The exact mechanisms involved in clinical cases of cutaneous adverse food reactions are rarely known. Commonly terms such as food allergy (to describe a repeatable immunologically mediated reaction to ingestion of a particular food) or food intolerance (non-immunological event) are used to differentiate between pathomechanisms. In dogs immunologically mediated adverse events dominate with respect to cutaneous manifestations. Food induced cutaneous reactions can be clinically indistinguishable from canine atopic dermatitis. Cellular responses and cytokine profiles in lesional skin are also similar. This has led to the terminology “food induced atopic dermatitis” (FIAD).

This presentation will focus on:

- Risk factors for food allergy
- Overview of pathogenesis of food allergy
- Link with atopic dermatitis
- When to suspect FIAD
- How to diagnose (and understanding our limitations)
- Management of FIAD

**RISK FACTORS FOR FOOD ALLERGY**

Food allergy is described as any immune mediated reaction to an ingested food. Food allergy is reported to affect between 2 -10% of people and 10 to 15% of pruritic dogs and cats but the true prevalence is unknown. Risk factors for the development of food allergy in children include gender (increased in males), ethnicity (increased in African Americans and Asians), genetics (familial associations and HLA linkage), atopic dermatitis co-morbidity, intestinal parasitism, increased hygiene, early use of oral antimicrobials, vitamin D deficiency, delayed exposure to dietary proteins (after 6 months), premature exposure to dietary proteins (less than 4 months), obesity, reduced omega 3 intake and tick bites.

In dogs associations are less clear. Diseases that disrupt the mucosal lining have been implicated but there are no sex, breed or age associations. Boxers, Cocker and Springer Spaniels, Collies, Dalmatians, German Shepherds, Lhasa Apsos, Miniature Schnauzers, Retrievers, Shar-Peis, Soft-Coated Wheaten Terriers, Dachshunds and West Highland White Terriers have been reported to be at increased risk of food allergy but this is not statistically confirmed.

**PATHOGENESIS OF FOOD ALLERGY**

The GALT (gut associated lymphoid tissue) has the onerous task of differentiating between nutrients and harmful and noxious agents. This is a big job as the gut has the largest surface area of any organ interfacing with the environment. The GALT consists of the Peyer’s patches (lymphoid follicles scattered through the intestinal mucosa), lymphocytes and plasma cells in the lamina propria, intraepithelial lymphocytes within enterocytes (IELs) and submucosal lymph nodes. Gastrointestinal tolerance is maintained by a combination of antigen exclusion and immune regulation (tolerance). The two major challenge periods are immediately after birth and at weaning.

Oral tolerance is an antigen specific active process by which antigens reaching systemic circulation are rendered immunologically inactive. Normal intestinal dendritic cells that present ingested antigens to naïve B and T cells within the lymphoid follicles lack co-stimulatory molecules resulting in hypo-responsive predominantly T regulatory cells. These intestinally derived T cell clones, when they enter systemic circulation via the mesenteric lymph nodes, will “home” (due to expression of specific
cellular adhesion molecules) to the intestinal lamina propria to await a secondary exposure to the same antigen. Repeat exposure requires intact antigens to reach the lamina propria and effector T cell clones there produce cytokines with a T reg or Th-2 bias (specifically IL10 and TGFβ) that promotes IgA production and suppresses development of Th-1 lymphocytes and IgG production. In other words promotes tolerance.

