

Diagnosis and management of food allergy and intolerance in dogs and cats

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SUMMARY: This paper reviews food allergy and intolerance in dogs and cats. Adverse reactions to ingested food components can affect many systems and can produce signs involving the skin, gastrointestinal tract, respiratory tract and central nervous system, and these clinical signs are reviewed. Most basic food ingredients have the potential to induce an allergic response, although most reactions are caused by proteins. In particular, dogs and cats can become sensitive to cow's milk, beef, fish or cereal. Food allergy and intolerance is rare in dogs and cats, although the incidence in practice is difficult to establish.

Clinical signs are quite variable, depending on the individual response, although the major clinical sign is pruritus. Diagnosis can be difficult, as there is no single test available to help the clinician to confirm or refute the presence of food sensitivity. Diagnosis is based on dietary investigation in the form of elimination diets and test meals. Elimination diets for dogs include lamb, chicken, rabbit, horse meat and fish as sources of protein, with rice or potatoes. Successful elimination diets for cats include lamb, chicken, rabbit or venison, with rice. Improvement in clinical signs while on the elimination diet is suggestive of food allergy. The diagnosis should be confirmed by feeding the original diet, with the development of clinical signs within 7 to 14 days of feeding.

Aust Vet J 71: 322 – 326

Introduction

The terminology applied to adverse reactions to food is confusing. An adverse reaction to food is a general term that can be applied to any clinically abnormal response attributed to the ingestion of a substance in the diet, and it does not presuppose an aetiology. There are a variety of subclassifications within this general term (Table 1). True food allergy or hypersensitivity is an immunologically-mediated phenomenon; it can be antibody-mediated, immune complex-mediated or cell-mediated. To confirm a diagnosis of food hypersensitivity, the antigen should be identified, a relationship between antigen exposure and reaction should be demonstrated, and the immunologic mechanism elucidated. If these criteria have not been met, then strictly speaking the term food hypersensitivity should not be used, and food intolerance, food idiosyncrasy or food sensitivity used instead. However, common usage in veterinary medicine has been to use 'food allergy' or 'hypersensitivity' for any abnormal reaction to food, regardless of the aetiology.

Reactions to ingested food components can affect many systems, and can produce signs involving the skin (Walton 1967; August 1985; White 1986; Muller *et al* 1989; Carlotti *et al* 1990; Rosser 1990; Jeffers *et al* 1991; Harvey 1993), gastrointestinal tract (Walton *et al* 1968; Walton 1979; Nelson *et al* 1984; Dakin 1988; Leib and August 1989), respiratory tract (Walton 1977, 1979) and central nervous system (Muller *et al* 1989).

Pathogenesis

Food Hypersensitivity

Animals are exposed daily to a great variety of potential dietary allergens, and yet most remain refractory to sensitisation. However,

after a period varying from a few weeks to several years, a small number of animals may enter a short asymptomatic phase during which mechanisms become mobilised against one particular foodstuff. Any subsequent ingestion of this foodstuff will cause development of clinical signs.

The pathogenesis of an adverse reaction to a food component involves the interaction of the component with a biological amplifi-

TABLE 1
Definitions of terminology applied to potential mechanisms of adverse reactions to food

Food hypersensitivity	An immune-mediated reaction to a food component; effect of the food unrelated to any physiological sensitivity
Food intolerance	An abnormal non-immune physiological response to a food component; of an idiosyncratic, metabolic, pharmacological or toxic nature
Food idiosyncrasy	An abnormal response to a food component in a sensitive individual, resembling hypersensitivity but not involving immune mechanisms. Often caused by an enzyme defect.
Pharmacological reactions to food	An abnormal response to a pharmacological compound in a food, or a component that causes release of a pharmacological substance from the body
Food poisoning	Adverse reactions to a specific toxin, for example, botulism
Enterometabolism	The absorption of metabolites produced as a result of bacterial fermentation in the colon

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This article is based on a paper presented to the Waltham Symposium *Clinical Nutrition in Practice*, which was held in Adelaide in May 1992 in conjunction with the AVA National Conference.

