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When Problem Behaviour Is Also a Clinical Signal: An Integrated Veterinary Framework for Distinguishing Pain, Fear, Learned Responses, Environmental Conflict, Neurological Dysfunction, and Underlying Medical Disease

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ABSTRACT

Problem behaviour in veterinary patients is often interpreted through a behavioural label before competing clinical explanations have been adequately separated. This is problematic because similar observable phenotypes can arise from pain, fear or anxiety, learned contingencies, environmental mismatch, social or handling conflict, neurological dysfunction, endocrine or other medical disease, or several causes acting concurrently. This article develops an original non-empirical clinical-behaviour framework for differentiating these possibilities without assuming that behavioural topography identifies aetiology. The synthesis treats history, direct observation, clinical examination, context change, diagnostic testing, and response to intervention as distinct evidence streams whose meaning depends on species, age, temporal pattern, setting, and measurement quality. Pain-associated behavioural change is separated from primary fear; learned maintenance is distinguished from initiating causes; environmental and handling effects are treated as context-dependent contributors rather than default diagnoses; and neurological or medical explanations remain active when temporal or clinical evidence warrants them. The principal contribution is a proposed behaviour–medicine differentiation architecture in which diagnostic confidence increases through convergence across independent evidence streams and is reduced by discordance, masking, observer effects, or context specificity. Therapeutic response is treated as evidence of modifiability rather than proof of original cause. The framework is intended to improve clinical reasoning and welfare-sensitive reassessment, not to provide a validated diagnostic score. Its principal limitations are evidence heterogeneity, concentration on companion dogs and cats, and absence of prospective external validation of the integrated architecture.

Keywords: Veterinary behaviour, Pain, Fear, Environmental mismatch, Neurological dysfunction, Clinical reasoning

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Introduction

Problem behaviour is clinically consequential because the same outward sign can sit at the intersection of behavioural medicine and physical disease. Reviews of dogs and cats describe pain, endocrine disorders, neurological disease, and other medical conditions as potential causes or modifiers of behavioural change, including when the medical contribution is not immediately obvious [1]. Treating the behaviour itself as the diagnosis can therefore prematurely narrow the differential. The central problem is not whether a sign is “medical”

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or “behavioural,” but how strongly available evidence supports one explanation, several interacting explanations, or continued uncertainty.

Behavioural topographies also need not be independent. In young dogs, clusters of apparently different problems have been interpreted as reflecting shared emotional commonalities, illustrating that several signs may be different expressions of a broader state rather than separate disorders [2]. Reactive and aggressive manifestations likewise covary with fear/anxiety and caregiver-related factors, showing that context surrounding the animal contributes information that a behaviour label alone cannot contain [3].

Medical attribution can be equally overconfident. In dogs with confirmed spontaneous hypothyroidism, levothyroxine increased activity at an early reassessment but did not produce broad significant behavioural change at six months [4]. This article therefore proposes a phenotype-first, evidence-convergence approach that keeps pain, fear, learning, environment, social handling, neurological dysfunction, and other medical disease simultaneously available until history, examination, testing, contextual pattern, and longitudinal response justify changing confidence.

Behavioral presentation as phenotype rather than diagnosis

A behavioural presentation is best treated initially as a phenotype: an observable pattern requiring description before aetiological interpretation. Medical conditions can alter behaviour directly or indirectly, so an apparently familiar pattern such as withdrawal, irritability, aggression, house-soiling, altered activity, or avoidance should not itself terminate diagnostic reasoning [1]. The clinical task is to specify what occurs, when it occurs, what precedes it, how long it lasts, what consequences follow, and whether the pattern changes across people, places, activities, or physiological states.

This descriptive separation matters because different surface problems can share emotional structure [2]. A cluster does not establish a diagnosis, but it warns against assuming that each topography maps uniquely to a cause. Caregiver observations also carry information about contexts unavailable during consultation, while caregiver characteristics and fear/anxiety can covary with reported reactive behaviour [3]. The proposed rule is therefore to treat caregiver history, direct observation, physical examination, and later test or treatment responses as separate evidence sources. Concordance raises confidence; discordance should reopen alternatives rather than be forced into the initial label. This phenotype-first rule is a proposed reasoning device, not a validated diagnostic classifier.

