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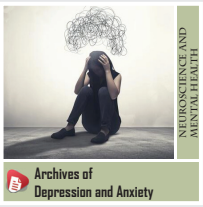

ARCHIVES OF Depression and Anxiety OPEN ACCESS JOURNAL

ISSN: 2455-5460 DOI: https://dx.doi.org/10.17352/ada

Review Article

Models of Depression for Preclinical Drug Discovery and Development: A Transitional Perspective

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Received: 28 August, 2025
Accepted: 08 September, 2025
Published: 09 September, 2025

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Keywords: Anti-depressants; Depression animal models; Chronic mild stress; Forced swimming test; Learned helplessness model; Tail suspension test; Chronic corticosterone exposure model

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Abstract

Animal models of depression are most frequently encountered within the pharmaceutical industry to screen molecules and detect a putative antidepressant activity of a drug. The multiplicity of approaches and models makes comprehensive statements difficult, but animal models are necessary. These are experimental arrangements where a simple system is utilized to represent a complex system.

Five of the most utilized behavioral animal models of depression, the mouse Forced Swimming Test (FST), the rat FST, the Tail Suspension Test (TST), the Chronic Mild Stress (CMS) model, the Learned Helplessness (LH) paradigm, and the chronic corticosterone exposure model, are discussed in this review. All these models present various symptoms of depression in animals suggested to resemble specific aspects of human illness. Their use enables the investigation of the underlying neurobiology of depression, as well as the mechanism of action of antidepressants and the screening of potential antidepressants. Apparently, the mouse FST is the most suitable animal of depression in predicting antidepressant response as it is easily and rapidly performed, robust, specific for antidepressant drugs, and reproducible. Moreover, it permits a good correlation with clinical studies in a translational approach. In this goal, another model is presented: The chronic corticosterone exposure, a more complete neuroendocrine model that seems closer to the conditions of depression in humans. Hyperactivation of the hypothalamic-pituitary-adrenal axis (HPA axis) is one of the most described alterations in patients with depression, as well as in rodent models of pathology.

Introduction

Depression, with over 280 million individuals affected worldwide (World Health Organization), ranks among the leading causes of global disability [1]. Despite advances in pharmacological treatments, many patients fail to respond adequately to existing therapies, highlighting the need for innovative therapeutic approaches. The DSM-5's revised criteria for diagnosing major depressive episodes emphasize symptoms such as anhedonia, psychomotor disturbances, and changes in appetite and sleep [2,3]. These updates present new challenges for preclinical models, which must evolve to capture the complexity of clinical symptoms and facilitate the development of more effective treatments.

Depression is a heterogeneous entity, and the need to further characterize the individual patient to personalize the management plan has been repeatedly emphasized [4]. This adds extra complexity to modeling depression in animal models.

Animal models are an important topic of pre-clinical research on the neurobiology of psychiatric disorders, and help in screening putative drugs for treating the disorder, and permit a better comprehension of the mechanisms implicated. An animal model may be defined by a simple experimental simulation of more complex systems that are less immediately accessible to examine and are thus based on a reductionist compromise [5]. The ideal animal model would not only replicate the essential features of depression but also reliably predict antidepressant activity in a novel compound. This review aims to provide a comprehensive overview of the most utilized animal models of depression, emphasizing their relevance in preclinical research and their contributions

Citation: Bourin M. Models of Depression for Preclinical Drug Discovery and Development: A Transitional Perspective. Arch Depress Anxiety. 2025;11(2):016-024. Available from: https://dx.doi.org/10.17352/2455-5460.000101

to the development of innovative therapeutic strategies. By addressing their strengths and limitations, this work seeks to highlight their role in bridging basic science with clinical advances in the treatment of depression.

Criteria for evaluating the validity of animal models of depression

Numerous animal models have been developed to study the pathophysiological mechanisms of depression and test new therapeutic strategies. However, the complexity of this disorder makes its modeling difficult. Different criteria have been defined to estimate the validity of an animal model, particularly for the study of neuropsychiatric diseases. Currently, 3 criteria are used, which include face, etiological, and predictive validity [6].

