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Climate change and the psychosomatic health of animals: Mechanisms, manifestations, and mitigation strategies

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Climate change is progressively reshaping environmental conditions in ways that significantly affect the psychosomatic health of animals across terrestrial and aquatic ecosystems. Rising temperatures, more frequent and prolonged heat waves, altered precipitation patterns, habitat instability, nutritional stress, and the expanding distribution of pathogens and vectors create persistent environmental pressures rather than isolated acute challenges. These stressors activate sustained neuroendocrine, immune, metabolic, and behavioral responses that may initially support adaptation but, under chronic exposure, shift toward maladaptive psychosomatic outcomes. Prolonged activation of the hypothalamic-pituitary-adrenal axis, autonomic imbalance, low-grade inflammation, oxidative stress, and disrupted gut-brain-immune communication collectively contribute to multisystem dysfunction. This review synthesizes current evidence on the biological pathways linking climate-related stressors with systemic dysregulation. Attention is given to neuroendocrine-immune interactions, inflammatory sensitization, metabolic strain under thermal load, and environmentally driven changes in behavioral ecology. We emphasize the role of early-life exposure and developmental programming in shaping long-term stress responsivity, as well as species-specific sensitivity thresholds and the cumulative burden of repeated environmental challenges. Clinical and subclinical manifestations of climate-induced psychosomatic disturbances are examined, including stress-associated gastrointestinal and dermatological disorders, impaired thermoregulation, reduced immune competence, reproductive disruption, and cognitive or affective abnormalities such as anxiety-like behavior and social withdrawal. Many of these conditions emerge gradually and may remain undetected without integrative monitoring. Finally, we outline practical mitigation strategies based on microclimate management, nutritional and metabolic resilience, behavioral stabilization, adaptive acclimation, and precision monitoring technologies for early detection of maladaptive trajectories. By integrating mechanistic insights with applied veterinary and management perspectives, this review highlights the need for interdisciplinary approaches to safeguard animal health and welfare in a rapidly changing climate.

Keywords: climate change; animal stress; psychosomatic health; neuroendocrine mechanisms; welfare; stressors.

Introduction

Anthropogenic climate change has become one of the most powerful and pervasive environmental drivers shaping animal physiology, behavior, and health in the twenty-first century. Rising mean global temperatures, increasing frequency and intensity of heatwaves, altered precipitation regimes, extreme weather events, and accelerating habitat instability collectively impose persistent and multifactorial stress on animals across terrestrial and aquatic ecosystems (IPCC, 2022). Unlike short-term environmental challenges, climate-related stressors are typically chronic, cumulative, and unpredictable, thereby exerting sustained pressure on regulatory systems responsible for maintaining physiological and behavioral homeostasis.

One of the most consistently documented biological consequences of environmental instability is the activation of central stress-response systems, particularly the hypothalamic-pituitary-adrenal (HPA) axis and associated neuroendocrine pathways. Repeated or prolonged stimulation of these systems leads to sustained glucocorticoid secretion, autonomic imbalance, and downstream effects on immune competence, metabolism, reproduction, and behavior (McEwen & Stellar, 1993; McEwen, 2007). Within this framework, the concept of allostatic load provides a unifying model for understanding how adaptive stress responses may transition into maladaptive physiological and behavioral outcomes when environmental demands exceed coping capacity (McEwen & Wingfield, 2010).

Climate change also disrupts the temporal organization of biological processes. Shifts in phenology, defined as changes in the timing of life-history events such as migration, breeding, flowering, or hatching, have been documented across a wide range of taxa. These shifts are frequently asynchronous among interacting species, resulting in mismatches between consumers and resources or between reproductive timing and optimal environmental conditions (Both & Visser, 2001; Visser & Both, 2005; Parmesan, 2006). Such phenological mismatches not only impair ecological interactions and fitness but also represent potent sources of chronic stress, particularly for species with limited behavioral or physiological plasticity.

From a psychosomatic perspective, chronic environmental stressors associated with climate change act simultaneously on emotional, neuroendocrine, immune, and somatic systems. Prolonged stress exposure has been shown to promote low-grade systemic inflammation, impair gut barrier integrity, alter neuroimmune communication, and reshape behavioral strategies, including increased vigilance, reduced exploration, altered feeding behavior, and social withdrawal (Koolhaas et al., 2011; Romero & Wingfield, 2015). These integrated responses form the biological substrate of psychosomatic pathology, wherein psychological or affective stressors drive or amplify somatic dysfunction.

While psychosomatic mechanisms have been extensively investigated in laboratory animals and humans, their relevance to domestic animals, production systems, and wildlife under climate-driven environmental change has only recently received focused scientific attention. Much of the earlier stress research was conducted under controlled experimental conditions, which do not fully capture the complexity, unpredictability, and cumulative nature of climate-related stressors experienced by free-living and managed animal populations (Romero et al., 2009). Consequently, there is a growing recognition that climate change represents not merely an ecological or production challenge, but a fundamental threat to animal psychosomatic health and welfare.

Integrating insights from stress physiology, behavioral ecology, veterinary medicine, and conservation biology is therefore essential for understanding how climate-related environmental instability translates into psychosomatic disorders across species. By synthesizing current evidence on neuroendocrine regulation, behavioral manifestations, immune alterations, and system-level dysfunction, this review aims to clarify the mechanisms through which climate change compromises psychosomatic health in animals and to provide a conceptual foundation for developing effective monitoring, prevention, and mitigation strategies in a rapidly changing world.