Reduced digestibility or reduced digestion of peptides, impairment of GIT mucous lining and enterocyte tight junctions, decreased secretory IgA levels and reduced peristalsis will increase antigen penetration. Once antigens penetrate then the mechanisms of presentation (enterocytes and dendritic cells in the lamina propria), the dose of the antigen, the ability to form immune complexes and remove the antigen via mononuclear phagocytes and the cytokine profile triggered by signalling induced by gut flora and functioning of the submucosal lymph nodes all play a role in outcome (potentially leading to loss of oral tolerance). Genetic profiling in people shows gene variants for IL12 R, Toll-like receptor 9, thymic stromal lymphopoietin and IL4 genes correlate with the development of food allergy. The gut microbiome impacts the cytokine profile and can trigger pro Th-2 (allergy) signalling. Sphingolipids in foods and milk can directly trigger natural killer (NK) T cells to produce Th-2 cytokines (pro-allergy) and induce allergic symptoms in some individuals. Heating of ovalbumen with glucose increases antigen processing and Th-2 cytokines while isoflavones in soy reduce the risk of peanut sensitisation. So the type of food, timing of challenge and the combinations of dietary proteins and the cooking/processing of the food may all influence the development of food allergy. Recent human data shows timing is critical for the development of tolerance with 4 months of age being the ideal time to expose infants to a wide range of dietary proteins.

Animal studies (pigs) also suggests the gut microbiome may profoundly impact on the development of oral tolerance. Studies in high IgE responding Beagles (a validated model for atopic / food allergies in people) have demonstrated that tolerance can be induced to ovalbumen (hen’s eggs) and cow’s milk by early ingestion of these proteins. This suggests the timing of introduction of dietary proteins may be important to the development or oral tolerance in animals as well. Increased numbers of duodenal T-reg cells has been correlated with oral tolerance in inflammatory bowel disease is dogs. IBD dogs have less duodenal T-reg cells compared with healthy controls, healthy dogs developed increased T-reg cell numbers with age (progressive tolerance) and nematode infestation increased T-reg cells. As in people, oral tolerance is likely a complex event affected by many factors including genetics, food type and timing of initial challenge and local cytokine milieu within the gut at the time of antigen presentation. Type I (IgE mediated), Type III (immune complex mediated) and delayed type IV reactions are suspected in dogs and cats but the exact mechanisms of cutaneous food reactions are usually not known.

What is obvious is that IgE triggered anaphylaxis and urticarial reactions to foods are rare in dogs and cats compared with people. This suggests that T cell mediated reactions may be important in our food induced cutaneous reactions. So how do food-sensitised T-cell clones that develop to ingested antigens, through a failure of gut tolerance, get to the skin? Re-homing to the gut is expected for these T-cell clones. So either there would need to be a failure in T-cell homing resulting in aberrant migration of sensitise T-cells to the skin OR there needs to be another mechanism for the development of cutaneous sensitisation to food antigens (not just a loss in oral tolerance).

Tick-bite induced food allergy is a special case where tick bites induce allergies to red meat. Alpha – gal is a carbohydrate carried by all mammals other than humans and the higher apes. This is ingested when a tick feeds on mammal’s blood and it introduces alpha-gal into the tick's GIT. The tick transfers this to the human host and induces sensitivity to alpha-gal. Ingestion of any mammal meat then leads to a hypersensitivity reaction. This alpha-gal sensitisation can also predispose to gelatin (in medications and vaccines) hypersensitivity reactions. The interesting thing is that sensitisation in this case is clearly via the skin and not a loss of oral tolerance. Could the skin be the primary target for sensitisation in cutaneous food allergies?
ASSOCIATION WITH ATOPIC DERMATITIS

In children one third of all atopic dermatitis sufferers are reported to have an allergy to food. In human medicine they tend not to discriminate between food induced eczematous reactions and non food triggered reactions and they are all covered by the umbrella term of atopic dermatitis. Food hypersensitivity has been induced experimentally (percutaneously) in the canine. Concurrent environmental and dietary triggers for canine atopic dermatitis are recognised in clinical practice but the true co-morbidity figure for canine and feline atopic dermatitis and cutaneous adverse food reaction is unknown.

So what is the link between food allergy and atopic dermatitis? Atopic dermatitis in children usually precedes the development of food allergy. A 5 year multi-centred study in infants (3-18months) showed that 15% of children with mild atopic dermatitis had concurrent food allergies and as the severity of the atopic dermatitis increased so did the percentage of food allergies. The diagnosis of food allergy in this study was based on proven immunological reaction to an ingested food and not just the presence of sensitisation (IgE on serology). The studies recommendation was that children with moderate to severe atopic dermatitis that persisted in spite of compliant atopic management be investigated for dietary allergies.