- -**Face validity:** This criterion is achieved when strong symptomatologic similarities are observed between the animal model and human pathology. Dysfunctions can manifest themselves at the behavioral level or be of a biochemical or anatomical nature. Table 1 shows the correspondence of symptoms observed in rodent models with those observed in patients with depression.
- -**Etiological validity or "construct validity":** This is the most complicated criterion to validate because it requires that the symptoms observed in the animal model be induced by neurobiological mechanisms similar to those involved in the patient, or that they result in similar psychological constructs. In the case of depression, the difficulty lies in the fact that the biological mechanisms of this pathology are not yet perfectly described. It is therefore difficult to take them all into account.
- -**Predictive validity:** This last criterion calls on the capacity of the model to respond to clinically effective treatments, and conversely, to demonstrate a lack of response to an ineffective treatment.

Ideally, a good animal model of human depression should fulfill, as much as possible, the criteria described above. To date, the available animal models do not achieve this objective due to the complexity of the disorder to be modeled [22]. Certain main symptoms, such as feelings of guilt or recurrent thoughts of death and suicide, are difficult, if not impossible, to assess in laboratory animals. Thus, most of the models used are validated by the beneficial action of antidepressants or by responses to stress. Considering that not all patients manifest the same symptoms, the necessity for an animal model of depression to present all the behavioral abnormalities linked to depression is questionable [23]. This part of the manuscript presents a non-exhaustive list of the main mouse models of depression described in the literature.

Main animal models of depression

In humans, stressful life events are closely associated with the onset of depressive episodes [24]. This is why most animal models of depression currently available are based on the administration of physical and/or psychosocial stress, either in the developmental period (perinatal/early) or adulthood. This gives rise to an anxiety-depressive type phenotype in animals (Table 2).

The connection between stress and depression is complex. The hypothesis suggesting that chronic stress can induce depression is questionable (yet it is the basis of most used models). According to this hypothesis, vulnerability to depression in humans could be compared to behavioral conditions induced by stress in animals. Sustained or chronic stress leads to elevated hormones such as cortisol, the "stress hormone," and reduced serotonin and other neurotransmitters in the brain, including dopamine, which has been linked to depression [31].

Table 1: Correspondence of symptoms observed in rodent models with those observed in patients with depression.

Symptoms associated with depression in humans	Phenotypes in mice	Phenotype assessment tools
Depressed mood	Not modellable	----
Loss of pleasure	Anhedonia	Sucrose preference test [7]
Sleep disorder	Disruption of sleep architecture	Electroencephalography
Psychomotor agitation or retardation	Modification of locomotion	- Actimetry test [8] - Open field test [9]- Rotarod test [10]
Fatigue	Abnormal locomotion	- Treadmill test [11]- Rotarod test- Grip test [12]
Cognitive impairment/ indecision	Alterations in spatial and working memory	- Barnes maze test [13] - Water Morris test [14] - Y maze test [9]
Poor self-esteem	Lack of grooming (carelessness)	- Assessment of the general conditions of the coat- Splash test [15]
Suicidal thoughts	Not modellable	----
Anxiety	Anxious behavior	-Open field test-Elevated plus maze test -Marble burial test [16] Light /Dark box test
Social unrest	Alteration of sociability	- Three-chamber test [18]- Preference for social novelty test [19]
Despair	Resignation	- Tail suspension test (FST) [20]- Forced swimming test (FST) [21]- Learned helplessness test (LHT) [22]
Weight gain or loss	Identical to humans	Weighting

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Table 2: Main mouse models of depression.