A comprehensive literature search was conducted using PubMed, Web of Science, Scopus, and Google Scholar databases for publications from 2000 to 2025. Key search terms included combinations of "climate change," "psychosomatic," "stress physiology," "animal welfare," "HPA axis," "behavioral indicators," "thermoregulation," "immune function," and "disease susceptibility." Additional sources were identified through citation tracking and expert recommendations. Selected studies included original research articles, systematic reviews, meta-analyses, and clinical guidelines that addressed behavioral, physiological, immunological, or ecological responses to environmental stressors associated with climate change. Extracted data were focused on: (a) behavioral indicators of stress and welfare, (b) physiological biomarkers including glucocorticoids, heart rate variability, and thermoregulatory parameters, (c) immune markers such as cytokine levels and leukocyte profiles, and (d) environmental and ecological variables including habitat changes, resource availability, and vector distributions. Findings were synthesized qualitatively to highlight mechanisms, manifestations, and mitigation strategies for climate-induced psychosomatic disturbances across species. Where applicable, results were categorized by species type (domestic vs. wild), stressor type (thermal, resource-based, ecological), and level of biological response (behavioral, physiological, immunological) to facilitate comparative assessment. Tables were created to summarize key empirical data on monitoring and management strategies.

Physiological and neuroendocrine pathways linking climate change to psychosomatic responses in animals

Climate change-related environmental stressors exert their effects on animal health primarily through dysregulation of central neuroendocrine systems that coordinate physiological and behavioral adaptation. Among these systems, the hypothalamic-pituitary-adrenal (HPA) axis represents the core integrative pathway linking external environmental challenges to internal homeostatic and allostatic responses. While homeostatic responses aim to maintain internal physiological variables within narrow limits, allostatic responses achieve stability through dynamic adjustments of set points in response to sustained or anticipated environmental demands and are largely mediated by neuroendocrine systems such as the HPA axis. Thermal extremes, dehydration, food scarcity, habitat disturbance, and social instability, common consequences of climate change, are potent activators of HPA-axis signaling across vertebrate taxa (Wingfield et al., 2011).

Acute activation of the HPA axis facilitates adaptive responses by mobilizing energy reserves, modulating cardiovascular tone, and enhancing vigilance. However, when climate-related stressors are prolonged or repeatedly encountered, glucocorticoid secretion may become chronically elevated or dysregulated. Such patterns have been documented in free-living animals exposed to persistent environmental instability, including heat stress, drought, and altered resource distribution (Wingfield & Sapolsky, 2003; Romero, 2004). Chronic glucocorticoid exposure is associated with the impaired ability of the brain to suppress HPA axis activity at the level of the hypothalamus and hippocampus, leading to sustained neuroendocrine imbalance. Prolonged elevation of circulating cortisol downregulates glucocorticoid receptor expression and disrupts receptor signaling efficiency within these regulatory brain regions, thereby weakening inhibitory control over corticotropin-releasing hormone and adrenocorticotropic hormone secretion. As a consequence, the hypothalamic-pituitary-adrenal axis becomes persistently hyperactivated, promoting a self-reinforcing cycle of hormonal dysregulation that contributes to structural neural alterations, heightened stress reactivity, and long-term allostatic overload.

Beyond their classical metabolic roles, glucocorticoids exert wide-ranging effects on immune regulation and neural plasticity. Prolonged HPA-axis activation suppresses important features of adaptive immunity while simultaneously promoting low-grade systemic inflammation through altered cytokine signaling and glucocorticoid resistance at immune cell targets (Dhabhar, 2014). These immune alterations provide an automatous link between chronic environmental stress and the emergence of psychosomatic conditions, in which inflammatory processes contribute to behavioral changes, gastrointestinal dysfunction, and reduced disease resistance.

Climate-induced stress also affects autonomic nervous system (ANS) regulation, particularly the balance between sympathetic and parasympathetic activity. Heat stress and resource unpredictability have been shown to favor sustained sympathetic dominance, characterized by elevated catecholamine release, increased heart rate, and reduced vagal tone (Korte et al., 2005). Persistent autonomic imbalance amplifies somatic vulnerability by impairing gastrointestinal motility, mucosal integrity, and cardiovascular regulation, thereby reinforcing psychosomatic feedback loops.

Importantly, neuroendocrine responses to climate stressors are not uniform across individuals or species. Life-history strategy, developmental stage, prior stress exposure, and genetic background significantly modulate physiological sensitivity and coping capacity (Wingfield et al., 2011). Early-life exposure to environmental instability, in particular, can induce long-lasting alterations in HPA-axis reactivity and stress responsivity, increasing susceptibility to psychosomatic disorders later in life (Lupien et al., 2009). These developmental effects are especially relevant under contemporary climate change, where animals increasingly encounter stressors during critical time of neuroendocrine maturation.

At the brain level, chronic stress alters the functional connectivity of limbic and prefrontal regions involved in emotional regulation, decision-making, and behavioral flexibility. Experimental and field-based studies indicate that sustained glucocorticoid exposure affects hippocampal neurogenesis, amygdala excitability, and prefrontal inhibitory control, thereby biasing behavior toward heightened fearfulness, reduced exploration, and impaired coping (McEwen et al., 2016). We suggest that such neurobehavioral shifts represent central components of psychosomatic pathology, as they directly influence feeding, social interaction, and environmental engagement.

Collectively, these findings demonstrate that climate change influences animal psychosomatic health through interconnected neuroendocrine, immune, and autonomic pathways. Rather than acting as isolated stressors, climate-related environmental changes enforce cumulative regulatory demands that progressively erode adaptive capacity of animals. Therefore, understanding these physiological and neuroendocrine mechanisms is essential for identifying vulnerable populations, developing biomarkers of stress-related dysfunction, and designing mitigation strategies that address both environmental conditions and internal regulatory resilience.

Behavioral and affective manifestations of climate-induced psychosomatic stress

Empirical evidence indicates that climate-induced environmental stressors are consistently associated with measurable changes in animal behavior and affective state, reflecting dysregulation of integrated neuroendocrine-somatic systems. Rather than be presented as discrete pathological entities, these manifestations often emerge as coordinated behavioral syndromes involving altered activity patterns and emotional reactivity, abnormality in feeding behavior and social interactions. Such changes represent functional expressions of psychosomatic stress, wherein affective dysregulation and somatic disturbances are tightly coupled.