The link between atopic dermatitis and food allergy may be the high rate of loss of function mutations of filaggrin (flg) leading to sensitisation to dietary proteins through the skin. Infants with flg mutations are 6 times more likely to get peanut and cow’s milk dietary allergies. Sensitisation to profilins in pollens can lead to sensitisation to ingested fruits and vegetable and is a major trigger for oral allergy in people. Sensitisation to grains is also commonly reported in atopic individuals. Flg polymorphisms and reduced flg expression have also been identified in canine atopic dermatitis.

HOW DO WE RECOGNISE CANINE FOOD ALLERGY?

There are some limitations in the published literature on food allergy. Most papers did not include any lesion scoring system, now considered standard, and validated in atopic dermatitis studies. Most also did not define how pruritus was assessed. Visual analogue scales for pruritus assessment have now been established and validated for dogs. Most did not have any process for checking owner compliance. All of these limitations reduce the strength of the evidence.

Observations from published literature

AGE OF ONSET

33 to 52% present before 1 year; Mean 2.4 years, youngest 4 months

DISTRIBUTION PATTERN

Ears, feet, inguinal and axillary areas the most commonly affected then muzzle, periorbital and limbs. Peri-anal area 11%-50% of cases, generalised 10%
Lateral contact areas, ventral chest and the flank the least commonly affected areas

LESIONS

Erythema without papules was most common symptom but papules seen in 22%.

OTITIS EXTERNA

Recurrent otitis externa (OE) 55%
Otitis externa first symptom 34%
CONCURRENT INFECTION

Recurrent pyoderma 66%
Recurrent Malassezia dermatitis 43%

SEVERITY OF PRURITUS AND RESPONSE TO CORTICOSTEROIDS

97% moderate to severe itch
Prednisolone response 39% complete resolution, 44% partial and 18% no response.

GASTROINTESTINAL SYMPTOMS

6% - 30% concurrent GIT symptoms

CONCURRENT ATOPIC DERMATITIS

18% - 30% had concurrent aeroallergies

Food allergy should be considered in all dogs with a classical ATOPIC presentation where the severity of the disease is moderate to severe and where there is no seasonal variation in intensity. Dogs with a CONTACT distribution of disease and no history of OE are less likely to be food allergic.

HOW DO WE DIAGNOSE FOOD ALLERGY?

A dietary elimination trial remains the only effective tool to definitively diagnose adverse food reactions (AFR) because skin and serologic tests, skin biopsy and gastroscopic food sensitivity testing have all been demonstrated inadequate for the purpose. The selection of an appropriate elimination test diet is dependent on the dietary history. Elimination diets used in veterinary medicine include commercial novel protein, commercial hydrolysed protein and home-prepared diets using fresh ingredients.

In the last decade, pet foods claiming to be useful as limited antigen diets have gained a significant market position with the aim of diagnosing AFR and controlling the related symptoms. The gold-standard method to diagnose an AFR consists in feeding the animal a limited antigen diet until clinical symptoms improve and then reintroducing the diet previously fed to demonstrate a relapse of symptoms. The objective of limited antigen diets is to avoid exposing animals to potential allergens during the 8- to 10-week elimination period. These diets can either be home-cooked or commercially produced. Both typically contain a single novel source of protein and a single source of carbohydrate, neither of which is usually contained in maintenance diets, and therefore, there is less probability of the animal ever having come into contact with them before. Whenever no clinical improvement is observed during the dietary trial, AFR is ruled out and other diseases such as atopic dermatitis or non-allergic pruritic disorders may be suspected.

