associated reduction of 30% in LTβ production, but no improvement in histopathology of biopsy samples (Aslan and Triadafilopoulos 1992). Diets rich in fish oil have also been shown to reduce the level of thromboxanes in the chronic stages of inflammation and to shorten the course of colonic disease by reducing the severity of lesions and their progression to chronicity (Vilaseca et al. 1990). The inclusion of fish oil in human patients with IBD increased the level of eicosapentaenoic acid and docosahexaenoic acid in mucosal lipid membranes within 3 wk of initiation. Arachidonic acid levels fell throughout this study. It was concluded that colonic lipid and eicosanoid production can be readily altered by dietary supplementation with fish oil (Hillier et al. 1991). In addition, after 3 wk of fish oil therapy, 9 of 10 patients had endoscopic and symptomatic improvement in their disease. Dietary supplementation of between 5:1 and 10:1 (n-6):(n-3) polyunsaturated fatty acids altered the lipid levels in canine skin within 6–12 wk of initiating the diet. There was a decrease in the proportions of LTβ and an increase in LTβ levels in skin and blood tissues (Vaughan et al. 1994). It has been suggested that diets with the optimal (n-6):(n-3) polyunsaturated fatty acid ratios may be used in many inflammatory conditions in dogs including IBD and colitis (Reinhart and Sunvold 1996). Further research into the role of these fatty acid ratios is required in dogs and cats with IBD to determine the extent of this beneficial response.

**MANAGEMENT OF IRRITABLE BOWEL SYNDROME**

Patients with IBS present the clinician with a considerable therapeutic challenge (Tams 1992) because there is no universal drug or dietary regimen available. Ideally, the underlying stress factor should be identified and removed. If this is not possible, treatment must be tailored to the individual and usually involves a combination of drug and dietary management because dietary management alone is rarely successful (Simpson 1995). It is interesting to note that rats with a previous history of pathologic colitis are more susceptible to stress effects on their enteric nervous function than would normally be the case (Collins et al. 1996). This is thought to predispose them to recurrence of their clinical disease.

The choice of drugs to be used in the treatment of IBS depends on whether chronic diarrhea or spastic colon and constipation are present. Motility-modifying drugs such as loperamide, diphenoxylate, mebaverine and hyoscine together with behavior-modifying drugs such as chlordiazepoxide and dicyclomine may be used (Murdock 1996, Simpson 1995, Tams 1992). These drugs may be required long term to maintain remission.

Dietary management usually involves the feeding of a low residue hypoallergenic diet with added insoluble (nonfermentable) fiber. The benefits of fiber in this situation include the alteration of abnormal intestinal myoelectrical activity and “normalization” of intestinal transit times (Tams 1992). However, diet alone rarely leads to a sustained clinical improvement.

**SUMMARY**

As our understanding of the etiology of inflammatory bowel disease improves, it is becoming clear that no single etiologic agent is responsible for colitis in dogs and cats. Multiple factors appear to play an important role in deciding whether immune-tolerance or inflammation occurs. It is equally clear that diet is playing an increasingly important role not only in the treatment of colitis but also in the prevention of disease. Our reliance on long-term drug therapy is declining, whereas our use of diet is increasing. It is likely that diet will continue to play a pivotal role in the treatment of colitis in the future.

**LITERATURE CITED**


Hiiller, K., Jewell, R., Dorrell, L. & Smith, C. L. (1991) Incorporation of fatty acids from fish oil and olive oil into colonic mucosal lipids and effects upon eicosanoid synthesis in inflammatory bowel disease. GUT 32: 1151


