The objective of this presentation is to learn how to diagnosis and manage a pruritic dog in a safe and cost effective manner.

When a dog is presented with a chief complaint of pruritus it is essential that you approach the problem in a systematic manner. Not only will this be the most cost effective approach but also it is also less likely that you will miss the underlying disease.

The simplified approach is as follows:

1. **History including signalment**
2. **Physical examination (skin AND ears)**
3. **Evaluate the distribution of the pruritus and lesions**
4. **Look for and treat the obvious- Perform the 3 slide technique**
   - Skin scrapings
   - Skin cytologies
   - +/- ear cytologies

- Treat for parasites empirically or confirmed
- Treat bacterial and/or *Malassezia* infection
- Treat otitis externa if present

- Complete response—therefore seasonal atopic dermatitis (AD), ectoparasites or endocrinopathy is the underlying disease

**/+/- oral prednisolone depending on the severity of the infection(s)**

- **Non-seasonal - do a food trial**
- **Partial or no response**
  - **Mild pruritus**
    - Good skin care (baths/clipping, moisturizers, clothing)
    - EFA
    - +/- antihistamines
  - **Moderate pruritus < 2 months/yr**
    - Good skin care
    - EFA
    - Discussion w/owner
    - AH vs. steroids
  - **Moderate pruritus > 2 months/yr**
    - Good skin care
    - EFA
    - Steroids for short term relief
    - mCSA for intermediate relief

- **Seasonal**
  - **Partial or no response**
  - **Moderate pruritus > 2 months/yr**

**RECHECK!!!**
DIAGNOSIS

Obtaining the signalment is the first step toward diagnosing the pruritic dog. Age, breed and sex can help point you in the right directions. Next is to obtain a detailed history including if, when and how the dog has been treated for previous skin disease and what medication(s) the dog is currently receiving. This is followed by a detailed dermatologic (including otic) examination including evaluation of the hair coat and the skin and the distribution of any lesions. At this point you should be able to make a list of differential diagnoses for this pruritic dog. A DIAGNOSIS OF PRURITUS IS LIKE MAKING A DIAGNOSIS OF VOMITING. It is a symptom so you need to follow the word “pruritus” with the phrase “due to”. Historical information of value includes the age of onset of the pruritus and the responsiveness of the pruritus to steroids.

COMMON CAUSES OF CANINE PRURITUS BASED ON AGE OF ONSET

<table>
<thead>
<tr>
<th></th>
<th>2-3 months old</th>
<th>5 months to 5 yrs</th>
<th>6 yrs +</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ectoparasites (fleas, sarcoptes Cheyletiella, Lice, etc) NOT DEMODEX</td>
<td>+++</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Atopic dermatitis</td>
<td>++</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Environmental triggered atopic dermatitis</td>
<td>+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Cutaneous adverse food reactions triggered AD</td>
<td>++</td>
<td>+++</td>
<td>++</td>
</tr>
</tbody>
</table>

Scale of + to +++ with + being very uncommon and +++ being very common

Another tool that is helpful in making a differential diagnoses list is the responsiveness of the pruritus to glucocorticoids (GC).

RESPONSIVENESS TO GLUCOCORTICOIDS

<table>
<thead>
<tr>
<th></th>
<th>Responsiveness to anti-inflammatory doses of steroids</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ectoparasites (fleas, sarcoptes Cheyletiella, Lice, etc) NOT DEMODEX</td>
<td>+ to +++ depending on which parasite (especially poor for sarcoptes)</td>
</tr>
<tr>
<td>Atopic dermatitis</td>
<td>+ to +++</td>
</tr>
<tr>
<td>Environmental triggered atopic dermatitis</td>
<td>+++</td>
</tr>
<tr>
<td>Cutaneous adverse food reactions atopic dermatitis</td>
<td>+ to +++</td>
</tr>
<tr>
<td>Pyoderma</td>
<td>+ to +++</td>
</tr>
<tr>
<td>Malassezia</td>
<td>+ to +++</td>
</tr>
</tbody>
</table>

Scale of + to +++ with + being poorly or unresponsive and +++ being very responsive.
Once you have made your differential diagnoses (ddx) you can then do diagnostic testing. By making a ddx list you can perform the most cost effective tests. Diseases that need to be ruled out and the tests needed are:

1. Ectoparasites (fleas, sarcoptes, Cheyletiella, lice, etc)
   a. Superficial skin scrapings, acetate tape impression (unstained); flea combing (helps dx Cheyletiella too), fecal, vacuuming, therapeutic trial w/an ectoparasiticidal agent.

