Dog Behavior, 2-2025, pp. 7-22 • DOI 10.4454/db.v11i2.211

A Comprehensive Review of Canine Anxiety: Pathogenesis, Behavioral Outcomes, and Treatment

Giovanni Lodrini[1,2*], Virginia Bellini[2], Alice Bevilacqua[2], Susanna Bova[2], Alessia Castagnoli[2], Carla Olmo[2], Valentina Gazzano[3]

[1] *Veterinary behaviorist freelance*
[2] *Dog trainer freelance*
[3] *Department of Veterinary Science, University of Pisa, Italy*

Abstract: Canine anxiety is a complex and multifactorial emotional condition characterized by the anticipation of perceived threats and associated with a wide range of clinical and behavioral manifestations. It can significantly impair animal welfare and the quality of the human–animal relationship, representing one of the most common reasons for referral in veterinary behavioral medicine. This paper provides an updated review of anxiety disorders in dogs, examining their pathogenesis through the interaction of genetic predisposition, life experiences, environmental factors, and concurrent clinical conditions. The main clinical forms of anxiety and the most frequent behavioral comorbidities, such as aggression, compulsive behaviors, separation-related problems, and social impairments are described. Particular attention is devoted to the neurobiological basis of canine anxiety, focusing on limbic neural circuits, the hypothalamic–pituitary–adrenal axis, and the key neurotransmitter systems involved in emotional regulation. The importance of an integrated therapeutic approach is emphasized, combining environmental management, behavioral modification strategies, and, when indicated, pharmacological or nutraceutical interventions. Overall, the review highlights the value of an evidence-based, multidisciplinary perspective for accurate assessment, effective treatment, and targeted prevention of anxiety disorders in dogs.

Key Words: canine anxiety disorders, dog behavior, psychopharmacology, behavioral medicine.

* *Corresponding Author*: giovanni.lodrini@tiscali.it

Introduction

Anxiety is an emotional state characterized by the apprehensive anticipation of a potential future threat, which may or may not actually occur. This anticipatory process leads to increased vigilance and arousal, resulting in a state of alertness that can interfere with normal canine behavior (Dias et al., 2013). Such a condition poses a significant threat to both the animal's psychophysical well-being and the quality of the human–animal relationship, to the extent that it represents one of the most frequent causes of relinquishment or requests for euthanasia (Talegon & Delgado, 2011).

In human medicine, the DSM-IV distinguishes two dimensions of anxiety: state anxiety and trait anxiety. State anxiety refers to a transient condition experienced at a specific moment, whereas trait anxiety reflects a stable predisposition to respond with anxiety across a wide range of situations (Beuzen & Belzung, 1995). A similar distinction can also be observed in dogs: in individuals with a high anxious trait, the emotional response tends to generalize, manifesting with greater intensity and frequency and across a broader range of contexts (Salonen et al., 2020).

Clinical Forms of Canine Anxiety

Anxiety can manifest in different forms, which vary in terms of duration, frequency, and triggering conditions.

Submitted, 12/15/2025
Accepted, 01/04/2026

Paroxysmal anxiety

This form presents as episodic, highly intense, and short-lasting episodes. Clinical signs may include tachycardia, tachypnea, tremors, diarrhea, and other gastrointestinal disturbances. It has been reported particularly in Irish Setters, Dobermans, and Toy Poodles (Pageat, 1999).

Intermittent anxiety

Episodes occur more frequently and are characterized by a clearly defined onset and resolution. This form is often associated with irritability- or fear-related aggression, tachycardia, tachypnea, gastrointestinal disturbances, and emotional urination (Pageat, 1999).

Permanent anxiety

In this form, the anxious state is constant and significantly compromises quality of life. The emotional condition becomes pathological due to its generalized nature, with increased stimulus reactivity, hypervigilance, heightened motor activity, and interference with normal social interactions (Blackwell et al., 2013).

Specific anxiety

Clinical signs emerge when the animal anticipates the occurrence of a specific stimulus perceived as threatening. This category includes noise fears (e.g., fireworks and loud sounds), separation anxiety, and isolation-related anxiety (Landsberg et al., 2024). Context-dependent situations, such as veterinary visits or car travel, may also act as powerful triggers. Clinical signs include tachycardia, tachypnea, mydriasis, hypersalivation, inappropriate elimination, and excessive motor activity (Piotti et al., 2019).

Generalized anxiety

Generalized anxiety is characterized by persistent reactivity accompanied by hypervigilance, increased exploratory behavior, and marked motor activity, interfering with normal social interactions. Unlike specific anxieties, symptoms may occur even in the absence of clear environmental triggers, particularly in low-stimulation contexts. The most common clinical signs include tremors, muscle tension, and palpitations (Baccolo, 2020). It is important to emphasize that the expression of generalized anxiety is strongly influenced by individual genetic predisposition and environmental characteristics (Overall, 2013). In dogs with generalized anxiety, behavioral comorbidities are frequent and include fear-related aggression, stress-induced or territorial aggression, panic attacks, excessive attention-seeking behaviors, displacement activities, and separation-related disorders. Moreover, specific phobias, such as fear of fireworks or thunderstorms, represent important predisposing and aggravating factors (Bamberger & Houpt, 2006; Gazzano & Ogi, 2020).

Etiology and Predisposing Factors

The development of anxiety disorders in dogs results from the interaction of genetic, environmental, experiential, and clinical factors. The heterogeneity of anxiety-related manifestations means that the relative contribution of these elements may vary considerably among individuals.

Genetic factors

A genetic predisposition to anxiety disorders is supported by multiple lines of evidence. Genes involved in glutamatergic and dopaminergic neurotransmission have been identified, suggesting a heritable component underlying vulnerability to anxiety (Sarviaho et al., 2019). Certain dog breeds, such as the Border Collie, German Shepherd, and Chihuahua, show an increased susceptibility to the development of anxiety-related behaviors, further supporting a genetic contribution to predisposition (Salonen et al., 2020).

Early experiences and the quality of the living environment play a crucial role in both the development and prevention of anxiety disorders.

Early experiences

Traumatic events during primary and secondary socialization periods increase the risk of anxiety disorders (Dietz et al., 2018; Fryers & Brugha, 2013). Early separation from the dam and inadequate maternal care are also considered predisposing factors, as they negatively affect the development of emotional regulation capacities.

Relationship quality and attachment

An insecure attachment style (anxious or avoidant) may lead to an unstable relationship with the owner, thereby increasing emotional vulnerability (Mariti et al., 2013).

