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A Systematic Clinical Approach To Handle Canine Pruritus To Combat Antimicrobial Resistance

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Abstract:

Canine pruritus is a multifaceted condition involving interactions between the nervous system, the skin barrier, and immune-driven inflammation. Key mediators such as IL-31 and various neuropeptides intensify itch signals and maintain the itch-scratch cycle, which further damages the skin and encourages secondary infections. This overview explains the major causes of pruritus, including parasites, infections, and allergies, and outlines a structured diagnostic process that prioritizes ruling out common, treatable conditions before investigating chronic allergic causes. Conventional management involving antibiotics and steroids needs thorough consideration, and novel regimen emphasizing precise therapies, such as cytokine-targeted drugs, alongside skin-barrier support and long-term, multimodal care to achieve sustained control of chronic itching must be adopted to help in combating antimicrobial resistance.

Keywords: Pruritus, Antimicrobial resistance, Pruritus algorithm, Etiotropic therapy, Canine

Introduction:

Pruritus (Itch) may be defined as an unpleasant sensation that provokes the desire to scratch. In dogs, it is often characterised by licking, intense scratching, rubbing, chewing and biting. It is not a disease but rather a clinical sign that may be linked to a complex etiology. Pruritus is the chief complaint of more than 30% of total canine dermatological consultations, which not only threatens the well-being of the animal but also drastically impacts the quality of life of the pet owner (Noli *et al*., 2011; Sousa, 2013). Skin is the largest organ of the animal body and has a complex physiology and composition. Pruritic skin has diminished ceramide levels, abnormal lipid organisation and increased transepidermal water loss, thereby hampering the skin barrier and making it susceptible to multiple secondary infections (Olivry, 2017). Thus, management of pruritus must include restoring skin health and not just suppressing itching (Idee *et al.*, 2022).

Many practitioners often prescribe antibiotics for any itch-associated dermatitis without thorough investigation of the underlying cause, and it can be a great risk factor for the emergence of antibiotic-resistant pathogens. Initiation of antibiotic usage started only in the 1940s; however, the recent emergence of antibiotic-resistant pathogens has surpassed the rate at which new drugs are discovered. The emergence of methicillin-resistant *Staphyllococcus pseudintermedius* associated with pyoderma and otitis in canines is alarming and is considered an emerging zoonotic pathogen of canine origin, which can cause fatal bacteremia and endocarditis in humans (Bunsow *et al.,* 2021). Thus, the systematic approach to handling a pruritic dog requires careful clinical evaluation, methodical diagnostic testing, and a thorough understanding of the mechanisms driving this complex sensory phenomenon before concluding with a therapeutic regimen.

Pathophysiology:

Pathophysiology of pruritus involves complex neural, inflammatory, and immunological mechanisms. Any agent (parasite, allergen or infection) acts as trigger to initiate an immune response to generate a pruritogenic stimulus. Specialized pruriceptors such as unmyelinated C-fibres and myelinated Aδ-fibers at the dermo-epidermal junction detect pruritogenic stimuli and transmit signals via dorsal root ganglia to the spinal cord and cerebral cortex (Nuttall *et al*., 2013). Multiple mediators induce itch. While histamine from mast cells is a recognized pruritogen, histamine-independent pathways are equally important in dogs. IL-31 produced by Th2 cells, directly stimulates sensory neurons and represents a critical pathway in allergic pruritus. Other mediators include proteases, neuropeptides (e.g. substance P, CGRP), leukotrienes, prostaglandins, and neurotrophins (Gonzales *et al*., 2016).

The "itch-scratch cycle" perpetuates pruritus through skin barrier disruption, inflammatory mediator release, and secondary infections. Chronic scratching causes epidermal hyperplasia, lichenification, and altered nerve fibre density, creating a self-sustaining cycle that persists beyond the initial trigger (Cevikbas & Lerner, 2020).