Controversy persists between the choices of a home-cooked diet and a commercial veterinary diet for the elimination trial. The commercial diets may offer the advantage of enhanced owner compliance because of minimal time in food preparation. Home-cooked diets can be unbalanced and may be inadequate in young, large breed, rapidly growing dogs if they are not carefully formulated. In addition, purchased minced meat from one animal source may be contaminated with minced meat from another meat source if the machine was not completely and thoroughly cleaned between uses. Rarely, dogs have been described that tolerate home-prepared ingredients from a specific protein source but not their commercially prepared versions. This finding raises concerns that heat processing may change the food allergen configuration, substances may leech into the food during industrial processing, or food additives may be an allergen source.
Although most veterinary dermatologists consider home-made diets the first choice when submitting a dog to a diagnostic food elimination trial, many owners prefer the convenience of commercial diets, and for this reason, several studies have assayed the efficacy of commercial limited antigen diets in diagnosing AFR. Choosing the correct commercial limited antigen diet is a very important step in diagnosing canine AFR and begins by collecting an accurate feeding history from the owner that reveals the ingredients the dog has already eaten. Therefore, careful reading of the ingredients on the label is of utmost importance in avoiding exposure to potential allergens. The diet must not contain any of the ingredients previously mentioned by the owner.

NOVEL PROTEIN DIETS

Commercially available novel protein diets usually contain a single protein and a source of carbohydrate that, ideally, most animals are unlikely to have received regularly in their diet. However, the novel protein commercial diets in Australia are rarely suitable for dogs and cats, because lamb, duck and turkey and fish are the major available options, all of which are common ingredients in a range of commercial foods. Generally, the closer the taxonomic relationship between meat sources, the higher the risk of cross-reactivity. Allergens from beef theoretically may cross-react with those from other ruminants. Thus, lamb and venison may not be the best unique ingredient protein sources because most animals have been previously exposed to beef. However, this finding has yet to be seen as a problem in veterinary medicine. There is evidence of common allergens in avian meats, and the use of duck diets in patients previously exposed to chicken may not be advisable. Cross-reactivity among meats of various proveniences has not been studied yet in dogs and cats.

While they are convenient, increase owner compliance, are nutritionally of higher value and more balanced compared to many home-cooked diets; there is increasing evidence that these diets are not infallible. In 2011 ELISA testing of 4 venison based elimination dry food diets showed 3 of the 4 diets contained soy or beef proteins not listed as ingredients (this may explain the high rate of relapse to the venison diet in the previous study mentioned above). A 2013 study evaluated 11 restricted protein and 1 hydrolysate diet promoted for use in elimination diets for the presence of undeclared animal proteins. Both DNA testing and microscopic examination of bone fragments in the diets was conducted. DNA testing showed in 8/12 diets there were undeclared proteins and microscopy showed 10/11 diets had bone fragments from non-declared species (the liver hydrolysate diet was not subjected to microscopy). Their conclusion was that failure to respond to a commercial elimination diet did not rule out food allergy and a home-cooked diet should be performed in those cases.

HYDROLYSED DIETS

Commercial hydrolysed protein diets (Table 1) represent a valid alternative to novel protein diets. These diets are enzymically modified to reduce the molecular weight of proteins to confer higher digestibility and lower allergenicity. It is not known, however what protein concentration, size, or structure is required to elicit an immunologic reaction in a food allergic dog.

In an early study, 21% of dogs known to be allergic to soy and corn reacted adversely to a hydrolyzed soy diet with a relapse of cutaneous signs, but 79% of these dogs were asymptomatic when the diet was fed for 2 weeks. Thus, a commercially available hydrolysate soy and corn diet was tolerated by most, but not all, of the dogs sensitized to the parent proteins. In a more recent study evaluating 12 dogs with cutaneous manifestations of allergic dermatitis after exposure to chicken meat, clinical signs improved in 11 dogs when fed a hydrolysed chicken diet, whereas 1 animal did not significantly improve. In another study of experimental soy allergic dogs, IgE binding to hydrolyzed soy peptides was found to be significantly reduced (but not absent) compared with binding to the native soy protein. So in summary, from 5–30% of dogs with confirmed AFR to a protein source also reacted to the hydrolysed version of that protein. This may be due to an insufficient degree of enzymatic proteolysis, which is an important variable for the successful outcome of a hydrolysed diet. Reactions to partial whey-hydrolysed formulas have been reported in cow’s milk-allergic children, and occasional reactions to even the most extensively hydrolysed casein or whey formulas have been
demonstrated in highly sensitive children. Another possibility is contamination with other proteins occurring during the production process of the diet. In all cases, a correct diagnosis of AFR would be compromised.