One of the most robust behavioral correlates of climate-related stress is altered activity and thermoregulatory behavior. Heat waves and elevated ambient temperatures have been shown to reduce locomotor activity, foraging efficiency, and exploratory behavior in both wild and domestic animals, while increasing resting time and heat-avoidance strategies (Angilletta et al., 2010; Fuller et al., 2010). These behavioral adjustments, although initially adaptive, may become maladaptive under prolonged exposure, contributing to negative energy balance, muscle wasting, and impaired immune competence.

Affective-like states associated with chronic environmental stress are increasingly documented through behavioral patterns such as increased fearfulness, heightened vigilance, and reduced behavioral flexibility. Field studies in birds and mammals demonstrate that individuals exposed to unstable or extreme climatic conditions exhibit exaggerated stress responses to novel stimuli, increased predator avoidance, and reduced risk-taking behavior (Wingfield et al., 2011; Breuner et al., 2013). These affective shifts are consistent with sustained activation of limbic circuits involved in threat detection and emotional responsiveness.

Feeding behavior represents another critical interface between affective state and somatic health. Climate-driven alterations in resource availability, combined with stress-induced neuroendocrine changes, frequently lead to disrupted feeding patterns, including hypophagia, altered meal timing, and selective feeding (Wingfield & Kitaysky, 2002). Such changes are mediated in part by glucocorticoid interactions with hypothalamic appetite-regulating pathways and gastrointestinal signaling, thus, reinforcing psychosomatic feedback loops linking emotional stress to digestive dysfunction.

Social behavior is particularly sensitive to environmental stressors associated with climate change. Increased temperatures, habitat compression, and resource scarcity intensify social competition and destabilize established dominance hierarchies. Empirical studies in social species report elevated aggression, reduced affiliative behaviors, and impaired parental care under conditions of climatic stress (Cain et al., 2010; Rubenstein, 2012). These social disruptions further exacerbate stress exposure, especially in subordinate individuals, thereby amplifying psychosomatic vulnerability.

In production and companion animals, climate-related stressors manifest behaviorally as increased restlessness and stereotypic behaviors, reduced play, and altered human-animal interactions. Heat stress in livestock, for example, is associated with behavioral indicators of discomfort and affective distress, alongside somatic consequences such as reduced feed intake, gastrointestinal disturbances, and decreased productivity (Polsky & von Keyserlingk, 2017). These observations underscore the translational relevance of behavioral markers as indicators of psychosomatic strain.

Collectively, empirical evidence supports the view that climate-induced environmental stress is reliably expressed through behavioral and affective changes that reflect underlying psychosomatic dysregulation. While these manifestations are often reversible in the short term, chronic or repeated exposure to climatic stressors increases the likelihood that adaptive behavioral responses transition into persistent affective disturbance and somatic dysfunction. Recognizing and quantifying these behavioral phenotypes is therefore essential for early detection of climate-related psychosomatic risk and for the development of evidence-based mitigation strategies.

Immune, gastrointestinal, and metabolic correlates of climate-related psychosomatic stress

Climate-induced psychosomatic stress exerts profound effects on somatic regulatory systems, particularly immune, gastrointestinal, and metabolic pathways that are highly sensitive to neuroendocrine modulation. Rather than representing independent pathological processes, alterations in these systems emerge as integrated consequences of sustained emotional and physiological stress exposure, reflecting disrupted communication between the brain and peripheral organs.

The immune system represents one of the primary somatic targets of chronic stress associated with climate-related environmental instability. Prolonged activation of the HPA axis and sympathetic nervous system alters leukocyte distribution, cytokine signaling, and inflammatory regulation. Empirical studies across vertebrate taxa also demonstrate that chronic environmental stress promotes the development of glucocorticoid resistance in immune cells, resulting in paradoxical patterns of low-grade systemic inflammation alongside impaired pathogen defense (Romero et al., 2009; Dhabhar, 2014). Such immune dysregulation provides a mechanistic substrate for psychosomatic vulnerability, as soon as inflammatory mediators directly influence neural circuits governing mood, motivation, and sickness behavior.

The gastrointestinal system constitutes a critical interface between emotional stress and somatic dysfunction under climate change. Heat stress, dehydration, and nutritional unpredictability disrupt gastrointestinal motility, mucosal barrier integrity, and gut microbial composition. Experimental and field-based evidence indicates that stress-induced alterations of the gut microbiota and increased intestinal permeability facilitate bidirectional signaling along the gut-brain axis, amplifying affective disturbances and systemic inflammatory responses (Foster et al., 2017; Karl et al., 2018). Understanding these processes helps explain why climate-related stress is frequently accompanied by feeding disturbances, gastrointestinal discomfort, and reduced nutrient assimilation.

Metabolic regulation may be also severely compromised under sustained climate-induced stress. Elevated ambient temperatures and resource instability impose increased energetic demands while simultaneously suppressing appetite and altering endocrine control of metabolism. Chronic glucocorticoid exposure promotes muscle protein catabolism, insulin resistance, and altered lipid metabolism, thereby shifting energy allocation away from growth, reproduction, and immune defense system (Sapolsky et al., 2000; McEwen & Wingfield, 2010). In production animals, these metabolic disruptions manifest as reduced feed efficiency, impaired body condition, lower productivity, and heightened disease susceptibility under heat stress conditions.

Importantly, immune, gastrointestinal, and metabolic alterations do not occur in isolation. These systems are interconnected through shared inflammatory mediators, neural pathways, and hormonal signals, forming self-reinforcing psychosomatic feedback loops. For example, stress-induced gut dysbiosis can exacerbate systemic inflammation, which in turn alters neuroendocrine regulation and behavioral state, perpetuating a cycle of emotional and somatic dysregulation. Climate change, by increasing the frequency and duration of environmental stressors, enhances the likelihood that such feedback loops become chronically activated.

Taken together, available evidence indicates that climate change contributes to psychosomatic disturbances in animals not through singular disease pathways, but via coordinated dysregulation of immune, gastrointestinal, and metabolic systems. These somatic correlates represent intermediate stages along a continuum from adaptive stress response to maladaptive psychosomatic pathology. Recognizing these system-level alterations is essential for identifying early indicators of climate-related health risk and for developing integrative mitigation strategies that address both environmental pressures and animals' resilience.