TABLE 2
Dietary items reported to have caused food sensitivity in the dog and cat

<i>Dog</i>	
Cow's milk	Wheat
Beef	Corn
Mutton	Soy
Pork	Rice flour
Chicken	Potatoes
Rabbit	Kidney beans
Horse meat	Canned foods
Fish (variety)	Dog biscuits
Eggs	Dog foods (including Hill's prescription diet canine d/d)
Oatmeal	Artificial food additives
<i>Cat</i>	
Cow's milk	Fish (variety)
Beef	Eggs
Mutton	Canned foods
Pork	Dry foods
Chicken	Cod-liver oil
Rabbit	Benzoic acid
Horse meat	

cation system that leads to inflammation and the development of clinical signs. The amplification system may include immunological mechanisms, complement pathway, eicosanoid synthesis, chemotaxis of phagocytes, and production of inflammatory mediators, such as kinins, leukotrienes and histamine. On occasion, there may be direct toxicity from the ingested component, without amplification, such as reactions to pharmacologic substances. Reaction to the products of colonic bacterial metabolism has been proposed in man (Hunter 1991). The immunological mechanisms of food hypersensitivity in dogs and cats probably involve both immediate (types I and III) and delayed (type IV) hypersensitivities (Halliwell and Gorman 1989). Immediate reactions to foods occur within minutes to hours, while delayed responses occur within several hours to days. However, it is not clear which mechanism, if any, predominates in the pathogenesis of food hypersensitivity, and it may be that more than one mechanism operates in some cases of an allergic reaction.

The mechanism by which allergens are absorbed by the intestine or allergen/antibody complexes later localise to the skin is unknown. The mechanism may depend on the type of hypersensitivity, the nature of the antigen, the concentration of free or cell-bound immunoglobulin, the distribution of mast cells, or other factors.

Food Intolerance

Food intolerance occurs if an individual lacks an enzyme necessary for a physiological or digestive process. For example, disaccharides such as lactose have to be hydrolysed to monosaccharides before they can be absorbed from the intestinal lumen. Certain cats and dogs produce reduced quantities of the enzyme lactase, and therefore have a limited ability to hydrolyse lactose in milk. Undigested lactose remains within the intestinal lumen, allowing proliferation of lactose-fermenting bacteria, and subsequently an osmotic diarrhoea develops. Another area of food intolerance recognised in man is reaction to pharmacological substances in food. Either the food itself contains

the chemical, in particular the vasoactive amines such as tyramine or histamine, or the food causes the release of inflammatory mediators, such as histamine. Fish can stimulate the release of histamine from mast cells, and may cause gastrointestinal upset in some cats. The importance of these reactions to pharmacological substances in food in dogs and cats is unknown, and would be very difficult to ascertain, particularly if the body's response to the substance was a migraine.

Antigenic Sources

Most food ingredients have the potential to induce an allergic response, although most reactions are caused by proteins. Dietary items reported to have caused food sensitivity in the dog and cat are shown in Table 2. More than half of the reported cases in cats were sensitive to cow's milk, beef or fish (Walton 1967; White and Sequoia 1989) and a similar proportion of the reported cases of dietary intolerance in dogs was sensitive to milk, beef or cereal, either alone or in combination (Walton 1967; Carlotti *et al* 1990; Jeffers *et al* 1991; Harvey 1993).

Incidence

Most authors agree that food hypersensitivity and intolerance is rare in dogs and cats (Muller *et al* 1989; Reedy and Miller 1989). The incidence of the condition is difficult to establish, and different figures have been published. It has been estimated that food sensitivity accounts for 1% of all canine and feline dermatoses in general practice (Walton 1967), and is the second most common allergic skin disorder in the cat (Reedy and Miller 1989). About 11% of feline miliary dermatitis cases may be caused by food sensitivity (Scott 1987). Food sensitivity may account for 10% of all non-seasonal dermatitis (Baker 1970) and 10% of all canine allergic skin diseases, excluding parasitic allergy (Scott 1978).

This apparent variation in incidence may be because skin disease is often multifactorial, with two or more factors combining, so that a clinical threshold is exceeded, and disease results. In a study of food allergy in cats, 3 of the 14 cats had concurrent flea bite hypersensitivity, flea collar hypersensitivity, or atopy, which all contributed to the pruritus and skin lesions (White and Sequoia 1989). Removal of one of the factors may be sufficient to reduce the challenge, dropping below the clinical threshold, resulting in improvement of signs. The variation in published incidence may therefore be a reflection of the order in which the possible aetiologies are investigated by the clinician during the diagnostic examination.

