Feline atopic dermatitis
What is similar to canine & human atopic dermatitis?

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Intro

✔ History
  ✔ Human
  ✔ Feline
  ✔ Canine
✔ Feline atopic disease
✔ Pathogenesis
✔ Epidemiology

✔ House dust mites
  ✔ background
  ✔ Clinical signs
✔ Diagnostics
✔ Treatment
✔ Future directions
House dust mites I

- *Dermatophagoides farinae (D.f.)*
- *Dermatophagoides pteronyssinus (D.p.)*
  - Allergens (HDMA):
    - *D.f. 1, D.f.2, D.f 15.....*
    - *D.p.1, D.p.2...*  
      
      (McCall et al Vet Immun & immunopathol.2001)

- Storage mites
  - *Tyrophagus putrescentiae (T.p.)*
  - *Acarus siro (A.s.)*

- Since 1964 important factor in human AD
  
  (H.Mosbech, Ugeskrift For Læger,1999 vol 161, p414-18)

- Canine since early 1970’s
- World wide present
- Humans more frequently AD/asthma with *D.p.*
- Dogs more AD with *D.f.*
- How is this in cats?
House dust mites II

- Color less small 0.3 mm long
- Feeds on dander from humans, dogs; cats?
- Optimum conditions 25 °C (77 F), 70~ RH%
- But they will adapt to low temperatures and can survive with lower RH%
- < 50 RH% w temp >20 °C (68 F)
- Immobilized at 4 °C
- Killed by washing at temperatures >55 °C (130 F)
Where in the homes?: fabric covered furniture, bedding = microenvironments where the RH% will provide water enough.

100 mites/gram of dust ~ 2-10 μg of group 1 HDMA = significantly risk for sensitizations in genetically predisposed individuals

Seasonal blooming in numbers of mites

HDMA still present after mites are dead!
Background

✓ Feline Atopic dermatitis
✓ 1st case of feline atopic dermatitis 1982
  (Reedy et al, JAAHA, 1982)
✓ Defined in 1989 by Halliwell as:
  (Vet. Clinical immunology p.232-52)

“An inherited predisposition to develop IgE antibodies to environmental allergens, resulting in allergic disease”
Feline atopic dermatitis

✓ Pruritic dermatologic disease
✓ No sex or breed predisposition?
✓ Age 6-24 months (6 months to 14 yrs)
✓ Major group of allergic cats
  (Scott, Miller & Kirk, p.602, P.J. Roosje et al Vet immun & Immunopathol. 2004)
  ✓ Non-seasonal (50-90%)
  ✓ Pollen allergies are rare

A clinical diagnosis!
Human atopic dermatitis
(atopic eczema)

“One definition”:
A genetic chronic relapsing inflammatory skin disease with a "Tendency to develop IgE antibodies to commonly encountered environmental allergens & subsequent of disease" frequently with elevated IgE levels.

- First described clinically in 1808
- In 1892 first association between hayfever & AD
- In 1923 Coca and Coke termed the term ATOPY & in 1939 the term Atopic dermatitis was coined to bring together all the group of atopic diseases.
- Type I hypersensitivity reaction (immediate)
- Considered “a genetic dysfunction of immune system”
  (V.S. Beltrani et al, Derm online J, Vol 9(2)2005)
- The exact pathogenesis still not clear........
Canine Atopic Dermatitis (cAD)

The ACVD Task Force on Canine Atopic Dermatitis, 2001

- Pruritic skin condition
- Relapsing dermatitis
  - Bacteria/Yeast
- HDM most common allergen
  (ACVD task force on cAD, 2001)
- Seasonal/non-seasonal
- Breed & familiar predilection

Definition: “A genetically-predisposed inflammatory & pruritic allergic skin disease with characteristic clinical features. It is associated most commonly with IgE antibodies to environmental allergens.”
Epidemiology of AD

**Canine**
- Approx. 10-15% of dog population
- Age: 4 months - 7 years (70% ≈ 1-3yr)
  - ↑ prevalence
  - More cases diagnosed?
- Genetic selection?
- More severe clinical manifestation?

**Feline**
- Incidence?
- Age: 6 months to 8 years
- Rare in older Fe.
- Increase in frequency?
- Increase in severity?

**Human**
- Children 10-20%
- Adults 1-3%
- Rare > 50 yr.
  - ↑ prevalence in developed countries
  - More severe clinical manifestation?
  - Hygiene hypothesis
Genetics and AD

- Genetic abnormalities reported in humans
- Little is known in dogs
- 3 cases of litter mates (K. Moriello, Vet Derm 2001)
- Abyssians (Bettenay, Proceeding AAVD, 1998)

**Canine AD**
- Familial history
- Strong breed predilection

**Human AD**
- Familial history
- Gene candidates

Not much is known in cats!
Pathogenesis: Inhalation Route
(allergic inhalant)

B-cell production of IgE

IgE migrate to tissue & bind to mast cells

Allergen is inhaled (ingested) & systemically absorbed

Slide modified after R. Marsalla
Pathogenesis:
Percutaneous route

Y IgE & mast cells

Green: Allergen

Blue: Activated mast cells

Allergen is captured by LC in the skin

B-cells produce IgE systemically?

Slide modified after R. Marsalla
TYPE I HYPERSENSIVITY

Allergen

IgE

FcεRI

Histamine

LT

IL-1, 2, 3, 4, 5, 13

IL-6, TNF-α

Blood vessel
Pathogenesis of AD?