Animal model	Characteristics	Main observations for the three criteria	References
Early stress	Maternal separation	- Face: lasting alteration of cognitive and emotional state, but absence of anhedonia - Construct: alteration of plasticity and HPA axis - Predictive: lack of effect of antidepressants	[25,26]
Unpredictable chronic stress	Exposure to a series of random stresses	- Face: robust and lasting alteration of emotional state - Construct: neurochemical alterations in plasticity and the HPA axis - Predictive: response to treatment	[26,27]
Learned helplessness	Exposure to electric shocks at the level of the paws	- Face: robust and lasting alteration of emotional states - Construct: neurochemical alterations and of the neutrophiles -Predictive: weak response to treatment with rapid effect of antidepressants	[28,29]
Restraint stress	Maintaining animals in a tube for 2 hours	- Face: alteration of emotional state - Construct: alteration of plasticity and the HPA axis - Predictive: weak response to treatment with rapid effect of antidepressants	[30]

In fact, the clinical symptoms of depression in humans are more complex than those induced by stress, which is more in the field of anxiety. The rodent models of anxiety are more built on fear. Animal models based on the hypothesis that depression is induced by stress include the mouse/rat Forced Swimming Test (FST), the Tail Suspension Test (TST), the Chronic Mild Stress CMS and the Learned Helplessness model (LH). In these models, animals are exposed to uncontrollable stress resulting in maladaptive behaviors [32].

Numerous studies of animal models indicate that the shock required to produce depressive symptoms must be uncontrollable since exposure of animals to equal amounts of controllable shock will not produce depressive symptomatology.

The mouse forced swimming test

The mouse FST is one of the most widely used preclinical models for evaluating depression-related behaviors in rodents. This model was initially developed to study the efficacy of classic antidepressant drugs (tricyclic antidepressants) and later on has been used for the study of Selective Serotonin Reuptake Inhibitors (SSRIs) [33].

In this model, mice are individually placed into glass cylinders (height, 25 cm; diameter, 10 cm) containing 10 cm of water maintained at 23 °C to 25 °C and left there for 6 min. After vigorous activity, swimming attempts cease, and the animal adopts a characteristic immobile posture. The animal is judged to be immobile when it floats in an upright position and makes only minimal movements to keep its head above water. This state of immobility has been named "behavioral despair", on the assumption that the animals have given up hope of escaping. Antidepressant drugs decrease the duration of immobility, which is recorded during the last 4 min of the 6-minute test period. FST is very useful for studying neurobiological mechanisms to better understand, through drug responses, what depression is in humans [34,35]. This behavioral test, far from the reality of depression clinical features, is a good translational approach [36].

FST is still a core behavioral model used to discover new drugs for treating depression. It was used to study Brain-Derived Neurotrophic Factor (BDNF), a potential antidepressant agent [37]. In this study, an infusion of BDNF was injected into the ventral tegmental area in mice. As a result, it induced a shorter latency to immobility compared to control animals [37]. Other researchers pointed out that they observed a significant decrease in immobility time compared to vehicle-infused controls after BDNF infusion when using FST [38]. FST has been used to study other potential mechanisms of action for antidepressants, such as K+ channel openers and K+ channel blockers [39-42].

FST is not only for screening antidepressant-like effects, but also for understanding the neurobiology of depression, particularly the function of monoamines. However, this model of depression is not only linked to monoamines. Electroconvulsive seizures, a traditional method of treating depression, were used on animals in FST [43] and were effective in increasing the swimming time.

FST can be used in genetically modified animals, which are useful for understanding the mechanisms of action of antidepressants using as well, specific ligands added to antidepressants [44-47].

In FST, behavioral despair does not correlate with the general mobility levels of the animal as measured in an open field test, and FST can reliably differentiate antidepressant treatments from other treatments that merely lead to increased mobility. It is therefore not a mere reflection of decreased physical energy or locomotion but indicates the level of psychomotor activity of the animal [48].

Among the limitations of the FST, we can mention its aversiveness. It is important to consider possible influences it might have on brain structure/function if brain analyses are to be carried out following this procedure [49].

