

## Feline Hyperesthesia Syndrome: Current Concepts in Pathophysiology - A short inside

### Sindromul de hiperestezie felină: concepte actuale în fiziopatologie - O scurtă prezentare

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#### Abstract

Feline hyperesthesia syndrome (FHS) is a poorly understood clinical condition characterized by episodic cutaneous hypersensitivity, dorso-lumbar skin rippling, agitation, excessive grooming, tail chasing, self-directed aggression, and paroxysmal behavioral episodes. Despite long-standing recognition, its pathophysiology remains unresolved, and no single explanatory model accounts for the full clinical spectrum. Proposed mechanisms include neurologic hyperexcitability, seizure-spectrum phenomena, neuropathic pain or sensory dysregulation, dermatologic triggers, compulsive-spectrum behaviors, and stress-related modulation. Individual clinical signs may also occur in healthy cats when isolated, underscoring the importance of evaluating overall behavioral patterns rather than single features. This review synthesizes current evidence into a mechanistic framework in which FHS is a multisystem syndrome likely reflecting variable contributions across neurologic, sensory, dermatologic, behavioral, and environmental domains. This heterogeneity may explain diagnostic variability and the inconsistent therapeutic responses reported with anticonvulsant, analgesic, dermatologic, and behavioral interventions).

#### Rezumat

Sindromul de hiperestezie felină (HFS) este o afecțiune clinică puțin înțeleasă, caracterizată prin hipersensibilitate cutanată episodică, ondulații dorso-lombare ale pielii, agitație, curățare excesivă, alergare după cozi, agresivitate autodirijată și episoade comportamentale paroxistice. În ciuda recunoașterii de lungă durată, fiziopatologia sa rămâne nerezolvată și niciun model explicativ unic nu explică întregul spectru clinic. Mecanismele propuse includ hiperexcitabilitatea neurologică, fenomenele din spectrul convulsiilor, durerea neuropatică sau disreglarea senzorială, factorii declanșatori dermatologici, comportamentele din spectrul compulsiv și modularea legată de stress. Semnele clinice individuale pot apărea și la pisicile sănătoase atunci când sunt izolate, subliniind importanța evaluării tiparelor comportamentale generale, mai degrabă decât a caracteristicilor individuale. Această analiză sintetizează dovezile actuale într-un cadru mecanistic în care FHS este un sindrom multisistemic care reflectă probabil contribuții variabile în domeniile neurologic, senzorial, dermatologic, comportamental și de mediu. Această heterogenitate poate explica variabilitatea diagnostică și răspunsurile terapeutice inconsistente raportate în cazul intervențiilor anticonvulsivante, analgezice, dermatologice și comportamentale.

#### Introduction

Feline hyperesthesia syndrome (FHS) is an episodic disorder characterized by cutaneous

hypersensitivity, dorso-lumbar skin rippling, sudden grooming or self-trauma, vocalization, agitation, and exaggerated response to touch

(Tuttle, 1980; Cornell Feline Health Center, 2021).

Despite decades of clinical recognition, its underlying mechanisms remain uncertain (Rusbridge, 2021).

Reported hypotheses include neurologic or seizure-like processes (Gaskell et al., 1977; Gómez Álvarez & Soler Arias, 2021), dermatologic or pain-related triggers (Ruiz-Suárez et al., 2021; Rusbridge, 2024), and behavioral or stress-associated contributions (Beaver, 1989; Ciribassi, 2009).

A major clinical challenge is the absence of definitive laboratory, imaging, or histopathologic markers, requiring diagnosis through pattern recognition and exclusion of alternative conditions (Cornell Feline Health Center, 2021).

Many features associated with FHS, including grooming and lumbar sensitivity, also occur in clinically normal cats, contributing to diagnostic ambiguity (Avril et al., 2025).

The lack of a consensus framework limits diagnostic consistency and contributes to variability in clinical phenotyping and case classification.

This review organizes current pathophysiologic concepts into a structured framework encompassing neurologic, sensory, dermatologic, behavioral, and stress-related domains (Gaskell et al., 1977; Finnerup et al., 2021; Ciribassi, 2009; Pauciulo et al., 2025), providing an integrated interpretation of observed clinical variability.