The “new” theory:
- T cell imbalances
- Biphasic “switch” of T helper cells:
  - Th1/Th2 balance

The “old” theory:
- Type I hypersensitivity IgE, mast cells, histamine, leukotriens

- Hygiene hypothesis
- Lack of parasites?

![Diagram showing acute and chronic AD with IL-4, IL-5, IL-2, γ-IFN, Th-1, Th-2, Cd4+ cells]

Acute AD

Chronic AD
Role of IgE-

**Canine AD**

✓ Increased expression of surface bound IgE on LC in lesional skin (Olivry et al, 1996)

✓ Most dogs with AD have detectable serological allergen-specific IgE

**Human AD**

✓ LC capture allergen via their high affinity IgE receptor (FcεRI)

No difference between atopic and non atopic cats in a small study

SPF cats

✓ 15 spayed female non clinical AD cats
✓ 6 months old
✓ Sampled in mid September 2005
✓ HESKA tested for 24 allergens
✓ 6 cats positive for 1 or more allergens
✓ 1 Positive for fleas
✓ 14 positive allergens in 480 tested
✓ NO HDMA found in the cage room
Role of missing IgE?

Canine AD
✓ IgE do not correlate with severity of disease
✓ No difference in serum total IgE between normal and atopic dogs
(M.A. Fraser et al, vet rec. 2003 & Deboer et al ACVD task force)
✓ Negative IDT & serology in some dogs with clinical AD

Human AD
✓ Genetic inability to produce IgE may develop AD

Intrinsic AD
✓ 10-30% of humans with clinical AD do not have increased IgE levels
✓ Negative IDT/Patch test/serology (total & specific)
If this is the explanation then......

Why do we see negative IDT & serology testing in some dogs with clinical signs of AD?

- Wrong season? testing
- Allergens not included in the panel test?
- Heterogeneity of IgE
- (Suppressive “Drugs”) EFA, etc.

Why do we see positive IDT and serology testing in normal dogs with no signs of AD?

- IgE heterogeneity
- Yet to develop clinical signs
- Positive IDT and serology testing: a secondary criteria for diagnosis
# Clinical signs of AD

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Human</th>
<th>k-9</th>
<th>Fe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pruritus</td>
<td>++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Otitis</td>
<td>*</td>
<td>++</td>
<td>-</td>
</tr>
<tr>
<td>2° Alopecia</td>
<td>?</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>2° Erythema =&gt; lichenification</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Eosinophilic granuloma</td>
<td>?</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>2° pyoderma</td>
<td>+/-</td>
<td>+/-</td>
<td>-</td>
</tr>
<tr>
<td>Miliary dermatitis</td>
<td>-</td>
<td>-</td>
<td>++</td>
</tr>
<tr>
<td>Rhinitis/conjunctivitis</td>
<td>+</td>
<td>(-)</td>
<td>++/-</td>
</tr>
<tr>
<td>Asthma</td>
<td>+</td>
<td>-</td>
<td>+/-</td>
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</tbody>
</table>
Clinical picture Dermatitis

Canine

Feline

Human

Miliary dermatitis
Clinical picture: abdomen

Canine

Feline

Human

Photo: MSU

Photo: MSU

Photo: MSU
Clinical picture: Feet

Canine

Feline

Human

[Image of Canine Foot]

[Image of Feline Foot]

[Image of Human Foot]

Photo: KEL

Photo: MSU

www.e-medicine
Clinical signs: face

www.e-medicine.org

Photo: MSU

www.e-medicine.org

Arthur Nouel
Diagnostics

**Anamnesis**

- ✓ History
  - ✓ Familial?
- ✓ Clinical signs
  - ✓ +/- Non seasonal pruritus
  - ✓ Alopecia (symmetrical)
  - ✓ Miliary dermatitis
  - ✓ Eosinophilic granulomas
- ✓ Response to prednisolone
  (C.Graham-Mize, WCVD5, 2004)

**Rule out**

- ✓ Parasites
- ✓ Fungal
- ✓ Bacterial
- ✓ Neoplasia/paraneoplasia
- ✓ Viral?
- ✓ Cutaneous adverse food reaction
Diagnostic tests

**Intra dermal test (IDT)**

- Flourescein 10%
- 4.4 mg/kg iv read with woods lamp
- To enhance the reading of the IDT

**IgE Serology**

- (RAST, ELISA, Liquid phase)
- n=59 cats HDM AD
- Age 1-13 yr (5.1yrs)
- Compared IgE (D.f & D.p)
- Grouped (6) based clinical signs
- n.s. for both Group I & II IgE)
Treatment of feline AD

- Avoidance ??
- Prednisolone (Graham-Mize)
- Allergen Specific Immuno Therapy (ASIT)
- Antihistamine
- Essential Fatty Acid (EFA)
  - (F. Kristensen et al, WCVD 2004)
- Cyclosporine (CSA)
- (Megestrol acetate)
Summary

1. CONSENSUS on the definition of AD
2. Acceptance of terminology
3. Uniform testing materials
4. Better understanding of the interplay between the genetics, allergens & the mechanism of disease

✓ Who do we test?
✓ Why do we test?
✓ How do we test?
✓ Does it make a difference?

✓ Questions?
Literature

- É. Guaguère & p. Prélaud: A practical guide to Feline Dermatology, Merial, p10.1-10.8
- Scott, Miller & Kirk: Small animal dermatology, 6th ed. P.601-608
- C.R. Norris et al: Production of polyclonal antisera against Feline immunoglobulin E & its use in ELISA in cats with experimental induced Asthma
- Fitzpatrick’s dermatology in Gen. med. P.1180-