On the other hand, immobility in the forced swim test is adaptive and does not reflect depression; the response should be considered for what it shows: a switch from active to passive behavior in the face of an acute stressor, aligned to cognitive functions underlying behavioral adaptation and survival [50]. (It seems there is debate about this point).

Here, I would expect a systematic analysis of validity criteria for this specific model (as mentioned in the previous comment about Table 2).

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The Tail Suspension Test (TST)

The TST is based on the observation that a mouse suspended by the tail shows alternate periods of agitation and immobility, similar but not identical to that observed in the mouse FST [51]. So, it is almost the same paradigm as the FST. In the TST, immobility is induced in mice simply by suspending them, using adhesive Scotch tape, to a hook connected to a strain gauge that picks up all movements of the mouse and transmits them to a central unit, which calculates the total duration of immobility during a 6-minute test [51].

This test has been automated (ITEMATIC-TST) and measures duration of immobility and the energy expended by each animal, the power of the movements [52], which can distinguish different classes of psychotropic activity [52].

The TST procedure bypasses several problems of the swimming model: the immobility is objectively measured, and no hypothermia is induced by immersion in cold water. The mouse TST can predict antidepressant activity of numerous components [51,52].

It will be shown later in this chapter that the combination of both tests (TST and FST) can help in the discrimination of mechanisms of action of antidepressants when used for screening.

Among the limitations of this model, we can mention interstrain variability in response to antidepressant drugs. This phenomenon has been reported in the most utilized behavioral animal models of depression: the Tail Suspension Test (TST) and the Forced Swimming Test (FST). Previous studies have revealed that drug sensitivity depends on the strain and test used [35]. Swiss mice are the most sensitive strain to detect serotonin and/or noradrenaline antidepressants, whereas C57BL/6J was the only strain sensitive to bupropion (dopaminergic agent) using the FST. In the TST, all antidepressants studied decreased the immobility time in Swiss and C57BL/6J strains [53].

Detection of an antidepressant-like activity could be performed using only one test (TST with Swiss mice or FST with Swiss and C57Bl/6 Rj mice), but both tests are necessary to conclude on the mechanism of action. We make it possible with a decision tree [54].

Due to the aversiveness of the FST and TST, it is important to consider possible influences it might have on brain structure/function if brain analyses are to be carried out following this procedure [48].

No analysis of validity criteria is offered here for this model

Chronic Mild Stress (CMS)

Chronic sequential exposure to a variety of mild stressors (chronic mild stress) has been found to decrease the consumption of and/or preference for a palatable weak sucrose solution in rats or mice [55].

In the CMS, animals are exposed to various types of stressors that change over weeks or months. Among stressors used in this model, we can mention overnight illumination, cage tilt, and change of cage mate, resulting in a decrease in sucrose preference for several weeks. This reflects a general decrease in the sensitivity to rewards or anhedonia. Along with a state of anhedonia, various other behavioral changes due to depression are shown, persisting weeks after stimulus cessation [56].

The model has predictive validity since the reversal of pathologic behavior requires 3-4 weeks of treatment, as in human depression. This feature was not included in the previous models.) This model can demonstrate a potential early onset of antidepressant treatment. Since this test has the advantage of chronicity, it is more similar to the treatment of depression, which takes several weeks to be active. Increasingly, CMS has been used as a model of modified epigenetic mechanisms leading to accelerated senescence and impaired cognitive performance in mice [57].

Learned helplessness model

The LH model is the most familiar simulation of depression and the most controversial. The model mimics some of the key features of depression, particularly those precipitated by unfavorable environmental stress. The model, described by Seligman, et al. [58], consists of exposing animals to unavoidable and uncontrollable stressors such as electric foot-shock, after which learning deficits on subsequent tests are observed, where animals are found to be unable to learn to avoid an aversive Stimulus and remain motionless and helpless in such a situation [58].

This state has been named “learned helplessness' and is not found in animals exposed to identical but controllable stress. It has been shown that the persistent immobility of the animal to respond is confined to the learned immobility that has been required during the unavoidable shock situation. Thus, the learned helplessness behavior does not generalize to other types of behavior that have been learned in the absence of the shock [58].