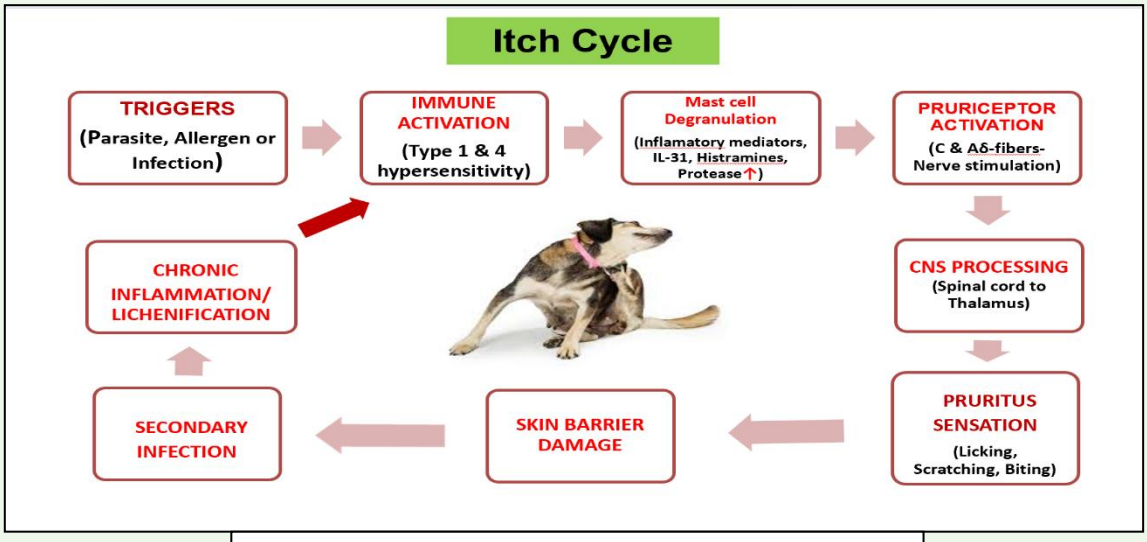

Fig. 1: Pathophysiology of pruritus — **The Itch Cycle**

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Etiology of Pruritus:

In dogs, pruritus may be commonly due to infestation (parasites), infection or allergy.

- **Parasitic Causes**: Ectoparasites are one of the leading causes of canine pruritus. Sarcoptic mange (*Sarcoptes scabiei*) causes severe pruritus through both mechanical irritation and allergic hypersensitivity to mite antigens. Flea allergy dermatitis (*Ctenocephalides canis*) results from a hypersensitivity reaction to salivary antigens injected during flea feeding, with even minimal flea exposure triggering intense pruritus in sensitized individuals. Other parasitic causes include *Cheyletiella* and *Otodectes* mites, ticks (*Rhipicephalus, Ixodes*), *Demodex spp*. (due to involvement of secondary bacterial infection), and various lice (*Trichodectes canis*, *Linognathus setosus*) (Sauve, 2023).
- **Infectious Causes**: Secondary bacterial and yeast infections frequently complicate underlying allergic skin diseases and contribute to pruritus severity. Staphylococcal pyoderma (*Staphylococcus pseudintermedius*), triggers pruritus through bacterial exotoxins, immune responses, and disruption of skin barrier function. Malassezia dermatitis (*Malassezia pachydermatitis*), resulting from overgrowth of commensal yeast, produces intense pruritus, particularly in warm, moist body regions (skin folds, armpits and ear). Dermatophytosis can cause variable pruritus depending on the degree of inflammation and individual host response (Olivry *et al*., 2017).
- **Allergic Causes**: Allergic dermatitis represents a major category of chronic pruritus. Canine atopic dermatitis (CAD) involves a genetic predisposition to develop IgE-mediated hypersensitivity reactions to multiple environmental allergens such as pollens, molds or house dust mites. The pathogenesis involves skin barrier dysfunction, allowing increased allergen penetration, combined with altered immune responses favouring Th2-type inflammation and IgE production. Cutaneous adverse food reactions (CARF) may involve either immunologic (true food allergy) or non-immunologic mechanisms, producing clinical signs indistinguishable from atopic dermatitis. Contact dermatitis, though less common, results from either irritant or allergic contact with environmental substances (Sousa, 2013).

Clinico-Diagnostic Approach to Pruritus:

Initial Assessment: The first step in approaching a pruritic dog involves obtaining a comprehensive history from the pet owner. An example with details is listed below-

Question/ Points	Significance
Signalment	**Breed**, age, and sex may be associated with certain disorders; Eg. CAD commonly affects breeds such as West Highland White Terriers, Labrador Retrievers, and German Shepherds, while Shar-Peis show increased predisposition to *Malassezia* dermatitis
Onset and Seasonality	Whether the itching was sudden or gradual, more in a particular season (suggestive of **atopy/environmental allergies**) or year-round (suggestive of **food allergy** or **non-seasonal parasite**).
Location	Ears, feet, underarms, and groin often suggest **atopy**; the genital, anal

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	and tail base are often associated with **flea allergy dermatitis**.
Response to Food/Medication	Recent changes in diet, deworming medications, etc, can rule out **infectious** causes
Severity	Canine Pruritus Severity Scale- on a scale of 1 to 10 (10 being the worst), how bad is the itch? Helps gauge the need for immediate pharmacological intervention.
History of other pets in the family or contact with other animals	Suggestive of a **contagious** cause like mites or fleas.

(Rybnicek *et al*., 2009; Tater, 2012)

Clinical Examination:

The examination should be done in a well-lit room with appropriate equipment for observation. A thorough clinical examination begins with **observing the animal's behaviour** and documenting any clinical signs involved- like scratching, licking, rubbing or biting. Special attention should be given to interdigital spaces, ear canals, axillae, inguinal regions, and mucocutaneous junctions, as these areas are commonly affected in allergic and parasitic conditions.