2. Endocrinopathy- note – these are NOT pruritic without a secondary infection (bacterial or fungal). Dogs that are presented for pruritus and have hypotrichosis/alopecia as a consequence of pruritus rarely would have a concurrent endocrinopathy except for iatrogenic hyperadrenocorticism
   a. CBC, serum chemistry profile and urinalysis. Depending on which endocrinopathy you suspect you will do thyroid testing and/or adrenal gland testing
      i. NOTE – It is very important to avoid testing thyroid function during and shortly after the administration of some medications because they may alter the thyroid status even after discontinuing them. GC can suppress thyroid function for 30-90 days and potentiated sulfas may suppress the thyroid for up to 30 days after discontinuing the drug

3. Pyoderma and/or Malassezia dermatitis
   a. Impression smears.

4. Dermatophytosis
   a. Woods lamp and fungal culture

5. Autoimmune (eg pemphigus foliaceus)

6. Atopic dermatitis (AD) – the diagnosis is a clinical diagnosis.
   a. Criteria that can help establish a diagnosis are
      i. Onset of signs under 3 years of age
      ii. Dog living mostly indoors
      iii. Glucocorticoid-responsive pruritus
      iv. Pruritus sine materia at onset (i.e. alesional pruritus)
      v. Affected front feet
      vi. Affected ear pinnae
      vii. Nonaffected ear margins
      viii. Nonaffected dorso-lumbar area
   b. If the dog meets 5 criteria- there is a specificity of 79% (21% false positives) and a sensitivity of 85% (15% false negative)
   c. If the dog meets 6 criteria- there is a specificity of 89% (11% false positives) but the and sensitivity decreases (more false negative) to 58% (42% false negative)
   d. How to use this data
      i. One minute atopic dermatitis test
         a. If you have a pruritic dog- you can be fairly certain that he/she has atopic dermatitis if you have ruled out ectoparasites and infection (bacterial, yeast/fungal). Serum testing/intradermal testing and/or food trials may be needed to ID flare factors but do not diagnose AD.

TREATMENT OF THE PRURITIC DOG

There are a variety of therapies for the symptomatic relief of pruritus, but before you do that you should think about addressing the underlying cause. You will be more effective in treating the pruritic patient if you find the “due to” rather than just treat the symptom (pruritus). If a specific diagnosis for the pruritus has not been established after the initial diagnostic tests have been performed and infection is present it is best to treat the infection for 14-21 days and then re-evaluate how much pruritus remains. DON'T use GC during this time since it would make interpretation of response to therapy impossible (was it the steroid or the antibiotic/antifungal therapy that resolved the pruritus?).
If the pruritus has resolved after only treating the secondary infections and/or ectoparasites it means that the ectoparasites or the secondary pyoderma/\textit{Malassezia} dermatitis was the major trigger of the pruritus at this time. This secondary infection was due to one (or more) of the following:

1. Ectoparasites
2. Seasonally triggered environmental allergen induced atopic dermatitis and the season has changed
3. Nonseasonally triggered environmental allergen induced atopic dermatitis that is not symptomatic when the infection is absent (threshold theory)
4. Atopic dermatitis that is triggered by a cutaneous food reaction- that is not symptomatic when infection is absent
5. An endocrinopathy (hypothyroidism, hyperadrenocorticism)

If a diagnosis has not been established through physical examination and laboratory testing and either there is not a secondary infection to begin with or it has been appropriately treated, the next step is a therapeutic ectoparasiticidal treatment (if not previously performed). Failing that, a food trial should be instituted. These later two treatments can be done simultaneously. If they are done simultaneously then if there is a positive response you can do a food “challenge” to determine which therapy was effective. By feeding the original diet and seeing the response you will be able to differential the underlying disease. A short course of GC at the beginning of the therapeutic trial may be done as long as you have eliminated the presence pyoderma, \textit{Malassezia}, demodex and dermatophytes.

At the end of these steps, if the pruritus has resolved w/o the concurrent administration of GC, you have identified your primary cause and can treat accordingly. If the dog has residual pruritus then the dog has environmental triggered atopic dermatitis.