Pharmacological modulation of climate-related behavioral and psychosomatic disturbances: mechanisms, applications, and limitations

Pharmacological interventions constitute an important component of contemporary veterinary behavioral medicine and are frequently employed to mitigate maladaptive emotional and behavioral responses associated with chronic stress. In the context of climate change, pharmacological agents may attenuate selected neurobehavioral and psychosomatic manifestations by modulating central neurochemical and neuroendocrine pathways involved in stress reactivity. However, pharmacotherapy does not eliminate the primary environmental drivers of climate-induced stress and should therefore be regarded as a supportive, rather than curative, strategy.

Most pharmacological approaches target neurotransmitter systems implicated in affective regulation and stress responsiveness, particularly serotonergic, noradrenergic, and GABAergic pathways. Selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants (TCAs) are widely used to reduce anxiety-related behaviors, compulsive disorders, and affective dysregulation in companion animals. Their therapeutic effects are mediated through enhanced serotonergic signaling within limbic and cortical networks involved in emotional control and stress appraisal (Landsberg et al., 2013). Although these agents demonstrate clinical efficacy across a range of behavioral conditions, direct empirical evidence linking their use specifically to climate-induced stress remains limited, with most applications relying on extrapolation from generalized anxiety and chronic stress models.

Short-acting pharmacological agents, including benzodiazepines and α_2-adrenergic agonists, are commonly applied for acute modulation of stress responses associated with environmental extremes, transport, or handling. These drugs reduce sympathetic nervous system activation and enhance inhibitory neurotransmission, thereby decreasing behavioral arousal and autonomic hyperreactivity (Overall, 2014). Nonetheless, their use is constrained by well-documented limitations, including sedation, tolerance development, paradoxical excitation, and potential interference with adaptive behavioral coping mechanisms. Under conditions of repeated or prolonged climate stress, reliance on short-term anxiolytics may obscure underlying welfare issues rather than resolve them.

Interest has recently increased in pharmacological and nutraceutical agents that influence circadian regulation, oxidative balance, and neuroendocrine stress pathways. Melatonin has received particular attention due to its role in circadian synchronization, modulation of HPA axis activity, and antioxidant defense. Experimental evidence in animal models demonstrates that melatonin administration can attenuate behavioral and endocrine responses to acute stress, including reductions in cortisol secretion and stress-related behavioral alterations (Guesdon et al., 2013; Lunkes et al., 2021). While these findings support a mechanistic rationale for melatonin use under stress conditions, data specifically addressing chronic climate-related stress scenarios remain sparse and species-dependent.

Beyond central nervous system effects, pharmacological modulation may indirectly influence immune, gastrointestinal, and metabolic correlates of psychosomatic stress. However, such effects are typically secondary and insufficient to counteract ongoing environmental challenges such as heat exposure, dehydration, or nutritional instability. Chronic pharmacological suppression of behavioral symptoms without concurrent environmental modification may delay recognition of maladaptive conditions and increase the risk of cumulative somatic dysfunction.

Accordingly, pharmacological modulation should be embedded within an integrative management framework that prioritizes environmental adaptation, husbandry modification, and behavioral intervention. Pharmacotherapy may be justified in cases of severe affective dysregulation or when stress exposure cannot be immediately mitigated. The use of medicines also requires careful case selection, defined therapeutic objectives, and ongoing reassessment. Future research should focus on delineating context-specific indications, long-term outcomes, and interaction effects between pharmacological agents and chronic climate-related stressors that may be especially indicated for domestic and farm animals.

Monitoring psychosomatic states in animals under climate change: what and how to measure

Effective assessment of psychosomatic health in animals exposed to climate-related stressors requires integrative monitoring frameworks that combine behavioral observations with physiological, endocrine, immunological, and molecular biomarkers. Climate change introduces chronic and fluctuating stress loads, through heat waves, altered photoperiods, resource instability, and extreme weather events, that affect emotional regulation, metabolic homeostasis, and somatic resilience. Consequently, traditional single-parameter welfare indicators are insufficient to capture the multisystemic nature of climate-induced psychosomatic dysregulation.

Behavioral phenotyping remains a primary non-invasive tool for detecting early psychosomatic disturbance. Heat stress, prolonged drought, and environmental unpredictability are consistently associated with increased anxiety-like behaviors, aggression, stereotypies, reduced exploratory activity, altered social interactions, and disrupted feeding rhythms across domestic and wild species (Koolhaas et al., 2011; Briefer & Comber, 2012). In livestock, thermal stress correlates with restlessness, excessive panting, reduced rumination, and social withdrawal, which precede and actually predict measurable physiological breakdown (Polsky & von Keyserlingk, 2017).

Recent developments in automated behavior tracking and machine-learning-based welfare assessment allow continuous quantification of locomotion patterns, posture changes, vocalization profiles, and social network dynamics under fluctuating climatic conditions (Valletta et al., 2017; Neethirajan, 2020). These tools are particularly valuable for long-term monitoring of subclinical psychosomatic stress accumulation.

The HPA axis higher activity remains a central indicator of psychosomatic load. Climate-related stressors consistently elevate glucocorticoid secretion, with both acute spikes during extreme events and chronic dysregulation under prolonged environmental pressure (Romero & Wingfield, 2015). Cortisol or corticosterone concentrations measured in saliva, feces, hair, feathers, or blood provide important evidence for and possible differentiation between short- and long-term stress exposure (Sheriff et al., 2011; Gupta et al., 2023). For example, hair and feather glucocorticoid analysis are particularly suited for climate research as it integrates cumulative stress over weeks to months, reflecting chronic psychosomatic burden rather than transient stress responses (Meyer & Novak, 2012). However, interpretation requires careful species-specific validation and consideration depending on seasonal endocrine rhythms.

Climate stress modifies autonomic balance, commonly shifting toward sympathetic dominance. Heart rate variability (HRV) has emerged as a robust indicator of emotional regulation capacity and psychosomatic resilience in animals (von Borell et al., 2007; Stubsjøen et al., 2015). Reduced HRV is consistently associated with chronic stress, anxiety-like states, inflammatory activation, and impaired thermoregulation.