There is no breed or sex predilection in dogs and cats with food sensitivity. In general, it is agreed that there is no age predilection; and age of onset can be from 6 months to 14 years (Walton 1967; White 1986; Muller *et al* 1989; Remy 1990). However, August (1985) states that clinical signs frequently appear before 9 months of age, and Rosser (1990) reported that 17 of his series of 51 dogs were less than 1 year old at the onset of signs. In a recent study of food sensitivity in 25 dogs in the UK, 13 (52%) of the dogs were less than 12 months old at the onset of clinical signs of diet-sensitive dermatitis (Harvey 1993). These findings suggest that dietary sensitivity should be considered in young animals with cutaneous signs suggestive of an allergic dermatosis. Food sensitivity typically is perennial, although seasonal signs could conceivably be seen if an animal were sensitive to a seasonally-available foodstuff. If the onset of clinical signs is noted in the spring or summer, potential misdiagnoses would include infestation with trombiculid mites, atopy or flea bite hypersensitivity.

Clinical Features

The major clinical sign of food sensitivity in dogs and cats is pruritus. The response to the pruritus results in a gradation of signs ranging from saliva staining of the hair to severe self-trauma and a variety of secondary skin lesions (Leib and August 1989; Muller *et al* 1989; Reedy and Miller 1989). There are no primary lesions or

clinical signs pathognomonic for dietary intolerance, and the condition can mimic various dermatoses.

In the dog the pruritus may be regional or generalised, and excoriation, alopecia, scale, crust and secondary superficial pyoderma are common (Walton 1967; August 1985; Muller *et al* 1989). Unilateral or bilateral otitis externa may be a feature and may occur in the absence of other signs of skin disease (White 1986; Carlotti *et al* 1990; Harvey 1993). In the series reported by Rosser (1990), 24% presented with otitis externa only. Occasionally dogs will be non-pruritic (Walton 1967; White 1986) and in some cases primary lesions such as oedema, erythema, papules and pustules may be seen (Walton 1967; White 1986; Muller *et al* 1989). Some cases may present with recurrent superficial pyoderma only (Leib and August 1989; Reedy and Miller 1989; Harvey 1993).

In cats, food sensitivity can manifest as one of several cutaneous patterns: facial and neck pruritus, localised or generalised scales or crusts, miliary dermatitis, symmetrical or localised areas of alopecia, eosinophilic plaques (Scott 1987; White and Sequoia 1989; Carlotti *et al* 1990), and otitis externa. Facial pruritus is the most common presenting sign, with crusting areas around the neck, and excoriation of the face (Baker 1975; Scott 1980; Stogdale *et al* 1982; White and Sequoia 1989; Wills 1991).

Some abnormal behavioural signs may be attributable to a food sensitivity in cats and dogs, but data are lacking. Epileptiform seizures have been reported in 3 dogs with dietary hypersensitivity (Muller *et al* 1989). Some animal behaviourists consider that diets high in protein may be associated with aggression in certain breeds of dog, but this is unlikely to be a true intolerance.

Concurrent gastrointestinal signs have been reported in 10 to 15% of cases of food hypersensitivity in dogs. Some cases of inflammatory bowel disease in dogs respond well to an elimination diet (Willard 1992). Gastrointestinal signs have also been reported in cats with food sensitivity, either concurrently with skin lesions or as a separate entity. It can manifest as vomiting (Dakin 1988), profuse watery diarrhoea (Walton *et al* 1968; Stogdale *et al* 1982) and inflammatory bowel disease, including lymphocytic-plasmacytic colitis (Nelson *et al* 1984; Tams 1988). In the 6 cases of lymphocytic-plasmacytic colitis reviewed by Nelson *et al* (1984), diffuse intestinal thickening could be felt in 2 cats. Fresh blood may be seen in the diarrhoeic faeces, and there may be increased frequency of defecation.

Diagnosis and Differential Diagnosis

The clinical signs of food sensitivity in the dog and cat are quite variable and unpredictable depending as they do on the individual response; diagnosis is therefore difficult. In the cat, it probably helps to group the differential diagnoses into the 4 major presenting signs: facial pruritus, scales and crusts, miliary dermatitis and self-inflicted hair loss (Table 3, from Foil 1988).