Pain-associated behavioral change

Pain can change what an animal does, avoids, tolerates, or appears to fear. In dogs with chronic musculoskeletal pain, multidomain welfare assessment identified differences extending beyond mobility into psychological, environmental, and procedural factors, with pain severity associated with selected mobility and fearful-stimulus measures [5]. These findings support pain as a competing explanation when new avoidance, reduced activity, irritability, handling resistance, altered posture, or context-specific reluctance develops alongside plausible painful disease.

Observation alone, however, is not specific. In a small acute-pain dog sample, qualitative behavioural assessment correlated with an established clinical pain scale, suggesting that integrated behavioural expression can add clinically relevant information when triangulated with structured assessment [6]. In cats, an expert-consensus ethogram retained acute-pain-relevant behaviours across posture, activity, affective-emotional expression, vocalisation, and other categories [7]. The breadth of these repertoires is precisely why a single behaviour should not be treated as pathognomonic.

The proposed distinction is between pain-compatible behaviour and pain-supported interpretation. Confidence should rise when behavioural change is temporally consistent with painful disease and converges with examination or structured pain assessment. Fear, sedation, illness, prior learning, and handling context remain alternatives when those signals do not converge.

Fear and anxiety

Fear and anxiety describe affective states, not their origin. In companion dogs, fear/anxiety is associated with reactive and aggressive manifestations, but the available cross-sectional evidence does not establish whether fear is primary, learned, pain-associated, environmentally elicited, or secondary to another condition [3]. A clinically useful formulation therefore separates evidence that an animal is fearful from evidence about why that state developed and what currently maintains it.

Pain is an important competing explanation because chronic painful states can affect psychological and behavioural welfare domains as well as mobility [5]. Acute pain can also alter integrated emotional expression observed by assessors [6]. These findings do not make fear-like behaviour a pain diagnostic; rather, they show that affective appearance and nociceptive state can overlap.

The proposed clinical question is consequently not "Is this fear or pain?" as a forced binary, but "What evidence supports fear, what evidence supports pain, and can both be operating?" Context specificity, onset after a painful event, altered tolerance of touch or movement, and persistence after appropriate pain management can each change confidence. Primary fear becomes more plausible when fear patterns generalise coherently without corresponding medical evidence, whereas pain or other disease remains active when clinical findings, temporal coupling, or domain-specific change point elsewhere.

Learned and reinforced responses

Learned behaviour can persist after the condition that initiated it has changed. Training and behaviour-modification evidence therefore matters to differential reasoning, but it should not be mistaken for proof of original cause. A review of aversive-based training found associations with adverse welfare-related outcomes while also emphasising substantial methodological heterogeneity in the evidence base [8]. This supports attention to reinforcement and punishment history while discouraging retrospective certainty based only on the current sign. Longitudinal Generation Pup data further show that owner-reported training approaches vary with caregiver and training-context factors over time [9]. A useful history should therefore ask not only what the animal does now, but what happened immediately before and after the behaviour, how people responded, which consequences were repeated, and whether those contingencies changed.

Counterconditioning studies provide a second caution. A recent systematic review found promising outcomes for some companion-dog problems but substantial heterogeneity in intervention definitions, samples, and outcomes [10]. Improvement during a learning-based intervention can demonstrate modifiability without establishing that learning was the sole initiating cause. The proposed framework therefore distinguishes an initiating trigger from a maintaining contingency: pain, fear, or conflict may initiate a response that later becomes partly maintained by predictable avoidance, attention, escape, or other consequences.

Environmental mismatch

Environmental mismatch occurs when an animal's surroundings, opportunities, predictability, or demands do not align with relevant behavioural needs or coping capacity. It is not a diagnosis and should not become a default explanation for every context-linked problem. In cats, provision of interactive play is associated with caregiver capability and opportunity, showing that species-relevant behavioural opportunities can be constrained by the human environment rather than by an intrinsic disorder alone [11].