Advances in wearable biosensors now permit continuous HRV, body temperature, and activity monitoring in farm animals, companion animals, and wildlife. They enable real-time assessment of climate-induced physiological strains (Neethirajan, 2020). Such multimodal physiological datasets provide early warning signals before somatic manifestations, when overt clinical pathology develops.

Psychosomatic dysregulation under climate stress is closely linked to low-grade systemic inflammation. Elevated pro-inflammatory cytokines (e.g., IL-6, TNF-α, IL-1β), acute phase proteins (haptoglobin, C-reactive protein), and oxidative stress markers are increasingly reported in animals exposed to heat stress, nutritional instability, and environmental disruption (Bernabucci et al., 2010; Bernabucci et al., 2014; Bagathet al., 2019). Monitoring inflammatory profiles offers critical insight into the somatic consequences of prolonged emotional and neuroendocrine strain. Chronic activation of immune pathways contributes to gastrointestinal dysfunction, reduced disease resistance, reproductive impairment, and behavioral depression-like phenotypes, reinforcing the psychosomatic feedback loop.

Climate stress frequently alters appetite regulation, energy expenditure, and gut barrier integrity. Changes in glucose metabolism, non-esterified fatty acids, ketone bodies, and cortisol-insulin dynamics reflect systemic stress adaptation or malfunction (Baumgard & Rhoads, 2013). Concurrently, growing evidence indicates climate-related shifts in gut microbiota composition, reduced microbial diversity, and increased intestinal permeability, all of which correlate with anxiety-like behavior and inflammatory activation (Xiong et al., 2020; Jiang et al., 2021; Kumar et al., 2021).

To enhance clarity and facilitate cross-species comparison, the main categories of indicators used to assess psychosomatic responses to climate-related stress can be systematized into behavioral, physiological, immune, and ecological domains. Table 1 provides an integrated overview of these indicator classes, the specific measures used within each of them, and their relevance to diagnosing acute and chronic stress in animals.

Table 1
Key behavioral, physiological, immune, and ecological indicators of climate-associated psychosomatic stress in animals

Indicator category	Specific indicators / measures	What they reflect	Methods of measurement	Typical species / applications
Behavioral indicators	Feed intake reduction; altered feeding time; decreased or increased locomotion; avoidance of social contact; shade-seeking; posture minimizing heat load; reduced activity	Early changes in affective state, thermoregulatory behavior, energy conservation, anxiety-like responses	Direct observation; automated video tracking; ethograms; accelerometers	Domestic livestock, poultry, companion animals, wildlife under field monitoring
Physiological indicators	Cortisol (saliva, blood, feces, urine); heart rate variability (HRV); respiratory rate; rectal or surface temperature	HPA axis activity; autonomic balance; heat load and thermoregulation; acute vs chronic stress	Biomarker assays; ECG telemetry; infrared thermography; metabolic sensors	All mammals, birds; especially production animals and wildlife in telemetry projects
Immune markers	IL-6, TNF-α; C-reactive protein (CRP); leukocyte differentials	Inflammation, stress-immune imbalance, immune suppression or activation	ELISA, hematology, cytokine panels	Mammals, birds; used in veterinary diagnostics and wildlife health assessments
Ecological / environmental indicators	Resource availability (water/food); habitat quality; vector presence; range shifts; microclimate data	External stressors shaping psychosomatic and physiological load; ecological risk	GPS tracking; remote sensing; climate loggers; habitat surveys	Wildlife, free-ranging domestic animals, grazing systems
Chronic stress marker	Hair cortisol concentration (HCC)	Long-term integrated glucocorticoid load over weeks–months	Hair sampling + cortisol assay	Domestic, laboratory, and wild mammals

The indicators shown in Table 1, combined into an integrated monitoring system, allow researchers and veterinarians to detect early stress signals, assess long-term physiological burden, and link individual behavioral changes to broader ecological pressures emerging under accelerating climate change. Psychosomatic monitoring increasingly integrates metabolomic profiling and microbiome analysis to capture the bidirectional gut-brain-immune axis affected by environmental stressors. To systematize the major behavioral and psychosomatic consequences of climate-related stress across taxa, Table 2 shows key manifestations, their underlying mechanisms, and representative evidence from current research.

Table 2
Key indicators for monitoring psychosomatic stress in animals under climate change

Domain	Indicator	Measurement method	Psychosomatic relevance	Key reference (APA, verified DOI)
Behavioral	Anxiety-like behavior, stereotypies, social withdrawal	Ethograms, automated video tracking	Early emotional dysregulation under chronic environmental stress	Koolhaas et al., 2011
Endocrine	Cortisol (saliva, feces, hair, blood)	Immunoassays (ELISA, LC–MS)	Chronic HPA-axis load reflecting cumulative stress	Gupta et al., 2023
Long-term stress	Hair/feather glucocorticoids	Segmental cortisol analysis	Integrated stress exposure over weeks–months	Meyer & Novak, 2012
Autonomic	Heart rate variability (HRV)	Wearable biosensors, ECG	Emotional regulation capacity, chronic stress burden	von Borell et al., 2007
Immune	Pro-inflammatory cytokines (IL-6, TNF-α, IL-1β)	ELISA, multiplex assays	Climate-driven inflammatory activation and psychosomatic pathology	Bagath et al., 2019
Acute phase response	Haptoglobin, CRP	Serum biochemical assays	Systemic stress-induced inflammation	Bernabucci et al., 2010
Metabolic	Glucose, NEFA, ketone bodies	Blood biochemical profiling	Energetic dysregulation under thermal stress	Baumgard & Rhoads, 2013
Gut integrity	Microbiome diversity, intestinal permeability	16S rRNA sequencing, biomarker assays	Gut–brain–immune axis disruption	Xionget al., 2020
Composite stress	Multisystem stress indices	Integrated behavioral + physiological datasets	Early detection of maladaptive trajectories	Madliger et al., 2018
Continuous monitoring	Activity, body temperature, HRV	Sensor platforms, AI analytics	Real-time climate stress load	Neethirajan, 2020