Social and environmental experiences

Limited exposure during the juvenile period, insufficient socialization with conspecifics, humans, or other species, as well as an impoverished environment, or conversely, an environment excessively rich in stimuli, represent additional predisposing factors.

Negative environmental stimuli also include intense and sudden noises (e.g., thunder and fireworks), abrupt changes in routine or domestic context, and prolonged periods of social isolation.

Physical and mental activity

A lack of physical exercise reduces an important protective factor against stress, as physical activity promotes serotonin release with anxiolytic and antidepressant effects. Similarly, the absence of structured activities and shared mental stimulation with the owner may contribute to the development of anxiety-related behaviors (Tiira et al., 2015).

Shelter environment

Residence in a shelter can represent a significant risk factor: unpredictability, sensory overload, social isolation, and chronic stress lead to persistent activation of the hypothalamic–pituitary–adrenal (HPA) axis, with increased cortisol levels and reduced oxytocin, even during routine procedures such as veterinary examinations. However, specific nutraceutical interventions have been shown to improve social skills and reduce anxiety-related behaviors in shelter-housed dogs, suggesting potential strategies for risk mitigation (Giuliano et al., 2024; Gazzano et al., 2025). Furthermore, suboptimal shelter management and prolonged stays have been shown to promote chronic stress and behavioral alterations, increasing vulnerability to anxiety disorders and reducing the likelihood of adoption (Iacopini & Gazzano, 2024).

Clinical conditions

Acute or chronic pain (Malkani et al., 2024; Sidel, 2025), neurological diseases (Camps et al., 2019), cognitive dysfunction (Gazzano et al., 2024), hormonal imbalances (Camps et al., 2019), and sensory impairments (Camps et al., 2019; Malkani et al., 2024) may contribute to the onset or persistence of anxiety. These conditions can act either as primary factors or as amplifiers of pre-existing vulnerability, significantly influencing the dog's emotional reactivity.

Neurobiological Basis of Anxiety

Recent advances in neuroscientific research, supported by animal models and clinical studies in dogs, have allowed a more precise characterization of the neural bases of anxiety. The main structures involved include the amygdala, hippocampus, prefrontal cortex, thalamus, and hypothalamus, key components of the limbic system responsible for emotional processing and the regulation of behavioral responses (LeDoux, 2000).

In parallel, neurotransmitter systems such as serotonin, GABA, noradrenaline, dopamine, and glutamate play a crucial role in modulating emotional circuits and mediating communication between cortical and subcortical brain regions (Millan, 2003). Evidence related to hemispheric lateralization further indicates a greater specialization of the right hemisphere in the processing of negative and high-intensity emotions (Rogers, 2010).

From an ethological perspective, activation of limbic circuits gives rise to stereotyped defensive responses such as fight, flight, and freezing, which are evolutionarily conserved and strongly influenced by the limbic system and the hypothalamic–pituitary–adrenal (HPA) axis (Kalin et al., 2004).

Neural Circuits and Neurotransmitter Systems

The functioning of the central nervous system relies on the interaction between neural circuits and neurotransmitter systems, two components that are closely integrated (Avery & Krichmar, 2017).

Neural circuits constitute the physical network through which information is transmitted and processed via excitatory and inhibitory connections, and they are involved in sensory, motor, and emotional responses (Tovote et al., 2015; Purves et al., 2018).

Neurotransmitters represent the fundamental biochemical component of synaptic communication and play a key role in modulating neural signal dynamics, thereby influencing information processing and the regulation of emotional and behavioral states. In dogs, alterations in neurotransmitter systems have been associated with the development of anxiety-related behavioral disorders, particularly through changes in the levels and balance of excitatory, inhibitory, and modulatory mediators (Riva et al., 2008). More recently, pharmacological models of control systems have highlighted how these neurotransmitters act within complex neural networks characterized by feedback loops and neuromodulatory mechanisms that regulate adaptive responses to stress (Gholami & Mortezaee, 2025).

Based on their primary function, neurotransmitters can be classified as:

- excitatory, such as glutamate;
- inhibitory, such as γ-aminobutyric acid (GABA);
- modulatory, including dopamine, serotonin, and acetylcholine.

While neural circuits define the direction and organization of the signal, neurotransmitters regulate its intensity, duration, and valence. Structural or biochemical alterations can disrupt emotional balance: circuit-level damage may affect neurotransmitter availability, whereas neurochemi-

cal deficits, such as reduced serotonin levels, can interfere with the functioning of limbic structures, thereby contributing to the emergence of anxiety states (Millan, 2003).

Brain Structures Involved in Anxiety

Anxiety in dogs arises from the dynamic interaction between specific brain structures and neurotransmitter systems that collectively regulate threat perception, emotional modulation, and the activation of behavioral responses. At the core of this system lies the limbic system, an anatomofunctional network responsible for emotional processing, memory, and emotional learning (Graef, 1994; Etkin et al., 2011; Bocchio et al., 2016; Barson et al., 2020). Neuroimaging and structural connectivity studies have shown that, in dogs with anxiety disorders, alterations in the limbic connectome are associated with ineffective regulation of emotional responses and increased reactivity to stress-related stimuli (Chen et al., 2023). These findings are consistent with neurobiological models of anxiety, in which dysfunctions within limbic circuits and neuromodulatory systems contribute to the persistence of anxiety states through impaired integration of emotional, cognitive, and neuroendocrine signals (Martin et al., 2008).

Within the limbic system, the amygdala represents the primary center for evaluating the threat value of stimuli. It integrates sensory information from the environment with previously stored emotional memories, enabling rapid attribution of emotional significance to events and the immediate activation of fear and defensive responses. This central role of the amygdala in emotional processing has been extensively documented in neurobiological studies describing its evolutionary origin and its function as a key hub in the regulation of emotions and motivated behaviors (Simic et al., 2021). Amygdalar activity depends on a dynamic balance between excitatory and inhibitory neurotransmitters: glutamate enhances reactivity and promotes the consolidation of fear memory, whereas GABA exerts a regulatory effect by limiting excessive activation. A predominance of glutamatergic activity may lead to persistent anxiety responses (Ressler & Mayberg, 2007), while adequate GABAergic activity contributes to the containment of emotional reactivity. Neuroimaging studies in dogs confirm increased amygdala activity in the presence of threatening stimuli (Coulik et al., 2014).