\textbf{Treatment of canine atopic dermatitis}

Treating a dog w/AD is like eating at a buffet. You can whatever you want but not everything that is offered. If you don’t like broccoli, then don’t eat it- but if you are hungry after your meal- then you will have to eat the broccoli. This concept is also applies to treating AD- you can have whatever you want but not everything. By explaining this to the owner, they can then feel involved in the treatment plan for their dog. Let’s choose the most user friendly, safest approach and if this fails, then they have to choose another option. Treatment options for dogs with atopic dermatitis include - (please note that these therapies are used as a preventative so they should be instituted before clinical signs recur):

1. Good skin care
   a. Restore barrier function
   b. Wiping the dog off after coming in from outdoors
   c. Clipping the hair coat to a short length (10 or 15 blade) which helps to decrease exposure to and contact with environmental triggers (allergic and irritant).
   d. Clothing all the time and boots outdoors
   e. Bathing with a hypoallergenic veterinary shampoo that contain moisturizers or barrier repair ingredients (eg shampoo that contain phytosphingosine) weekly
      i. Dermalyte shampoo® - Dechra - contain moisturizers
   f. Follow the bath w/a humectant (eg Humilac® - Virbac) or barrier repair ingredient (Douxo’s phytosphingosine containing products- eg Seborrhea spray, Calm spray)
      i. In humans – best applied w/in 2 minutes after finishing the bath for maximum effect
   g. Topical moisturizers
      i. In humans – best applied w/in 2 minutes after finishing the bath for maximum effect
      ii. Allerderm spot on®- Virbac, Dermoscent Essential 6 Spot-On Skin Care®-Aventix or Douxo Seborrhea Spot-on® Sogeval are examples
   h. Fatty acid supplementation- try an omega 3 product for 3 months and if there is no improvement, try a product with a combination of an omega 3 and 6
      i. Omega 3
         1. 18 mg/kg of EPA daily
      ii. Omega 6/3- double the bottle dose OR
      iii. High fatty acid diets (eg J/D)
   i. Bathing is helpful to decrease antigen load and bacterial colonization
2. Identify and prevent/manage the triggers (ectoparasites, food, infection (bacterial/Malassezia))
   a. If the dog has environmental triggered atopic dermatitis, allergen specific immunotherapy is appropriate if the symptoms are present for more than 2 or 3 months/year and is severe enough to need corticosteroids or cyclosporine for symptomatic control.
   b. If the dog has a food trigger- avoid those foods
   c. Good flea control especially if the dog has flea bite hypersensitivity

3. During acute flares- treating infection and inflammation is necessary. Therapy would include antibiotics, antifungals and glucocorticoids along w/the above recommendations

4. Treatment options for symptomatic relief of dogs w/atopic dermatitis w/o secondary infection are
   a. Glucocorticoids
   b. mCSA
      a. Antihistamines/tricyclic antidepressants – there are a variety of antihistamines available that may help mildly pruritic dogs.

Summary from the ACVD task force on AD

Treatment of acute flares of canine atopic dermatitis

1. Identification and avoidance of flare factors:
   a. Identification and elimination, whenever possible, of allergenic flare factors (fleas, food and environmental allergens)
   b. Evaluation of use of antimicrobial therapy if clinical signs of infection or colonization with bacteria or yeast are present on the skin or in the ears

2. Improvement in skin and coat hygiene and care:
   a. Bathing with a nonirritating shampoo

3. Reduction of pruritus and skin lesions with pharmacological agents:
   a. Treatment with topical glucocorticoids, especially for localized lesions, as needed to control signs
   b. Treatment with oral glucocorticoids, especially for widespread or severe lesions, as needed to control signs

Treatment of chronic canine atopic dermatitis

1. Identification and avoidance of flare factors:
   a) Dietary restriction-provocation trials in dogs with nonseasonal signs
   b) Implementation of an effective flea control regimen in areas where fleas are present
   c) Performance of allergen-specific intradermal and/or IgE serological tests to identify possible environmental allergen flare factors
   d) Possible implementation of house dust mite control measures, if relevant and feasible
   e) Evaluation of use of antimicrobial therapy if signs of infection or colonization with bacteria or yeast are present on the skin or in the ears

2. Improvement in skin and coat hygiene and care:
   a) Bathing with a nonirritating shampoo or an antiseborrheic/antimicrobial shampoo, depending on the skin lesions seen
   b) Dietary supplementation with essential fatty acids

3. Reduction of pruritus and skin lesions with pharmacological agents:
   a) Treatment with topical glucocorticoids or tacrolimus, especially for localized lesions, as needed to control signs
   b) Treatment with oral glucocorticoids, cyclosporine or subcutaneous interferon, especially for widespread or severe lesions, as needed to control signs. These agents would not normally be combined together.
   c) Use of steroid-sparing agents, such as essential fatty acids, Chinese herbs and antihistamines, if glucocorticoids are being used as a long term treatment option.

4. Implementation of strategies to prevent recurrence of signs
   a) Avoidance of known flare factors, as identified above
b) Consideration of preventive pharmacotherapy, if feasible and relevant

c) Implementation of allergen-specific immunotherapy, if feasible. This can be used alongside all the above treatment options in an attempt to provide long term amelioration of the aberrant immune response
