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Psychosomatic disorders in animals: Interplay of affect, cognition, and reflex regulation

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Psychosomatic disorders represent a complex category of conditions characterized by the interaction between psychological processes and somatic functions in animals. Unlike traditional disease models that separate mental and physical health, the psychosomatic approach involves the recognition of dynamic regulations across cognitive, emotional, autonomic, and physiological systems. This review summarizes contemporary knowledge on the definition, classification, etiology, pathogenesis, diagnosis, and treatment of psychosomatic disorders in animals in veterinary contexts. Emphasis is placed on how cognitive processes (such as learning, expectations, and perceptions), affective states (including chronic fear, frustration, or social deprivation), and reflexive mechanisms (e.g., autonomic reactivity and visceral responses) interact to produce and sustain pathological outcomes. Biological risk factors, such as neuroendocrine dysregulation, immune dysfunction, and neural interruption, are examined alongside environmental, social, and developmental contributors. The review also explores species-specific diagnostic challenges and current assessment tools, including behavioral scoring systems, salivary cortisol level, thermography, and heart rate variability. Therapeutic approaches are discussed in terms of providing environmental enrichment, behavioral modification, pharmacological treatment, and integrative clinical models grounded in behavior and welfare science. Interdisciplinary approaches including affective neuroscience, psychoneuroimmunology, and ethology are proposed as essential for advancing both the scientific understanding, prevention and clinical management of these disorders. This work highlights the importance of understanding psychosomatic disorders not merely as isolated clinical conditions, but as complex physiological phenomena shaped by the animal's emotional, cognitive, and environmental experiences.

Keywords: psychosomatic disorders; animal behavior and welfare; cognitive-affective regulation; autonomic reactivity; veterinary behavioral medicine; biopsychosocial model.

Introduction

Psychosomatic disorders (PSD) in animals represent a complex and multifactorial group of conditions in which psychological or emotional processes causes somatic dysfunctions. The term "psychosomatic" refers to the interaction between mind (psyche) and body (soma), wherein psychological factors such as stress, anxiety, fear, frustration, or social deprivation contribute significantly to the onset and progression of physical illnesses. While well established in human medicine, this integrative concept is still emerging in veterinary behavioral medicine and ethological research. In animals, PSDs are often underdiagnosed or misattributed due to the absence of verbal self-reporting and the complexity of distinguishing between primary somatic diseases and somatic symptoms with psychological origins. These disorders frequently manifest through various physiological systems – most commonly the gastrointestinal, dermatological, cardiovascular, and respiratory systems – and are often accompanied by behavioral changes, including self-injury, stereotypic movements, indifference, hypervigilance, or aggression (Overall, 2013; McMillan, 2017).

The recognition of PSD in animals necessitates the integration of affective neuroscience, comparative cognition, and reflexive physiology. Affective components – such as fear or chronic anxiety – can trigger persistent activation of the hypothalamic–pituitary-adrenal (HPA) axis, leading to maladaptive stress responses that compromise immune, gastrointestinal, and cardiovascular function (Broom & Zanella, 2004; Mormède et al., 2007). Cognitive processes, including learning history, perceived predictability and controllability of the environment, and memory of adverse experiences, play a key role in modulating the stress response and behavioral expression of distress (Proops et al., 2013; Düpjan et al., 2017). Reflexive mechanisms, such as autonomic regulation and conditioned physiological responses, mediate the body's immediate reactions and the development of pathological changes under chronic psychological strain. The importance of addressing PSD in animals extends into broad welfare considerations. In both companion and production animals, prolonged psychosomatic dysregulation not only affects individual health and longevity but may also impair social functioning, learning ability, reproduction, and human-animal interaction. Moreover, species-specific manifestations of PSD call for an ethologically informed approach to differential diagnosis and treatment, incorporating behavioral assessments, physiological biomarkers, and environmental analysis.

The scope of PSDs also intersects with the domains of psychoneuroimmunology, neuroendocrinology, and behavioral pharmacology. Recent studies have demonstrated correlations between chronic stress, elevated glucocorticoid levels, immune dysregulation, and tissue pathologies in several species including dogs, cats, pigs, and horses (Koolhaas et al., 2011; Palme, 2019). This convergence of behavioral and physiological dysfunction underscores the necessity of integrative approaches in veterinary medicine, combining behavioral therapy, environmental modification, and targeted pharmacological support. Thus, psychosomatic disorders in animals are not merely somatic diseases with coincidental behavioral components, but rather systemic pathologies rooted in complex interactions among affect, cognition, and reflexive control. The recognition and comprehensive management of such conditions is essential for advancing both animal health and welfare, and for bridging veterinary medicine with neuroscience and behavioral science. The study of PSD in animals has to be done taking into account diverse scientific domains including behavioral

neuroscience, comparative psychology, veterinary medicine, psychoneuroendocrinology, and ethology. To construct an understanding of PSD, one must consider the interplay of internal psychological states and physiological responses within an organism exposed to complex environmental stimuli. While empirical studies in nonhuman animals have long contributed to modeling human psychosomatic and neuropsychiatric conditions, the application of these models back to veterinary practice and animal welfare science is relatively recent and still is conceptually underdeveloped.

A foundational theoretical framework underpinning PSD is the biopsychosocial model originally developed in human medicine (Engel, 1977). This model posits that health and disease result from the dynamic interaction between biological, psychological, and social factors. Applied to animals, this implies that stress-related pathologies should not be viewed solely as outcomes of infectious agents, genetic predisposition, or physiological injury, but also as shaped by affective experiences, social context, and environmental conditions (Moberg, 2000). In this view, psychological distress in animals – manifesting through fear, frustration, or learned helplessness – may act as a primary etiological or aggravating factor in somatic dysfunction. Closely related is the allostatic load model (McEwen & Wingfield, 2003), which emphasizes how chronic activation of adaptive physiological systems, such as the HPA axis and autonomic nervous system, leads to wear and tear on the body. Animals experiencing prolonged social instability, inadequate stimulation, or unresolved trauma may exhibit physiological deterioration through mechanisms such as immune suppression, gastrointestinal disruption, or cardiovascular dysregulation. This model reinforces the importance of interpreting somatic illness in the context of cumulative stress and behavioral plasticity.

Affective neuroscience, a field pioneered by Panksepp (1998), provides a neurobiological foundation for understanding how primary emotional systems modulate animal behavior and physiological states. Fundamental affective systems – including fear, panic/grief, rage, and seeking – are evolutionarily conserved and interact with both cognitive and reflexive processes. Dysregulation within these systems may lead to chronic affective arousal, which in turn alters somatic function through neuroendocrine and autonomic channels. For example, persistent fear activation may result in sympathetic overdrive and ulcerogenic states, while chronic separation distress may contribute to gastrointestinal hypomotility and behavioral withdrawal. This approach challenges the outdated Cartesian dualism that separated mind and body, emphasizing instead that emotional processing has direct physiological consequences. It also highlights the evolutionary continuity between humans and other animals in emotional architecture, supporting the validity of PSD across species.

Cognitive-behavioral frameworks, widely applied in human psychosomatic medicine, offer essential insights into how animals perceive, interpret, and adapt to environment. Concepts such as predictability, controllability, and cognitive bias have been explored in animal models to explain how chronic stress and learned helplessness influence both behavioral and somatic outcomes (Mineka & Hendersen, 1985; Harding et al., 2004). For instance, when animals are repeatedly exposed to aversive stimuli without the possibility of avoidance, they may develop passive coping strategies, associated not only with behavioral signs of depression but also with suppressed immune function and increased morbidity. Furthermore, cognitive assessments in animals, such as judgment bias tests, which measure whether an animal interprets ambiguous situations in a positive or negative way, are now used to assess their emotional states. These methods help connect what we can observe in behavior with the underlying psychological and brain processes. They may also enhance the capacity to detect early indicators of psychosomatic imbalance before it develops clinically.

An often-underappreciated domain within PSD research is the role of reflexive regulation. Reflexes – including baroreceptor, gastrointestinal, and nociceptive pathways – mediate involuntary responses to internal and external irritants. Chronic emotional states can recalibrate these reflex arcs, resulting in maladaptive feedback loops such as visceral hypersensitivity, cardiac arrhythmias, or altered pain thresholds. The vagus nerve plays a central role in the so-called "inflammatory reflex," linking emotional experience with immune modulation (Tracey, 2002). Understanding the reflexive nature of PSD supports the integration of somatic therapies (e.g., vagal stimulation, physical modulation) with behavioral interventions. An ethological perspective is also essential to interpret PSD manifestations across species. For instance, confinement stress in pigs may result in stereotypies and gastric ulcers, whereas social isolation in dogs may lead to separation-related behaviors and dermatological self-trauma. Recognizing these distinctions is vital for appropriate diagnosis, treatment, and welfare assessment. By acknowledging the bidirectional pathways between brain and body, and the mediating roles of emotion, cognition, and reflexes, veterinary science can evolve toward more comprehensive and compassionate models of animal care, diseases' prevention and treatment.

The integration of cognitive, affective, and reflexive mechanisms is essential for understanding the etiology, manifestation, and clinical management of psychosomatic disorders in animals. These three domains, though traditionally examined in isolation within their respective scientific fields, constitute an interdependent triad that shapes how animals perceive, process, and respond to their internal and external environments. Their integration provides a more comprehensive and biologically valid framework for addressing PSD, supporting both clinical interventions and welfare-oriented management. Cognition enables animals to assess their surroundings, anticipate events, form associations, and evaluate threats or opportunities. Cognitive processes influence how stressors are estimated and whether they are perceived as predictable, controllable, or inescapable. These estimations directly affect neuroendocrine responses, particularly the activation of the HPA axis and sympathetic-adrenal-medullary system. For example, animals with prior learning experiences of helplessness (e.g., inability to escape from aversive stimuli) often exhibit stronger physiological stress responses and increased vulnerability to psychosomatic dysfunction (Dess & Foltin, 2001; Koolhaas et al., 2011). Cognitive mechanisms also modulate attention and memory processes that can sustain or amplify emotional reactions. An animal that anticipates negative outcomes in a given situation, based on previous experience, may exhibit heightened anxiety or aggression, which, if prolonged, translates into physiological dysregulation. Thus, cognition serves as an interpretive filter that either buffers or exacerbates the somatic impact of psychological stressors.

Affect refers to the underlying emotional states and motivational systems that guide behavior and physiological preparedness. Affective states such as fear, frustration, or social grief are not merely behavioral expressions but represent coordinated neural, hormonal, and autonomic processes that lead the organism for adaptive responses. In pathological contexts, these affective systems can become dysregulated, resulting in chronic activation of stress pathways, impaired immune function, and maladaptive behaviors (Panksepp, 2005; McMillan, 2005). Affective experiences shape not only behavioral tendencyes but also modulate somatic outcomes. For instance, in dogs and cats, emotional distress has been linked to conditions such as idiopathic cystitis, inflammatory bowel disease, and dermatological self-trauma, conditions that often worsen in the absence of environmental or social enrichment (Buffington et al., 2006; Overall, 2013). These disorders are best understood not as isolated somatic phenomena, but as expressions of persistent affective disturbance interacting with physiological systems. Reflexive processes, including autonomic and neuroendocrine reflex arcs, constitute the most immediate interface between psychological stimuli and somatic systems. Reflexive mechanisms such as heart rate variability, gastrointestinal motility, salivation, pupil dilation, and cutaneous vasomotor responses are tightly regulated by emotional and cognitive states. Chronic affective disturbances may recalibrate these reflexes, leading to a breakdown of homeostasis and contributing to somatic symptomatology (von Borell et al., 2007).

The review presented herein is based on a structured literature search designed to retrieve recent, high-quality, and peer-reviewed scientific publications addressing psychosomatic disorders in animals from an interdisciplinary perspective. The search strategy included searching PubMed, Scopus, Web of Science, and CAB Abstracts. Publications from 1990 to 2025 were prioritized to ensure contemporary relevance. Seminal works and theoretical contributions older than 10 years were included selectively if they remain foundational or are frequently cited

in current literature. Non-peer-reviewed material, studies lacking clear methodological transparency, or works focusing solely on human psychosomatic medicine without translational relevance to animal health were excluded. All sources were evaluated for relevance, methodological rigor, and conceptual clarity to support a scientifically grounded and interdisciplinary review.

Conceptual foundations of psychosomatic disorders in animals

The conceptualization of PSD in animals has evolved through a scientific achievement in comparative psychology, veterinary behavioral medicine, neurobiology, and ethology. While the origins of psychosomatic thought can be traced back to antiquity in human medicine, most notably in Hippocratic theories of bodily imbalance influenced by emotional states, the extension of such ideas to nonhuman animals is a relatively recent advancement shaped by changing perceptions of animal sentience, emotion, and cognition.

Historically, animals were widely viewed as automata, biological machines lacking subjective experience, a view reinforced by Cartesian dualism which postulated a fundamental distinction between mind and body, and further between humans and other species. This framework left little room for acknowledging emotional causation in animal disease. Veterinary medicine during the 19th and early 20th centuries focused primarily on infectious diseases, parasitology, and structural pathology, with limited interest in behavioral or emotional determinants of health (Rollin, 1990). Yet anecdotal observations by veterinarians and animal caregivers, particularly in horses, dogs, and zoo animals, often noted physical illnesses in conjunction with behavioral anomalies such as aggression, withdrawal (lack of social interaction or engagement), or repetitive behaviors. These were frequently attributed to “nervous temperament” or "hysteria" but lacked formal frameworks for interpretation.

A major theoretical shift occurred with the introduction of the stress model by Hans Selye in the 1930s. Selye’s general adaptation syndrome (GAS) proposed that organisms respond to prolonged stress with a sequence of physiological changes that can culminate in disease (Selye, 1936). Although initially applied to humans and laboratory rodents, the universality of the stress response soon led to its adoption in animal physiology and veterinary science. The concept that chronic psychological stress could damage somatic systems laid the foundation for later PSD models. Selye’s work prompted significant interest in the hypothalamic-pituitary-adrenal (HPA) axis, which became a central focus of research in animal welfare and behavior. Over subsequent decades, studies demonstrated that sustained HPA activation was associated with gastrointestinal ulcers, immune suppression, reproductive failure, and dermatological symptoms in various species, including pigs, cattle, horses, and primates (Moberg, 2000).

Parallel advances in behavioral science and comparative psychology during the mid-20th century further enriched the theoretical landscape for PSD in animals. Research by Harlow, Mason, and others revealed that social deprivation, maternal separation, and learned helplessness could produce both psychological distress and somatic dysfunction in primates and dogs (Harlow & Suomi, 1971; Seligman, 1975). These findings challenged prevailing assumptions and offered experimental evidence that emotional experiences could trigger physiological breakdown. By the late 20th century, this body of research formed into a behavioral model of psychosomatic interaction, emphasizing the organism's perception of its environment and its behavioral coping strategies as key mediators of health. These models are drawn from operant learning theory, emotional regulation theory, and ethological studies of naturalistic behavior with the aim of developing a clearer understanding of how animals experience and respond to stress.

The turn of the 21st century witnessed an accelerating integration of disciplines, including neuroendocrinology, immunology, and affective neuroscience, into the study of animal behavior and health. Jaak Panksepp’s foundational work on the neurobiology of emotion established that core emotional systems are evolutionarily conserved and functionally homologous across mammals (Panksepp, 1998). These systems, including fear, seeking, care, and rage, have clear physiological consequences that align with known somatic disorders. His work prioritized the study of emotion as a physiological driver in animals, offering a scientific basis for PSD frameworks.