In a recent systematic review of 11 studies examining the evidence in favour of reduced immunologic and clinical allergenicity of hydrolysates in dogs with CAFR, it was concluded that hydrolysate-containing diets were probably best used in dogs with no suspected hypersensitivity to the original source of hydrolysed peptides. In other words if the parent protein is not a novel protein then the diet is not suitable for diagnostic purposes.

So based on the above we need to change manufacturing standards to minimise risks of cross contamination of foods and look at testing the end product for purity (this is starting to happen) or we are back at feeding a home-cooked NOVEL protein and NOVEL carbohydrate source (one the dog has not been previously exposed to) for a minimum of 6 to 10 weeks.

A new commercial hydrolysate diet Anallergenic® composed of purified corn starch and hydrolysed chicken, turkey, duck feathers has recently become available. The aim of this diet is to ensure all hydrolysed peptides are of very low molecular weight (< 1 kDa) in order to ensure they are non-immunogenic. This diet is essentially purified corn starch (protein removed) and hydrolysed chicken, turkey, duck feathers. To minimise risks of diet contamination there is complete cleaning of manufacturing lines before production, single diet production and post-production quality control (PCR to check for poultry and beef proteins) and chromatography to ensure anallergenic molecular weight of the hydrolysis.

Independent studies showing usefulness of these diets for elimination diets are still lacking. There are two company sponsored trials involving a total of 34 dogs using a non-validated pruritus score and a validated CADESI 03 lesional score, that showed significant improvement in dog’s global score when this diet was fed to dogs with suspected cutaneous food reactions (many of which had previously failed on hypoallergenic diets). Palatability was reported to be high. These were low evidence studies based on design but warrants further evaluation.

<table>
<thead>
<tr>
<th>Table 1: Hydrolysed Diet</th>
<th>Constituents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Royal Canin Anallergenic® canine</td>
<td>hydrolysed chicken, turkey, duck feathers, fish oil, purified corn starch (protein removed)</td>
</tr>
<tr>
<td>Royal Canin Hypoallergenic® canine dry and canned</td>
<td>hydrolysed soya protein isolate, hydrolysed poultry liver, rice, beet pulp, poultry fat, borage oil</td>
</tr>
<tr>
<td>Hill’s Prescription Diet z/d ultra ® dry and canned</td>
<td>hydrolysed chicken, corn starch, cellulose and soybean oil</td>
</tr>
<tr>
<td>Hill’s Prescription Diet z/d ® canine low allergen</td>
<td>hydrolysed chicken, potato, potato starch, cellulose and soybean oil</td>
</tr>
<tr>
<td>Nestle-Purina HA ® (NZ only)</td>
<td>hydrolysed soy, corn starch, cellulose, vegetable gums, coconut oil, canola oil, corn oil</td>
</tr>
</tbody>
</table>
HOME COOKED DIETS: ARE THEY INFALLIBLE? DO WE KNOW WHAT IS NOVEL?

In vitro molecular studies in dogs have demonstrated specific food allergens include chicken serum albumin, bovine serum albumin, bovine IgG, ovine IgG, muscle phosphoglucomutase and two soy proteins. Immunoglobulin G has been shown to be a potential cause of cross reactivity to lamb due to high homology in beef allergic dogs. Five other lesser IgE binding antigens have identified in beef and lamb.29,30,31

Cross reactivity means that not having been exposed to that meat source before does not mean sensitivity cannot exist. Cross reactivity occurs when an adaptive immune response to one antigen causes reactivity to other structural related proteins. Homology of greater than 70% is considered necessary for IgE mediated events.32 Less is known about T cell cross reactivity. Cross reactivity and reactions to conserved molecules across different food groups are commonly documented in people and challenge what we think we know about “novel” foods when considering elimination diets. What do we really know about cross reactions to dietary proteins in dogs in cats and the potential for pollen sensitisation to increase the risk of food sensitisation in animals? The answer is not much and certainly much less than in humans.