Alongside the amygdala, the hippocampus plays a crucial role in contextual memory and in the ability to associate a stimulus with its environmental relevance. In particular, the hippocampus is involved in the retrieval of detailed contextual representations, allowing the individual to discriminate between similar situations based on previous experience and to appropriately modulate emotional responses (Wiltgen et al., 2010). Functional interactions between the hippocampus and the amygdala are critical in the learning and maintenance of contextual fear, with a dynamic shift in mnemonic processing between these two structures depending on the phase of acquisition, consolidation, and retrieval of emotional memories (Chaaya et al., 2018). Glutamate and acetylcholine are involved in memory consolidation and retrieval processes; functional alterations at this level may impair the proper discrimination between real and perceived threats, thereby promoting inappropriate anxiety responses (McEwen et al., 2016).

Another key node is the hypothalamus, which is involved in the integration of emotional processing and the endocrine stress response. Through the release of corticotropin-releasing hormone (CRH), it activates the hypothalamic–pituitary–adrenal (HPA) axis, leading to increased cortisol secretion. Elevated cortisol levels, typical of chronic stress conditions, have also been observed in dogs affected by anxiety disorders (Hydbring-Sandberg et al., 2004).

Within the brainstem, the locus coeruleus represents the primary source of noradrenaline, a key neurotransmitter involved in modulating arousal, alertness, and responsiveness to stimuli. Hyperactivity of the noradrenergic system can lead to states of hypervigilance and increased sensitivity to external stimuli, which are hallmark features of anxiety disorders (Caestecker et al., 2025).

The cognitive modulation of emotions is mediated by the prefrontal cortex (PFC), which integrates sensory, mnemonic, and emotional information with decision-making and executive processes. The PFC exerts inhibitory control over the amygdala and contributes to the regulation of emotional responses. Under conditions of prolonged stress, reduced efficiency of prefrontal circuits may impair the dog's ability to modulate impulsive responses, thereby promoting the onset or persistence of anxiety (Horowitz, 2017).

In addition to the systems already discussed, other neurotransmitters also play a significant role in the regulation of emotional states. In particular, neuromodulatory mechanisms rely on complex interactions between intracellular signaling pathways and neural networks, through which multiple chemical mediators contribute to modulating the intensity, duration, and contextual relevance of emotional responses. Recent evidence highlights how cross-talk among cellular signaling pathways represents a key element in the fine regulation of emotions and in behavioral adaptation to environmental stimuli (Tsuboi et al., 2024).

Serotonin (5-HT) is involved in the modulation of mood, impulsivity, and emotional stability through receptors widely distributed in the amygdala, hippocampus, and prefrontal cortex. Dopamine, which plays a central role in learning, motivation, and social behavior, contributes to anxiety regulation through circuits connecting frontal, limbic, and mesolimbic regions; reduced dopaminergic activity has been associated with emotional vulnerability and diminished adaptive capacities (Landsberg et al., 2024).

Taken together, these brain structures and their associated neurochemical systems constitute a highly integrated network in which anatomical and biochemical components cooperate in regulating emotional and behavioral responses. Functional, structural, or neurotransmitter-related alterations affecting even a single node within this network may impair the dog's ability to accurately interpret and cope with environmental stimuli, thereby promoting the onset or maintenance of anxiety states. Neuroanatomical and neurochemical evidence indicates that anxiety disorders in dogs arise from distributed dysfunctions rather than from isolated alterations within a single brain structure (Vermeire et al., 2011; Riva et al., 2008).

Behavioral Outcomes and Comorbidities

Most canine behavioral disorders are accompanied by anxiety (Ibanez & Anzola, 2011). In particular, anxiety is commonly observed in dogs with separation-related problems, noise sensitivity, generalized fear, compulsive behaviors, and aggressive behavior (Salonen et al., 2020).

Separation-Related Problems

Separation-related problems represent a multifaceted condition in which dogs exhibit significant distress when left alone at home. From a behavioral perspective, clinical signs range from vocalization and destructive behavior to inappropriate elimination (urination and defecation) and excessive motor activity. These behaviors typically occur within 30 minutes of the owner's departure (Lund & Jorgensen, 1999; Sherman et al., 2008).

The behaviors displayed reflect responses to different emotional states, such as fear, phobia, frustration, or anger, and to different situational contexts, including separation from the attachment figure versus social isolation or solitude. Although emotional and situational components vary among individual cases, an anxiety component is almost always present. Dogs anticipate the occurrence of the problematic event (separation or being left alone) by relying on environmental cues previously associated with that event. Consequently, behavioral and physiological changes associated with anxiety often arise before the owner's departure and prior to the onset of actual isolation (Amat et al., 2014).

The most severe condition occurs when, at the moment of the owner's departure, anxiety is accompanied by a phobic response. In such cases, the stress experienced by the dog reaches extremely high levels, and the resulting behavioral responses—such as escape attempts or frantic searching for the owner—may pose a serious risk to the animal's safety and even its life (Davis et al., 2010).

Anxiety-Related Aggression

Aggressive behavior is part of the canine ethogram and, like all behaviors, should be considered physiological when it has adaptive value, allowing the animal to achieve a goal such as self-protection, defense of offspring, or safeguarding resources. When physiological, aggressive behavior is characterized by an ordered and complete sequence of phases and is proportional in both intensity and duration to the context and to the stimulus perceived as threatening (Gazzano, 2013). The emotional states commonly associated with this behavior include anger, frustration, and fear.

In anxious dogs living under conditions of chronic stress, a lowering of the threshold for aggressive responses is frequently observed, as heightened emotional arousal increases reactivity to stimuli (Arata et al., 2014). Anxiety represents the most frequent comorbidity in fear-related aggression, stress-induced aggression, inter-dog aggression, and territorial aggression (Tiira et al., 2016).

Compulsive Behaviors and Anxiety

Compulsive behaviors are repetitive, non-adaptive, and context-inappropriate actions in which the animal loses control over both the initiation and termination of the behavior, and which interfere with normal social activities (Kaulfuss et al., 2010). These behaviors typically arise when dogs experience frustration resulting from the inability to satisfy their motivational needs and/or when they are exposed to conflict generated by the simultaneous motivation to perform two opposing behaviors (Protopopova et al., 2014).

Predisposing factors include an anxious personality, poor frustration tolerance, and heightened excitability; together, these factors create the conditions for the development of compulsive disorders (Bowen & Fatjó, 2024).

Treatment Options

The management of anxiety in dogs requires consideration of three core elements: the environment in which the dog lives, behavioral modification strategies, and pharmacological therapy.

Environment

Regardless of the nature of the stimulus that triggered the anxious state, it is essential that the dog lives in an "enriched" environment. This includes a structured daily routine and regular engagement by the owner in activities such as olfactory games, cognitive stimulation, play, and physical exercise (Bousono Garcia et al., 1999). The owner also plays a fundamental role in determining environmental quality: knowledge of canine communication, learning processes, and ethological needs allows the dog to live in an organized environment characterized by clear and consistent rules.