Contemporary PSD theory is formed by models such as the allostatic load concept, biopsychosocial integration, and psychoneuroimmunology. These frameworks reveal that repeated or prolonged emotional dysregulation, modulated by cognitive evaluation and expressed through autonomic and neuroendocrine pathways, results in a physiological burden that manifests by somatic damage (Dantzer, 2001; McEwen & Wingfield, 2003). Such models have been applied not only in laboratory species but also in clinical cases involving companion and captive animals. Today, the recognition of psychosomatic disorders in animals is supported by converging evidence from behavioral pathology, chronic disease studies, and welfare science. This change reflects a shift in veterinary and animal research from focusing narrowly on individual pathological factors to considering health and illness as the result of interactions among multiple physiological, behavioral, and environmental factors. As this theoretical foundation continues to mature, it opens new avenues for interdisciplinary research, clinical innovation, and full reevaluation of how animals are diagnosed and treated.

Interdisciplinary perspectives on psychosomatic disorders in animals

The complexity of PSD in animals demands an integrative, interdisciplinary approach that crosses traditional disciplinary boundaries. A strict biomedical perspective, while valuable for identifying structural or infectious causes of disease, is insufficient for explaining disorders in which psychological and environmental factors significantly influence somatic function. Accordingly, recent advances in veterinary behavioral science, affective neuroscience, psychoneuroimmunology, and systems biology have contributed to the development of comprehensive models that account for the multidimensional nature of PSD. Some of the most influential contemporary frameworks include the biopsychosocial model, approaches from affective neuroscience, and integrative concepts such as allostatic load, which describes the cumulative physiological burden of chronic stress, and neurovisceral integration, all of which emphasize the coordinated regulation of brain and body systems in maintaining health.

Originally formulated by George Engel (1977) to address limitations in the reductionist biomedical model of human illness, the biopsychosocial model claims that biological, psychological, and social factors all contribute to disease and health. In the context of animal health, this model provides a critical lens through which PSD can be understood not merely as a result of somatic dysfunction but as an emergent outcome of the interaction between internal physiological states, individual behavioral coping capacities, and environmental conditions (Moberg, 2000). In applied veterinary settings, the biopsychosocial concept allows for the systematic assessment of biological factors (e.g., genetics, hormonal profiles, immune function), psychological variables (e.g., temperament, affective state, stress history), environmental and social factors (e.g., housing conditions, social isolation, human-animal interaction). This model has proven particularly useful in evaluating chronic disorders such as feline idiopathic cystitis, stress-induced colitis in horses, and dermatological self-trauma in dogs – conditions where the underlying etiology often cannot be fully explained by structural pathology alone (McMillan, 2005; Westropp et al., 2006).

Affective neuroscience, pioneered by Jaak Panksepp, provides a neurobiological basis for understanding how primary emotional systems mediate animal behavior and physiological responses (Panksepp, 1998). These core systems, seeking, fear, rage, panic/grief, play, lust, and care, are conserved across mammalian species and influence both adaptive and maladaptive patterns of emotional and physiological regulation. In the context of PSD, chronic dysregulation of affective systems such as fear, panic, or rage may result in somatic damage through persistent activation of the HPA axis, sympathetic nervous system, or inflammatory pathways (Bateson, 2011). Understanding the neurofunctional anatomy of affect allows clinicians and researchers to interpret behavior and somatic signs not merely as coincident phenomena, but as causally linked events revealed through

shared neural substrates. Psychoneuroimmunology explores the interactions among psychological processes, the nervous system, and immune function. Research in this field has revealed how chronic stress and emotional dysregulation impair immune competence, alter cytokine profiles, and facilitate inflammatory responses (Dantzer, 2001). These findings are particularly relevant in animals subjected to prolonged environmental stress, such as in sheltering dogs or zoo animals kept in suboptimal conditions.

Complementing this, the allostatic load model, developed by McEwen & Wingfield (2003), describes how repeated or sustained efforts to maintain physiological stability (allostasis) in response to stressors can lead to cumulative biological "wear and tear." This model is essential for conceptualizing how emotionally mediated stress, if unmanaged, transitions from functional adaptation to pathological states. Animals that cope poorly with stress (low resilience) or have strong emotional reactions to external challenges are more likely to develop long-term stress-related diseases such as inflammatory bowel disease, heart problems, or hormonal imbalances.

Another emerging interdisciplinary model is the neurovisceral integration framework, which emphasizes the connection between central autonomic control structures (e.g., prefrontal cortex, amygdala, hypothalamus) and peripheral somatic responses (Thayer & Lane, 2000). In animals, this model has been useful for interpreting vagally mediated reflexes such as heart rate variability, gastrointestinal motility, and immune function as indices of emotional state and somatic regulation.

Reflexive systems, long considered automatic and peripheral, are currently increasingly understood as modifiable by emotional and cognitive states. This conceptual shift is supported by evidence from affective neuroscience showing that spinal and autonomic reflexes, such as the nociceptive flexion reflexes, are significantly influenced by emotional states: negative affect enhances reflex magnitude, while positive emotional states suppress it (Bradley et al., 1992). These effects are mediated by descending cortical and limbic modulation of brainstem reflex pathways, highlighting that reflexes are not merely fixed responses, but regulated processes influenced by affective and cognitive inputs. In both humans and animals, reflex mediated disorders such as stress-induced vomiting, tachycardia, and diarrhea often occur in the absence of primary organic disease, particularly under acute or chronic emotional dysregulation (Valera et al., 2012; Travain et al., 2015). For instance, in functional gastrointestinal disorders in humans, psychological stress and anxiety disrupt the gut-brain axis, altering vagal and sympathetic reflex control of visceral organs and leading to symptoms such as nausea and bowel irregularity without detectable pathology (de Jong et al., 2000). In veterinary contexts, similar pathophysiological patterns are observed. Emotional instability in dogs, pigs, and horses has been linked to reflex somatic outputs, including autonomic hyperresponsiveness and gastrointestinal motility disturbances, that resolve with stress reduction or behaviorral therapy, further reinforcing the bidirectional relationship between emotional states and reflexive somatic functions (Valera et al., 2012; Travain et al., 2015).

Ethology contributes a crucial dimension to these models by grounding them in species-specific behavior. Animals' adaptive responses to environmental stimuli, their social structures, and natural histories form both emotional valence and stress susceptibility. An interdisciplinary approach that synthesizes neurobiology, behavioral science, immunology, and ethology not only deepens our understanding of PSD but also enhances diagnostic accuracy and therapeutic efficacy in veterinary and welfare settings.

Comparative overview with human psychosomatic conditions

The study of PSD in animals is mainly formed by the rich body of literature on psychosomatic medicine in humans. Although direct extrapolations must be approached cautiously due to differences in language, consciousness, and different contexts, there are substantial cross-species resemblances in emotional neurobiology, stress physiology, and disease mechanisms. These parallels not only validate the conceptualization of PSD in animals but also underscore the translational potential of veterinary research to human health, and vice versa. Both humans and nonhuman animals exhibit conserved neuroendocrine and autonomic responses to psychological stress. Central to these responses is the HPA axis, which, when persistently activated, contributes to a cascade of metabolic, immunological, and neurophysiological disruptions. Chronic HPA activation has been implicated in both human psychosomatic disorders, such as irritable bowel syndrome (IBS), chronic fatigue syndrome, and analogous conditions in animals, including feline idiopathic cystitis, stress-induced colitis in horses, and compulsive disorders in dogs (Chrousos, 2009; Stella et al., 2013). Similarly, dysregulation of the autonomic nervous system (ANS), particularly reduced vagal tone and sympathetic overdrive, is a well-documented contributor to somatic symptomatology in both humans and animals. Heart rate variability, a measure of parasympathetic flexibility, has been increasingly used in veterinary contexts as a physiological correlate of emotional regulation and somatic integrity (von Borell et al., 2007).

A growing number of human disorders are classified as functional somatic syndromes. That is because many physical symptoms are not fully explained by structural or biochemical pathology. These include conditions such as tension-type headaches, functional gastrointestinal disorders (e.g., IBS), somatoform illnesses (disorders showing physical dysfunction without clear organic pathology, often linked to chronic stress or emotional dysregulation), and psychosomatic dermatological diseases (Barsky & Borus, 1999). In animals, similar clinical presentations are increasingly recognized, although nomenclature and diagnostic frameworks are less standardized. Here are three examples. IBS in humans shares pathophysiological features with stress colitis in dogs and horses, characterized by altered motility, visceral hypersensitivity, and mucosal immune activation that are often exacerbated by anxiety and environmental instability (Paterson & Hall, 2015; Gao et al., 2019; Margolis et al., 2021). Atopic dermatitis with psychosomatic components in humans has parallels with dogs exhibiting psychogenic alopecia, overgrooming, or acral lick dermatitis without clear dermatopathological causes (Overall, 2013). Somatoform disorders in humans resemble some cases of stereotypies and compulsive behaviors in captive or socially deprived animals, where behavioral pathology results in physical harm or physiological compromise (DeNapoli et al., 2000; Garner et al., 2003; Lutz, 2014). These examples are supported by similar pharmacological responsiveness. For instance, both tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRIs) are used across species to manage anxiety-related somatic syndromes (Luescher & McKeown, 2009).

Unlike humans, animals cannot verbally articulate subjective symptoms such as pain, fatigue, or discomfort, which presents a unique diagnostic challenge. Consequently, the diagnosis of PSD in animals relies on behavioral indicators (e.g., withdrawal, aggression, stereotypy), physiological biomarkers (e.g., cortisol levels, HRV, inflammatory markers), exclusion of structural pathology, and full evaluation of environmental and social factors. These criteria parallel those used in diagnosing functional somatic disorders in humans, as soon as subjective reports must often be added by exclusion and contextual analysis.

From an ethical standpoint, recognizing PSD in animals provides a stricter approach to welfare. Just as human psychosomatic patients are vulnerable to misdiagnosis or dismissal, animals with PSD, when their condition is not properly diagnosed, may be subject to inappropriate treatment, e.g., unnecessary pharmacological interventions or punitive behavioral correction. The comparative study of PSD strengthens the foundations of the One Health initiative, the interdisciplinary movement linking human, animal, and environmental health. Animal PSD models offer valuable insights into the interplay of affective regulation, social environment, and somatic disease, especially in naturalistic settings not easily replicated in human studies. Additionally, advancements in human psychosomatic research provide conceptual and methodological tools for refining animal models of emotional-somatic comorbidity. Furthermore, domestic animals, particularly companion animals, serve as both sentinels (they can show early signs or symptoms of stress and environmental problems that also affect humans) and surrogate for human psychosocial stress, especially in shared environments. This reinforces the importance of

studying psychosomatic disorders not merely as clinical syndromes, but as biopsychosocial phenomena that integrate neurophysiological, emotional, and environmental factors across species boundaries.

Biological causes of PSD in animals (genetics, neurochemistry, endocrine system)

Psychosomatic disorders in animals are shaped by an interplay of biological, psychological, and environmental factors. Among these, biological predispositions, including genetic makeup, neurochemical regulation, and endocrine dynamics, form a critical substrate upon which environmental and experiential factors reveal their influence. Understanding these biological factors is essential for understanding why certain individuals are more susceptible to PSD and for developing both preventive and therapeutic approaches. Evidence suggests that heritable traits influence vulnerability to emotional dysregulation and related somatic consequences in animals. Selectively bred traits such as heightened fearfulness, reactivity, or compulsivity are observed in particular breeds or lines. For instance, German Shepherds, Border Collies, and Cavalier King Charles Spaniels demonstrate increased prevalence of noise phobia, separation-related behaviors, and compulsive disorders, respectively (Overall, 2013; Ogata, 2016). Table 1 summarizes key biological contributors to PSD, emphasizing their interrelated roles in affective, cognitive, and somatic regulation.

Table 1
Genetic, neurochemical, and endocrine factors contributing to PSD in animals

Factor	Mechanism	Species / Breeds	Associated Clinical Manifestations	Key References
Genetic predisposition	Inherited traits (e.g., fearfulness, compulsivity); polymorphisms in MAOA, SLC6A4 genes	German Shepherd, Border Collie, Bull Terrier	Anxiety, noise phobia, compulsive behaviors, GI dysregulation	Overall, 2013; Ogata, 2016; Tiira et al., 2019
Serotonin (5-HT)	Affective regulation, inhibition of gut motility, behavioral inhibition	Dog, cat, rodent	Diarrhea, nausea, anxiety, compulsivity	Steiner et al., 2012; Mayer et al., 2015
Dopamine (DA)	Reward and motivational signaling, compulsivity	Bull Terrier, cat	Tail chasing, stereotypies, self-licking	Dodman et al., 2010
GABA / Glutamate	Balance of arousal/inhibition; stress and anxiety modulation	Dog, primate	Hyperarousal, aggression, tension behaviors	Crowell-Davis et al., 2003
HPA Axis (CRH-ACTH-Cortisol)	Stress response, energy mobilization, immune and gut modulation	Dog, horse, rodent	Vomiting, diarrhea, dermatoses, immunosuppression	McEwen & Wingfield, 2003; Tiira & Lohi, 2014
Thyroid dysfunction	Low metabolic rate, altered mood regulation	Dog (hypothyroid breeds)	Lethargy, aggression, dermal issues	Ferguson, 2008
Sex hormones	Arousal, mood, behavioral thresholds	Dog, cat	Aggression, anxiety, cyclic behavior changes	McMillan, 2017

Genetic polymorphisms related to neurotransmission have been associated with behavioral and stress-related pathologies. In dogs, variations in the monoamine oxidase A (MAOA) gene and serotonin transporter (SLC6A4) gene have been linked to aggression, anxiety, and compulsive behaviors, which often manifest with somatic symptoms such as dermatological self-trauma or gastrointestinal dysregulation (Tiira et al., 2019). Similar findings in rodents and primates support the evolutionary conservation of genetic influences on emotional-somatic interactions (Lesch et al., 1996; Hovatta & Barlow, 2008).

At the neurochemical level, several neurotransmitter systems play central roles in modulating affective state, stress reactivity, and somatic responses. Serotonin (5-HT) is heavily implicated in mood regulation, behavioral inhibition, and gastrointestinal function. Dysregulated serotonergic activity is a hallmark of both affective disorders and stress-induced somatic complaints, such as diarrhea, nausea, and altered gut motility (Steiner et al., 2012). SSRIs are commonly used to treat behavioral disorders in dogs and cats with concurrent somatic symptoms, underscoring serotonin's integrative role. Dopamine (DA), central to motivational salience (helps animals to decide what matters in their environment and drives them to act on it) and reward processing influences behaviors relevant to compulsivity and stereotypy. Abnormal dopaminergic signaling has been associated with tail-chasing in Bull Terriers and compulsive licking in dogs and cats, behaviors often leading to self-inflicted injury (Dodman et al., 2010). Gamma-aminobutyric acid (GABA) and glutamate, as primary inhibitory and stimulatory neurotransmitters respectively, modulate emotional arousal, anxiety, and stress coping. Pharmacological agents targeting GABAergic systems (e.g., benzodiazepines) are widely used for short-term anxiolysis in veterinary practice and have indirect somatic effects via reduction in sympathetic output (Crowell-Davis et al., 2003). These neurotransmitter systems interact dynamically with peripheral organs via the gut-brain axis, further linking affective state to somatic outcomes (Mayer et al., 2015).

Table 2 integrates physiological systems involved in psychosomatic symptomatology, highlighting their diagnostic and therapeutic implications.