The molecular basis of food allergy and the potential for cross reactions are becoming increasingly understood in people. The presence of sensitisation to carbohydrate epitopes like alpha-gal means that in people there is the potential to be sensitised to ALL mammal proteins and mammal epithelia (cat and dog dander) due to expression of alpha – gal. Other documented examples of cross reactivity in people that MAY be relevant to dogs include:

- cow’s milk with beef due to bovine serum albumin (BSA);
- beef with lamb due to BSA and IgG;
- potential cross reactivity between pork, beef and lamb due to conserved carbohydrate epitopes;
- cross reaction between mammal proteins due to albumins and immunoglobulin homology;
- eggs with poultry meat due to alpha-livetans and avian serum albumin;
- conserved reaction between fish species due to parvalbumen (levels vary with species and 6 to 50X lower in tuna and lower again heat processed canned tuna)
- grass pollen profilins show 75% homology with those of tomato, watermelon, banana, sunflower, carrot and a long list of fruit and vegetables (our pan pollen allergic atopic dogs are thought to be sensitised to profilins);
- rice allergy due to sensitisation to Asteracea pollens (daisy, dandelions) and the potential then to cross react with non heat labile lipid transfer proteins in many fruits and multiple vegetable groups and nuts;
- birch pollen allergies and cross reaction with apple and carrot.3,32,33,34

How then can we be sure, when feeding a home-cooked diet with a novel protein and carbohydrate source that they are truly novel? With so much not yet known about the molecular nature of food allergy in dogs and cats OR the molecular nature of pollinosis in dogs and cats (thus the potential for pollen sensitisation to influence dietary sensitisation) some caution should be exercised about the superiority of home-cooked diets over commercial diets. They probably both have limitations due to lack of knowledge.
HOW I DO AN ELIMINATION DIET?

I encourage owners to do a “novel” protein and carbohydrate diet. In patients that I strongly suspect FIAD that fail a compliant “novel” home-cooked food trial I will repeat the trial with a second phylogenetically distinct “novel” protein. In people there appears to be very little cross reactivity between mammals to poultry to fish (but lots of potential for cross reaction within phylogenetic lines). I often use horse or donkey meat or kangaroo as my initial novel protein source depending on the previous dietary history. Recently it has been suggested that crocodile meat is an ideal source if available as no cross reaction has been identified between crocodile and pork, beef, chicken or fish.35.

During the dietary trial other controllable triggers for itch need to be controlled. Topical antimicrobials and topical flea control recommended during the elimination diet (beware flavoured medications and avoid gelatine capsules as may contain pork or beef and be aware that not all ingredients are listed by the manufacturer). The safest way is avoid oral medications during the elimination diet.

Apoquel® (oclacitinib maleate), when available and depending on excipients, may be useful for the first 3 to 4 weeks of the dietary trial to help manage the pruritus. Assessment of the itch is performed between week 6 and 8 (2 to 4 weeks after cessation of anti-pruritics). Clients are given a visual analogue scale to record itch levels and encouraged to record the level prior to starting the trial and then weekly. This is freely available at the following link


Animals should be weighed each 14 days to ensure neither under or over feeding. Energy levels will vary depending on the patients breed and life style. Home cooked diets are not balanced but serious health issues are not expected for the duration of the food trial. They are not recommended though for long term feeding.