An environment designed in this way supports neurotransmitter regulation of the dog's emotional and psychological state. Increased release of serotonin and dopamine promotes positive mood, reduces anxiety, and improves learning performance. In parallel, elevated circulating endorphin levels and reduced cortisol concentrations enhance overall well-being, improve stress

coping efficiency, and support more effective cognitive functioning. These effects are crucial in increasing the likelihood of success of behavioral modification techniques (Albright & Ng, 2022).

It should also be emphasized that facilitating the expression of these activities and providing a stable daily routine enhances the dog's perception of control over its environment. Moreover, the predictability of events reduces the likelihood of anxiety development and maintenance (Ibanez & Anzola, 2011).

Behavioral Modification

General principles applicable to anxiety treatment include:
- Avoiding punishment, particularly positive punishment;
- Minimizing exposure to the triggering stimulus, ensuring that any exposure occurs in a gradual and controlled manner;
- Replacing anxiety-related behaviors by modifying the dog's underlying emotional state.

Behavioral modification techniques vary depending on the stimulus responsible for the dog's anxiety. Habituation, desensitization, and counterconditioning are among the most commonly used approaches. While habituation and desensitization aim to increase the response threshold to a given stimulus, counterconditioning seeks to alter the dog's perception—and therefore emotional response—to a stimulus perceived as negative, thereby promoting an alternative behavior to the problematic one (Overall, 1997).

Both habituation and desensitization involve presenting the triggering stimulus at progressively increasing levels of intensity that do not elicit the emotional state responsible for the problematic behavior, along with reinforcing desirable behaviors as they are learned (Mills, 2006).

Counterconditioning, often preceded or accompanied by desensitization, is completed by teaching the dog a behavior that is opposite or incompatible with the behavior expressing fear, discomfort, or anger—emotional states that commonly precede anxiety (Overall, 1997).

For example, in separation-related problems, treatment typically includes desensitization to the owner's departure, habituation to being alone, and counterconditioning of solitude itself (Butler et al., 2001). In generalized anxiety, once the triggering stimulus has been identified, desensitization and counterconditioning are applied to that stimulus (Overall, 1997).

Pharmacological Therapy

Pharmacological treatment of anxiety in dogs is based on the modulation of the main neurotransmitter systems involved in emotional regulation. Since anxiety disorders are characterized by alterations in the balance between excitatory and inhibitory neurotransmission, anxiolytic drugs act by restoring neurochemical homeostasis, either by enhancing inhibitory systems or by reducing excessive activation of fear-related neural circuits (Mills & Simpson, 2006).

The use of pharmacological agents varies according to the type of anxiety, the severity of clinical signs, and the neural circuits predominantly involved. In general, medications can be classified into two main categories:
- Long-term medications, administered on a daily basis, which require several weeks to achieve full therapeutic efficacy and are essential for managing the underlying disorder.
- "As-needed" medications, used in specific triggering situations (e.g., noise phobias, acute events, veterinary visits) (Crowell-Davis et al., 2019).

These two categories may be used either separately or in combination, provided that potentially hazardous drug interactions are avoided. In particular, combinations that increase the risk of adverse effects, such as serotonin syndrome resulting from the concurrent use of multiple serotoner-

gic agents, should be carefully avoided (Overall, 2005; Mills, 1995).

Long-Term (Maintenance) Treatments

1. Monoamine Oxidase Inhibitors (MAOIs) (Selegiline)

Selegiline is an irreversible inhibitor of monoamine oxidase B (MAO-B), leading to increased levels of dopamine, serotonin, and noradrenaline. In addition to its anxiolytic effects, selegiline has been shown to reduce neuronal oxidative stress and enhance free radical detoxification mechanisms (Crowell-Davis, 2019). Pageat (2007) reported clinical efficacy of selegiline in dogs unresponsive to fluoxetine, with concurrent reductions in stress-related hyperprolactinemia. Due to the risk of severe drug interactions, careful consideration must be given to washout periods when transitioning between MAOIs and other serotonergic medications.

2. Selective Serotonin Reuptake Inhibitors (SSRIs) (Fluoxetine, Sertraline, Paroxetine, Fluvoxamine)

Selective serotonin reuptake inhibitors (SSRIs) increase synaptic serotonin availability by inhibiting its reuptake. They are widely used in dogs for the management of anxiety, aggression, compulsive disorders, and phobias. Among this class, fluoxetine is the most commonly prescribed drug in veterinary medicine and is particularly indicated for chronic anxiety disorders and separation-related problems (Landsberg et al., 2008). SSRIs typically require 4–6 weeks to achieve full therapeutic efficacy. Adverse effects may include gastrointestinal disturbances, sedation, tremors, and agitation. Caution is warranted due to the risk of serotonin syndrome when SSRIs are combined with other serotonergic agents, such as monoamine oxidase inhibitors or high doses of tricyclic antidepressants (Mills, 1995). In addition, SSRIs may inhibit hepatic cytochrome P450 enzymes, potentially increasing the plasma concentrations and toxicity of concurrently administered drugs (Landsberg et al., 2024).

3. Tricyclic Antidepressants (TCAs) (Clomipramine, Amitriptyline, Imipramine, Doxepin)

Tricyclic antidepressants (TCAs) inhibit the reuptake of serotonin and noradrenaline, thereby increasing the synaptic availability of these neurotransmitters. Among this class, clomipramine is considered the most effective agent for the treatment of anxiety disorders in dogs (Hewson, 1997). TCAs exert their effects on multiple brain regions involved in emotional regulation, including the thalamus, amygdala, cortex, and hippocampus (Kurata et al., 1986). Their use is commonly associated with adverse effects such as sedation, tachycardia, hypotension, constipation, and reductions in circulating thyroid hormone levels. In addition, TCAs should not be co-administered with monoamine oxidase inhibitors such as selegiline, or with certain antiparasitic agents including amitraz, due to the risk of serious pharmacological interactions (Martin, 2010).

Situational and Adjunctive Treatments

4. α2-Adrenergic Agonists (Dexmedetomidine)

Dexmedetomidine acts on α2-adrenergic receptors within the limbic system and the locus coeruleus, reducing noradrenaline release and thereby attenuating arousal and stress responses. It is primarily indicated for the management of acute anxiety states and noise-related phobias and is typically administered 30–45 minutes before the anticipated triggering event (Korpivaara et al., 2017).

Reported adverse effects include vomiting and transient pallor of the mucous membranes, generally mild and self-limiting (Sinclair, 2003).