The HPA axis is the central coordinator of the physiological stress response. When activated by perceived threats (cognitive, emotional, or physical) it results in the secretion of corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), and cortisol. Chronic HPA activation leads to immune suppression, gastrointestinal dysfunction, and metabolic derangements, all common in PSD (McEwen & Wingfield, 2003). In companion animals, elevated baseline or reactive cortisol levels have been associated with behavioral disorders such as separation anxiety, noise phobia, and generalized anxiety disorder, all of which often involve somatic comorbidities including vomiting, diarrhea, and dermatologic manifestations (Tiira & Lohi, 2014). Beyond the HPA axis, other endocrine pathways, including the thyroid axis, gonadal hormones, and the sympatho-adrenomedullary system, modulate mood, behavior, and somatic health. For example, hypothyroidism in dogs can manifest with both lethargy and dermatologic symptoms, complicating differential diagnoses between endocrine and psychosomatic pathology (Ferguson, 2008).

In sum, biological factors, ranging from gene expression patterns and neurotransmitter availability to endocrine signaling cascades, provide the foundational architecture for psychosomatic vulnerability. These elements do not act in isolation but interact with environmental conditions, individual learning history, and affective processing to produce the complex clinical picture seen in PSD. The biological groundwork described here forms the basis upon which cognitive, emotional, and reflexive systems interact to mediate health or dysfunction. The biological underpinnings of PSD in animals, encompassing genetic predisposition, neurochemical regulation, and endocrine modulation, establish a foundational frame for understanding individual susceptibility to stress-related somatic dysfunction. However, these biological factors constantly interact with the psychological state of the animal. Emotional experiences such as anxiety, fear, and frustration not only modulate physiological systems in real time but also condition long-term behavioral and somatic outcomes. Thus, to fully capture the etiology of PSD, it is essential to examine the psychological dimensions that translate affect into bodily disorder.

Psychological causes of PSD in animals (anxiety, fear, frustration)

Emotional dysregulation is central to the development and manifestation of psychosomatic disorders in animals. Psychological stressors, especially when chronic, unpredictable, or uncontrollable, contribute significantly to the onset and exacerbation of somatic patholo-

gy. Among the most consequential affective states are anxiety, fear, and frustration – each of which activates specific neural circuits and physiological pathways, with cascading effects on animal health. Anxiety describes a heightened state of anticipatory arousal in the absence of immediate threat. Unlike fear, which is a response to present danger, anxiety is sustained, often diffuse, and heavily mediated by cognition and memory (Steimer, 2002). In animals, anxiety is showed by behavioral markers such as pacing, vocalization, hypervigilance, and displacement behaviors.

Table 2
Neurobiological systems and their psychosomatic relevance in companion animals

System	Core functions	Stress-linked pathology	Relevance to PSD	Example treatments / interventions
Serotonergic system	Mood regulation, GI motility, sleep-wake cycle	IBS-like symptoms, compulsivity, behavioral inhibition	Links affective dysregulation to GI and behavioral symptoms	SSRIs (fluoxetine, clomipramine)
Dopaminergic system	Motivation, reward processing, action selection	Stereotypy, self-directed aggression	Dysregulation leads to compulsive and repetitive behaviors	Dopamine stabilizers, enrichment
GABAergic / Glutamatergic systems	Neural excitation-inhibition balance, emotional modulation	Anxiety, impulsiveness, seizure threshold changes	Central to fear responses, emotional lability	Benzodiazepines, gabapentin
HPA axis	Stress hormone release (CRH, ACTH, cortisol); systemic stress modulation	Immunosuppression, GI dysfunction, dermatoses	Chronic activation drives physical manifestations of stress	Behavioral therapy, adaptogens, environmental control
Thyroid and adrenal hormones	Energy regulation, arousal, metabolic and mood balance	Hypothyroidism-linked lethargy, alopecia	Endocrine dysfunction mimicking or compounding PSD symptoms	Hormonal replacement, behaviorally-informed diagnosis

Prolonged anxiety activates the HPA axis and sympathetic nervous system, leading to hypercortisolemia, altered autonomic tone, and downstream effects on immunity, digestion, and reproduction. Studies in dogs with generalized anxiety disorders show elevated salivary cortisol, decreased heart rate variability (HRV), and greater susceptibility to gastrointestinal disturbances and skin conditions (Dreschel & Granger, 2005; Part et al., 2014). Furthermore, trait anxiety (a stable, inherent tendency to experience anxiety), shaped by early-life experience and genetic predisposition, has been shown to sensitize animals to environmental triggers and increase the likelihood of developing stress-related somatic symptoms. Shelter animals, particularly those with histories of neglect or inconsistent socialization, are at heightened risk (McMillan, 2017).

Fear is an adaptive response to immediate threats. However, when fear responses become generalized or resistant to reduction, they can contribute to chronic somatic dysfunction. The amygdala plays a key role in the encoding of fear memories and orchestrating downstream autonomic and neuroendocrine responses. In animals, persistent or inappropriately triggered fear responses can lead to enduring physiological changes. Experimental studies demonstrate that fear-conditioned rodents show increased visceral sensitivity, elevated inflammatory markers, and dysregulation of gut microbiota – mechanisms similarly implicated in functional somatic syndromes (Moloney et al., 2016). In horses and dogs, phobias (e.g., noise, veterinary settings) have been linked to stress-induced colitis, diarrhea, and dermatologic manifestations (Mills, 2005; Dreschel, 2010). Importantly, chronic fear compromises learning and coping behavior, leading to behavioral rigidity and reduced flexibility – factors that exacerbate PSD by impairing the animal's capacity to regulate stress responses effectively (LeDoux & Pine, 2016).

Frustration arises when an animal is blocked from achieving a motivated goal, such as access to food, social contact, or locomotor freedom. It is often accompanied by behavioral signs such as redirected aggression, stereotypic behaviors, and vocalization. Chronic frustration can precipitate conflict behaviors, ambiguous or contradictory actions reflecting internal tension, which are frequently associated with self-directed somatic damage, such as overgrooming, feather plucking, or tail chasing (Garner et al., 2003).

Displacement behaviors, such as excessive licking or scratching in the absence of dermatological pathology, may serve as somatic outlets for emotional conflict. These behaviors often become habitual and self-reinforcing, resulting in physical damage and reinforcing the psychosomatic cycle (Luescher & McKeown, 2009). Moreover, frustration is not merely behavioral; neurobiological studies indicate that frustrated states activate dopaminergic and noradrenergic circuits associated with arousal, aggression, and autonomic imbalance – pathways that influence both psychological and somatic outcomes (Fuchs & Flugge, 2002).

To better illustrate the link between emotional triggers and their physiological and behavioral consequences, Table 3 summarizes the main psychological states blamed in psychosomatic disorders, their underlying neurobiological mechanisms, and associated clinical expressions in animals.

Table 3
Psychological triggers of PSD in animals and their behavioral and somatic expressions

Emotional state	Triggering conditions	Neurobiological mechanisms	Somatic manifestations	Behavioral indicators	Key references
Anxiety	Anticipation of uncertain or uncontrollable events (e.g., separation, novel stimuli)	HPA axis activation; increased cortisol; reduced HRV	Diarrhea, vomiting, dermatitis, immune suppression	Pacing, vocalization, hypervigilance, displacement behaviors	Steimer, 2002; Dreschel & Granger, 2005; Part et al., 2014
Fear	Immediate or remembered threats (e.g., noise, handling, vet visits)	Amygdala hyperactivation; sympathetic arousal	Colitis, GI dysmotility, tachycardia, elevated inflammatory markers	Avoidance, trembling, freezing, escape attempts	Mills, 2005; LeDoux & Pine, 2016; Moloney et al., 2016
Frustration	Blocked access to expected rewards (e.g., food, movement, social contact)	Dopaminergic and noradrenergic activation; limbic-cortical dysregulation	Gastrointestinal upset, skin overgrooming, stress-related alopecia	Stereotypies, redirected aggression, self-injury	Fuchs & Flugge, 2002; Garner et al., 2003; Luescher & McKeown, 2009

The psychological states of anxiety, fear, and frustration are neither abstract nor transient in their impact. Each engages specific neural substrates and hormonal cascades that alter immune function, gastrointestinal integrity, cardiovascular regulation, and metabolic balance. Moreover, these affective states often co-occur and reinforce each other, forming persistent feedback loops that sustain or exacerbate somatic symptoms. Affective dysregulation should therefore be considered as an important etiological factor in the diagnosis and treatment of PSD in animals, not merely a secondary reaction to physical illness.

While biological and psychological factors provide the internal conditions predisposing animals to PSD, the external environment acts as both a trigger and modulator of disease expression. The quality

of the animal's surroundings, particularly in terms of social structure, spatial complexity (the physical richness and structural variety of the environment that allows animals to explore, move, and engage naturally), predictability, and human interaction, play a crucial role in either mitigating or exacerbating affective distress and related somatic pathology. Environmental and social factors do not act in isolation but influence and potentiate the activation of psychological and biological mechanisms underlying PSD.

Environmental and social factors contributing to the development of PSD (isolation, captivity stress)

The external conditions in which animals live, both physical and social, are central determinants of health and welfare. In both domestic and captive species, isolation, restricted environments, predictability deficits, and social deprivation have been repeatedly identified as major risk factors for affective disturbances that give rise to psychosomatic disorders. These environmental stressors not only contribute to maladaptive behaviors but also initiate and maintain physiological dysfunction. Social species, including canines, primates, equines, and many avian taxa, rely on stable social bonds for emotional security, behavioral regulation, and neurodevelopment. Early-life isolation, weaning without maternal contact, or prolonged periods without conspecifics in adulthood are associated with increased anxiety-like behaviors, hypervigilance and behavioral pathologies, including compulsive self-directed behaviors (Hennessy et al., 2009; McMillan, 2017).

In dogs, prolonged isolation during their development can result in persistent emotional dysregulation and somatic comorbidities such as chronic gastrointestinal upset, dermatologic conditions, or urinary incontinence – common complaints in clinical behavioral referrals (Tiira & Lohi, 2015). In primates, early maternal separation has been shown to alter HPA axis functioning and immune system responsiveness, producing long-term vulnerability to psychosomatic functions (Sanchez et al., 2015).

In zoos and laboratory settings, captivity stress is a serious challenge, arising mainly from spatial restriction, lack of cognitive stimulation, and control deficits. Animals kept in barren or monotonous environments often develop stereotypic behaviors, indicative of affective and physiological distress. These repetitive, functionless behaviors, such as pacing, head weaving, or overgrooming, are often associated with immune dysregulation, gastrointestinal disorders, and skin lesions, consistent with negative psychosomatic processes (Mason & Latham, 2004; Novak et al., 2017).

Even in domestic environments, inadequate external enrichment can elicit chronic stress responses. For example, indoor-only cats lacking vertical space, hiding opportunities, or predictable routines may develop idiopathic cystitis or overgrooming behaviors due to underlying affective stress (Buffington et al., 2006). Horses subjected to stall confinement and restricted foraging exhibit increased gastric ulceration, stereotypy, and heightened startle responses that all are clinical outcomes consistent with psychosomatic etiology (Visser et al., 2008).

Environmental predictability and the capacity for control are critical variables mediating the affective and somatic impact of captivity and confinement in animals. Learned helplessness, as demonstrated in rodent and primate models, occurs when animals are exposed to stressors without the ability to predict or escape them, resulting in depressive-like behaviors and physiological suppression of immune and endocrine responses (Seligman, 1975; Mineka & Hendersen, 1985).

Conversely, environments that allow animals to engage cognitively, with opportunities for problem-solving, exploration, and agency, can attenuate the physiological consequences of stress. Enrichment interventions that increase environmental complexity have been shown to reduce both behavioral pathology and physiological stress indicators in multiple species (Young, 2003; Meehan & Mench, 2007).

In companion and laboratory animals, human-animal interactions significantly influence the stress profile and health outcomes of animals. Poor handling, inconsistent routines, or aversive training techniques increase fear and frustration, contributing to the affective instability underpinning PSD. On the other hand, predictable, positive, and responsive human interaction can buffer against the adverse effects of social deprivation and environmental monotony (Hemsworth & Coleman, 2011). Miscommunication or conflict in interspecies social contexts – such as the misreading of signals or punishment during stress – can also serve as chronic social stressors. These interactions often manifest in behaviorally mediated somatic symptoms such as vomiting, diarrhea, alopecia, or urinary retention (Overall, 2013).

Specific roles of cognition, affection, and reflexes in psychosomatic disorders

The multifactorial etiology of psychosomatic disorders in animals cannot be adequately understood without reference to the dynamic interplay between cognitive, affective, and reflexive systems. These three regulatory domains – closely interwoven across neuroanatomical and functional levels – serve as integrative mechanisms through which internal and external stimuli are interpreted, evaluated, and expressed somatically. While previous subsections have discussed these elements as parts of broader biological, psychological, and environmental factors, the present subsection aims to explicitly dissect how cognitive processing (e.g., expectation, learning), affective states (e.g., emotional responses), and reflexive mechanisms (e.g., autonomic reactivity) independently and collectively mediate vulnerability and response to psychosomatic stress.

Cognition in animals encompasses processes of perception, memory, associative learning, and expectation. These functions are critical to predictive coding, a neurocognitive framework in which the brain generates models of likely outcomes based on prior experience (Friston, 2010). When there is a mismatch between expected and actual outcomes – particularly in socially or physically salient contexts – it may result in stress responses or the activation of defensive systems. Studies in dogs and rodents demonstrate that animals anticipate aversive or rewarding outcomes based on cues, and sustained exposure to unpredictable environments leads to anxiety-like behavior and physiological arousal (Harding et al., 2004; Burman et al., 2008). Learned helplessness, as demonstrated in primates and canines, occurs when the animal perceives an absence of contingency between action and outcome, leading to behavioral passivity and physiological dysregulation (Maier & Seligman, 2016). Additionally, maladaptive conditioning, such as the pairing of neutral stimuli with aversive events (e.g., veterinary clinic with pain), may lead to the persistence of stress-related somatic symptoms – gastrointestinal upset, urinary retention, tachycardia – even in the absence of the original threat (Steimer, 2002).

Emotions in animals are increasingly recognized as biologically rooted, evolutionary processes with measurable behavioral and physiological components. Emotions not only influence decision-making and attention but also initiate autonomic and neuroendocrine responses that shape somatic functioning (Panksepp, 2011; Anderson & Adolphs, 2014). For example, negative affective states such as fear, frustration, and anxiety are commonly associated with increased HPA-axis activity, elevated heart rate, reduced gastrointestinal motility, and immune modulation that are the hallmarks of many psychosomatic conditions. In dogs with separation anxiety, affective arousal leads to vomiting, diarrhea, and dermatologic self-injury, all in the absence of primary somatic disease (Dreschel, 2010). On the other hand, positive emotional states, when elicited through play, social bonding, or enrichment, are associated with parasympathetic activation and homeostatic regulation that is the evidence of the bidirectional influence of affection on somatic systems (Boissy et al., 2007; Mendl et al., 2010).

The autonomic nervous system (ANS) is a key mediator between central affective-cognitive processes and peripheral physiological function. Reflexive responses, which are largely involuntary, regulate cardiovascular tone, gastrointestinal activity, respiratory rate, and immune surveillance in response to internal and external stimuli. Hyperactivation of the sympathetic division during chronic stress leads to elevated norepinephrine and cortisol levels, promoting vasoconstriction, gastric acid secretion, and mucosal barrier breakdown with this contributing to the development of ulcers, colitis, and dermatitis

(McEwen & Wingfield, 2003). Conversely, the parasympathetic branch, particularly the vagus nerve, plays a critical role in downregulating inflammation and restoring homeostasis. That is the fact that explains the interest in vagal tone as a biomarker for resilience to PSD (Thayer & Lane, 2000). In practical terms, reflex-mediated somatic symptoms, such as vomiting in response to stress, colonic dysmotility during anxiety, or cardiovascular instability during fear, illustrate how the reflex system executes and reinforces affective-cognitive states at the somatic level (Goehler et al., 2005).