Population-level dog data similarly show that aggressive behaviour covaries with multiple demographic, environmental, and behavioural factors [12]. Such associations identify plausible modifiers, not deterministic patient-level causes. Environmental inquiry should therefore examine opportunities for species-typical activity, rest and retreat, predictability, social density, stimulation, resource access, and whether the problematic behaviour changes when those conditions change.

The immediate handling environment can also produce behaviour. In a randomised study of shelter dogs, more restrictive examination restraints elicited more negative behavioural responses and higher fear scores than passive restraint [13]. A context-specific sign may therefore reflect situational conflict rather than a stable trait. The proposed adjudication principle is to ask whether altering the environment changes the phenotype while medically relevant alternatives remain under consideration.

Table 1 treats environmental change as a hypothesis test: a reproducible contextual response can increase plausibility while pain, fear, learning, neurological dysfunction, and medical disease remain open.

Table 1. Environmental hypotheses tested by controlled changes in opportunity, loading, handling, and context

Contextual pattern	Environmental contrast or change	Finding that increases contextual plausibility	Causal claim still not justified
Restricted species-relevant opportunity	Provide feasible play, exploration, retreat, or resource access	The phenotype attenuates when the opportunity becomes usable	Environmental restriction need not be the sole initiating cause

Environmental loading around aggression	Compare the behavior across social or environmental configurations	Expression changes reproducibly with the loading condition	Population associations cannot diagnose the individual animal
Restrictive examination conflict	Use lower-conflict handling or passive restraint when clinically safe	Fear, avoidance, or escape decreases under reduced restraint	Improvement does not exclude pain, prior learning, or generalized fear
Context-responsive phenotype	Recreate safe contrasting contexts while holding other factors as stable as possible	Emergence or attenuation is repeatable across contexts	Context sensitivity demonstrates contribution, not etiological closure

Social and handling-related conflict

Behaviour directed toward people is easily overinterpreted as a stable trait when it may instead reflect conflict between the animal, the interaction, and the way that interaction is conducted. Restrictive handling can acutely increase avoidance, escape, and fear-related behaviour during veterinary examination [13]. The clinically relevant distinction is therefore between behaviour generalised across contexts and behaviour that emerges mainly when movement, restraint, proximity, or loss of control reaches a particular threshold.

History is essential because caregiver and training practices change the contingencies surrounding human contact. Longitudinal evidence shows that training approaches vary with caregiver and training-setting factors [9]. Current defensive behaviour cannot reconstruct those past exposures with certainty, but history can identify plausible learned expectations and repeated triggers.

Reactive and aggressive manifestations also covary with fear/anxiety and caregiver-related factors [3]. These associations should not be converted into caregiver blame or a single psychosocial diagnosis. The proposed approach is to compare the phenotype across handlers, locations, restraint levels, and predictable versus unexpected contact while concurrently assessing pain and medical contributors when indicated. Improvement with lower-conflict handling supports context sensitivity; persistence across contexts, or emergence with touch and movement in medically plausible patterns, keeps broader fear, pain, neurological, and medical explanations active.

Neurological dysfunction

Neurological disease can alter behaviour through cognition, perception, motor function, episodic events, or changes in arousal, but behavioural signs alone rarely specify the neurological process. In older dogs, standard canine cognitive-dysfunction screening questionnaires correlate with one another yet are not identical, and behavioural dimensions including fear and pain sensitivity can covary with cognitive scores [14]. Screening can therefore organise suspicion without replacing neurological, sensory, medical, and pain assessment.

Temporal structure is particularly important in epilepsy. Behavioural phenomena may occur interictally, before or after seizures, during prodromal periods, or in patterns that could otherwise be interpreted as primary fear or aggression [15]. A timeline anchored to episodes, recovery, medication changes, sleep, and altered awareness can therefore carry diagnostic information that a cross-sectional behavioural label cannot.

Evidence around canine cognitive dysfunction also illustrates the cost of nonstandard case definitions. A scoping review of non-pharmacological interventions found substantial heterogeneity in inclusion criteria, testing methods, dog populations, and outcome approaches [16]. The proposed framework consequently treats cognitive scales, neurological history, direct examination, caregiver observations, and response over time as converging but non-equivalent evidence streams. Neurological confidence should increase when temporal, cognitive, sensory, motor, or episodic findings cohere; uncertainty should remain visible when those signals conflict.