There is no single test available to confirm or refute the presence of food sensitivity. Intradermal skin testing with food extracts is unreliable as a diagnostic test (Kunkle and Horner 1992). A radio-allergosorbent test (RAST) and an ELISA are commercially available in some countries to evaluate canine serum for food allergens by identifying antigen-specific IgE, but there is doubt about their reliability and credibility (Ackerman 1988; Jeffers *et al* 1991). More recently, similar systems have been introduced for cats, but have not yet been thoroughly evaluated.

Diagnosis of food sensitivity should be based on an accurate clinical history, full clinical examination and dietary investigation in the form of elimination diets and test meals (Muller *et al* 1989). Elimination diets should be individualised, whenever possible, on the basis of dietary history. Drinking water should always be available, but distilled or bottled water may be preferable to tap water in this diagnostic phase, as fungal contaminants and chemicals in tap water have been cited as a cause of food sensitivity in the dog.

Study of a detailed list of the animal's diet will allow the identification of foods that have not been fed before, and that could be used to formulate an elimination diet that will be hypoallergenic for the individual. Elimination diets for dogs include lamb, chicken, rabbit, horse meat and fish as sources of protein, with rice or potatoes as a source of carbohydrate. Most of these home-prepared elimination diets are not nutritionally complete unless balanced with appropriate vitamins and minerals (Roudebush and Cowell 1992). It is recommended that immature animals should not be fed such a diet for more than 3 weeks unless supplemented with a source of minerals and vitamins. Successful elimination diets for cats include boiled, baked or microwaved lamb, chicken, rabbit or venison with rice. Chicken has been used successfully in elimination diets for cats with food sensitivity (Baker 1975; Scott 1980; Reedy 1982; Holzworth 1987), and in dogs (Harvey 1993). If the diet is not complete and balanced, vitamin and mineral supplements may need to be given, as well as taurine supplementation for cats. A diet of 100 g chicken and 100 g rice will provide sufficient calories for a moderately active 4 kg cat for a day. If the chicken is microwaved or boiled, 100 g cooked chicken will still provide sufficient taurine for maintenance requirements in an adult cat. If the meat is boiled, the gravy should also be fed, as taurine can leach into the cooking water.

TABLE 3
Differential diagnosis of dietary hypersensitivity in the cat
(after Foil 1988)

	Facial pruritus	Scales or crusts	Miliary dermatitis	Self-inflicted hair loss
Dermatophytosis	*	*	*	*
<i>Ectoparasitic diseases</i>				
flea allergy	*	*	*	*
cheyletiellosis		*	*	*
trombiculidiasis		*	*	
demodicosis	*	*		*
pediculosis		*	*	
notoedric mange	*	*		*
tick bite reaction		*		
otodectic mange	*		*	
<i>Immunological diseases</i>				
food sensitivity	*	*	*	*
eosinophilic granuloma	*	*		
contact dermatitis	*	*		
drug sensitivity	*	*	*	
atopy	*	*	*	*
intestinal parasite hypersensitivity			*	
<i>Viral diseases</i>				
poxvirus	*	*		
herpesvirus		*		
calicivirus		*		
pseudorabies	*	*		
<i>Other diseases</i>				
neurodermatitis		*		*
cat bite		*		
pyoderma	*	*	*	
feline acne	*			
idiopathic			*	
biotin deficiency		*	*	
fatty acid deficiency		*	*	

If possible, cats should be restricted indoors as this will help to ensure that those with multiple feeding stations and those that hunt are unable to consume alternative foodstuffs. The period for which the elimination diet should be fed has been stated to be 3 weeks (Walton 1977; White 1986; Muller *et al* 1989). However, Rosser (1990) reported a prospective study of 51 dogs and found that 13 responded in this period, while 17 required 4 to 6 weeks, 12 required 6 to 7 weeks and 9 required 8 to 10 weeks to improve on the elimination diet. A recent report on a prospective study of 13 cats with food sensitivity suggests that some cats will not show maximum improvement in clinical signs until 9 weeks on the elimination diet (Rosser 1993). Fatty acid supplements containing fish oils should not be fed at this time, as fish is a commonly reported allergen in the cat (White and Sequoia 1989). The current recommendation is that an elimination diet be fed for 60 days before a diagnosis of food sensitivity may be ruled out.

A commercially-available restriction diet can be used in this diagnostic period, but it has been shown that some dogs with food sensitivity that improve with a home-prepared diet of lamb and rice, develop exacerbation of clinical signs when fed the equivalent canned prescription diet (White 1986; Rosser 1990; Jeffers *et al* 1991). This occurrence has been recorded in one cat (White and Sequoia 1989). The reason is not understood. It may be that the animal is sensitive to the allergen only in its form in prepared petfood, that is, processing of the food could affect allergen presentation.