Cognition, affection, and reflexes are not separate mechanisms but components of a regulatory triad. Cognitive interpretations of threat or novelty, shaped by learning and context, give rise to affective responses that, in turn, activate reflexive somatic adjustments. Over time, chronic activation or dysregulation within this triad can lead to pathological feedback loops, such as a fearful anticipation of pain leading to gastrointestinal dysfunction, which reinforces the original fear response. This perspective underscores the necessity of multimodal assessment and treatment strategies in PSD, addressing not only physical symptoms but also emotional history, cognitive expectations, and autonomic tone. The following Table 4 summarizes the respective contributions of cognitive, affective, and reflexive domains to psychosomatic expression in animals, highlighting their mechanisms, outcomes, and clinical relevance.

Table 4
Integrative roles of cognition, affection, and reflexes in psychosomatic disorders in animals

Regulatory domain	Key functions	Mechanisms involved	PSD-linked somatic outcomes	Representative behavioral expressions	Illustrative examples / references
Cognition (e.g., expectation, learning, prediction)	Risk appraisal, learning, anticipation	Predictive coding, associative learning, memory	GI dysregulation, tachycardia, urinary retention	Avoidance, learned helplessness, anticipatory stress	Harding et al., 2004; Maier & Seligman, 2016
Affection (e.g., emotional arousal, mood states)	Emotional reactivity, behavioral prioritization	Amygdala activation, HPA axis stimulation, neuromodulation	Vomiting, diarrhea, immunosuppression, skin lesions	Agitation, withdrawal, excessive grooming, aggression	Dreschel, 2010; Panksepp, 2011; Anderson & Adolphs, 2014
Reflexes (e.g., autonomic reactivity)	Physiological homeostasis, rapid somatic response	ANS output, vagal tone, neuroimmune interaction	Stress colitis, ulcers, cardiovascular instability	Panting, collapse, trembling, stress-induced vomiting	Thayer & Lane, 2000; McEwen & Wingfield, 2003; Goehler et al., 2005
Interaction of Domains	Integrated stress processing	Cognitive-affective-reflexive feedback loops	Chronic inflammation, metabolic dysfunction, behavior-somatic cycles	Stereotypies, somatic self-injury, fear-based avoidance	Boissy et al., 2007; Friston, 2010; Mendl et al., 2010

Thus, the etiology of PSD in animals emerges from a complex network of biological, psychological, and environmental factors that dynamically interact. Genetic predispositions, including breed-specific neurochemical and endocrine sensitivity, set the stage for differential vulnerability. These innate factors are modulated by psychological experiences, particularly those involving fear, anxiety, and frustration, which exert sustained effects on neuroendocrine regulation and immune competence. In parallel, environmental conditions, such as social isolation, captivity stress, unpredictability, and human-animal relational quality, play a pivotal role in either exacerbating or mitigating affective dysregulation and its somatic consequences. Importantly, cognitive processes (e.g., expectation, learning), affective states (e.g., emotional arousal), and reflexive autonomic responses (e.g., sympathetic activation) work as integrative systems through which external stimuli are interpreted and expressed physically.

Together, these findings underscore the need for an integrative etiological model of PSD in animals, a model that does not isolate pathology within either the body or the mind but rather accounts for the reciprocal regulation between cognitive-affective experience and physiological function. This understanding holds direct implications for diagnosis, prognosis, and treatment of animal diseases. It demands interdisciplinary approaches that take into account the biopsychosocial complexity of animal health and welfare.

Classification of psychosomatic disorders in animals

A precise and functional classification of PSD in animals is essential for clinical recognition, differential diagnosis, and development of targeted interventions. Given the interdependence of cognitive, affective, and physiological systems in animals, PSD can affect virtually all organ systems. It often mimics or overlaps with primary somatic disease. In this section of the article, we provide an overview of the most affected systems, the patterns of symptom presentation, and the mechanisms through which emotional and cognitive dysregulation manifest somatically. A classification based on systemic involvement will offer a practical framework for veterinary behavioral medicine and comparative pathophysiology.

Psychosomatic disorders in animals follow system-specific pathways, often with functional disruption rather than structural pathology being developed. Among the most affected systems are the gastrointestinal, dermatologic, and cardiovascular systems, each reflecting the somatic endpoints of affective and cognitive dysregulation. The gastrointestinal tract is highly sensitive to emotional states due to dense innervation by the enteric nervous system (ENS), its bidirectional communication with the central nervous system via the gut-brain axis, and its high responsiveness to stress hormones and inflammatory cytokines. In dogs and cats, chronic stress is frequently associated with vomiting, diarrhea, constipation, anorexia, or coprophagia, even in the absence of identifiable organic disease (Dreschel, 2010; German et al., 2010). In horses, stress-induced colitis, often linked to transportation, confinement, or social separation, manifests in similar clinical signs and is exacerbated by cortisol-induced changes in gut permeability and motility (Cunha et al., 2019). Such disorders parallel human irritable bowel syndrome (IBS), with overlapping features: visceral hypersensitivity, dysmotility, and central sensitization. In both humans and animals, chronic anxiety and early-life stressors are known predisposing factors (Moloney et al., 2016).

The skin, as an immunologically active organ and visible site, frequently reflects emotional dysregulation through both neurogenic inflammation and behavioral self-injury. Conditions such as psychogenic alopecia, acral lick dermatitis, overgrooming in cats, and feather-plucking in birds are among the most documented dermatologic manifestations of PSD (Luescher & McKeown, 2009; Mills et al., 2013). These behaviors may initially serve as coping strategies but later they often become compulsive, leading to secondary infections, tissue damage, and chronic inflammation. Stress-induced activation of mast cells, increased histamine release, and HPA dysregulation further contribute to the pathophysiology of these conditions (Mueller & Tzivian, 2019). Importantly, many of these disorders respond poorly to dermatologic treatment alone and require behavioral modification and psychotropic intervention. For example, dogs with separation anxiety frequently present with both gastrointestinal disturbances and dermatologic self-injury, such as excessive licking or chewing, often requiring integrated behavioral and medical veterinary care (Dreschel, 2010; Overall, 2013).

Though less frequently reported, cardiovascular involvement in PSD has been documented, particularly in species predisposed to stress reactivity (e.g., dogs, primates, rabbits). Emotional stress can provoke arrhythmias, tachycardia, blood pressure dysregulation, and in some cases, stress-induced cardiomyopathy. In canine patients, chronic an-

xiety and fear have been linked to reduced heart rate variability, a biomarker of autonomic imbalance and poor parasympathetic tone (Maros et al., 2013). This dysregulation may be associated with syncope (transient loss of consciousness due to reduced cerebral blood flow), exercise intolerance, or sudden cardiac events in susceptible animals. Case reports have documented transient alterations in cardiac function following acute emotional stress, resembling the stress-induced cardiomyopathy seen in humans (Fukuda et al., 2015).

While these system-specific manifestations appear distinct, they frequently co-occur, reflecting having shared regulatory mechanisms. For instance, a dog with separation anxiety may exhibit both dermatologic self-injury and gastrointestinal upset, illustrating the wide-ranging effects of a dysregulated emotional state on somatic systems (Ogata & Dodman, 2011; Landsberg et al., 2012; Overall, 2013). Moreover, these patterns highlight the importance of interdisciplinary diagnostics, where behavioral, neurological, and internal medicine expertise must interact to differentiate primary organic disease from psychosomatic etiology.

Table 5 shows a classification of psychosomatic disorders in animals by system involvement, behavioral indicators, and underlying psychological mechanisms. This integrative framework allows differential diagnosis and interdisciplinary treatment planning.

Table 5
System-based classification of psychosomatic disorders in animals

System affected	Common PSD manifestations	Associated behaviors	Underlying affective/cognitive mechanisms	Examples / references
Gastrointestinal	Vomiting, diarrhea, constipation, anorexia, coprophagia, stress colitis	Pica, food refusal, house soiling	Chronic anxiety, unpredictability, separation stress, learned helplessness	German et al., 2010; Moloney et al., 2016; Cunha et al., 2019
Dermatologic	Psychogenic alopecia, acral lick dermatitis, feather plucking, overgrooming	Self-licking, tail-biting, excessive scratching	Social frustration, compulsivity, displacement, sensory deprivation	Luescher & McKeown, 2009; Dreschel, 2010; Mills et al., 2013
Cardiovascular	Tachycardia, arrhythmia, hypotension, stress-induced cardiomyopathy	Panting, collapse, exercise intolerance	Affective arousal, chronic fear, loss of control	Maros et al., 2013; Fukuda et al., 2015
Neuroendocrine / Immune	Immune suppression, altered cortisol rhythms, increased inflammatory markers	Flattened affect, loss of reactivity	Emotional blunting, chronic stress exposure	McEwen & Wingfield, 2003; Tiira & Lohi, 2014
Multisystemic / Combined	Co-occurrence of GI, dermatologic, cardiovascular, immune dysfunctions	Multiple self-directed or stereotypic behaviors	Long-term emotional dysregulation, trauma history	Ogata & Dodman, 2011; Overall, 2013

The classification of PSD in animals is incomplete without paying attention to the behavioral expressions that precede, accompany, or result from underlying physiological dysfunction. These indicators often serve as early warning signs of affective-cognitive dysregulation and frequently co-occur with systemic manifestations such as dermatologic injury, gastrointestinal upset, or immune suppression. This subsection highlights three major categories of behavioral markers commonly observed in PSD: self-harm behaviors, stereotypies, and social or environmental deprivations.

Self-directed aggression, including acral licking, tail biting, excessive scratching, or feather plucking, often emerges in response to emotional arousal, social frustration, or sensory deprivation. These behaviors typically arise in the absence of external threats and are not driven by organic pathology, although they may later result in secondary somatic injury such as ulceration, infection, or alopecia (Luescher & McKeown, 2009; Overall, 2013). Such behaviors are frequently associated with compulsive or obsessive behavioral syndromes and are considered to function as maladaptive coping mechanisms, particularly in environments lacking possibility for control or predictability (Mills et al., 2013). For example, cats with anxiety or conflict-related stress may develop psychogenic alopecia, while dogs with separation-related problems may engage in excessive paw licking or tail chewing (Dreschel, 2010; Ogata & Dodman, 2011). Importantly, self-harm behaviors are often interpreted incorrectly as dermatologic disease, leading to applying of ineffective pharmacologic interventions unless their psychological basis was addressed.

Stereotypies are defined as repetitive, invariant, and seemingly purposeless behaviors that persist across time and context. Common examples in animals include circling, pacing, bar-biting, flank-sucking, head weaving, and repetitive vocalizations. These behaviors are particularly prevalent in captive animals, such as zoo mammals and laboratory rodents, as well as in domestic animals with restricted environmental or social complexity (Mason & Latham, 2004; Novak et al., 2017). The development of stereotypies is often associated with early-life stress, environmental monotony, or inaccessible behavioral needs (e.g., for locomotion, foraging, or social interaction). Stereotypies may co-occur with or predispose to somatic dysfunctions. That is because they may have common pathophysiological mechanisms involving autonomic dysregulation, HPA axis hyperactivity, or impaired immune modulation (McMillan, 2017). In some species, such as horses and pigs, stereotypic behaviors are associated with gastrointestinal lesions, including ulcers and colitis, suggesting a bidirectional relationship between behavioral pathology and physical disease (Visser et al., 2008).

Another important but often overlooked class of behavioral indicators in animals with PSD is withdrawal from social interaction and environmental engagement. Animals experiencing chronic emotional stress or learned helplessness may exhibit reduced play behavior, loss of exploratory drive, decreased social behavior, and lowered affective responses (Paul et al., 2005; Mendl et al., 2010). These behaviors reflect a depressive-like phenotype and often precede somatic decline. Social withdrawal has been particularly well documented in non-human primates exposed to early maternal separation, in dogs suffering from chronic anxiety or neglect, and in farm animals housed in barren or overcrowded conditions (Hemsworth & Coleman, 2011; Sanchez et al., 2015). In such cases, physiological markers such as suppressed immune function, altered cortisol rhythms, and gastrointestinal dysregulation frequently occurred. Unlike overt stereotypies or self-harm, withdrawal symptoms are subtle and require proper interpretation within a species-typical ethogram to be clinically informative.

Thus, behavioral indicators of PSD in animals are not merely superficial signs of distress but diagnostically meaningful phenomena that offer insight into the internal affective-cognitive state of the individual. Their presence calls for comprehensive biopsychosocial assessment, as well as caution against only symptomatic treatment approaches. Moreover, the co-occurrence of behavioral pathology and somatic illness highlights the need for interdisciplinary collaboration between veterinarians, behaviorists, and ethologists in both clinical and welfare settings. This co-occurrence also clearly indicates the high possibility of PSD development in the affected animals.

Pathogenesis and neurobiological mechanisms of PSD in animals

Understanding the pathogenesis of PSD in animals requires an integrative perspective on how stress interacts with cognition, emotion, and reflexes to disrupt physiological equilibrium. The biological mechanisms underlying these disorders are primarily mediated through neuroendocrine, autonomic, and neuroimmune systems. They all collectively translate environmental and affective stimuli into somatic outcomes. These pathways operate within a dynamic feedback loop that links psychological appraisal (cognitive evaluation of a stimulus), emotional arousal (physiological activation that follows psychological appraisal, in other words – the autonomic and endocrine changes that prepare the body for action), and visceral regulation (how bodily systems react in the response). In this article we will

explore the central neurobiological mechanisms involved in PSD, focusing on the HPA axis, autonomic nervous system (ANS), neuroimmune responses, and the modulatory influence of cognitive and affective states on somatic function. The discussion will highlight the translational relevance of findings from human and animal models, emphasizing how species-specific behavioral and physiological traits shape the expression of psychosomatic phenomena in animals.

Table 6 summarizes neurobiological mechanisms implicated in the development of PSD in animals. These systems interact to mediate the transformation of stress and emotional dysregulation into somatic pathology.

Table 6
Neurobiological mechanisms in the pathogenesis of psychosomatic disorders in animals

System / mechanism	Primary function	Pathological alterations in PSD	Clinical manifestations	Key references
HPA axis	Regulates stress response via CRH, ACTH, and cortisol secretion	Chronic activation; glucocorticoid receptor desensitization; disrupted feedback inhibition	GI dysfunction, immunosuppression, metabolic imbalance, behavioral rigidity	McEwen, 2008; Koolhaas et al., 2011
Autonomic nervous system	Maintains visceral homeostasis; coordinates sympathetic-parasympathetic balance	Sympathetic overdrive; reduced vagal tone; altered HRV	Tachycardia, GI dysmotility, cardiovascular instability, stereotypic behavior	Thayer & Lane, 2009; Maros et al., 2013
Neuroimmune interface	Mediates crosstalk between nervous and immune systems	Cytokine dysregulation; glucocorticoid resistance; microglial priming	Lethargy, anorexia, anhedonia, increased infection risk, psychodermatoses	Dantzer et al., 2008; Capuron & Miller, 2011
Microglial activation	Maintains neural immune surveillance; modulates synaptic plasticity	Proinflammatory shift in limbic regions (e.g., hippocampus, amygdala)	Cognitive impairment, emotional dysregulation, behavioral inflexibility	Kreisel et al., 2014; Calcia et al., 2016
Cytokine signaling (IL-1β, TNF-α, IL-6)	Triggers immune response; modulates brain–body communication	Chronic elevation; CNS penetration; disruption of neural circuits	Sickness behavior (withdrawal, apathy), visceral hypersensitivity, stress-related inflammation	Kelley et al., 2003; Frank et al., 2019

The HPA axis and the ANS constitute the two primary effector systems of the vertebrate stress response. In the norm their coordinated activity enables an organism to maintain homeostasis in the face of environmental or internal challenges. In psychosomatic disorders, however, chronic or dysregulated activation of these systems leads to allostatic overload, resulting in pathological alterations in both behavior and somatic function. The HPA axis is centrally involved in mediating the neuroendocrine response to psychological and physical stressors. Activation begins in the hypothalamus, which secretes corticotropin-releasing hormone (CRH), stimulating the anterior pituitary to release adrenocorticotropic hormone (ACTH), which in turn prompts the adrenal cortex to secrete glucocorticoids (primarily cortisol or corticosterone, depending on species). Under acute stress, this cascade supports adaptive responses that are mobilizing energy reserves, suppressing inflammation, and modulating attention. However, in animals subjected to chronic stress, particularly in the absence of control or predictability, maladaptive activation of the HPA axis is triggered. This leads to persistent elevations or suppression of glucocorticoid levels, receptor downregulation, and feedback resistance (cortisol signaling back to the brain to reduce further release of stress hormones) (McEwen, 2008; Koolhaas et al., 2011).