Endocrine, metabolic, and other medical causes

Endocrine, metabolic, and systemic disease can alter activity, irritability, social tolerance, elimination, and responsiveness, yet these changes are rarely etiologically specific. The veterinary task is therefore not to attach a medical label to nonspecific behaviour, but to decide whether signalment, history, physical examination, or concurrent physiological signs justify targeted investigation. Reviews of canine and feline behavioural medicine support retaining endocrine, neurological, painful, and other medical disorders within the differential when the temporal pattern and clinical context are compatible [1].

At the same time, the presence of disease does not establish that it explains the behavioural phenotype. In dogs with confirmed hypothyroidism, levothyroxine increased activity at an early reassessment but did not produce significant broad behavioural change at six months [4]. This discordance is clinically informative: successful treatment of a medical abnormality may leave learned, fear-related, environmental, or other maintaining processes intact. The proposed framework therefore treats medical findings as weighted evidence rather than causal closure. Testing should be guided by plausible clinical indications, and persistent behaviour after appropriate disease management should trigger reassessment of competing or concurrent explanations rather than escalation of the original medical hypothesis.

Multiple concurrent causes

Single-cause explanations are often attractive because they simplify treatment, but behavioural phenotypes can emerge from overlapping vulnerabilities, triggers, and maintaining conditions. In a large dog dataset, repetitive behaviour was associated with aggression, ADHD-like traits, and environmental factors, illustrating that behavioural comorbidity and contextual correlates can coexist [17]. Such population associations do not show that all associated factors are causal in one patient, but they argue against forcing complex presentations into mutually exclusive categories.

Clinical burden can also cross conventional behaviour–medicine boundaries. Dogs classified with hypersensitivity-hyperactivity syndrome and their owners showed poorer reported well-being and additional health or burden indicators than matched controls, although the small cross-sectional design does not establish causal direction [18]. Likewise, a nine-dog uncontrolled pilot in drug-resistant epilepsy reported behavioural changes after fecal microbiota transplantation, but the design is appropriately interpreted as hypothesis-generating rather than mechanistic confirmation [19].

The proposed framework therefore distinguishes initiating causes, amplifying conditions, and maintaining processes. Pain may initiate defensive avoidance; repeated escape may reinforce it; restrictive handling may amplify it; neurological disease may alter thresholds; and caregiver responses may change its persistence. These roles can coexist without having equal evidential weight.

Table 2 separates causal roles across the timeline so that persistence after treatment is not mistaken for proof that the initiating cause remains active.

Table 2. Concurrent causes separated by their roles as initiators, amplifiers, and maintainers

Analytical question	Initiating cause	Amplifying condition	Maintaining process
Position in the timeline	Precedes the first recognizable phenotype	Changes severity or probability after the phenotype exists	Sustains the response after the original trigger has changed
Examples available in the framework	Pain, primary fear, neurological dysfunction, or other medical disease	Restrictive handling, environmental conflict, social conditions, or arousal	Escape, attention, avoidance, or other repeated learned contingencies
Evidence pattern	Onset is temporally and clinically coherent with the candidate cause	Severity varies with the amplifying context	Behavior persists in the presence of stable, repeatable consequences
Why one intervention may produce only partial change	The initiator can resolve while another role remains active	Removing the amplifier reduces severity without treating the primary cause	Learning-based treatment can change persistence without identifying the original cause
Reassessment focus	Did the targeted underlying domain change?	Did the phenotype change when the context was reduced?	Did the behavior change gradually with altered contingencies, and what competing causes remain?

History and context as diagnostic evidence

History is not background decoration; it is a structured evidence stream. A useful account should reconstruct onset, first recognizable context, changes in frequency or intensity, preceding medical events, training and handling exposure, consequences that follow the behaviour, and whether the phenotype appears across people, places, activities, and physiological states. Longitudinal training research shows that caregiver practices and exposure can vary over time, making a static history insufficient when learned contingencies are plausible [9].