### **Clinical phenotype relevant to pathophysiology**

The clinical phenotype of FHS includes dorso-lumbar skin rippling, sudden agitation or running, exaggerated responses to palpation, tail chasing, excessive grooming, self-trauma, and episodic vocalization (Tuttle, 1980; Cornell Feline Health Center, 2021).

These features overlap with recognized indicators of pain in cats, necessitating careful exclusion of underlying painful conditions (Merola & Mills, 2016; Steagall & Monteiro, 2019; Evangelista et al., 2019). The paroxysmal and clustered nature of episodes warrants

consideration of neurologic, pain-related, and behavioral differentials.

Clinical signs frequently localize to the dorso-lumbar region and tail base (Amengual Batle et al., 2019; Rusbridge, 2024).

Importantly, similar behaviors may occur in healthy cats, limiting diagnostic specificity (Avril et al., 2025).

Thus, FHS is best defined by a pattern-based diagnosis following exclusion of mimics (Cornell Feline Health Center, 2021).

### **Current concepts in pathophysiology**

The literature describes several partially explanatory frameworks addressing different aspects of the FHS presentation, although none is sufficient to account for the full clinical spectrum (Amengual Batle et al., 2019; Rusbridge, 2021).

Reported presentations include neurologic-like, pain-associated, dermatologic, and behavioral phenotypes (Gaskell et al., 1977; Ruiz-Suárez et al., 2021; Ciribassi, 2009; Pauciulo et al., 2025).

The following subsections examine these proposed models individually.

### **Neurologic / seizure-spectrum hypothesis**

FHS has been hypothesized to involve focal neurologic dysfunction or seizure-spectrum activity, based on its paroxysmal onset, abrupt termination, and repetitive motor phenomena resembling focal seizures (Gaskell et al., 1977; Gómez Álvarez & Soler Arias, 2021).

Some clinicians classify it within a seizure-related spectrum, and anticonvulsants are occasionally used (Cornell Feline Health Center, 2021). A retrospective case series reported clinical improvement with anticonvulsants or gabapentin in some cats, although no standardized protocols or electroencephalographic confirmation were available (Amengual Batle et al., 2019).

These findings suggest neuronal hyperexcitability may contribute in a subset of cases, but do not support a universal epileptic mechanism.

### **Neuropathic pain/sensory dysregulation hypothesis**

Clinical observations of exaggerated tactile responses and dorso-lumbar sensitivity support a role for altered nociceptive processing (Merola & Mills, 2016; Cornell Feline Health Center, 2021).

These signs overlap substantially with established behavioral indicators of feline pain, including grooming changes, agitation, and aversion to palpation (Steagall & Monteiro, 2019).

Support for a sensory dysregulation component is further suggested by reported remission in cats treated with gabapentin in a retrospective series (Amengual Batle et al., 2019).

Gabapentin modulates voltage-gated calcium channels and reduces excitatory neurotransmission, mechanisms implicated in neuropathic pain and central sensitization (Finnerup et al., 2021; Rusbridge, 2024).

Neuroimmune signaling via chemokine-mediated neuron–glia interactions may further modulate nociceptive amplification, providing a plausible link between peripheral inflammations and altered sensory processing (White et al., 2007).

While these observations support a sensory dysregulation component in some cases, they do not confirm this as the universal mechanism for all FHS presentations.

### **Dermatologic trigger hypothesis**

Dermatologic disease may act as both a mimic and a potential trigger of FHS-like signs.

A case report described chronic superficial perivascular dermatitis associated with FHS-like clinical signs (Ruiz-Suárez et al., 2021).

Itch and pain share overlapping neuroanatomical pathways, including shared spinal processing and central representation (Finnerup et al., 2021). Accordingly, pruritic or inflammatory skin disease may produce behavioral signs resembling FHS.

Dermatologic evaluation is therefore essential, including exclusion of allergic,

parasitic, infectious, and traumatic conditions (Robertson et al., 2022).

However, this single case provides limited evidence and does not establish causality, though it suggests a possible association between cutaneous inflammation and sensory–behavioral expression.

### **Behavioral/compulsive-spectrum hypothesis**

FHS has been associated with repetitive grooming, tail chasing, and self-directed behaviors overlapping with compulsive-spectrum disorders (Overall, 1997; Landsberg et al., 2013).