Empirical studies in dogs with anxiety disorders, socially isolated primates, and stereotypic horses have shown altered cortisol rhythms, impaired feedback inhibition, and associations with somatic dysfunctions such as gastrointestinal disease and immunosuppression (Carlstead et al., 1993; Beerda et al., 1999; Moberg, 2000). In parallel with the HPA axis, the ANS, comprising the sympathetic and parasympathetic branches, rapidly adjusts visceral functions in response to perceived threats. Sympathetic activation promotes cardiovascular, respiratory, and metabolic readiness, while parasympathetic activity supports recovery and regulation. In PSD, however, chronic sympathetic dominance leads to reduced vagal tone, elevated heart rate, and altered gastrointestinal motility (von Borell et al., 2007; Maros et al., 2013). Dogs with separation anxiety, for instance, exhibit persistently elevated heart rate and reduced heart rate variability, markers of autonomic imbalance and poor stress resistance (Palestrini et al., 2010). In horses, confinement and restricted movement contribute to elevated sympathetic tone and stereotypies, correlated with gastric dysfunction and immunosuppression (Visser et al., 2008). Moreover, neurovisceral integration theory suggests that ANS function, particularly vagal modulation, is tightly coupled with affective regulation and cognitive flexibility. That implies that animals with dysregulated ANS functions may also display impairments in behavioral adaptability (Thayer & Lane, 2009).

Thus, the chronic dysregulation of the HPA axis and ANS undermines physiological stability across multiple systems. Elevated cortisol impairs gastrointestinal integrity, suppresses immune responses, and alters neurotransmitter function. Simultaneously, sympathetic overactivity increases cardiovascular risk and promotes visceral hypersensitivity (Ulrich-Lai & Herman, 2009). Together, these disruptions create a physiological substrate upon which psychological distress is expressed as somatic pathology, leading to PSD. Understanding these mechanisms allows biomarker-informed diagnostics (e.g., HRV, salivary cortisol). It also helps to develop preventive and treatment measures targeting stress-response systems, such as environmental enrichment, anxiolytic pharmacotherapy, and behavioral modification.

The intimate bidirectional relationship between the nervous and immune systems also plays an important role in the pathogenesis of PSD in animals. The concept of neuroimmune modulation acknowledges that psychological states, particularly those related to chronic stress, anxiety, or dysregulated affect, can influence immune responsiveness, alter inflammatory processes, and contribute to somatic disease even in the absence of pathogens or structural abnormalities. Conversely, we may suggest that peripheral immune activation can affect brain function and behavior, contributing to affective and cognitive dysregulation. With this it is important to highlight the key molecular and physiological mechanisms that link stress physiology with immune system modulation, focusing on glucocorticoids, proinflammatory cytokines, microglial activation, and immune-brain signaling pathways as mediators of psychosomatic phenomena in animals.

One of the principal pathways through which stress affects the immune system is via glucocorticoid secretion following activation of the HPA axis. In the short term, glucocorticoids, such as cortisol, exert immunosuppressive effects by inhibiting cytokine production, reducing lymphocyte proliferation, and preventing exaggerated inflammatory responses (Sapolsky et al., 2000; McEwen, 2008). However, in animals exposed to chronic psychological stress, this regulation becomes disrupted. Prolonged elevations in glucocorticoids can paradoxically lead to glucocorticoid resistance in immune cells, resulting in low-grade systemic inflammation, impaired wound healing, and increased susceptibility to infections or autoimmune phenomena (Segerstrom & Miller, 2004). These mechanisms have been implicated in a range of stress-related conditions in animals, including psychogenic dermatopathies, colitis, and stress-induced immunosuppression (Moberg, 2000; Dhabhar, 2009).

Proinflammatory cytokines such as interleukin-1β (IL-1β), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6) are released

both peripherally and centrally in response to immune challenge or chronic stress. These molecules are not only markers of immune activity but also potent modulators of behavior. When cytokines reach the central nervous system, via neural (vagal) or humoral routes, they influence neural circuits involved in motivation, arousal, appetite, and social behavior (Kelley et al., 2003; Dantzer et al., 2008). In animals, this mechanism contributes to what is called sickness behavior: a coordinated set of adaptive behavioral responses that include lethargy, anhedonia (inability to experience pleasure), anorexia, and social withdrawal. Although originally adaptive, when cytokine signaling becomes persistent or dysregulated, it can contribute to or mimic the clinical manifestations of PSD (Capuron & Miller, 2011). In this context, the immune system functions as a mediator of emotionally and cognitively driven behavioral and organic somatic outcomes.

Within the central nervous system, microglia, the brain's resident immune cells, respond to stress and inflammation by adopting either protective or neurotoxic phenotypes. Chronic psychosocial stress has been shown to drive microglia toward proinflammatory states by increasing neuronal excitability and disrupting synaptic plasticity, particularly in limbic structures such as the hippocampus and amygdala (Kreisel et al., 2014; Calcia et al., 2016). In animal models, these neuroimmune changes have been linked to affective dysregulation, cognitive deficits, and increased vulnerability to somatic disease (Frank et al., 2019). Although direct studies in companion animals remain limited, indirect evidence from behavioral and neuroendocrine assessments supports a similar role for neuroimmune mechanisms in veterinary contexts (Prolo et al., 2005; McMillan, 2017).

Thus, understanding neuroimmune mechanisms in animals with suspected PSD has important implications for diagnosis, biomarker development, and therapeutic options. Peripheral markers such as cytokine profiles, acute-phase proteins, and leukocyte ratios may serve as indicators of chronic stress or affectively driven immune modulation (Carere et al., 2010). Furthermore, interventions that modulate the neuroimmune interface, such as environmental enrichment, anti-inflammatory agents, or behavioral therapies that reduce allostatic load, may indirectly improve somatic outcomes in PSD-affected animals.

Modulatory effects of affective states and cognitive appraisal

Psychosomatic disorders in animals are not solely the consequence of adverse responses to stressors, but are also modulated by central and efferent influences, including affective states and cognitive evaluations. The mechanisms by which animals process, anticipate, and emotionally interpret their environment can significantly shape the intensity, duration, and somatic consequences of stress exposure. This section examines how emotions (affection), cognition, and their neurobiological interrelationships influence the onset and progression of PSD.

In non-human animals, affective states, ranging from fear and anxiety to frustration and depression, can substantially alter somatic function. Chronic negative emotional states are associated with heightened physiological reactivity, such as increased heart rate, elevated glucocorticoid levels, immune suppression, and altered gastrointestinal activity (Paul et al., 2005; Mendl et al., 2010).

Emotional responses are mediated through limbic structures, particularly the amygdala, hippocampus, and prefrontal cortex, which interact with the HPA axis and the autonomic nervous system (Panksepp, 2004; McEwen & Morrison, 2013). Activation of fear circuits, for instance, can lead to hyperactivation and persistent stress state, increasing susceptibility to PSD through sustained allostatic load. Importantly, individual differences in emotional temperament, e.g., behavioral inhibition, impulsivity, or neophobia, have been linked to different susceptibility to stress-related pathology in animals (Koolhaas et al., 1999; Carere & Locurto, 2011). Animals with poor emotional regulation capacities, usually those reared in socially restricted or unpredictable environments, often display enhanced "tear and wear" somatic responses to psychological stress.

Cognition refers to an animal's interpretation of environmental events and its ability to predict or maintain control over stressors. According to appraisal theories of stress, the perception of a stimulus as controllable, predictable, or escapable significantly attenuates its negative physiological impact (Lazarus & Folkman, 1984; Koolhaas et al., 2011). Animals capable of anticipating stressors, such as routine social separation or veterinary handling, may exhibit either habituation (adaptive modulation) or sensitization (maladaptive potentiation), depending on prior learning, their temperament, and context meaning. This cognitive mediation affects stress response magnitude via modulation of limbic-prefrontal circuit and stress effector systems (Paul et al., 2005; Bateson et al., 2011). Evidence from animal welfare research has demonstrated that a lack of environmental predictability or unclear social cues increases emotional instability and contributes to stereotypies, immune dysfunction, and gastrointestinal disorders (Boissy et al., 2007). Conversely, environmental enrichment and cognitive stimulation are associated with improved emotional stability and reduced somatic dysregulation (Young, 2003).

When animals repeatedly encounter aversive stimuli that they cannot predict or escape, they may develop learned helplessness. It is a state of behavioral passivity characterized by dulled HPA reactivity, immune dysregulation, and depressive behaviors (Maier & Seligman, 2016). This condition mirrors human depression and contributes to the development of PSD signs, such as anorexia, social withdrawal, or somatic painfulness. Learned helplessness has been experimentally induced in dogs and rodents and is associated with dysregulated neurotransmitter systems (serotonin, norepinephrine), reduced hippocampal neurogenesis, and altered neuroimmune responses (Sherman & Franklin, 2017; Willner, 2017). In practical veterinary settings, animals subjected to chronic pain, confinement, or harsh training methods may exhibit similar profiles, emphasizing the need for addressing cognitive-affective components of the disease.

Together, affective states and cognitive appraisal represent essential mediators of how animals evaluate their environment and generate somatic outcomes. These central-effective processes not only shape behavioral and physiological responses but also determine individual trajectories of defense or susceptibility to psychosomatic diseases. Incorporating assessments of affective and cognitive functioning, such as decision-making tests or behavioral profiling, enhances diagnostic precision and improves treatment outcomes in animals with psychosomatic disorders.

Role of stress-related reflexes in somatic dysfunction

Reflexes are rapid, involuntary responses to internal or external stimuli. They constitute fundamental components of the vertebrate nervous system's capacity for maintaining homeostasis. In the context of PSD, stress-related reflexes act as both mediators and markers of interaction between environmental load and somatic function. Reflexes encompass autonomic, neuroendocrine, and somato-visceral responses, and their dysregulation is increasingly recognized as a key mechanism linking stress to disease states.

Stress-responsive reflexes are centrally coordinated by the brainstem, hypothalamus, and limbic forebrain. They typically involve the ANS, particularly the sympathetic-adrenal-medullary axis, and vagal outputs of the parasympathetic system. Activation of these circuits results in characteristic changes: increased heart rate and blood pressure, bronchodilation, gastrointestinal stasis, piloerection, and pupil dilation that are classical elements of the "fight-or-flight" response (Ulrich-Lai & Herman, 2009). Reflex pathways also include viscerosensory input apparatus, such as baroreceptors, chemoreceptors, and nociceptors. They direct stress-induced visceral sensations to central structures, thereby closing the regulatory loop. This dynamic feedback system allows fine-tuned adjustments to stressors, but when hyperactivated or chronically stimulated, it leads to maladaptive outputs (LeDoux, 2000; Koob, 2008).

The vagal nerve plays a crucial inhibitory role in balancing sympathetic overdrive, modulating immune responses, and supporting gastrointestinal integrity. Decreased vagal tone, often measured via HRV, is associated with increased vulnerability to PSD, particularly in animals with high stress loads or chronic emotional disturbance (von Borell et al., 2007; Maros et al., 2013). In dogs, for example, low HRV indexes correlate with noise phobia, separation anxiety and stereotypic behaviors. All of them are known to co-inside with soma-

tic disturbances such as diarrhea, skin lesions or recurrent vomiting (Palestrini et al., 2010). Reflex slowing of gut motility, due to parasympathetic inhibition and sympathetic activation, is particularly relevant in stress-induced colitis, gastric ulcers, and vomiting syndromes, as reported in canine and equine medicine (Nagel et al., 2019; Sjögren et al., 2021).

Repeated exposure to aversive stimuli can lead to conditioned somato-visceral reflexes, wherein innocuous stimuli (e.g., confinement, handling, transportation) elicit exaggerated physiological responses such as hyperventilation, tachycardia, defecation, or even seizures in neurologically intact animals. That means that after repeated exposure to unpleasant experiences, an animal can become so sensitized that even harmless routine situations like being confined, handled, or transported (which normally wouldn't cause distress) can trigger strong physiological stress reactions. These responses are often mediated via the amygdala-hypothalamus-brainstem axis and persist even when the original stressor is no longer present (Maier & Watkins, 2005). This mechanism is a basis for a variety of behavioral syndromes with somatic presentations, such as refusal to eat, urinary retention, or self-mutilation, all of which have no clear organic cause but reflect dysfunctional reflex regulation secondary to emotional trauma or chronic stress condition (McMillan, 2017).

As animals experience cumulative stress, the threshold for triggering aversive reflexes may be lowered. This phenomenon is known as sensitization. Sensitized animals react to minimal stimuli with excessive autonomic and endocrine output, with that exacerbating somatic deterioration. This is seen in stress myopathies, tachyarrhythmias, and inflammatory responses that are often following behavioral stressors in previously healthy animals (Moberg, 2000; Dhabhar, 2009). Furthermore, sustained activation of stress reflexes contributes to autonomic imbalance, with dominance of sympathetic output and impaired vagal restoration. This imbalance not only disrupts homeostatic mechanisms but creates a ground for stress-sensitized somatic diseases, perpetuating a vicious cycle of chronic psychosomatic syndromes (Thayer & Lane, 2009; McEwen & Gianaros, 2011).

Monitoring stress-related reflex markers, such as pupil dilation, HRV, cutaneous reflex thresholds, or gastrointestinal motility changes, offers valuable diagnostic and prognostic information in suspected cases of PSD in animals. Treatments targeting reflex normalization (e.g., biofeedback, anxiolytics, prokinetics, or vagal nerve stimulation) may represent effective approaches for restoring physiological balance (Rosenthal & Bhatia, 2015; Yap et al., 2020). Veterinary behavioral medicine thus may benefit from incorporating reflex-based diagnostics and treatments into standard somatic care. This is particularly true in animals with recurrent, stress-induced, or idiopathic illnesses.

Taken together, it may be concluded that PSD in animals cannot be explained solely through either bodily or tissue perspectives. Rather, these disorders emerge from the dynamic interplay of somatic, neuroendocrine, immune, cognitive, affective, and reflexive systems. Each of them influences one another in a context-dependent manner. This multi-based understanding calls for the appropriation of integrated diagnostic and therapeutic approaches in veterinary medical practice.