Context changes what is observed. Restrictive veterinary handling can acutely increase fear, avoidance, and escape responses [13]. A behaviour seen only under restraint therefore carries different evidential meaning from

the same behaviour occurring spontaneously or across unrelated settings. Neurological timing adds another layer: in canine epilepsy, behaviour may occur in interictal, peri-ictal, or prodromal relationships to seizure activity [15]. The proposed approach is to build a time–context map before assigning high confidence to any cause. Consistency across contexts can strengthen a stable explanation; reliable context dependence can strengthen a situational one; and temporal coupling with pain, disease episodes, medication changes, or seizures can redirect the differential. Caregiver history remains indispensable but should be corroborated when feasible rather than treated as error-free ground truth.

A behavior–medicine differentiation framework

The proposed behavior–medicine differentiation framework treats diagnostic reasoning as adjudication among competing explanations rather than passage through a single pipeline. Four evidence streams are kept analytically separate: caregiver history, direct behavioural observation, clinical examination or targeted testing, and longitudinal response to contextual or therapeutic change. Confidence increases when independent streams converge and decreases when they conflict, are weakly measured, or are compatible with several causes.

Observer interpretation itself can be a source of error. In a large cat-owner study, anthropomorphic beliefs were associated with less accurate interpretation of some feline emotional cues [20]. Multimodal information can partly reduce ambiguity: people interpreted combined visual and vocal feline signals more accurately than some unimodal signals, although this was a communication experiment rather than a clinical validation study [21]. Age and living context must also condition interpretation because older-dog surveys document concurrent behavioural, sensory, mobility, sleep, and elimination changes, with some variation related to housing or body-related factors [22].

The framework therefore proposes a multidomain state architecture in which pain, fear, learned maintenance, environmental conflict, social or handling conflict, neurological dysfunction, and other medical disease remain separately visible but cross-connected. Masking or adaptation may reduce overt signs without removing underlying cost; context may expose or suppress a phenotype; duration can shift an initiating cause into a maintaining process; and reassessment can move confidence between domains. No numerical score or threshold is proposed.

The architecture must therefore display competing causes together with convergence, discordance, masking, context, time, and reassessment **(Figure 1)**.

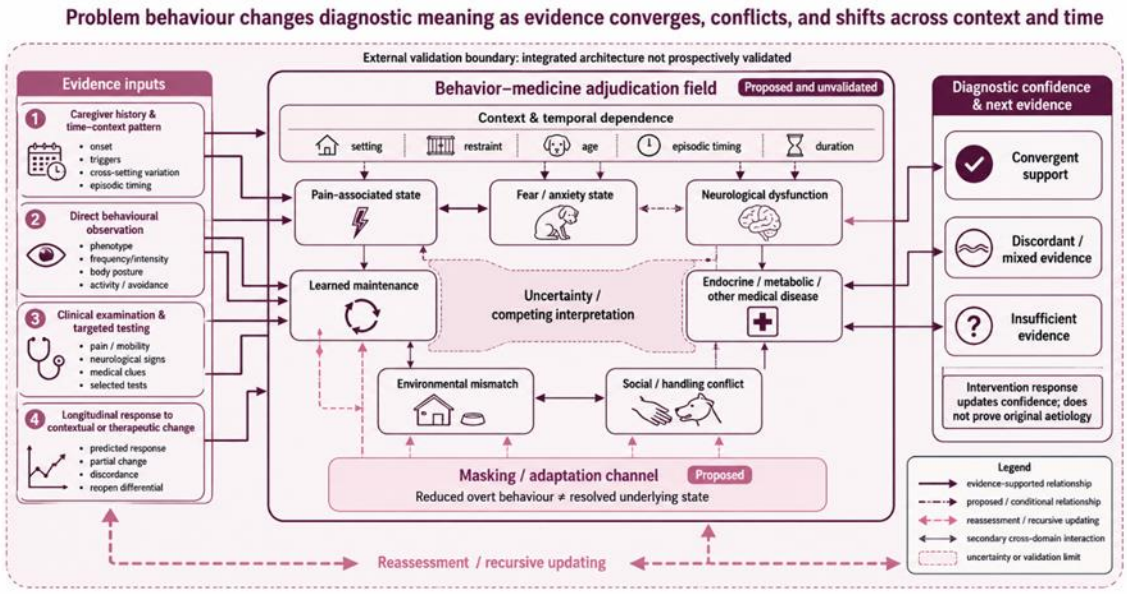

Figure 1. Diagnostic confidence shifts when competing causes converge, diverge, mask one another, or change across context and time