5. β-Adrenergic Blockers (Pindolol, Propranolol)

β-blockers reduce the peripheral manifestations of anxiety by antagonizing β-adrenergic receptors,

thereby alleviating somatic symptoms such as tachycardia, tremors, and gastrointestinal hypermotility. Although they do not directly address the emotional component of anxiety, they may be useful as part of integrated treatment protocols aimed at controlling autonomic symptoms (Notari, 2005).

6. Benzodiazepines (Alprazolam, Diazepam, Flurazepam, Oxazepam, Clonazepam)

Benzodiazepines (including alprazolam, diazepam, flurazepam, oxazepam, and clonazepam) act as positive allosteric modulators of the GABAAA receptor, rapidly reducing neuronal excitability. Owing to their ability to readily reach key brain regions involved in anxiety—such as the limbic system, cortex, thalamus, and hypothalamus—they exert anxiolytic, sedative, and muscle-relaxant effects. For this reason, benzodiazepines are primarily indicated as as-needed medications and are often used in combination with long-term pharmacological treatments (Mealey, 2019). However, their use may be associated with adverse effects, including behavioral disinhibition with possible exacerbation of aggressive responses, dose-dependent sedation, and interference with learning processes. Moreover, prolonged administration carries a risk of dependence and withdrawal phenomena, limiting their suitability for long-term monotherapy in dogs with anxiety disorders (Herron et al., 2008).

7. Gabapentinoids (Gabapentin, Pregabalin, Carbamazepine)

Gabapentin, a structural analogue of GABA, was originally developed as an analgesic for neuropathic pain but has also demonstrated efficacy in the management of anxiety, noise phobias, and compulsive disorders in dogs (Di Cesare et al., 2023). Clinical effects are typically observed within 60–90 minutes of administration, and gabapentin is frequently used in combination with TCAs or SSRIs.

Although its precise anxiolytic mechanism remains incompletely understood, it is hypothesized to involve a reduction in activity within fear-related neural circuits (Kirby-Madden et al., 2024). Adverse effects are generally mild and mainly consist of sedation at higher doses.

8. Serotonin Antagonist and Reuptake Inhibitors (SARIs): Trazodone

Trazodone, a serotonin antagonist and reuptake inhibitor, may be used both as an as-needed medication and as an adjunct to long-term maintenance therapies. Increased serotonergic activity within the prefrontal cortex is believed to contribute to its anxiolytic effects (Gruen, 2008).

Adverse effects primarily include gastrointestinal disturbances, and there is a risk of serotonin syndrome when trazodone is combined with SSRIs or MAOIs.

In recent years, scientific interest has also expanded to integrated supportive strategies, including the use of nutraceuticals and interventions aimed at modulating the microbiota–gut–brain axis, which are considered potential adjunctive approaches in the management of anxiety disorders. However, the currently available evidence indicates heterogeneous outcomes and highlights the need for a cautious, multimodal clinical approach, in which such interventions complement, rather than replace, established behavioral and pharmacological strategies (Sacchettino et al., 2025).

Prevention of Anxiety Disorders in Dogs

Prevention of anxiety disorders in dogs is based on targeted interventions aimed at reducing exposure to major risk factors and promoting balanced emotional development. Central elements include environmental stability, predictability of daily routines, physical and mental well-being, and the establishment of a secure relationship with the owner.

A first area of intervention concerns responsible selection and breeding practices, favoring individuals with good emotional stability and without marked anxiety-related traits, thereby limiting the transmission of genetic vulnerabilities (Salonen et al., 2020; Pongrácz et al., 2019).

A crucial role is played by early and gradual socialization, which involves positive exposure to people, conspecifics, other animals, environments, and a variety of stimuli during sensitive developmental periods. Such experiences promote the development of social competencies and greater emotional resilience (Howell et al., 2015).

A predictable routine helps reduce uncertainty and anxiety related to the unpredictability of daily events, while regular physical activity represents an important protective factor due to its positive effects on mood regulation. In addition to exercise, mental stimulation and structured activities shared with the owner—such as training, cognitive games, and olfactory activities—support the development of self-confidence and emotional security (Tiira & Lohi, 2015).

The quality of the human–dog relationship plays a decisive role: consistent interactions that are sensitive to the animal's needs foster the formation of a secure attachment bond, which acts as a protective factor against the development of anxiety responses (Mariti et al., 2013).

Finally, gradual management of potentially stressful stimuli, such as intense noises, handling, or environmental changes, allows dogs to process these experiences adaptively, reducing the likelihood that they become triggers for anxiety-related behaviors.

Conclusion

Anxiety disorders in dogs represent a complex and multifactorial condition that can significantly compromise animal welfare and the quality of the human–animal relationship. Anxiety manifestations, ranging from acute episodes to persistent states, arise from the interaction between genetic predispositions, life experiences, and environmental conditions.

Early recognition of the different clinical forms of anxiety is essential for the implementation of targeted interventions. Distinguishing among paroxysmal, intermittent, permanent, specific, and generalized anxiety allows clinicians to appropriately guide diagnostic and therapeutic strategies.

Optimal management requires a multidisciplinary approach that integrates evaluation of the living environment, modification of problematic behaviors, and, when necessary, pharmacological support. Environmental enrichment, predictability of daily routines, and reduction of stressors represent essential components of non-pharmacological interventions. Behavioral techniques such as habituation, desensitization, and counterconditioning constitute core tools of behavioral therapy, while carefully selected and monitored pharmacotherapy may be indispensable in more severe or treatment-resistant cases.

Prevention also plays a crucial role and relies on appropriate socialization, consistent environmental and routine management, adequate levels of physical and mental activity, and, upstream, responsible breeding practices aimed at reducing the transmission of anxiety-related predispositions.

In conclusion, the management of anxiety in dogs requires a comprehensive, individualized, and evidence-based approach that takes into account the characteristics of each animal and its experiences. Collaboration among owners, veterinarians, and behavioral medicine professionals is essential to promote animal welfare and to strengthen a balanced and secure human–dog relationship.

References

Albright J.D., Ng Z.Y. Measurement of neurotransmitters excreted in the urine of behaviorally healthy dogs in home and boarding kennel conditions. Journal of Veterinary Behavior. 48:74–77; 2022.

Altamura A.C., Moro A.R., Percudani M. Clinical pharmacokinetics of fluoxetine. Clinical Pharmacokinetics. 26(3):201–214; 1994.

Amat M., Camps T., Brech S.L., Manteca X. Separation anxiety in dogs: the implications of predictability and contextual fear for behavioural treatment. Animal Welfare. 23:263–266; 2014.