These conditions involve repetitive behaviors arising from affective dysregulation, frustration, or impaired coping mechanisms (Overall, 1997; Heath, 2018).

Behavioral interventions and pharmacologic therapy may improve selected cases (Ciribassi, 2009), and multimodal management has shown benefit in recent studies (Pauciulo et al., 2025).

However, FHS cannot be classified as a primary compulsive disorder based on current evidence.

### **Stress and environmental modulation**

Stress is widely considered a modulating factor that can exacerbate clinical expression in predisposed cats.

Environmental instability, conflict, and reduced coping opportunities may lower thresholds for episodic manifestations (Heath, 2018; Pauciulo et al., 2025).

Clinical reports support improvement following environmental modification and behavioral stabilization (Pauciulo et al., 2025).

Consequently, stress is best conceptualized as a modulating amplifier that exacerbates expression in predisposed individuals, rather than as an independent etiologic factor.

### **An integrated mechanistic model**

FHS can be conceptualized as a clinical condition in which multiple neurologic, sensory,

dermatologic, behavioral, and environmental domains contribute variably among individual cases (Amengual Batle et al., 2019; Rusbridge, 2021).

Accordingly, clinical presentation reflects domain-specific influences rather than a single unified mechanism.

Predisposing influences may include individual temperament, stress sensitivity, and variation in sensory reactivity (Heath, 2018; Avril et al., 2025).

Triggering factors may include tactile stimulation, pain, inflammation, environmental disruption, or social stressors (Pauciulo et al., 2025).

Depending on the dominant mechanism, clinical expression may resemble neurologic hyperexcitability, pain-associated behaviors, compulsive motor patterns, or stress-related arousal states (Gaskell et al., 1977; Finnerup et al., 2021; Ciribassi, 2009; Rusbridge, 2024).

This results in overlapping clinical presentations driven by variable underlying mechanisms - a shared phenotype arising from diverse causation.

### Implications for diagnosis and treatment

Given its heterogeneity, FHS should not be managed as a single disease entity (Amengual Batle et al., 2019; Rusbridge, 2021).

Diagnostic evaluation should prioritize exclusion of dermatologic disease, musculoskeletal pain, trauma, parasitism, allergic disease, and other identifiable causes (Robertson et al., 2022; Cornell Feline Health Center, 2021).

Treatment should be mechanism-guided.

Sensory-predominant cases may respond to gabapentin-based therapy (Finnerup et al., 2021), dermatologic cases require targeted skin treatment (Ruiz-Suárez et al., 2021), and behavioral or stress-linked cases may benefit from environmental and pharmacologic intervention (Ciribassi, 2009; Pauciulo et al., 2025).

Combined multimodal approaches are appropriate when overlap exists.

This mechanism-guided approach provides a practical framework for current

clinical management, though prospective validation is needed.

### Knowledge gaps and future directions

Three key gaps persist:

1. definition heterogeneity due to lack of standardized diagnostic criteria,
2. evidence limitations due to reliance on case reports and small series, and
3. methodological constraints due to absence of standardized prospective phenotyping frameworks (Rusbridge, 2021; Amengual Batle et al., 2019).

Future studies should prioritize standardized case definitions and integrated phenotyping incorporating behavioral, dermatologic, pain, and neurologic assessments to improve classification and mechanistic clarity (Finnerup et al., 2021; Robertson et al., 2022).

Prospective treatment trials stratified by mechanistic subtype, long-term cohort studies to establish natural history and sustained efficacy, and investigation of potential genetic or breed-related risk factors would all strengthen evidence for mechanism-guided therapy and clarify whether FHS represents distinct sub disorders.

### Conclusion

Feline hyperesthesia syndrome (FHS) remains an incompletely characterized clinical syndrome without a single unifying pathophysiological mechanism.

This review supports interpreting FHS as a heterogeneous syndrome arising from variable contributions across neurologic, sensory, dermatologic, behavioral, and environmental domains.

Clinically, management should be guided by phenotypic stratification - identifying the predominant contributing domain in each case to direct targeted diagnostic evaluation and multimodal therapy.

Future work should prioritize standardized diagnostic criteria and prospective multidimensional phenotyping to refine case

definition and improve mechanistic understanding.

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