Diagnostic testing and therapeutic trials

Diagnostic tests and therapeutic trials are most useful when they answer a pre-specified question and have an interpretable expected response. A positive response should modify confidence in the targeted state or mechanism,

not be treated as retrospective proof of the original cause. In cats with transport and veterinary-visit anxiety, a randomized blinded field study found lower anxiety ratings with pregabalin than placebo, supporting pharmacological modifiability of that fear state [23]. It does not establish why the fear developed.

Environmental manipulation can provide a parallel test of context sensitivity. A randomized double-blinded study evaluated environmental enrichment in dogs recovering after hemilaminectomy for acute intervertebral disc extrusion, embedding environmental change within a known medical state [24]. Such designs illustrate why medical and environmental contributions need not be mutually exclusive.

Combined interventions further complicate causal attribution. In sensitized cats undergoing a claw-trimming desensitization–counterconditioning protocol, gabapentin accelerated progression relative to placebo, while transient sedation or ataxia was reported [25]. Improvement may therefore reflect reduced arousal, learning, repeated exposure, or their interaction. The proposed rule is that a trial should define the hypothesis, expected domain-specific response, safety constraints, observation window, and alternative explanations before treatment begins.

Table 3 compares three evidence-generating strategies by the hypothesis tested, the meaning of a response, and the causal claim that must remain prohibited.

Table 3. Diagnostic tests and therapeutic trials compared by the inference each can and cannot support

Interpretive question	Targeted clinical test	Context or pharmacologic trial	Learning-based trial
Hypothesis tested	Is a clinically plausible disease or dysfunction present?	Is the phenotype modifiable through a specified state or context?	Is a learned contingency maintaining part of the phenotype?
Result that raises confidence	A disease-relevant result that fits the pre-test clinical pattern	A predicted state-specific change within the planned observation window	Progressive change with adequate exposure and treatment fidelity
What the result can establish	The medical explanation becomes more or less plausible	The targeted state or context contributes to modifiability	The maintenance hypothesis gains or loses support
What it cannot establish	An abnormality may be incidental or insufficient to explain the behavior	Response does not prove why the problem began	Improvement does not identify the initiating cause
If phenotype and targeted domain diverge	Reassess whether disease is causal, concurrent, or incidental	Consider coexisting pain, fear, learning, environment, or adverse effects	Reopen medical, affective, neurological, and contextual alternatives

Reassessment after intervention

Reassessment converts a static differential into a longitudinal one. Before intervention, the clinician should specify what change would be expected if the working explanation were substantially correct: improved mobility after pain control, reduced fear under lower-conflict handling, reduced context-specific anxiety after anxiolytic support, or gradual change under learning-based treatment. A response that matches the predicted domain increases confidence but does not by itself prove the initiating cause.

Discordance is equally informative. In treated hypothyroid dogs, increased activity did not translate into broad behavioural normalization [4]. Counterconditioning evidence also remains heterogeneous across interventions and outcomes [10], and pharmacologically facilitated learning in cats occurs alongside repeated exposure and possible sedative effects [25]. These patterns support separating response from causal certainty.

The proposed framework therefore uses three reassessment questions: Did the targeted domain change? Did the problematic phenotype change? Did unintended or alternative-domain changes emerge? Complete, partial, absent, or context-limited responses should shift confidence differently. Persistence after apparently adequate treatment can indicate an incorrect hypothesis, an additional cause, a maintaining learned process, insufficient treatment exposure, or measurement error. Reassessment should therefore reopen the differential rather than simply intensify the initial intervention.