Seligman and co-authors have suggested that animals learn that responding to uncontrollable shock is futile and that the cognitive and motivational deficits produced in this paradigm are parallel to human clinical depression. The helpless animal enters a learning situation with a generalized associative set in which its actions are without consequence. It therefore responds less, or not at all. In addition to an acquisition deficit, other features of the helpless animal parallel clinical dimensions of depression, deficits in motivation, and emotion. Changes in activity, food intake, and weight have also been reported [58].

There is no established learned helplessness protocol that includes the trans-situational feature for mice. In this model, helplessness did not correlate with immobility time or latency. Mice that underwent the LH procedure showed behavior in the tail suspension test similar to that of naive home cage controls [58].

Please, check the redaction, I am not sure I get the idea of some pieces (apparently, this is the limitation).

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The chronic corticosterone exposure, a more complete neuroendocrine model [59]

A model is proposed here that seems closer to the conditions of depression in humans. Hyperactivation of the hypothalamic-pituitary-adrenal axis (HPA axis) is one of the most described alterations in patients with depression, as well as in rodent models of the pathology [60].

This depressive phenotype is frequently accompanied by disturbances in plasma corticosterone (CORT) levels in animals, considered an indicator of stress [60]. Corticosteroids are released following activation of the HPA axis in response to acute stress. Corticosteroids, and more particularly glucocorticoids, make it possible to develop a physiological response via the establishment of an inhibitory feedback, allowing the activity of the HPA axis to be returned to the basal state [61].

On the contrary, in pathological conditions of chronic exposure to a stressful event, large and continuous quantities of corticosterone are released due to the removal of this "inhibitory feedback," which results in the desensitization of GR receptors to glucocorticoids. This hyperactivity of the HPA axis then leads to an uncontrolled release of stress hormones, leading to anatomo-functional alterations at the cerebral level [62].

Based on these observations, CORT is considered a biochemical marker of depression, but this hormone is mainly used as a pharmacological tool to induce anxiety-depressive type behaviors in animals, particularly in rodents [23]. Thus, a widely used mouse model of depression is based on repeated administration of CORT [63]. Different protocols may be used. The duration of exposure, dose, or route of administration of CORT may vary [64]. Administration of CORT can take place orally (in drinking water) [65], intraperitoneally [66], or using subcutaneous CORT pellet implants [67]. We must note that the model based on chronic exposure to CORT for 8 weeks in drinking water validates the criteria stated above and therefore appears to be a good model of pathology.

Face validity criterion

Concerning the face validity criterion, numerous measurable symptoms in rodents are found in this model, such as an increase in anxiety [68] and learning deficits [69] or even disturbances in sleep architecture [70]. Added to this is also weight gain associated with an increase in food and drink consumption. Anhedonia was also characterized in this model after 8 weeks of exposure [71]. These data suggest that the CORT exposure model has strong face validity. However, this model appears to exhibit a sex-dependent effect. Different doses of CORT were tested in drinking water (7, 35 and 70 µg/ml) in female C57BL6 mice; females were not affected by low or high doses of this hormone. On the contrary, at the optimal dose of 35 µg/ml, females show an attenuated emotional response compared to male mice [72].

Construct validity criterion

This criterion is also validated in the CORT model because it is based on the alteration of the HPA axis. Indeed, animals exposed to CORT display a dysregulation of HPA axis activity similar to that observed in patients with depression [73]. Furthermore, this model induces neurobiological alterations, also found in patients suffering from major depression, and which may be at the origin of the symptoms observed. For example, several studies highlight the impact of CORT on the activity of monoaminergic systems. Concerning the serotonergic system, it has been shown in rodents by intracerebral microdialysis that acute exposure to CORT induces an increase in extracellular concentrations of 5-HT in the ventral hippocampus [74], which would result from the increased expression of tryptophan hydroxylase, the enzyme that synthesizes 5-HT [75].