Occasionally, the pruritus may be so severe that it may be necessary to try to break the 'itch-scratch-lick' cycle with a short course of sedation, glucocorticosteroids or antihistamines. Cats are more resistant than dogs to the side-effects of glucocorticosteroids, and they can tolerate higher doses. Oral prednisolone can be given at 2.2 mg/kg every other day. Antihistamine therapy is of benefit in some pruritic cats, and the most commonly recommended is oral chlorpheniramine at 2 to 4 mg twice daily. While the pruritus is being controlled with drugs, the cat can be started on the elimination diet. If the allergen has been eliminated, it should be possible to reduce the drug dose after 7 to 10 days without the pruritus returning. The diet should be fed for at least 2 weeks after the medication has stopped.

The major clinical sign being evaluated during the elimination diet is the pruritus. If food sensitivity was the cause of the pruritus, then the degree of pruritus should decrease markedly in 7 to 21 days. If skin lesions are present, they may take longer to heal. Animals with diarrhoea should improve in 3 to 5 days. If there is no improvement while on the elimination diet, this could be because:

- the animal is pruritic for a reason other than dietary sensitivity.
- the animal has a concurrent hypersensitivity (for example, flea-bite hypersensitivity, atopy or flea-collar hypersensitivity).
- the animal is allergic to the protein source in the elimination diet.
- poor owner (or family) compliance.

Improvement in clinical signs while on the elimination diet is suggestive of food sensitivity. The diagnosis should be confirmed by feeding the original diet (or the diet the animal was on at the time it originally presented with clinical signs). Exacerbation of clinical signs should occur within 7 to 14 days of feeding (Rosser 1990; Harvey 1993). This procedure establishes two things. Firstly, it demonstrates that there is a dietary aetiology, that is, the improvement was not coincidental, and secondly, it allows identification of the duration between challenge and clinical relapse. This information may be useful when producing a calendar detailing challenge items, order of challenge and duration of challenge (Table 4). Unfortunately, many owners are unwilling to challenge with the original diet. For owners who do not want to take the case further, it is worthwhile extending the range of prepared pet foods that the animal can tolerate. A different product could be fed every 14 days, and clinical response assessed (Rosser 1990; Harvey 1993).

TABLE 4
Summary of the procedure for elimination diet and challenge studies

1.	Identify a home-prepared elimination diet.
2.	Feed this diet for at least 3 weeks.
3.	If there is improvement, then replace with the original diet.
4.	Deterioration suggests diet-related disease and identifies duration between challenge and relapse.
5.	'Rescue' with elimination diet and attempt to replace with a commercial elimination diet.
6.	Sequential addition of individual dietary items for at least 2 weeks each, or as guided by note 4.
7.	Advise a commercial diet based on the results of the challenge studies. In a few instances it may be necessary to feed a home-prepared diet that is nutritionally balanced.

Management

Most cases can be managed with a commercial diet, either a grocery brand or a veterinary prescribed diet. If the client is co-operative, then the presumptive food allergen can be investigated. First, the diet is gradually changed from a home-prepared elimination diet to a commercial diet of selected protein. This not only provides a complete and nutritionally-balanced diet, but is also more convenient for the owner and increases owner cooperation. This diet acts as the basal diet for test meals, summarised in Table 4. The clinical response to the diet should be assessed. Then individual foodstuffs are introduced into the diet. The recommended length of time to challenge with the test diet is 7 days for each dietary component (August 1985; Muller *et al* 1989). However, Rosser (1990) found that 7.7% of his canine cases required 7 to 14 days of feeding dietary components for a positive response. In most cases, if the animal is not pruritic after 7 to 14 days, then the next item can be introduced. The most commonly reported allergenic foods should be tried first. In the dog, these are cow's milk, cheese, beef, cereal and egg (Harvey 1993), and in the cat, cow's milk, beef and fish (Wills 1991). If the offending allergen is re-introduced, then there will be a clinical relapse in 1 hour to 7 days; when the allergen is removed, clinical signs will improve.

Prognosis

The prognosis for food sensitivity in the dog and cat is good, if the specific allergen can be avoided. Sometimes it is not possible to identify the offending item, in which case the nutritionally-balanced elimination diet can be maintained, or a grocery brand tried. Most food sensitive dogs and cats can be managed on a commercial diet.