Clinical and welfare implications

Diagnostic interpretation changes welfare because the method used to investigate or suppress behaviour can itself alter the animal's state. In cats undergoing mock spay-recheck examinations, video telemedicine was associated with fewer negative responses than the in-clinic condition and with lower caregiver-perceived stress and greater

accessibility, although remote assessment cannot replace physical examination when clinically required [26]. The implication is not that difficult patients should avoid examination, but that the diagnostic environment can be designed to reduce avoidable conflict.

Behaviour-management choices can also affect relationships and welfare beyond the target sign. Reward-based training has been associated with a tendency toward more secure dog–owner attachment, although observational design limits causal inference [27]. In a separate comparative study, dogs trained with more aversive methods showed more stress-related behaviours and poorer welfare indicators than reward-trained dogs [28].

The proposed framework therefore defines clinical success more broadly than suppression of the presenting behaviour. Relevant outcomes include reduced underlying distress, preserved or improved function, safer interaction, feasible care, and avoidance of unnecessary diagnostic or training burden. Welfare consequences should be reassessed alongside behavioural change, especially when an intervention reduces visible behaviour by limiting expression rather than resolving the state driving it.

Failure modes

The first failure mode is premature closure: assigning a familiar behavioural or medical label and interpreting subsequent evidence through that label. Confirmed disease can coexist with persistent behaviour, as shown by limited broad behavioural change after levothyroxine treatment in hypothyroid dogs [4]. A second failure mode is single-cue attribution. Qualitative or ethogram-based pain behaviours are clinically useful, but their presence does not make pain the only compatible explanation [6].

A third failure mode is therapeutic-response-as-diagnosis. Improvement with counterconditioning, anxiolytic medication, environmental change, or exploratory biological treatment can show modifiability while leaving the initiating cause unresolved. The uncontrolled epilepsy pilot involving fecal microbiota transplantation is a particularly clear reminder that pre/post improvement does not establish mechanism [19]. A fourth failure mode is ignoring context and observer effects; interpretation of feline emotional cues can vary systematically with the observer's assumptions [20].

The proposed safeguards are therefore explicit: describe before diagnosing; separate evidence sources; document competing explanations; avoid indiscriminate testing; define what a therapeutic trial can and cannot establish; and reopen the differential when evidence is discordant. These safeguards are reasoning constraints, not validated rules of clinical performance. Their value remains to be tested prospectively across species, settings, and practitioner populations.

Validation priorities

The integrated framework requires validation at several levels before it can be treated as a clinical decision instrument. First, its observable components need construct and criterion validity where suitable comparators exist. Development of the Feline Grimace Scale illustrates how a behaviour-based clinical tool can be examined for discrimination, reliability, criterion association, responsiveness, and decision thresholds while retaining population-specific limitations [29]. The broader framework would require analogous testing without assuming a single gold standard across etiologies.

Second, measurement should become more objective where that improves repeatability without confusing technical detection with clinical meaning. Digitally enhanced dog behavioural testing demonstrates the feasibility of augmenting conventional assessment, but any digital measure must be validated for the specific construct, species, setting, and decision it is intended to support [30].

Third, observer and setting effects must be measured rather than treated as random noise. In canine behavioural testing, tester identity or type was systematically associated with some outcomes [31]. Future validation should include standardized or blinded assessment where feasible, inter-rater reliability, repeated cross-context observation, multisite recruitment, longitudinal follow-up, and external validation. Prospective studies should test diagnostic agreement, decision consequences, welfare outcomes, and reassessment trajectories; none can currently be assumed.

Conclusion

Problem behaviour should be approached as a clinical phenotype whose meaning remains provisional until competing behavioural, environmental, neurological, painful, and other medical explanations have been weighed

against one another. The framework developed here proposes that diagnostic confidence should arise from convergence among history, direct observation, clinical examination or targeted testing, context, and longitudinal response rather than from behavioural topography or treatment response alone.

Its central contribution is to keep causes analytically separate while allowing them to coexist, interact, change over time, and leave different traces across contexts. Discordance is therefore useful: a finding that fails to match the working explanation should reopen rather than simply intensify that explanation. The framework is intentionally non-numeric and remains unvalidated. Its strongest current evidence base concerns companion dogs and cats, and transfer to other species or practice settings requires dedicated study. Prospective testing is required before the architecture can be considered a validated clinical decision tool.

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