While analyzing the data we should remember that behavioral and psychosomatic outcomes may vary across species but they consistently reflect interactions among neuroendocrine stress responses, immune activation, and environmental pressures. Given the multifactorial nature of climate-induced psychosomatic disorders, contemporary research emphasizes composite indices that integrate behavioral scores, endocrine load, autonomic balance, and inflammatory status (Madliger et al., 2018). Such systems move beyond binary welfare assessments toward dynamic resilience profiling, identifying individuals or populations at heightened risk under progressive climatic pressure. Longitudinal designs are particularly critical, as psychosomatic dysregulation often emerges gradually through cumulative stress exposure rather than discrete climatic events. Combining continuous sensor data with periodic biomarker sampling will enable early detection of maladaptation trajectories and support the development of targeted mitigation strategies.

Prevention of climate-related psychosomatic stress disorders in animals

Prevention of psychosomatic stress disorders under climate change requires multilevel interventions targeting environmental exposure, physiological resilience, and emotional regulation. Unlike acute climatic insults, which may trigger transient stress responses, chronic climate-driven pressures, such as sustained heat load, resource unpredictability, and environmental instability, progressively dysregulate neuroendocrine, immune, and metabolic systems, thereby increasing vulnerability to psychosomatic pathology. Consequently, preventive strategies must focus not only on reducing physical stressors but also on stabilizing psychophysiological homeostasis.

Thermal mitigation remains the primary preventive approach against climate-related psychosomatic stress. Shade provision, ventilation, evaporative cooling systems, optimized housing orientation, and reduced stocking density consistently lower physiological stress indicators and behavioral distress across livestock species (Bernabucci et al., 2014; Polsky & von Keyserlingk, 2017). Such interventions reduce cortisol secretion, preserve adequate feed intake, and attenuate inflammatory activation associated with prolonged heat exposure.

In extensive systems, landscape-level modifications, such as shelter belts, water access optimization, and grazing rotation, buffer climatic extremes and reduce sustained autonomic arousal. These measures align with adaptive husbandry recommendations promoted by the Food and Agriculture Organization of the United Nations in climate-resilient livestock systems.

Nutritional modulation plays a central role in preventing psychosomatic dysregulation during thermal and environmental stress. Heat stress consistently reduces appetite while increasing oxidative load and metabolic inefficiency (Baumgard & Rhoads, 2013). Dietary supplementation with antioxidants (vitamin E, selenium, polyphenols), electrolytes, and rumen-protected fats improves thermotolerance, limits oxidative damage, and stabilizes neuroendocrine responses (Shi et al., 2020). Moreover, targeted amino acid supplementation (e.g., methionine, glutamine) can support immune function and enhance intestinal barrier integrity, which in turn may attenuate peripheral inflammatory signaling associated with altered behavior and increased vulnerability to stress-related somatic disturbances (Ma & Ma, 2019; Trzeciak & Herbet, 2021). These nutritional interventions function not as curative measures but as resilience enhancers that attenuate cumulative psychosomatic load.

Environmental predictability and cognitive stimulation significantly moderate stress susceptibility under climate pressure. Enrichment strategies, including shaded resting areas, social stability, manipulable objects, and foraging complexity, reduce anxiety-like behavior, normalize HPA-axis activity, and improve autonomic regulation (Boissy et al., 2007). It was found that in companion animals and managed wildlife, structured routines, thermal comfort zones, and controlled exposure to environmental variability promote emotional coping capacity. The findings support the concept that psychosomatic vulnerability under climate change is partially mediated by perceived environmental controllability, not solely physical exposure.

Gradual heat acclimation protocols enhance thermoregulatory efficiency, cardiovascular stability, and hormonal adaptation. Controlled pre-exposure to moderate heat loads improves sweat gland activity, plasma volume regulation, and cortisol responsiveness, thereby lowering psychosomatic strain during extreme events (Horowitz et al., 2015; Horowitz, 2017). Importantly, acclimation must remain within adaptive thresholds, as excessive or prolonged exposure exacerbates inflammatory burden and emotional dysregulation rather than promoting resilience.

Preventive effectiveness increases substantially when integrated with continuous physiological and behavioral monitoring systems. Composite indices incorporating cortisol trends, HRV metrics, activity patterns, and inflammatory biomarkers enable early identification of maladaptive trajectories before clinical disease emerges (Fuller et al., 2010; Glover, 2018; Madliger et al., 2018; Seebacher et al., 2023).

Table 3 summarizes contemporary strategies of prevention or mitigation of psychosomatic stress in animals under climate change. The strategies combine infrastructure, management, veterinary care, habitat conservation, selective breeding, and the application of digital monitoring technologies. Integrating these approaches provides a comprehensive framework to enhance welfare, reduce the risk of diseases, and support survival resilience in both domestic and wild animal species.

Table 3
Preventive and mitigation strategies for psychosomatic stress in animals under climate change

Strategy domain	Intervention	Targeted psychosomatic mechanism	Documented effects	Key reference (APA)
Microclimate control	Shade structures, ventilation, evaporative cooling	Reduction of HPA-axis activation and thermal load	Lower cortisol, improved feed intake, reduced distress behaviors	Bernabucci et al., 2014
Stocking management	Reduced density, optimized housing orientation	Decreased social stress and autonomic arousal	Improved welfare scores, stabilized physiological stress markers	Polsky & von Keyserlingk, 2017
Nutritional modulation	Antioxidants (vitamin E, selenium), electrolytes	Oxidative stress reduction, endocrine stabilization	Enhanced thermotolerance, reduced inflammatory activation	Shi et al., 2020
Energy metabolism support	Rumen-protected fats, adjusted feeding schedules	Maintenance of metabolic homeostasis	Improved energetic efficiency under heat stress	Baumgard & Rhoads, 2013
Gut integrity support	Functional lipids, amino acids (glutamine, methionine)	Reduced gut permeability and inflammatory signaling	Enhanced immune resilience and stress tolerance	Liu et al., 2012
Environmental enrichment	Predictable routines, social stability, manipulable objects	Emotional regulation, reduced anxiety-like behavior	Normalized HPA activity, improved autonomic balance	Boissy et al., 2007
Physiological acclimation	Gradual heat exposure protocols	Improved thermoregulatory efficiency	Lower hormonal stress response during heat waves	Horowitz, 2017
Long-term adaptation planning	Housing redesign, climate-resilient husbandry	Cumulative stress load reduction	Sustained welfare improvement	Renaudeau et al., 2012
Health surveillance	Integrated behavioral and physiological monitoring	Early detection of maladaptation	Prevention of clinical psychosomatic disorders	Collier et al., 2017
Production system adjustment	Seasonal workload and feeding shifts	Reduced chronic metabolic strain	Improved performance stability	West, 2003