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(Accepted for publication 25 February 1993)

Feline lower urinary tract disease: dietary considerations

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Feline lower urinary tract disease (LUTD) is generally suspected when cats are presented with a history of dysuria, haematuria, pollakiuria with or without urethral obstruction. Although epidemiological studies on LUTD in Australian cats have not been done, the incidence of haematuria, dysuria and/or urethral obstruction in overseas cats has been reported to be less than 1% per annum (Fennell 1975; Walker *et al* 1977; Tomey and Follis 1978; Lawler *et al* 1985). However, proportional morbidity has been observed to be as high as 5% in cat-only clinics in Australia (VM Menrath, personal communication), which is consistent with overseas reports of 1 to 10% (Foster 1961; Engle 1977; Walker *et al* 1977; Willeberg 1981). Although feline LUTD can be due to a number of causes, including traumatic, neurogenic, anatomical, neoplastic, infectious and urolithic, most are idiopathic in origin (Barsanti *et al* 1982; Osborne *et al* 1984, 1989; Kruger and Osborne 1990).

Based on clinical signs, idiopathic LUTD in cats can be divided into non-obstructed and obstructed groups. Whether the two groups have common aetiologies (Schechter 1910; Osborne *et al* 1989) or are made up of a number of subsets, each with its own aetiology, is unknown. The non-obstructed idiopathic LUTD group, which usually present with haemorrhagic cystitis in the absence of bacterial infection, is seen mainly in female cats in Australia, although an approximately equal sex distribution is reported overseas (Schechter 1970; Osborne *et al* 1989). These cats often have a history of recurring episodes of pollakiuria and/or dysuria. The urine usually contains frank blood, and the duration of the haematuria may vary from a few hours to many days. Blood clots may be seen occasionally in the voided urine. Urinalysis shows haematuria with erythrocytes at various stages of degeneration, suggesting chronic bleeding. The urine is usually bacteriologically sterile (Schechter 1970; Barsanti *et al* 1982) and pyuria is not a feature of this disease, so that the urinalysis is more suggestive of a bleeding disorder than of an inflammatory disease. Crystalluria is absent in most cases and when

present, the crystals are of normal size and number. The urinary bladder wall in cats with haematuria may show petechial and/or ecchymotic haemorrhages and histological signs consistent with chronic cystitis.

The obstructed idiopathic LUTD group, which is commonly referred to as having feline urethral obstruction (Bovee *et al* 1979), is almost always male cats (Engle 1977; Osborne *et al* 1984, 1989). These cats are presented with overdistended urinary bladders and in varying stages of post-renal renal failure (Burrows and Bovee 1978). The urethral obstruction is usually caused by a sabulous plug composed of colloidal matrix into which are embedded crystals, usually struvite crystals (Jackson 1971; Osborne *et al* 1989). In about 5% of cases, the urethral plug lacks crystalline material and is known as a non-crystalline matrix plug (Jackson 1971). The factor(s) involved in the formation of urethral plugs are unknown and may be different for the different plugs (Osborne *et al* 1989). Since minerals other than struvite may be found in some urethral plugs, matrix *per se* may play an important primary role in the formation of some plugs (Osborne *et al* 1984, 1989). Urinalysis in cats with obstructed LUTD shows varying degrees of haematuria depending on the duration of the obstruction (Jackson 1971). Pyuria is variable, but crystalluria is often seen, and on many occasions large crystals are found in the urine (Carbone 1965). The significance of these large crystals is unknown. Urinary pH and specific gravity (SG) values, at presentation, are variable, but pH is usually acidic and SG generally approaches 1.020 with prolonged obstruction (Carbone 1965; Rich and Kirk 1969; Jackson 1971).

Historically, diets or components of diets have been implicated in the pathogenesis of some forms of LUTD in cats, notably urolithiasis (Jackson 1971, 1972; Rich *et al* 1974; Walker *et al* 1977; Lewis *et al* 1978; Buffington *et al* 1985). Based on this research approach, veterinary-dispensed diets have been formulated to, at least theoretically, overcome the dietary factors that have been considered important in struvite crystal formation (Lewis and Morris 1984; Cook 1985). Such diets should be nutritionally complete as they need to be fed exclusively so that the cat's total dietary intake can be

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