Where doing a home cooked food trial is not possible due to owner lifestyle I would prefer a compliant commercial food trial than a poorly compliant home cooked food trial. In these circumstances, for now, I would go with SKD ® Prime crocodile and tapioca OR the Royal Canin Anallergenic® diet. In growing puppies I would stick to commercial based diets and if there is a failure, I would repeat later at the end of the rapid growth phase. In a dog that I strongly suspected FIAD that failed to respond to a compliant commercial based diet I strongly encourage repeating the trial with a home cooked trial.

Response to dietary trialling must be CONFIRMED by controlled re-challenge. Initial re-challenge is an all in repeat of the original diet. This means a positive flare confirms food allergy but not the exact nature of the offending allergen(s). Re-examination as soon as possible after a flare is good to rule out relapsing infections that can occur with re-challenge. In the absence of infection animals usually settle when back on the strict diet after 7 to 14 days. At that point a SINGLE INGREDIENT re-challenge is performed. I will usually give the owners a predetermined order of re-challenge based on what is most likely to be the trigger down to the most unlikely based on their previous dietary history. Remember that cooking/processing and the combination can alter the allergenicity of foods so identifying the exact trigger is not always easy. The duration of each introduction is based on the timing of the confirmed flare but in most cases this will be about 7 days (obviously if the flare occurred a day 10 then the single item re-challenge would be extended to 2 weeks per new ingredient). For mammal proteins and kangaroo I will ensure that the re-challenge includes raw meats as well as cooked as the allergenicity can vary with cooking. When a flare occurs on single item re-challenge the process repeats, re-examine if possible, back to strict diet, settle (if not examined at time of flare then must re-examine if does not settle within the previous time frame as this usually indicates infection or flare was co- incidental and not food triggered) then continue on to ensure ALL dietary triggers identified and assess for cross reaction within the same animal group.
Balanceit.com can be a useful resource for assisting in preparation of home-cooked food trials as can Dr Nick Cave at Massey University. They have a web site and I would highly recommend the Massey service.

WHEN TO REFER OR DO IgE SEROLOGY

The focus today is on food induced atopic dermatitis but it must be remembered that most dogs presenting with typical atopic symptoms will have airborne allergies (pollen and dust mite allergies) rather than food induced allergy. To do a commercial trial and fail then 2 x phylogenetically distinct home cooked trials and fail would take 18 to 24 weeks. It is hard to keep people motivated for this length of time when a 5 -15% success rate is all you are offering. So for me, in primary access cases, failure on either a COMPLIANT home-cooked or commercial based trial would be a trigger for referral (or doing IgE serology for aeroallergens if referral not geographically accessible). In those cases you have reduced the likelihood of food allergy further but not eliminated the possibility. In such a cases if IgE testing (intradermal and/or serology) were negative then repeat trialling as previously discussed could be considered.

HOW TO WE TREAT FOOD ALLERGY?

In a word, AVOIDANCE. If the offending allergen(s) have been identified then finding a balanced, convenient, commercial diet that does not contain these allergens is sought. A controlled re-challenge is performed. Remember if an allergen is not listed in the ingredients, it means it has not been added, but it does not mean it is not present. If the diet is tolerated, no skin reactions and palatable to the patient, then I would stay with it long term. Accidental contamination during processing is still possible unless made in an exclusive facility utilising only those ingredients listed or post production testing for purity of ingredients is performed.

In infants now there is a trend towards early intervention to try and restore tolerance via use of probiotics, oral immunotherapy with heat treated (baked) or hydrolysed allergens and incremental dosing. Oral immunotherapy requires frequent dosing. Cessation for several weeks can lead to a loss of tolerance. 36

CONCLUSION

Dietary triggers of pruritus should be considered in all non-seasonal atopic dogs especially where the pruritus is moderate to severe and drug reliance is not reduced by standard atopic therapies. Our understanding of food allergy is superficial and as such we need to be open minded about what is the ideal elimination diet and be prepared, in some cases, to repeat the elimination diet. Probiotics may play a role in prevention of development of food allergy and re-induction of oral tolerance. Hydrolysed diets may turn out to have a role in re-induction of tolerance as well as being useful for diagnostic purposes.
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