The table highlights interventions that unite environmental, physiological, genetic, and technological approaches which are most effective for reducing the impacts of climate-related psychosomatic stress on animal health and welfare. Such preventive surveillance aligns with climate adaptation frameworks advocated by Intergovernmental Panel on Climate Change, which emphasize anticipatory management over reactive treatment in biological systems facing environmental change.

While environmental modification and resilience enhancement substantially reduce psychosomatic burden, prevention cannot fully eliminate climate-related pathology under escalating global temperatures and extreme weather frequency. Genetic susceptibility, early-life stress exposure, and cumulative environmental insults modulate individual outcomes, necessitating ethical reassessment of husbandry intensity in climate-vulnerable regions. Thus, psychosomatic prevention should be viewed as a dynamic risk-reduction strategy rather than absolute protection.

Integrated strategies for mitigating climate-related psychosomatic stress disorders in animals

Effective mitigation of climate-related psychosomatic stress disorders requires coordinated intervention across environmental, physiological, behavioral, and management domains. Isolated preventive measures, such as cooling infrastructure or nutritional supplementation, provide partial protection but fail to address the multisystem dysregulation characteristic of chronic climate stress. Contemporary evidence supports integrative frameworks that simultaneously reduce physical stress exposure, enhance biological resilience, and stabilize emotional regulation. Such systems-based mitigation aligns with adaptive biological management principles promoted by the Food and Agriculture Organization of the United Nations and climate risk frameworks developed by the Intergovernmental Panel on Climate Change, which emphasize anticipatory, multi-layered intervention strategies to enhance resilience rather than relying on reactive treatment of stress-related outcomes (FAO, 2018; IPCC, 2022).

Environmental modification remains the foundational layer of mitigation, but its effectiveness increases substantially when coupled with metabolic and endocrine support. Cooling systems, shade provision, and housing optimization consistently reduce thermal load, yet animals remain vulnerable to oxidative stress and metabolic disruption if nutritional resilience is not simultaneously addressed (Baumgard & Rhoads, 2013; Bernabucci et al., 2014).

Integrated programs combining microclimate control with antioxidant supplementation, electrolyte balance, and energy-dense feeding strategies demonstrate superior stabilization of cortisol dynamics, inflammatory markers, and behavioral calmness compared with environmental modification alone (Collier et al., 2019; Shi et al., 2020). This synergy illustrates the necessity of targeting both stress exposure and internal stress-processing capacity. Climate stress disrupts emotional regulation by increasing environmental unpredictability and physical discomfort. Enrichment strategies embedded within climate-adapted housing, such as shaded social resting zones, structured feeding schedules, and manipulable substrates, buffer anxiety-like responses while supporting autonomic stability (Boissy et al., 2007).

Importantly, enrichment is most effective when thermal comfort is already ensured; emotional buffering cannot compensate for sustained physiological distress. Thus, behavioral strategies function as amplifiers of resilience rather than primary mitigation tools. Emerging evidence highlights the central role of intestinal integrity and immune regulation in psychosomatic adaptation to climate stress. Heat exposure increases gut permeability, endotoxin translocation, and systemic inflammatory activation, which in turn exacerbate HPA-axis dysregulation and depressive-like behavioral states (Liu et al., 2012). Integrated mitigation, therefore, increasingly incorporates functional lipids, amino acid support, and dietary strategies that preserve epithelial barrier function alongside conventional cooling and nutritional measures. This approach attenuates the inflammatory feedback loop linking somatic stress to emotional dysregulation.

Gradual physiological conditioning to moderate thermal stress improves cardiovascular efficiency, sweat gland function, and hormonal responsiveness, reducing psychosomatic vulnerability during extreme climate events (Horowitzet al., 2015; Horowitz, 2017). However, acclimation must be paired with adaptive workload planning, seasonal production adjustments, and rest scheduling to prevent cumulative exhaustion. Long-term system redesign, including climate-resilient housing, adjusted breeding cycles, and resource distribution, further minimizes chronic stress accumulation (Renaudeau et al., 2012).

Mitigation effectiveness increases substantially when embedded within real-time surveillance frameworks. Behavioral metrics, heart rate variability, cortisol trends, and inflammatory biomarkers enable early detection of maladaptive trajectories, allowing dynamic adjustment of environmental, nutritional, and management interventions (Collier et al., 2017). This feedback-based mitigation model transforms psychosomatic care from static prevention into adaptive biological regulation.

While integrated strategies markedly reduce psychosomatic risk, they cannot fully offset the biological consequences of escalating climate extremes. Genetic susceptibility, early-life stress exposure, and cumulative environmental load constrain adaptive capacity. Ethical and sustainability considerations therefore increasingly intersect with psychosomatic mitigation, particularly in regions facing persistent thermal stress. Integrated mitigation should thus be viewed as risk minimization within ecological limits rather than absolute protection.

Ultimately, the most effective mitigation strategies are those that combine biological, behavioral, environmental, and technological dimensions into a unified system. Such integrated frameworks support early detection, proactive intervention, and the need for development of long-term resilience in both domestic and wild animal populations. As climate change continues reshaping ecosystems and animal physiology, cross-disciplinary collaboration among veterinarians, ecologists, ethologists, animal scientists, and data specialists becomes essential. The collaboration ensures sustaining animal welfare and prevents the negative cascading effects of psychosomatic disturbances on animals' health, productivity, and biodiversity.