Then, the skin should be systematically examined for **gross primary and secondary lesions**. Primary lesions, such as papules, pustules, wheals, or vesicles, provide crucial diagnostic information about the underlying pathology, while secondary lesions, like excoriations, lichenification, hyperpigmentation, and alopecia, result from self-trauma or chronic pruritus. Further, **skin cytology** examination- scrapings may reveal the presence of ectoparasites; skin swabs/smear- infections (bacterial or fungal agents).

Diagnosis through the Pruritus Algorithm:

The algorithm is designed to systematically eliminate differential diagnoses (Steinhoff *et al*., 2011). The algorithm begins with excluding parasitic causes, then addressing infectious complications, and finally investigating allergic and other etiologies of pruritus (Miller *et al*., 2023).

- **Phase 1: Ruling out Parasites**

  Fleas and mites are the priority, as they are often easily treated. A single flea bite can trigger a severe allergic reaction (Flea Allergy Dermatitis), hence, even in the absence of visible fleas, a therapeutic trial with effective ectoparasiticides is often warranted. Sarcoptic mange should be considered in any intensely pruritic dog, particularly when the face, ear margins, elbows, and hocks are affected. A deep skin scraping or hair pluck may be suggestive to look for other mites microscopically.

- **Phase 2: Identification and Treatment of Infectious agents**

  Staphylococcus pseudintermedius or *Malassezia* spp. can be identified by cytological examination utilizing tape strips, direct impression smears, or cotton swab samples stained with Diff-Quik or Gram stain. Since the production of pruritogenic mediators from these secondary infections greatly increases the intensity of itching, treatment is necessary with appropriate drugs to establish proper control of pruritus.

- **Phase 3: Investigation and addressing of Allergies**

  Once parasites and infections have been addressed or ruled out, allergic pruritus becomes the primary consideration.

  → **Contact Dermatitis:** History of types of disinfectants, floor cleaners, soaps or shampoos used should be thoroughly assessed. Changing to a pet-friendly composition can address this form of pruritus.

  → **Food Allergy:** This requires a strict, 8-12 week Elimination Diet Trial using a novel protein or hydrolysed protein diet, which is the only scientifically validated way to diagnose a food allergy at present.

  → **Environmental Allergy (Atopy):** If food allergy is ruled out, the dog is likely suffering from atopic dermatitis, reacting to pollens, dust mites, or moulds. Management involves a multimodal approach combining barrier support, immunotherapy, and systemic anti-pruritic medications.

Therapeutic Strategy:

Treatment must address the underlying cause as well as provide symptomatic relief simultaneously. Conventional management of pruritus relied heavily on antihistamines, steroids and often along with broad-spectrum antibiotics. However, recent studies discouraged the existing trend of itch control in pets (Moriello, 2018). Thereupon, according to Olivry (2017) and Bruet *et al.* (2022), a modern therapeutic approach should be precise and must incorporate the strategies given below:

1. **Strict ectoparasite control:** Proper grooming and management; appropriate ectoparasitic drugs- for FAD all animals in contact should be treated, although there are no visible signs of fleas.
2. **Antimicrobial therapy:** For itch associated only with secondary bacterial or fungal infections. Generally indicated for deep pyoderma.
3. **Elimination diet trials:** If food allergy is suspected
4. **Targeted anti-pruritic medications:** Novel drugs such as oclacitinib (JAK inhibitor) or lokivetmab (anti-IL-31 monoclonal antibody) that specifically interrupt pruritus pathways.
5. **Allergen-specific immunotherapy:** For confirmed atopic dermatitis
6. **Topical therapy:** To restore skin barrier function. Due to increasing risk of antimicrobial resistance, topical formulations of herbal origin or preparations containing antiseptics like chlorhexidine must be considered rather than antibiotic therapy for mild infections. Moreover, systemic antibiotics must be reserved for only deep pyoderma or recurring causes.
7. **Long-term management:** Nutraceuticals containing essential fatty acids, particularly omega-3 fatty acids, are used to help strengthen the lipid barrier of the skin, making it less permeable to allergens and improving coat quality in long-term management of pruritus. Hypo-allergenic diets (eg, Hill's Prescription- Derm Complete) are recommended for most chronic pruritus associated with food and

environmental allergens.

Conclusion:

Approaching a pruritic dog requires keen observation, patience, and understanding the intricate pathophysiology of pruritus and its diverse etiologies. It is vital for owners to understand that chronic pruritus is rarely cured; rather, it is managed. By consistently providing accurate historical data, adhering strictly to diagnostic plans and committing to long-term multimodal therapy, owners can significantly break the debilitating itch-scratch cycle and ensure their furry companion enjoys a comfortable, itch-free life. And at the same time, clinicians should adhere strictly to mandatory systematic diagnosis and devise only an etiotropic therapeutic regimen. This can significantly curb the traditional reliance on antibiotic drugs and ultimately help in combating the menace of antimicrobial resistance, and promote global public health.

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