Arata S., Takeuchi Y., Inoue M., Mori Y. Reactivity to stimuli is a temperamental factor contributing to canine aggression. PLoS ONE. 9(6):e100767; 2014.

Avery M.C., Krichmar J.L. Neuromodulatory systems and their interactions: a review of models, theories, and experiments. Frontiers in Neural Circuits. 11:108; 2017. https://doi.org/10.3389/fncir.2017.00108.

Baccolo D. Disturbi d'ansia e bias cognitivi nel cane domestico. Università di Parma; 2020.

Bamberger M., Houpt K.A. Signalment factors, comorbidity and trends in behavior diagnosis in dogs: 1644 cases (1991–2001). Journal of the American Veterinary Medical Association. 229:1591–1601; 2006.

Barson J., Mack N., Gao W. The paraventricular nucleus of the thalamus is an important node in the emotional processing network. Frontiers in Behavioral Neuroscience. 14; 2020. https://doi.org/10.3389/fnbeh.2020.598469.

Beuzen A., Belzung C. Link between emotional memory and anxiety states: a study by principal component analysis. Physiology & Behavior. 57:111–118; 1995.

Blackwell E.J., Bradshaw J.W.S., Casey R.A. Fear responses to noises in domestic dogs: prevalence, risk factors and co-occurrence with other fear-related behaviour. Applied Animal Behaviour Science. 145:15–25; 2013.

Bocchio M., McHugh S., Bannerman D., Sharp T., Capogna M. Serotonin, amygdala and fear: assembling the puzzle. Frontiers in Neural Circuits. 10; 2016. https://doi.org/10.3389/fncir.2016.00024.

Boissy A. Fear and fearfulness in animals. Quarterly Review of Biology. 70(2):165–191; 1995.

Botler R., Sargisson R., Douglas E. The efficacy of systematic desensitization for treating the separation-related problem behaviour of domestic dogs. Journal of Applied Animal Behaviour Science. 129:136–145; 2011.

Bousoño García M., Sáiz P.A., González M.P., Bobes J. Protocolo diagnóstico de los estados de ansiedad. Medicine. 7(106):4969–4971; 1999.

Bowen J., Fatjó J. Repetitive behaviours in dogs. Veterinary Clinics of North America: Small Animal Practice. 54:71–85; 2024.

Chaaya N., Battle A.R., Johnson L.R. An update on contextual fear memory mechanisms: transition between amygdala and hippocampus. Neuroscience & Biobehavioral Reviews. 92:43–54; 2018. https://doi.org/10.1016/j.neubiorev.2018.05.013.

Camps T., Amat M., Manteca X. A review of medical conditions and behavioral problems in dogs and cats. Animals. 9(12):1133; 2019. https://doi.org/10.3390/ani9121133.

Chen A., Xu Y., Christian E., Wu G.-R., De Witte S., Vanhove C., Saunders J., Peremans K., Baeken C. Structural connectome alterations in anxious dogs. Scientific Reports. 13:37121; 2023. https://doi.org/10.1038/s41598-023-37121-0.

Coulik P., et al. Functional MRI of the canine brain during visual stimulation. Journal of Veterinary Behavior. 9(5):272–277; 2014.

Crowell-Davis S.L., Murray T.F., Mattos de Souza Dantas L. Veterinary psychopharmacology. Wiley; 2019.

Davis M., Walker D.L., Miles L., Grillon C. Phasic vs sustained fear in rats and humans: role of the extended amygdala in fear vs anxiety. Neuropsychopharmacology. 35:105–135; 2010.

Di Cesare F., Montanaro G., Negro V., Ravasio G., Villa R., Draghi S., Cagnardi P. Gabapentin: clinical use and pharmacokinetics in dogs, cats and horses. Animals. 13(12):2045; 2023.

Dias B.G., Banerjee S., Goodman J., Ressler K.J. Towards new approaches to disorders of fear and anxiety. Current Opinion in Neurobiology. 23:346–352; 2013.

Dietz L., Arnold A.-M.K., Goerlich-Jansson V.C., Vinke C.M. The importance of early life experiences for the development of behavioural disorders in domestic dogs. Behaviour. 155:83–114; 2018

Etkin A., Egner T., Kalisch R. Emotional processing in anterior cingulate and medial prefrontal cortex. Trends in Cognitive Sciences. 15:85–93; 2011. https://doi.org/10.1016/j.tics.2010.11.004.

Fryers T., Brugha T. Childhood determinants of adult psychiatric disorder. Clinical Practice and Epidemiology in Mental Health. 9:1–50; 2013.

Gazzano A. Manuale di etologia del cane. Edizioni ETS; 2013. 9:103–105.

Gazzano V., Curadi M.C., Baragli P., Mariti C., Cecchi F., Cavallo S., Sacchettino L., Gazzano A. Physiological and Behavioral Evaluation of Shelter Dogs During Veterinary Routine Health Checks. Veterinary Sciences. 12(6):583; 2025. https://doi.org/10.3390/vetsci12060583

Gazzano V., Capsoni S., Cavallo S., Curadi M.C., Casini L., Gazzano A., Cecchi F. Preliminary insights into β-amyloid, phospho-tau and inbreeding in Labrador Retriever dogs. Dog Behavior. 10(3):19–26; 2024. https://doi.org/10.4454/db.v10i3.196.

Gazzano V., Ogi A. Canine phobia. Dog Behavior. 6(2); 2020.

Gholami M., Mortezaee K. Control systems pharmacology: neuromodulation, feedback loops and brain network regulation. ACS Chemical Neuroscience. 16(19):3653–3664; 2025. https://doi.org/10.1021/acschemneuro.5c00426.

Giuliano V.O., Sacchettino L., Gazzano V., Ciccarelli D., Napolitano F., d'Angelo D. Supplementation with nutraceutical Calmina® (BuonaPET) improves anxiety status in shelter dogs: a pilot study. Dog Behavior. 10(3):1–10; 2024.

Graeff F. Neuroanatomy and neurotransmitter regulation of defensive behaviors and related emotions in mammals. Brazilian Journal of Medical and Biological Research. 27(4):811–829; 1994.

Gruen M.E. Use of Trazodone as an adjunctive agent in the treatment of canine anxiety disorders: 56 cases (1995–2007). Journal of the American Veterinary Medical Association. 233(12):1902–1907; 2008.

Haug L.I. Canine aggression toward unfamiliar people and dogs. Veterinary Clinics of North America: Small Animal Practice. 38(5):1023–1040; 2008.