General discussion

Climate change emerges not merely as a physical environmental challenge but as a chronic multisystem stressor that progressively reshapes neuroendocrine regulation, immune function, metabolism, and emotional stability in animals. The evidence synthesized across physiological, behavioral, and molecular domains demonstrates that psychosomatic stress disorders under climate pressure arise through cumulative dysregulation rather than isolated acute events. Heat stress, environmental unpredictability, nutritional instability, and habitat modification converge to amplify HPA-axis activation, autonomic imbalance, inflammatory load, and behavioral maladaptation.

A central theme across contemporary research is the bidirectional nature of psychosomatic mechanisms. Neuroendocrine stress responses exacerbate immune and metabolic dysfunction by promoting sustained glucocorticoid release, altered cytokine signaling, and metabolic imbalance. In parallel, chronic systemic inflammation and disruption of the intestinal barrier increase the translocation of microbial products into circulation, which further activates inflammatory pathways and neuroimmune communication. These peripheral inflammatory signals affect brain regions involved in emotional regulation and stress processing, thereby intensifying emotional instability, anxiety-like states, and vulnerability to subsequent stressors. Together, these processes form a bidirectional feedback loop in which stress physiology amplifies somatic dysfunction, while inflammatory and gut-derived signals perpetuate neuroendocrine dysregulation (Liu et al., 2012; Baumgard & Rhoads, 2013). All of this reinforces the inadequacy of unidimensional welfare assessments and supports the integrative monitoring frameworks presented earlier in this article.

Preventive and mitigation strategies demonstrate greatest efficacy when implemented as coordinated systems rather than isolated interventions. Environmental buffering alone reduces thermal exposure but fails to prevent oxidative stress, metabolic inefficiency, and emotional strain unless paired with nutritional resilience, behavioral stabilization, and physiological conditioning (Bernabucci et al., 2014; Horowitz, 2017; Shi et al., 2020). The success of integrated approaches underscores that psychosomatic vulnerability under climate change reflects both external stress load and internal regulatory capacity.

Importantly, emerging monitoring technologies, such as continuous behavioral analytics, wearable physiological sensors, and biomarker profiling, enable early detection of maladaptive trajectories before overt clinical pathology develops (Collier et al., 2017). This transition from reactive treatment to anticipatory regulation represents a fundamental shift in climate-resilient animal management.

Nevertheless, biological limits to adaptation remain evident. Genetic predisposition, early-life stress exposure, cumulative inflammatory burden, and escalating frequency of extreme climate events constrain long-term resilience. Even optimally managed systems cannot fully offset the physiological costs of sustained environmental warming. Ethical considerations therefore increasingly interfere with psychosomatic health, particularly in intensive production systems situated within climate-vulnerable regions. From a broader perspective, the psychosomatic framework aligns with adaptive management principles advanced by the Food and Agriculture Organization of the United Nations and climate risk projections developed by the Intergovernmental Panel on Climate Change, which emphasize the importance of consideration and understanding of systemic resilience, anticipatory intervention, and ecological limits to adaptation (FAO, 2018;

IPCC, 2022). Finally, in this article, there were reviewed advances in biomarkers and diagnostic tools, showing that non-invasive endocrine indicators, heart-rate variability, immune cell profiles, and automated behavior-tracking systems may provide accurate means for early detection of stress-related homeostasis dysregulation. In general, it was found that integration of physiological metrics with behavioral observation and precision-livestock technologies represents a promising direction for both research and clinical application.

Conclusion

Climate-related psychosomatic stress disorders represent an emerging and underrecognized dimension of animal health in the context of global environmental change. The convergence of neuroendocrine dysregulation, inflammatory activation, metabolic disruption, and behavioral maladaptation forms a self-reinforcing pathological network rather than independent physiological disturbances. Psychosomatic stress disorders may also be viewed as a multifactorial condition driven by chronic exposure of animals to environmental, social, and anthropogenic challenges. Physiological dysregulation, particularly of the HPA axis and autonomic nervous system, links stress to increased disease vulnerability. Behavioral indicators serve as the most practical early markers of chronic stress. Early diagnosis allows timely intervention in both domestic and wild animals. Social environment plays a critical role in shaping stress resilience, highlighting the importance of stable group structures and welfare-oriented management. Anthropogenic factors, including habitat loss, pollution, and disruptive human activity, further elevate stress risk, with climate change emerging as a dominant driver of stress-related pathology. These changes necessitate the development and adoption the preventive strategies that are effective to cope with extreme environmental conditions.

The growing availability of biomarkers and digital monitoring tools offers good opportunities for early detection of psychosomatic disturbances and the implementation of personalized welfare management. This approach forms the foundation for mitigating the impact of chronic climate driven stress and opens the door for safeguarding both individual animal health and population resilience.

Effective protection of animal welfare under climate change requires: integrated monitoring systems capturing behavioral, endocrine, autonomic, and immune indicators; multilevel preventive strategies combining environmental modification, nutritional resilience, emotional buffering, and adaptive acclimation; dynamic mitigation frameworks informed by continuous physiological feedback rather than static management protocols.

Future research should prioritize longitudinal designs, mechanistic exploration of gut-brain-immune interactions under climate stress, and species-specific resilience profiling. Without such integrative approaches, psychosomatic morbidity will likely escalate alongside global temperature rise and environmental instability.

Overall, the presented data indicates that preventing and mitigating climate-driven psychosomatic pathology requires a multidisciplinary approach that integrates behavioral science, neurobiology, environmental management, and climate-adaptive strategies. For veterinary practitioners, wildlife biologists, and animal welfare professionals, the priority task should not be only treating stress-related disorders but also redesigning environments and management systems that actively promote resilience, adaptive capacity, and long-term animals' well-being. We strongly believe that the future of preventive veterinary medicine lies in the synergy of ecology, physiology, and technological innovation.

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