Diagnosis and clinical assessment of psychosomatic disorders in animals

Diagnosing psychosomatic disorders in animals remains a complex and evolving area that demands the intersection of behavioral medicine, internal medicine, and neurophysiology. Unlike for structural or infectious pathologies, for PSD characteristic is the presence of somatic symptoms without a clearly identifiable organic cause. The signs are often emerging within the presence of chronic stress, emotional dysregulation, or behavioral conflict. Given the absence of pathognomonic markers, diagnosis relies on careful exclusion of physical disease, detailed behavioral history, and the use of integrative assessment strategies tailored to species, context, and individual variability. This section provides an evidence-based overview of current and emerging diagnostic approaches, including behavioral phenotyping, physiological biomarkers, and species-specific considerations, while emphasizing the need for a structured, multidisciplinary methodology. A major foundational principle in diagnosing PSD in animals is that it is a diagnosis of exclusion, requiring the veterinarian to first rule out identifiable somatic causes using comprehensive clinical, laboratory, and imaging assessments (Overall, 2013; Mills et al., 2014). Only when physical illness, infection, injury, or structural abnormalities are excluded should a possible psychosomatic etiology be taken into account.

History-taking must be extensive, context-sensitive, and species-specific. It has to be capturing not only medical details but also information about the animal's environment, routine, social structure, recent events, and behavioral changes. Crucial information includes onset and pattern of symptoms (acute, recurrent, situational), relation to environmental or social triggers (e.g., relocation, loss of conspecific, change in caregiver), behavioral signs (withdrawal, hypervigilance, aggression, compulsivity), housing conditions and enrichment, feeding and handling routines ets. Owner or caretaker interviews should use structured behavior questionnaires, such as the Canine Behavioral Assessment and Research Questionnaire (C-BARQ) or similar validated instruments to systematically assess mood, stress reactivity, and behavioral tendencies (Hsu & Serpell, 2003; Mendl et al., 2010). In zoos or laboratory settings daily observation data and ethograms may provide deeper insight into behavioral deviations.

Standard diagnostic workup should include full physical and neurological examination, complete blood count, biochemistry panel, urinalysis, and species-appropriate imaging modalities (e.g., radiography, ultrasonography). Additional tests, such as thyroid function, cortisol levels, serologic assays, and endoscopy, may be needed depending on clinical presentation. In cases of suspected PSD with gastrointestinal symptoms (e.g., vomiting, diarrhea, anorexia), exclusion of inflammatory bowel disease, neoplasia, parasitism, or food hypersensitivity is essential. Similarly, dermatologic signs (e.g., alopecia, excoriations, pruritus) must be evaluated for presence of ectoparasites, fungal infections, or allergic dermatoses (White, 2011; Paterson, 2019).

Even when no organic cause is found, clinicians must be still cautious not to assume psychogenic origin prematurely. The absence of lesions or laboratory abnormalities does not confirm PSD. It rather shifts diagnostic focus toward behavioral and environmental causality. The concept of functional somatic syndromes, well described in human medicine (e.g., fibromyalgia, irritable bowel syndrome), is increasingly applicable in veterinary contexts, particularly for animals in chronic confinement, social isolation, or stress-prone environments (Prolo et al., 2005; McMillan, 2017). It means that animals can also exhibit persistent, non-specific somatic distress (widespread musculoskeletal pain, heightened pain sensitivity (hyperalgesia), and associated symptoms such as fatigue, sleep disturbances, cognitive difficulties, and emotional distress) resulting from chronic stress or emotional dysregulation, even in the absence of detectable pathology.

Finally, given the multifactorial nature of PSD, diagnostic accuracy may be improved when clinical veterinarians closely work with veterinary behaviorists, animal welfare scientists, and what is also important, with owners, trainers, and zookeepers. Such interdisciplinary collaboration allows the merger of data from medical, behavioral, and contextual sources, enabling a more robust and ethically sound diagnosis (Mills & Hall, 2014).

Use of behavioral assessments and scoring systems

In the diagnosis of PSD in animals, the integration of behavioral assessments with quantitative scoring systems is essential for identifying abnormal emotional states, maladaptive responses to stress, and persistent behavioral deviations with potential somatic consequences. These tools provide structured, replicable means to characterize the affective, cognitive, and reflexive profiles of animals in clinical and research settings. Behavioral assessments complement physiological and medical diagnostics by elucidating how animals perceive, respond to, and cope with environmental stimuli. It is crucial to have such insights while dealing with disorders where stress and emotion underlie somatic expressions (Mills et al., 2014; McMillan, 2017).

This section reviews standardized instruments, observational strategies, and key behavioral metrics relevant to the assessment of PSD.

Standardized owner- or caregiver-administered questionnaires allow systematic evaluation of behavioral history and temperament. Among the validated tools these three are the most widely used. Canine Behavioral Assessment and Research Questionnaire (C-BARQ) was developed to assess a range of behavioral traits in dogs, including fearfulness, aggression, separation-related behaviors, and trainability. C-BARQ is widely used in both clinical behavior practice and epidemiological studies (Hsu & Serpell, 2003). Feline Behavioral Assessment and Research Questionnaire (Fe-BARQ), a feline-adapted version is used for assessing anxiety, aggression, compulsive behaviors, and inappropriate elimination (Serpell & Duffy, 2016). Equine Behavior Assessment and Research Questionnaire (E-BARQ), developed most recently to quantify learning ability, compliance, anxiety, and sensory responsiveness in horses (Fenner et al., 2020). These tools enable early identification of behavioral syndromes that may predispose animals to PSD. The syndromes include chronic fear, impulsiveness or maladaptive emotional bond between an animal and a caregiver, conspecific, or social environment. All of them are linked to somatic stress markers such as gastrointestinal dysmotility or immunosuppression.

In settings where owner reports are unavailable or insufficient (e.g., zoos, farms, shelters), ethograms, standardized lists of species-specific behaviors, are critical. Structured behavioral observation allows assessment of frequency and duration of abnormal behaviors (e.g., pacing, excessive grooming, vocalization), withdrawal or hypoactivity in response to human or conspecific interaction, altered feeding, grooming, or sleeping patterns that are indicative of emotional distress, reflexive or stress-induced behaviors – startle reactions, urination, or defecation upon handling. Protocols such as the Five Domains Model and Qualitative Behavioral Assessment (QBA) provide frameworks for interpreting affective states and welfare-related behaviors in animals, combining objective ethology with subjective evaluation of demeanor and mood (Wemelsfelder et al., 2000; Mellor et al., 2020).

More recently, cognitive bias testing has emerged as a robust method to assess affective states and the form of their modulation by environmental or therapeutic factors. These tests measure whether an animal interprets ambiguous stimuli optimistically or pessimistically that may reflect its internal emotional state (Harding et al., 2004; Baciadonna & McElligott, 2015). For example, dogs exposed to unpredictable environments show pessimistic bias (it takes longer to approach unfamiliar objects), which correlates with increased anxiety and stress reactivity (Burman et al., 2011). This method has been applied to assess the effects of enrichment, social support, or pharmacological interventions in mitigating PSD symptoms. Certain conditions with psychosomatic components, such as feline idiopathic cystitis, equine gastric ulcer syndrome, and canine compulsive disorder, now include scoring systems that combine behavioral and clinical indicators. The Feline Stress Score (FSS) evaluates acute and chronic stress responses in cats based on posture, vocalization, and activity (Kessler & Turner, 1997). The Horse Grimace Scale (HGS) and Dog Grimace Scale estimate pain or discomfort that may be modulated by affective state (Dalla Costa et al., 2014). All of these tools support differential diagnosis by highlighting the role of affective disturbance and corresponding behavioral conflict in somatic symptomatology.

While behavioral assessments provide valuable data, their limitations include observer bias, variability across species and individuals, and incomplete validation for some tools. Moreover, behavioral manifestations may be subtle or intermittent, necessitating repeated assessments and contextual interpretation (Mills & Hall, 2014). For maximal clinical utility, these instruments must be integrated with physical examination, owner consultation, and longitudinal follow-up. When used systematically, they may contribute to early recognition of PSD risk, inform preventive or therapeutic strategies, and support welfare monitoring in clinical, shelter, and zoological environments.

Emerging diagnostic tools for psychosomatic disorders in animals

As PSD in animals increasingly demands multidimensional diagnostic approaches, the integration of non-invasive physiological and neurobiological tools has become a central focus in veterinary behavioral science. Emerging technologies offer objective biomarkers for emotional dysregulation, autonomic imbalance, and stress-induced somatic change, complementing conventional behavioral and clinical assessments. Here we review key emerging tools, such as salivary cortisol measurement, infrared thermography, and neuroimaging techniques, which provide novel insights into stress-related physiological and neural processes underlying PSD in animals.

Cortisol, the principal glucocorticoid in many mammals, is a validated biomarker of HPA axis activation and a proxy for stress reactivity. Salivary cortisol measurement is particularly advantageous due to its non-invasive, stress-minimizing, and species-flexible nature. Studies in dogs, cats, horses, and non-human primates have demonstrated that elevated salivary cortisol levels are associated with acute stress, chronic anxiety, and exposure to aversive stimuli, while not pronounced responses may indicate chronic HPA dysregulation or learned helplessness (Beerda et al., 1997; Dreschel & Granger, 2005; Peeters et al., 2011). However, one must keep in mind that cortisol dynamics are also influenced by circadian rhythms, temperament, and previous experience. Therefore, repeated sampling, contextual interpretation, and comparison with behavioral data are essential for diagnostic relevance (Vincent & Michell, 1992; Mills et al., 2014).

Infrared thermography (IRT) detects changes in cutaneous blood flow through the measurement of skin surface temperature, which reflects autonomic nervous system activity, especially sympathetic vasoconstriction or vasodilation. Stress-induced activation of the sympathetic system typically results in peripheral cooling, particularly of the nasal planum, ears, and ocular region. For instance, dogs subjected to social stress or handling display nasal temperature drops, while horses exhibit periorbital cooling during painful or aversive procedures (Valera et al., 2012; Travain et al., 2015). IRT offers a real-time, contact-free method to detect subtle emotional changes preceding overt behavioral signs. It is particularly useful in assessing reactivity or distress in animals unable to express behavioral indicators clearly, such as prey species or highly stoic individuals.

Although still rare in routine veterinary settings, functional neuroimaging (fMRI, PET, SPECT) is emerging as a research tool to visualize brain activity patterns associated with emotion, pain, cognition, and behavioral pathology in animals. In canine studies, fMRI has been used to map amygdala and caudate activation in response to human voices or training cues (Berns et al., 2015). In non-human primates, brain imaging studies have shown that chronic stress disrupts the function of limbic regions and weakens the connections between the prefrontal cortex and the limbic system. These changes are associated with stress-related physical disorders (Kalin et al., 2005). Thus, while technical and ethical constraints limit routine use of these research tools, future advancements in portable imaging, machine learning analysis, and awake-animal protocols may facilitate their broader application in diagnosing affect-related somatic syndromes and monitoring treatment effects.

Additional diagnostic innovations include heart rate variability (HRV) as a marker of vagal tone and emotional regulation, electrodermal activity (EDA) reflecting sympathetic arousal, behavior-linked wearable sensors for tracking movement, posture, and environmental responses, machine vision systems that quantify facial expressions or body language (e.g., DogFACS, EquiFACS). These tools enable the development of multi-parameter stress profiling, integrating behavioral, autonomic, endocrine, and cognitive data for individualized PSD assessment (Zupan et al., 2016; Dolensek et al., 2020). Thus, the adoption of emerging diagnostic tools provides a critical bridge between observable behavior and underlying physiological states, improving the precision, timeliness, and ethical soundness of PSD diagnostics in animals. We believe that future clinical application depends on continued validation, standardization, and interdisciplinary cooperation between veterinarians, neuroscientists, and behaviorists.

Table 7 presents an integrated diagnostic framework for identifying PSD in animals. It gives a ground to state that diagnostic accuracy is enhanced by combining clinical exclusion of physical disease, stru-

ctured behavioral assessment, physiological biomarker analysis, and interdisciplinary interpretation. Furthermore, emerging diagnostic tools, such as cognitive bias testing and infrared thermography, even further improve the sensitivity and specificity of PSD detection across different species and environmental settings.

Table 7
Multidimensional diagnostic framework for psychosomatic disorders in animals

Diagnostic domain	Tools and methods	Targeted parameters	Interpretive value	Species/context examples
Clinical Exclusion of Organic Disease	Physical exam, CBC, biochemistry, urinalysis, imaging (USG, X-ray, MRI), endocrine panels	Rule-out of infection, inflammation, neoplasia, metabolic or structural disease	Essential first-line exclusion to identify functional vs. organic etiology	GI signs in cats without IBD or parasites may suggest psychogenic vomiting
Behavioral History and Interview	Structured owner/caregiver interview; questionnaires (C-BARQ, Fe-BARQ, E-BARQ); contextual analysis	Onset, triggers, routines, environment, social structure	Establishes behavioral patterns, chronicity, stress context	Separation anxiety history in dogs linked to GI symptoms and self-harm
Ethological Observation	Ethograms, daily logs, Five Domains Model, Qualitative Behavioral Assessment (QBA)	Frequency and intensity of abnormal behaviors; affective demeanor	Non-verbal assessment in shelters, zoos, farms; useful when owners not present	Pacing, hypoactivity, altered grooming in captive primates or horses
Quantitative Behavioral Scoring	C-BARQ, Fe-BARQ, E-BARQ; Feline Stress Score (FSS); Dog/Horse Grimace Scales	Anxiety, compulsivity, pain-related affect, stress indices	Standardized comparison across individuals, situations, and interventions	High FSS correlates with poor welfare and GI dysmotility in shelter cats
Cognitive Bias Testing	Ambiguous cue response tasks	Optimism/pessimism; emotional valence; behavioral flexibility	Reveals affective state and chronic stress impact	Dogs under stress show "pessimistic" interpretation of ambiguous stimuli
Biomarker Analysis	Salivary cortisol, fecal glucocorticoids, blood cytokines, HRV monitoring	HPA axis activity, autonomic balance, immune markers	Objective indexes of stress and emotion; requires repeated/contextual sampling	Blunted cortisol response in chronically stressed animals; low HRV in anxious dogs
Thermal and Physiological Imaging	Infrared thermography (IRT); thermographic stress mapping	Peripheral vasoconstriction, autonomic arousal	Early, non-invasive stress indicator prior to overt behavior	Nasal cooling in dogs under social stress; periocular drops in stressed horses
Functional Neuroimaging (Research Use)	fMRI, PET, SPECT	Brain activation patterns (limbic, cortical); connectivity	Experimental mapping of emotional-cognitive networks in behavior and somatic outcomes	Amygdala overactivation in fearful dogs; limbic disruption in chronically stressed primates
Wearables and Digital Monitoring	Accelerometers, GPS, EDA sensors, facial expression coding (e.g., DogFACS, EquiFACS)	Activity, posture, sympathetic arousal, micro-expression metrics	Real-time monitoring for subtle behavioral or stress-related changes	Inactivity spikes before stereotypies; equine facial tension preceding GI signs
Multidisciplinary Integration	Collaboration among vet clinicians, behaviorists, ethologists, owners, keepers	Cross-domain data synthesis	Enables biopsychosocial diagnosis; reduces risk of misattributing somatic signs to physical-only pathology	Interdisciplinary case review clarifies psychogenic origin of dermatologic or GI signs in dogs or cats

Species-specific diagnostic challenges

The diagnosis of PSD in animals requires a nuanced understanding of species-specific behavioral repertoires, stress physiology, and communication modalities. While the core pathophysiological mechanisms underlying PSD, such as chronic stress, emotional dysregulation, and autonomic imbalance, may be conserved across vertebrate species, clinical manifestations, diagnostic accessibility, and contextual interpretation vary considerably between different animal groups. In this section we emphasize that recognizing these differences is essential for improving diagnostic sensitivity, avoiding misinterpretation, and ensuring species-appropriate care.