Herron M., Shofer F.S., Reisner I.S. Retrospective evaluation of the effects of diazepam in dogs with anxiety-related behaviour problems. Journal of the American Veterinary Medical Association. 233:1420–1424; 2008.

Hewson C.J. Clomipramine in dogs: pharmacokinetics, neurochemical effects and efficacy in compulsive disorder. University of Guelph; 1997.

Howell T.J., King T., Bennett P.C. Puppy parties and beyond: the role of early age socialization practices on adult dog behavior. Veterinary Medicine (Auckland). 6:143–153; 2015.

Horowitz A. Smelling themselves: dogs investigate their own odours longer when modified in an olfactory mirror test. Behavioural Processes. 143:17–24; 2017.

Hydbring-Sandberg E., von Walter L.W., Höglund K., et al. Physiological reactions to fear provocations in dogs. Journal of Endocrinology. 180(3):439–448; 2004.

Iacopini L., Gazzano V. Analysis of the management of a shelter dog kennel. Dog Behavior. 9(3):41–45; 2024. https://doi.org/10.4454/db.v9i3.183.

Ibáñez M., Anzola B. Anxiety disorders in dogs. In: Anxiety Disorders. InTech; 2011.

Kalin N.H., Shelton S.E., Davidson R.J. The role of the central nucleus of the amygdala in mediating fear and anxiety in the primate. Journal of Neuroscience. 24(24):5506–5515; 2004.

Kaulfuss P., Würbel H., Failing K. Studies on classifying abnormal-repetitive behaviours in dogs. Proceedings of the European Veterinary Behaviour Meeting. 92–96; 2010.

Kaur G., Voith V.L., Schmidt P.L. The use of fluoxetine by veterinarians in dogs and cats: a preliminary survey. Veterinary Record Open. 3:e000146; 2016.

Kirby-Madden T., Thompson C.W., Herron M. Effect of Gabapentin on the treatment of behavioral disorders in dogs: a retrospective evaluation. Animals. 14(10):1462; 2024.

Korpivaara M., Laapas K., Huhtinen M., Schöning B., Overall K. Dexmedetomidine oromucosal gel for noise-associated acute anxiety and fear in dogs. Veterinary Record. 181; 2017. https://doi.org/10.1136/vr.104045.

Kurata K., Kishitani K., Kido H., Kurachi M., Yamaguchi N. A pharmacokinetic study of clomipramine in regions of brain. Psychiatry and Clinical Neurosciences. 40:631–638; 1986.

Landsberg G.M., Melese P., Sherman B.L., Neilson J.C., Zimmerman A., Clarke T.P. Effectiveness of fluoxetine chewable tablets in the treatment of canine separation anxiety. Journal of Veterinary Behavior. 3(1):12–19; 2008.

Landsberg G., Radosta L., Ackerman L. Behavior problems of the dog and cat. Elsevier; 2024.

LeDoux J.E. Emotion circuits in the brain. Annual Review of Neuroscience. 23:155–184; 2000.

Lund D.J., Jorgensen M.C. Behavior patterns and time course of activity in dogs with separation problems. Applied Animal Behaviour Science. 63:219–236; 1999.

Malkani R., Paramasivam S., Wolfensohn S. How does chronic pain impact the lives of dogs: an investigation of factors that are associated with pain using the Animal Welfare Assessment Grid. Frontiers in Veterinary Science. 11:1374858; 2024. https://doi.org/10.3389/fvets.2024.1374858.

Mariti C., Ricci E., Zilocchi M., Gazzano A. Owners as a secure base for their dogs. Behaviour. 150(11):1275–1294; 2013.

Martin E.I., Ressler K.J., Binder E., Nemeroff C.B. The neurobiology of anxiety disorders: brain imaging, genetics and psychoneuroendocrinology. Behavioural Pharmacology. 20(5–6):460–484; 2008.

Martin K.M. Effect of clomipramine on the electrocardiogram and serum thyroid concentrations of healthy cats. Journal of Veterinary Behavior. 5:123–129; 2010.

McEwen B.S., Nasca C., Gray J.D. Stress effects on neuronal structure: hippocampus, amygdala, and prefrontal cortex. Neuropsychopharmacology. 41(1):3–23; 2016.

Mealey K.L. Pharmacotherapeutics for veterinary dispensing. In: Overall K.L. (Ed.), Small Animal Behavior. Elsevier; 2019. pp. 379–391.

Mertens P.A. Reproductive and sexual behavioral problems in dogs. Theriogenology. 66(3):606–609; 2006.

Millan M.J. The neurobiology and control of anxious states. Progress in Neurobiology. 70:83–244; 2003.

Mills D., Simpson B. Agentes psicotropicos. In: Horwitz D., Mills D., Heath S. (Eds.), Manual de comportamiento en pequeños animales. España; 2006.

Mills K.C. Serotonin syndrome. American Family Physician. 52:1475–1482; 1995.

Notari L. Combined use of selegiline and behaviour modifications in cases involving fear and phobias. In: Mills D., Levine E., Landsberg G. (Eds.), Current Issues and Research in Veterinary Behavioural Medicine. Purdue University Press; 2005. pp. 267–269.

Overall K.L. Clinical behavioral medicine for small animals. Mosby-Year Book; 1997.

Overall K.L. Why these drugs work: using neuropharmacology in a manner that addresses how dogs and cats learn. Small Animal Behavior. 103–106; 2005.

Overall K.L. Manual of clinical behavioral medicine for dogs and cats. Elsevier; 2013.

Pageat P. Patologia comportamentale del cane. France Ed.; 1999.

Pageat P., Lafont C., Falewee C., Bonnafous L., Fontbonne A., Alvinerie M. An evaluation of serum prolactin in anxious dogs and response to treatment with selegiline or fluoxetine. Applied Animal Behaviour Science. 105(4):342–350; 2007. https://doi.org/10.1016/j.applanim.2006.11.004

Piotti P., Uccheddu S., Alliani M., Mariti C., Nuti V., Ogi A., Pierantoni L., Gazzano A. Management of specific fears and anxiety in the behavioral medicine of companion animals: punctual use of psychoactive medications. Dog Behavior. 5(2):23–30; 2019. https://doi.org/10.4454/db.v5i2.109

Pongraz P., Gomez S.A., Lenkei R. Separation-related behaviour indicates the effect of functional breed selection in dogs (Canis familiaris). Applied Animal Behaviour Science. 222:104884; 2019.

Protopopova A., Hall N.J., Wynne C.D. Association between increased behavioral persistence and stereotypy in pet dogs. Behavioural Processes. 106:77–81; 2014.