In terms of other monoaminergic systems, we note that chronic exposure (21 days) to CORT induces anxiety and anhedonia. These behaviors are associated with increased dopamine beta-hydroxylase (DBH), the enzyme responsible for converting dopamine (DA) to norepinephrine (NA) in the Locus coeruleus (LC) and increased expression of the norepinephrine transporter in the hippocampus, amygdala, and prefrontal cortex [76].

Another brain plasticity process strongly regulated by CORT is adult hippocampal neurogenesis. Indeed, chronic exposure to CORT reduces cell proliferation in the dentate gyrus of the hippocampus, and this effect is counteracted by antidepressants [77]. In addition, exposure to CORT via the subcutaneous route reduces the survival of new neurons [78], which remains unchanged when CORT is administered in drinking water. This reveals the importance of the route of administration of CORT on the neurogenic effects of CORT. Finally, CORT also acts on cell proliferation and the maturation of new hippocampal neurons. Indeed, the dendritic arborization of immature granule cells that survive in the DG of CORT-treated rats is less complex, while the dendritic complexity of mature granule cells remains unchanged [79].

Predictive validity criterion

The literature shows that many classes of antidepressants are effective in restoring behavioral deficits of the CORT model. For example, a 3-week chronic treatment with fluoxetine (18 mg/kg), a Selective Serotonin Reuptake Inhibitor (SSRI), can counteract the deleterious effects of CORT on anxiety, resignation, or carelessness. Other classes of antidepressants, notably tricyclics (TCAs), also have beneficial effects in this model. This is the case of imipramine, which has anxiolytic effects, selective inhibitors of noradrenaline reuptake such as reboxetine (IRN), effective in the resignation task, and venlafaxine, a mixed inhibitor of 5-HT and NA reuptake (SNRI), which reduces anxiety and restores preference over sucrose in a test measuring anhedonia [80]. Unconventional antidepressants, such as ketamine, attenuate the pro-depressant effects of CORT [81].

All of these data indicate that the CORT model is a model adapted to the study of depressive pathology. Although it has certain defects, such as the absence of a reduction in serotonergic tone, this model largely meets the validity criteria

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and is based on hyperactivity of the HPA axis, as is the case in human pathology [82].

Alternative models of depression

Automating behavioral tests, as is often proposed, seems illusory or even counterproductive, as observing behavior brings us closer to the human clinic. However, to date, this approach has failed to lead to the development of new treatments, and the biological mechanisms of depression are still poorly understood. It is difficult to compare all the animal models of depression as they vary widely in the manner of inducing abnormal behavior, in the aspects of behavior chosen for study, and in the time course of antidepressant action. This difficulty can be problematic in exporting data from the various laboratories. Other factors hinder comparing these models, such as strain, age, seasonal variations, light cycles utilized, etc. [83-85]. These different parameters can lead to observational differences between laboratories for the same drugs.

Conclusion

The perfect animal model of depression does not yet exist. No single animal model reviewed here is a precise paragon of depression as seen in humans, and questions concerning the utilization of a battery of tests/or instead of a single model, to determine antidepressant activity have been raised. As different aspects of depression are measured in each model and the different models possibly represent a different category of depression, the question remains whether a true comparison between models of a compound's antidepressant activity is possible [86].

However, the screening of drugs in these paradigms allows for a better understanding of the mode of action of antidepressants, the neurobiology of depression [87], as well as the discovery of new and more effective antidepressants [64]. The progress in knowledge of these animal models leads to translational psychopharmacology. That means the researchers can understand, through the models, the clinical features and make the synthesis we need to discover new drugs and even to better understand mood disorders.

The full potential of animal models of depression has not yet been realized, and they represent an under-explored opportunity for drug development, mainly because there are not enough intellectual links between preclinical and clinical researchers. Such opportunities arise from the molecular dissection of the biological features of the models. Finally, it is difficult to predict the potential activity of an antidepressant while having an animal model that closely resembles depression.

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