In dogs, broad behavioral patterns and strong human-animal bonds may facilitate the recognition of emotional distress and behavioral pathology. However, breed-specific behaviors, such as compulsive tail chasing in Bull Terriers or noise sensitivity in herding breeds, may mask or mimic signs of PSD (Dodman et al., 2010; Overall, 2013). Moreover, anthropomorphic biases by owners can lead to over- or under-reporting of symptoms. In cats, solitary tendencies and inclination for withdrawal under stress complicate behavioral assessment. Many feline PSD manifestations are somatic, including feline idiopathic cystitis, inflammatory bowel symptoms, and psychogenic alopecia, with subtle or absent behavioral signs (Buffington, 2011). Diagnosis in cats often relies on ruling out organic disease and evaluating environmental stressors, since they typically do not display obvious emotional or behavioral signs.

Horses exhibit a range of stereotypies (crib-biting, weaving, box walking) and somatic syndromes (equine gastric ulcer syndrome, chronic colic) linked to confinement and stress (McBride & Cuddeford, 2001). However, their stoic pain responses and high prey vigilance may suppress overt behavioral expression. Diagnosis is further complicated by management factors (e.g., stabling, social isolation, feeding regimen) and a tendency to attribute behavioral problems to "training issues" rather than underlying distress. It is suggested that quantitative tools such as the E-BARQ and facial grimace scales aid the early detection of behavioral disturbances in horses (Dalla Costa et al., 2014; Fenner et al., 2020).

PSD diagnostics in livestock (cattle, pigs, sheep) are rarely performed at the individual level due to production pressures and herd-based management. However, increasing evidence links chronic stress and impoverished environments to immunosuppression, gastrointestinal dysfunction, and stereotypic behaviors, particularly in intensive systems (Meagher et al., 2019; Broom, 2001). Practical challenges here include the lack of validated behavioral scales, limited human-animal interaction, and underreporting of distress behaviors. Emerging modern research focuses on automated behavior monitoring, heart rate variability, and stress biomarker panels to improve misbehavioral detection.

In captive exotic species, PSD diagnosis is especially difficult due to limited ethological baselines, species-specific communication deficits, and lack of normative physiological data. Animals may display non-specific behaviors (e.g., pacing, overgrooming, self-injury) that require detailed ethograms and long-term observation to be interpreted meaningfully (Mason et al., 2007). Laboratory rodents and primates, despite controlled environments, often experience stress-induced stereotypies, impaired immune responses, or cognitive inflexibility, highlighting the need for refined housing, enrichment, and the use of cumulative welfare assessment tools (Balcombe et al., 2004; Novak et al., 2013). In avian species, particularly parrots, PSD often manifests as feather-plucking, self-mutilation, or gastrointestinal stasis, typically in response to social deprivation or environmental restriction. However, their highly social and vocal nature makes standard

stress assessments difficult to calibrate without extensive species-specific knowledge (van Zeeland et al., 2009). In reptiles, limited behavioral expression and poorly understood stress physiology render making PSD diagnosis speculative. Chronic environmental mismatch (e.g., thermal gradients, humidity) may lead to inappetence, dermatologic issues, or immobility, all of which can reflect both physical and affective compromise (Warwick et al., 2013). Thus, the diagnostic process for PSD in animals must be strongly supported by the knowledge about species-specific ethology, ecological needs, and neurobehavioral constraints. Misdiagnosis, whether through anthropomorphism, underestimation of pain or stress, or neglect of environmental context, can lead to incorrect diagnosing and consequently to ineffective or even harmful interventions. Advancing species-appropriate diagnostic standards require collaborative research, proved welfare-focused tools, and continuous refinement of behavioral and physiological markers across taxa.

Prevention and treatment strategies for psychosomatic disorders in animals

Effective prevention and treatment of PSD in animals must address the multifactorial etiology and interrelation of emotional, cognitive, and reflexive regulation. In PSD cases the interventions should not be limited to pure somatic renovation but aim at restoring homeostasis in neuroendocrine, immune, and behavioral systems, mitigating the underlying causes of stress, and enhancing resilience. Unlike purely somatic diseases, PSDs are context-dependent, influenced by an animal's perception of safety, social support, predictability, and control over its environment. That is why comprehensive therapeutic strategies are required. They should include environmental enrichment, behavioral interventions, and, when appropriate, pharmacological modulation of affective and cognitive pathways. Also, important to these approaches is a shift from reactive treatment to proactive prevention, guided by an understanding of species-specific needs and welfare indicators. Here there will be outlined evidence-based strategies for the prevention and treatment of PSD in animals, beginning with environmental and management-level interventions.

Environmental enrichment is a cornerstone of both the prevention and amelioration of PSD. Defined as the enhancement of an animal's living conditions to promote natural behaviors, cognitive engagement, and emotional stability, enrichment interventions are particularly effective in reducing chronic stress, which is a central driver of psychosomatic pathology. A lack of stimulation, predictability, and control, especially in captive, domesticated, or socially restricted animals, results in dysregulation of the HPA axis, leading to maladaptive affective states and physiological disturbances (Meehan & Mench, 2007). Environmental enrichment counteracts the problem by improving the animal's sense of control, reducing boredom and frustration, and by supporting adaptive coping mechanisms. For instance, predictable routines, accessible places to escape, foraging opportunities, and interactive objects have been shown to decrease the incidence of stereotypies, vocalization, and stress-induced somatic signs in dogs, cats, horses, rodents, and zoo animals (Young, 2003; Wells, 2009). Importantly, the buffering effect of enrichment appears to modulate both baseline cortisol levels and acute stress reactivity, which indicate physiological as well as behavioral benefits (Gottlieb et al., 2019).

Effective enrichment strategies vary by species and context but generally fall into five categories: physical enrichment (e.g., varied substrates, climbing structures, hiding places), social enrichment (e.g., conspecific interaction, caregiver bonding), cognitive enrichment (e.g., problem-solving tasks, novelty exposure), sensory enrichment (e.g., olfactory stimulation, auditory variation) and feeding enrichment (e.g., puzzle feeders, scatter feeding). In dogs, enrichment that includes nose work games, structured play, and training exercises reduces behavioral manifestation of anxiety and improves sleep and gastrointestinal regularity, which is a common PSD-associated somatic issues (Rooney et al., 2009). In laboratory animals, enriched cages with tunnels, shelters, and manipulable objects decrease gastrointestinal ulceration, immunosuppression, and odd grooming (Van de Weerd & Day, 2009).

Enrichment programs must be species-appropriate, goal-directed, and preferably tightly customized to an animal's preferences, history, and behavioral profile. Enrichment that is inappropriate or excessively stimulating may itself become a stressor, especially in animals with heightened arousal or cognitive impairment (Olsson & Dahlborn, 2002). Best enrichment practices include varying enrichment type and presentation to maintain novelty, ensuring opportunities for choice and control, monitoring behavioral and physiological responses to enrichment and integrating enrichment into routine husbandry and treatment protocols. Enrichment not only mitigates existing PSD signs but also may prevent their onset by enhancing neuroplasticity and buffering stress pathways. Rodent studies show that early-life environmental enrichment develops resilience to later stressors by reducing both affective dysfunction and visceral hypersensitivity that are the core features of PSD (Francis et al., 2002). Similar findings are emerging in canine and equine populations, where enriched environments in early life are linked to reduced aggression, improved sociability, and enhanced behavioral flexibility (Clark et al., 1997).

Thus, environmental enrichment is a powerful, cost-effective, and ethically sound strategy for preventing and alleviating psychosomatic disorders across animal species. Its role in modulating affective states, cognitive engagement, and autonomic stability makes it irreplaceable in both clinical and welfare-aimed practice. As our understanding of PSD deepens, so must the design, implementation, and evaluation methods of enrichment protocols that have to be tailored to individual animals and specific environmental contexts.

Behavioral therapy (desensitization, counterconditioning, cognitive stimulation)

Behavioral therapy is an obligatory component of PSD treatment in animals, as it directly addresses the affective dysregulation, maladaptive learning, and stress-induced behavioral patterns that underlie and perpetuate somatic dysfunction. Unlike purely pharmacological approaches, behavioral interventions target the causal psyche-dependent mechanisms and foster adaptive emotional responses, restoring functional coping strategies and facilitating the reducing of somatic injury. Here are outlined the major components of behavioral therapy such as systematic desensitization, counterconditioning, and cognitive stimulation, which are applicable across companion, captive, and farmed animal populations.

Systematic desensitization is a gradual process by which an animal is exposed to feared or stress-inducing stimuli at intensities below its threshold of reactivity, allowing for habituation and affective reassociation. It is particularly effective in treating PSD linked to specific trauma, phobias, and conditioned autonomic responses (Overall, 2013). For example, dogs with noise phobia or post-traumatic stress symptoms may exhibit gastrointestinal upset, excessive grooming, or somatic tension in response to triggering cues. When these cues are introduced incrementally, beginning with low-intensity sound playback or contextual simulation, physiological stress markers such as salivary cortisol and heart rate usually decrease, indicating limbic normalization (Sherman & Mills, 2008). Key principles of desensitization include maintaining sub-threshold intensity to avoid further sensitization. It is important to combine the sub-threshold stimuli with positive reinforcement to support calm behavior while monitoring for signs of stress (panting, avoidance, displacement behaviors).

Counterconditioning involves pairing the aversive stimulus with a positive outcome (e.g., food, play, touch), thereby transforming the animal's emotional attitude to that stimulus. Unlike mere exposure, it actively reshapes the affective memory associated with the trigger. In horses with handling-related PSD (e.g., stress-induced bodily unrest, mouth ulcers), pairing haltering with high-value food rewards and gentle tactile engagement has been shown to reduce cardiovascular reactivity and oral self-injury (Sankey et al., 2010). In captive primates, counterconditioning is employed to reduce grooming overuse, withdrawal, and stress-induced alopecia (Nevison et al., 1999). Effective counterconditioning in animals depends on timely pairing (positive consequence must immediately follow stimulus). There also have to be enough repetition events to establish a stable new association

and context control while avoiding unpredictable variables that might reinstate fear.

Cognitive stimulation plays a dual role in PSD management: (1) reducing learned helplessness and behavioral rigidity, and (2) enhancing resilience by activating dopaminergic and prefrontal circuits that support adaptive emotion regulation (Rosenzweig & Bennett, 1996). Forms of cognitive stimulation include problem-solving tasks for animals to engage in (e.g., puzzle feeders, scent trails), novel object exploration under low-stress conditions, choice-based environments, supporting free self-directed behavior and ability to express behavioral variation.

In dogs, cognitive training and enrichment have been linked to improved affective tone, reduction in somatic complaints (e.g., nausea, pacing), and enhanced HRV (Mills et al., 2014). In pigs and rodents, cognitive challenges have been shown to mitigate stereotypies and reduce stress-related visceral pathology, including gastric ulceration and altered immune responses, thereby supporting both therapeutic and preventive influence of the environmental enrichment (de Jong et al., 2000; Spinka, 2006; Van de Weerd & Day, 2009). Notably, cognitive interventions should be graded to ability of animals to avoid frustration (in compromised individuals). Practically, the interventions have to be monitored for signs of engagement or avoidance and integrated with affective support, ensuring emotional safety during challenge exposure.

Thus, behavioral therapy, when tailored to the individual's cognitive, affective, and reflexive profile, may represent a cornerstone of PSD treatment. Desensitization and counterconditioning dismantle maladaptive associations, while cognitive stimulation builds resilience and neuroplasticity, supporting long-term recovery. Definitely, these approaches require expertise in behavioral analysis, and stress physiology, emphasizing the necessity of interdisciplinary care teams' efforts in animal behavioral medicine.

Pharmacological approaches (anxiolytics, antidepressants, adaptogens)

Pharmacological interventions play an important supportive role in the management of PSD in animals, particularly when affective dysregulation, maladaptive arousal, or chronic stress activation compromises the animal's capacity to respond to environmental or behavioral therapies. In these cases, pharmacological modulation of neurotransmitter systems can stabilize mood, attenuate physiological stress responses, and enable learning processes that are critical for behavioral recovery (Overall, 2013; Landsberg et al., 2015). Here we examine three principal categories of pharmacologic agents used in veterinary behavioral medicine: anxiolytics, antidepressants, and adaptogens.

Anxiolytics are commonly used to reduce hyperarousal and autonomic reactivity, especially in acute stress or phobia-related PSD presentations. Benzodiazepines, such as diazepam and alprazolam, act on GABA-A receptors to produce rapid anxiolysis, muscle relaxation, and mild sedation (Crowell-Davis et al., 2005). They are often indicated in noise phobia, separation distress, or acute gastrointestinal motility disorders with a strong emotional component (Simpson, 2008). However, benzodiazepines must be used cautiously due to the risk of paradoxical excitation, possible dependence development, and amnesic disinhibition (loss of behavioral restraint combined with memory impairment), particularly in felines and some working breeds (Overall, 2013). Trazodone, a serotonin antagonist and reuptake inhibitor (SARI), has emerged as a safer alternative for situational anxiety in dogs. It has been shown to improve hospital-related stress tolerance and reduce somatic distress markers, including vomiting and pacing (Gruen et al., 2014).

Selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs) are the mainstream medicine for chronic affective disorders contributing to PSD, such as compulsive behaviors, chronic colitis with stress components, or psychogenic dermatoses (Hewson et al., 1998). Fluoxetine is the most widely studied SSRI in veterinary medicine. It is effective in treating canine compulsive disorder, feline urine spraying, and stress-related alopecia, by enhancing serotonergic tone and reducing limbic hyperresponsivity (Simpson, 2008; Landsberg et al., 2015). Clomipramine, a TCA with both serotonergic and noradrenergic activity, has demonstrated efficacy in canine separation anxiety, stereotypies, and feather plucking in birds (Luescher & McKeown, 2009). It is also associated with a reduction in stress-induced gastrointestinal disturbances through central autonomic modulation. Importantly, most of the antidepressants require chronic administration (typically 3–6 weeks) to achieve clinical effects and they should be paired with behavioral therapy for optimal outcomes. Side effects, including gastrointestinal upset, sedation, or anticholinergic effects, must be monitored, and constant dose titration is recommended.

Adaptogens are naturally derived substances that support the regulation of the HPA axis and improve resistance to stress without inducing sedation or overstimulation. They have gained increasing interest as adjunctive treatments in animals with subclinical PSD presentations or as preventive interventions in high-risk situations (Panossian & Wikman, 2010). L-theanine, an amino acid found in green tea, modulates glutamate and GABA transmission and has shown anxiolytic effects in dogs and cats, with reductions in heart rate and cortisol levels under stress (Pike et al., 2015). Zylkene® (alpha-casozepine), a milk-derived peptide, mimics GABA-A receptor modulation and has been associated with behavioral improvement in noise phobia and relocation stress in shelter animals (Beata et al., 2007). Herbal preparations such as *Valeriana officinalis*, *Withania somnifera*, and *Rhodiola rosea* have demonstrated anti-stress and neuroprotective effects in rodent models, reducing corticosterone levels, improving cognitive flexibility, and mitigating stress-induced somatic dysfunctions (Panossian et al., 2009). However, rigorous clinical trials in animals are limited, and standardization of dosing and formulation remains a challenge for veterinary practitioners.

Hence, pharmacological approaches, when applied judiciously and in conjunction with behavioral and environmental therapies, are valuable tools for stabilizing affective dysregulation, enhancing cognitive engagement, and interrupting destructive reflex-somatic feedback loops in animals with PSD. So far, selection of agents should be individualized, based on the animal's species, history, and comorbid conditions, and continually evaluated for efficacy and tolerability. We claim that ongoing research is needed to validate emerging compounds and establish evidence-based integrative protocols for treatment of animals with PSD.