Purves D., Augustine G.J., Fitzpatrick D., Hall W.C., LaMantia A.-S., McNamara J.O., Williams S.M. Neuroscience. 6th ed. Oxford University Press; 2018.

Ressler K.J., Mayberg H.S. Targeting abnormal neural circuits in mood and anxiety disorders. Nature Neuroscience. 10(9):1116–1124; 2007.

Caestecker S., Lescrauwaet E., Boon P., Carrette E., Raedt R., Vonck K. The Locus Coeruleus–Noradrenergic System in the Healthy and Diseased Brain: A Narrative Review. European Journal of Neurology. 32(9):e70337; 2025. https://doi.org/10.1111/ene.70337

Riva J., Bondiolotti G., Michelazzi M., Pagliuca G., Taddei S. Anxiety-related behavioural disorders and neurotransmitters in dogs. Applied Animal Behaviour Science. 113:310–328; 2008. https://doi.org/10.1016/j.applanim.2008.01.020.

Rogers L.J. Relevance of brain and behavioural lateralization to animal welfare. Applied Animal Behaviour Science. 127:1–11; 2010.

Sacchettino L., Gazzano V., Mayer M., Giuliano V.O., Napolitano F., Ogi A., d'Angelo D. Looking beyond the mind: the gut microbiome as a potential new avenue for treating behavioral disorders in dogs. Current state of the art and potential future perspectives. Dog Behavior. 11(1):1–12; 2025. https://doi.org/10.4454/db.v11i1.200.

Salonen M., Sulkama S, Mikkola S., Puurunen J., Hakanen E., Tiira K., Araujo C., Lohi H. Prevalence,

comorbidity, and breed differences in canine anxiety in 13,700 Finnish pet dogs. Scientific Reports. 10(1):2962; 2020.
Sarviaho R., Hakosalo O., Tiira K., Sulkama S., Salmela E., Hytönen M.K., Sillanpää M.J., Lohi H. Two novel genomic regions associated with fearfulness in dogs overlap human neuropsychiatric loci. Translational Psychiatry. 9:18; 2019.
Sherman B.L., Mills D.S. Canine anxieties and phobias: an update on separation anxiety and noise aversions. Veterinary Clinics of North America: Small Animal Practice. 38(5):1081–1106; 2008.
Sidel S.B. Acute onset of generalized anxiety disorder related to chronic orthopedic pain in a Bernese Mountain Dog: a case study. Animal Behaviour and Welfare Cases. abwcases20250020; 2025. https://doi.org/10.1079/abwcases.2025.0020.
Simic G., Tkalcic M., Vukic V., Mulc D., Špoljar A., Šimic I., Hof P.R. Understanding emotions: origin and roles of the amygdala. Biomolecules. 11(6):823; 2021. https://doi.org/10.3390/biom11060823.
Sinclair M.D. A review of the physiological effects of alpha2-agonists related to the clinical use of medetomidine in small animal practice. Canadian Veterinary Journal. 44:885–897; 2003.
Talegon M.I., Delgado B.A. Anxiety disorders in dogs. Psychiatric Veterinary Review. 2011.
Tiira K., Lohi H. Early life experiences and exercise associate with canine anxieties. PLoS ONE. 10(11); 2015.
Tiira K., Sulkama S., Lohi H. Prevalence, comorbidity and behavioural variation in canine anxiety. Journal of Veterinary Behavior. 16:36–44; 2016.
Tovote P., Fadok J.P., Lüthi A. Neuronal circuits for fear and anxiety. Nature Reviews Neuroscience. 16:317–331; 2015. https://doi.org/10.1038/nrn3945.
Tsuboi D., Nagai T., Yoshimoto J., Kaibuchi K. Neuromodulator regulation and emotions: insights from the crosstalk of cell signaling. Frontiers in Molecular Neuroscience. 17:1376762; 2024. https://doi.org/10.3389/fnmol.2024.1376762.
Vermeire S., Audenaert K., Vandermeulen E., De Meester R., Van Bree H., Dobbeleir A., Peremans K. What's in a brain: neuroanatomy and neurochemistry of anxiety disorders in dogs. Vlaams Diergeneeskundig Tijdschrift. 80(3):175–184; 2011. https://doi.org/10.21825/vdt.87268.
Wiltgen B.J., Zhou M., Cai Y., Balaji J., Karlsson M.G., Parivash S.N., Li W., Silva A.J. The hippocampus plays a selective role in the retrieval of detailed contextual memories. Current Biology. 20(15):1336–1344; 2010. https://doi.org/10.1016/j.cub.2010.06.028.

Ansia nel cane: una revisione completa su patogenesi, manifestazioni comportamentali e possibilità di trattamento

Giovanni Lodrini[1,2*], Virginia Bellini[2], Alice Bevilacqua[2], Susanna Bova[2], Alessia Castagnoli[2], Carla Olmo[2], Valentina Gazzano[3]

[1] *Veterinary behaviorist freelance*
[2] *Dog trainer freelance*
[3] *Department of Veterinary Science, University of Pisa, Italy*

Sintesi

L'ansia nel cane rappresenta una condizione emotiva complessa e multifattoriale, caratterizzata dall'anticipazione di eventi percepiti come minacciosi e associata a un'ampia varietà di manifestazioni cliniche e comportamentali. Essa può compromettere in modo significativo il benessere psico-fisico dell'animale e la qualità della relazione uomo–animale, costituendo una delle principali cause di consulto in medicina comportamentale veterinaria. Il presente lavoro offre una revisione aggiornata dei disturbi d'ansia nel cane, analizzandone la patogenesi alla luce dell'interazione tra predisposizione genetica, esperienze di vita, qualità dell'ambiente e condizioni cliniche concomitanti. Vengono descritte le principali forme cliniche dell'ansia e le comorbilità comportamentali più frequenti, tra cui aggressività, comportamenti compulsivi, disturbi da separazione e alterazioni della socialità. Particolare attenzione è dedicata alle

basi neurobiologiche dell'ansia, con riferimento ai circuiti limbici coinvolti, all'asse ipotalamo–ipofisi–surrene e ai principali sistemi neurotrasmettitoriali responsabili della regolazione emotiva. Il lavoro sottolinea l'importanza di un approccio terapeutico integrato, che combini interventi ambientali, tecniche di modificazione comportamentale e, quando indicato, supporto farmacologico o nutraceutico. Nel complesso, emerge il valore di una prospettiva multidisciplinare e basata sull'evidenza per una valutazione accurata, una gestione efficace e una prevenzione mirata dei disturbi d'ansia nel cane.