Combined therapeutic models incorporating affective and cognitive dimensions

As was already noted, psychosomatic disorders in animals are complex conditions that emerge from the dynamic interplay of emotional states, cognitive processes, and reflexive physiological responses. While separate interventions, such as behavioral therapy, pharmacological treatment, or environmental enrichment, can yield significant improvement, a multimodal, integrative approach, that simultaneously targets affective regulation, cognitive restructuring, and somatic feedback loops offers superior and often more durable therapeutic outcomes (Crowell-Davis & Murray, 2006; Mills et al., 2014). In animals with established PSD, unidimensional treatments frequently fail to disrupt the acting and reinforcing cycles between stress, perception, and bodily dysfunction. For instance, in dogs with separation-related gastrointestinal distress, pharmacologic anxiolysis may suppress acute symptoms, but without addressing the underlying attachment insecurity or learned helplessness, the condition may often recur (Sherman & Mills, 2008). Similarly, enrichment alone may be insufficient if cognitive rigidity or emotional trauma blocks engagement with the environment (Olsson & Dahlborn, 2002). By combining cognitive and affective therapeutic elements, interventions can more effectively reduce limbic overactivation and sympathetic tone. This mutual effort can also enhance cognitive flexibility and learning of new associations, foster behavioral resilience through adaptive coping mechanisms and prevent relapses through neuroplastic reinforcement of healthy behaviors.

Here are some examples of combined models approaches in different animal species. Effective management for canine compulsive

disorder (CCD) integrates fluoxetine (to stabilize serotonergic tone), counterconditioning (to reduce stimulus-induced arousal), and cognitive enrichment (to restore behavioral diversity). This multimodal approach has been shown to significantly reduce the frequency and intensity of compulsive behaviors compared to monotherapy (Simpson, 2008; Ogata & Dodman, 2011). Feline idiopathic cystitis (FIC) is a stress-sensitive condition linked to both autonomic imbalance and affective instability. Integrated care typically includes environmental enrichment (e.g., vertical space, choice control), pheromone therapy, interactive play, and nutritional modulation (Buffington et al., 2006). It was shown that these strategies not only reduce urinary signs but also improve social behavior and reduce avoidance responses. Treatment protocols for captive parrots with feather-damaging behavior combine behavioral desensitization, SSRIs or anxiolytics, and cognitive foraging challenges to restore species-specific activity patterns, reduce anxiety, and modify dysfunctional grooming habits (Van Zeeland et al., 2009).

Thus, successful implementation of combined therapeutic models requires four main approaches. Multidimensional assessment as a thorough evaluation of behavioral history, environmental context, and physiological indicators to inform intervention selection (Landsberg et al., 2015). Individualized planning as a modulation of intervention intensity, dosing, and modalities to the animal's personality, history, and stress resilience. Iterative feedback as an ongoing assessment of response to treatment, with readiness to adjust treatment modalities according to animals' reaction and their current state. Interprofessional collaboration as an engagement of veterinarians, behaviorists, and caregivers in producing a unified care protocol that, along with somatic renewal, reinforces animals' learning and emotional safety.

Combined models offer not only therapeutic efficacy but also uphold high ethical care standards by minimizing distress, minimizing pharmacologic dependency, and supporting positive affective states as part of welfare enhancement (Yeates & Main, 2009). However, practitioners must be aware of avoiding overstimulation, inconsistency of application, or inadequate caregiver compliance. All of those omissions may impede progress or cause iatrogenic stress. Hence, only integrated therapeutic models that combine affective stabilization with cognitive and environmental interventions represent a best-practice framework in the management of psychosomatic disorders in animals. By acknowledging the interdependence of emotion, cognition, and somatic expression, we can promote sustainable recovery, improve animal welfare, and align with the modern biopsychosocial understanding of PSD in animals.

Summary of current knowledge on the cognitive-affective-reflexive regulation of PSD

Psychosomatic disorders in animals represent a complex class of conditions in which behavioral, cognitive, emotional, and physiological systems interact to produce morphologically and clinically relevant somatic outcomes. The contemporary understanding of PSD has evolved from a one-sided focus on either organic pathology or behavioral symptomatology to a more nuanced somatic-psychosocial framework that counts on neural, affective, and reflexive regulation mechanisms across species. At the cognitive level, it was established that learning processes, including classical and operant conditioning, habituation, and cognitive appraisal, play pivotal roles in the development, generalization, and persistence of psychosomatic responses (Sherman & Mills, 2008; Mills et al., 2014). Animals develop maladaptive affective reactions and associations that perpetuate somatic dysfunction in response to internal or external cues. Cognitive inflexibility and compromised neural executive control, often observed in chronically stressed animals, further constrain adaptive coping and somatic recovery.

Affective mechanisms have been increasingly recognized as central to PSD pathogenesis. Namely, emotional dysregulation, especially chronic anxiety, fear, or frustration, modifies neuroendocrine outputs via the HPA axis and the limbic system, leading to physiologic changes such as altered immune activity, disrupted gastrointestinal motility, and autonomic imbalance (Broom, 2011; Landsberg et al., 2015). Emotional experiences also exert neural upstream and downstream effects on memory capacity and behavior selection that reinforce maladaptive patterns.

Reflexive processes, traditionally viewed as automatic and isolated, are now understood to be dynamically modulated by both cognitive and affective inputs. Reflex-based clinical signs such as rapid heartbeat, vomiting, diarrhea, or repetitive movements are common in animals with PSD and are closely linked to their emotional state and past experiences (Bradley et al., 1992; Travain et al., 2015). This dynamic interaction between higher standing neural centers and autonomic or somatic reflex pathways indicates the necessity to treat PSD as a multidimensional disorder, where interconnection between perception, emotion, and bodily function shapes clinical presentation and response to treatment.

Modern research data, though unevenly distributed across species and disciplines, converges on a commonly shared model. Namely, PSD in animals arises from dysregulation in neural circuits that integrate cognition, emotion, and physiology. This dysregulation can be advanced or supported by environmental impoverishment, trauma, social instability, or neurochemical imbalances. It is typically expressed through both behavioral and somatic symptom bundles. Importantly, recovery most likely may occur only through reintegration of these systems via environmental enrichment, behavioral therapy, pharmacologic modulation, and caregiver engagement. In summary, current evidences support a common mechanism to all models of psychosomatic disorders. It applies to different animal species, emphasizing how mental processes, emotions, and bodily reflexes interact to shape both behavior and physical health. Recognizing these interconnections improves diagnostic sensitivity and treatment efficacy of PSD in animals. It also aligns veterinary practice with the best welfare practices and scientifically grounded understanding of animal health.

Importance of interdisciplinary and integrative research approaches

The scientific investigation of PSD in animals demands an interdisciplinary approach. This approach bridges classical veterinary medicine, neuroscience, ethology, behavioral science, and welfare studies. The multidimensional nature of PSD that involves interactions among cognitive, emotional, autonomic, and somatic systems, cannot be fully understood or effectively managed if only one single discipline is used (Broom, 2011; Mills et al., 2014). Historically, veterinary pathology has prioritized structural and biochemical aspects of disease, often underestimating or overlooking the role of behavioral and environmental contributors. Yet, evidence from affective neuroscience and psychoneuroimmunology demonstrates that emotions and cognitive appraisals apply direct effects on somatic health via modulation of neuroendocrine, autonomic, and immune pathways (McEwen, 2007; Dantzer, 2018). Likewise, studies in comparative psychology and animal cognition provide frameworks for understanding how individual learning histories, coping styles, and alleged ability to control shapes disease susceptibility and recovery outcomes (Boissy et al., 2007; Bensky et al., 2013).

Meanwhile, ethology and welfare science contribute their own essential insights. By characterizing species-specific behavioral needs, social relations, and environmental acceptances, these disciplines help the identification of chronic stressors and deprivation conditions that may trigger PSD (Olsson & Westlund, 2007; Fraser, 2008). For instance, stereotypies, stress-induced alopecia, or stress colitis in captive or companion animals are rarely explainable without reference to both individual psychological processing and the ecological mismatch between the environment and the species' behavioral needs. From a clinical perspective, integrative models that combine behavioral assessment, physiological diagnostics, pharmacological interventions, and environmental modifications are more effective than one-sided approach (Crowell-Davis & Murray, 2006; Landsberg et al., 2015). These models duplicate the biopsychosocial model in human medicine, which has revolutionized the understanding of functional disorders, chronic pain, and stress-mediated pathologies (Engel, 1977; Gatchel et al., 2007). Translating this model to veterinary practice re-

quires coordinated input from veterinarians, behaviorists, neuroscientists, ethologists, and welfare professionals. Each of them can contribute important complementary insights into the animal's condition.

Moreover, interdisciplinary frameworks facilitate the development of translational methodologies, such as the application of cognitive bias testing, thermography, neuroimaging, or salivary cortisol assays, to detect and monitor affective-somatic disorders (Paul et al., 2005; Travain et al., 2015). These tools demonstrate how integrating knowledge across disciplines not only deepens our understanding of affective-somatic interactions but also promotes innovation in diagnostic, preventive, and therapeutic strategies.

In summary, PSD research and management in animals reside at the intersection of multiple scientific fields. An integrative approach is not only methodologically advantageous but also ethically sound. It promotes a systemic view on PSD and ensures that interventions address both physical and psychological aspects of the animal's health. Embracing such an interdisciplinary approach is essential for advancing both scientific understanding and clinical efficacy when dealing with psychosomatic pathology in veterinary medicine.

Future research perspectives: neuropharmacology, behavioral biomarkers, and welfare-based clinical models

As the field of veterinary behavioral medicine continues to evolve, future research on PSD in animals must address critical knowledge gaps through a translational, multidimensional, and welfare-driven lens. Here we envision at least three particularly promising domains: (1) development of objective behavioral and physiological biomarkers, (2) clinical models grounded in individualized, species-specific welfare frameworks and (3) neuropharmacological mechanisms and interventions. While psychopharmacological agents such as selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants, and anxiolytics have shown clinical utility in managing PSD, their mechanism of action still remains incompletely understood in non-human species (Simpson, 2008; Landsberg et al., 2015). Future research should prioritize receptor-specific studies on serotonergic, dopaminergic, GABAergic, and glutamatergic systems in species-relevant models. On this way the important direction of the research also would be the assessments of prolonged drug effects on affective states, autonomic balance, and neuroplasticity, studying of comparative pharmacodynamics, examining interspecies differences in perception, metabolism, and behavioral outcomes. Moreover, emerging classes of compounds, including neurosteroids, endocannabinoid modulators, and natural adaptogens, warrant exploration for their potential role in modulating stress responsivity, emotional regulation, and reflexive autonomic function (Mason & Rushen, 2006; Hekman et al., 2012).

Reliable biomarkers are essential for early detection, individualized intervention planning, and objective monitoring of treatment outcomes in animals with PSD. Future research must validate and standardize tools across different species and settings. In our opinion, promising areas for future research include studying the efficacy of a range of behavioral, physiological, and neurobiological markers. Behavioral biomarkers such as attentional bias, latency to approach, and cognitive flexibility have already been proposed as indicators of affective-cognitive dynamics (Paul et al., 2005; Baciadonna & McElligott, 2015). Physiological indices, including salivary cortisol, HRV, eye temperature via thermography, and fecal glucocorticoid metabolites, in their turn, offer non-invasive measures of affective-autonomic interaction (Valera et al., 2012; Travain et al., 2015). Additionally, neuroimaging and neurophysiological tools such as functional MRI, EEG, and evoked potentials can help identify network-level dysfunctions in animals affected by chronic psychosomatic disorders (Adrian et al., 2018). Also important, multi-modal approaches that combine behavioral data with physiological metrics are likely to enhance diagnostic accuracy and provide deeper insight into the causes of progression or remission of psychosomatic symptomatology.

Veterinary behavioral medicine must continue evolving toward researching clinical PSD models and give equal priority to psychological well-being and physical health. Future frameworks for addressing PSD should adopt formulation of individualized case protocols that incorporate the animal's history, emotional valence, cognitive style, and reflex reactivity (Fraser, 2008; Yeates & Main, 2009). It is also essential to develop and integrate species-specific welfare benchmarks, including dependability from natural behavioral repertoires (ethogram fidelity), needs and opportunities for voluntary control, and compatibility within social environments. In addition, preventive strategies should be explored through early-life interventions. In particular, it is necessary to study how early environmental enrichment, social bonding, and cognitive engagement, may reduce long-term vulnerability to psychosomatic dysregulation.

The design of such models should reflect feedback-informed care and promote dynamic monitoring over time. That may additionally acknowledge that recovery from PSD is a process of neural, emotional, and behavioral rehabilitation rather than simple symptom suppression. Hence, by integrating advanced neuropharmacology, validated biomarkers, and welfare-centered clinical paradigms, the field can generate more effective, humane, and scientifically grounded approaches to one of the important evolving domains in veterinary medicine.

Conclusions

Psychosomatic disorders in animals constitute a multidimensional category of pathology, in which cognitive, affective, and reflexive systems interact to shape both somatic health and behavioral outcomes. Growing evidence from veterinary medicine, comparative neuroscience, and behavioral science underscores the systemic connections of mind and body. The traditional dichotomy between "organic" and "psychogenic" disease is becoming increasingly outdated. Across species, cognitive processes such as learning, expectation, and perceived control shape behavioral patterns and physiological outcomes. Affective states, especially chronic fear, anxiety, frustration, or emotional deprivation, modulate neuroendocrine function and immune responses, with somatic consequences ranging from gastrointestinal dysmotility to dermatologic disorders and cardiovascular dysregulation. Reflexive systems, once considered automatic and insulated from emotional input, are now understood to be flexible and responsive to affective and cognitive input.

In terms of pathogenesis, key mechanisms of PSD include HPA axis hyperactivation, autonomic imbalance, and disrupted communication between neural and immune systems, which collectively alter somatic function in the absence of structural pathology. Importantly, these mechanisms are not species-specific but reflect conserved vertebrate responses to stress and affective dysregulation, making PSD an ideal domain for translational and comparative research.

Diagnosis remains challenging due to the multifactorial and often subclinical presentation of PSD. However, current advances in behavioral assessment tools, biomarkers (e.g., salivary cortisol, thermography, HRV), and species-specific scoring systems offer greater diagnostic precision and objectivity. Similarly, therapeutic approaches have evolved from isolated pharmacological interventions to multimodal tactics that include environmental enrichment, cognitive-behavioral therapy, and targeted pharmacotherapy.

A unifying theme throughout the review is the critical role of interdisciplinary collaboration. Namely, effective understanding and management of PSD requires coordinated insights from veterinary clinicians, ethologists, neuroscientists, animal welfare scientists, and behaviorists. Furthermore, the application of welfare-based clinical models, attentive to species-specific needs, emotional states, and environmental contexts, represents not only an ethical obligation but a scientifically grounded necessity.

There were identified three promising domains for further exploration. They include the development of objective behavioral and physiological biomarkers, the creation of clinical models grounded in individualized, species-specific welfare frameworks, and the investigation of underlying neuropharmacological mechanisms and potential models for therapeutic interventions. Future research directions must also emphasize a pressing need to move beyond symptomatic care toward approaches that advance adaptive emotional regulation, cognitive adaptive capacity, and behavioral adaptability. This calls for

investment not only in laboratory and clinical research, but also in developing practical strategies for implementation across diverse settings, including shelters, farms, zoos, research facilities, and companion animal care environments.

Finaly, PSD in animals is not merely a collection of syndromes, it is rather a window into the dynamic interconnections of body, brain, and behavior. Contemporary research illuminates both the adaptive capacities and vulnerabilities of animals in human-managed environments. The problem calls for a future-oriented, integrative scientific approach that connects research across species, brings together diverse disciplines, and supports coherent, welfare-centered systems of care.

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