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Narrative Review

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Emotional mood disorders research: A narrative review

Abstract

This narrative review briefly summarizes studies in current literature (2024-2025) on the five most common emotional mood disorders including depression, generalized anxiety disorder, panic disorder, post-traumatic stress disorder and obsessive-compulsive disorder. These studies are primarily focused on potential underlying biological mechanisms and interventions. These disorders are often comorbid likely because some of the underlying mechanisms and interventions are shared by the different disorders. These include reduced functional connectivity in different regions of the brain as a mechanism and transcranial brain stimulation and cognitive behavioral therapy as interventions. Methodological limitations of this literature include the frequent sampling of patients as opposed to community samples, the broad age range of the groups, the questionably reliable self-report data and the lack of randomized controlled groups for the interventions.

Keywords: panic disorder, depression, cognitive behavioral therapy, yoga, antidepressants

Volume 16 Issue 5 - 2025

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Received: September 26, 2025 | **Published:** October 06, 2025

Introduction

Narrative review

Emotional mood disorders have been defined as mental health conditions characterized by significant disturbances in emotions and behavior that interfere with daily function and well-being. The five most common emotional mood disorders include depression, generalized anxiety disorder, panic disorder, post-traumatic stress disorder and obsessive-compulsive disorder. Common symptoms of emotional disorders include mood swings, sadness, worry, difficulty concentrating, social withdrawal, irritability, aggressivity, suicidality, eating and sleep disturbances. Potential causes of emotional disorders include a combination of genetic, biological, psychological and social factors. Changes in brain function including altered activity of various neural circuits in the brain are also involved.

In this narrative review, brief summaries are given on studies that were published in 2024-2025 on the five most common emotional mood disorders. This research was found on PubMed and PsycINFO by entering the specific emotional disorder and the years 2024-2025 as search terms. Exclusion criteria included proposed protocols, pilot studies and non-English papers. The current literature on these emotional disorders has primarily focused on potential underlying biological mechanisms and interventions for the disorder that are also the focus of this narrative review.

Major depression disorder

Major depression disorder is characterized by persistent feelings of sadness, hopelessness, a loss of interest in activities and suicidality. Physical symptoms include altered energy levels, sleep and appetite changes. Treatment-resistant depression affects approximately 30% of individuals with major depression disorder, and those individuals with that problem are often included as participants in the major depression studies.[1] Most of the current research on major depressive disorder is focused on potential underlying biological mechanisms and therapeutic interventions.

Potential underlying biological mechanisms

A few potential underlying biological mechanisms have been identified in this literature on major depression disorder. These include lower heart rate variability, elevated cortisol related to lower hippocampal volume and increased or decreased volume in different regions of the brain.

Lower heart rate variability has been reported in a study comparing major depressive disorder patients (N=128) versus healthy controls (N= 222).[2] Significantly lower heart rate variability was noted in those with major depressive disorder. These results were not surprising as lower heart variability has frequently been associated with depression.[3]

In a study on cortisol plasma levels associated with hippocampal volume in those with major depression (N= 27 with major depression versus 32 healthy controls), a significant interaction effect emerged between elevated overnight cortisol levels and low hippocampal volume in those with major depression.[4] That relationship was related to the severity of depression symptoms which was not surprising given that depression has been associated with both elevated cortisol and low hippocampal volume in other studies. The low hippocampal volume was likely an effect of chronic depression(Table 1).

Table 1 Depression disorder potential underlying biological mechanisms and interventions (and first authors)

Mechanisms	First authors
Low heart rate variability (low vagal activity)	Tan
Elevated o vernight cortisol levels and low hippocampal volume	Rabi
Greater volume in left precuneus, lentiform nucleus and prefrontal	Pan
Less volume in right cingulate gyrus and cerebellar region	Pan
Therapeutic interventions	
Ketamine	Ramezani
Transcranial magnetic stimulation	Rakesh, Hassanzadehet
Deep brain stimulation	Reddy
Bright light therapy	Tong
Cognitive behavioral therapy	Chan
Yoga	Moosburner, Field

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In a systematic review and meta-analysis of fMRI-based data, 10 studies were selected that widely varied on duration of depression from 3 to 60 months (N=477 participants including 231 depression patients and 246 healthy individuals.[5] Brain regions with significantly **increased volume** included the left precuneus, the lentiform nucleus and the left prefrontal lobe, all regions involved in emotional distress. Conversely, **reduced volume** was noted in the left post central gyrus, left cerebellar area, the lingual gyrus and the right cingulate gyrus, also regions involved in emotional distress. That both increased and decreased volume were noted in regions involved in emotional distress is difficult to interpret, although those contradictory findings possibly relate to variability in depression duration.

Therapeutic interventions

Several different therapeutic interventions have appeared in this current literature on major depression disorder. They include ketamine, transcranial magnetic stimulation, deep brain stimulation, bright light therapy, cognitive behavioral therapy and yoga.

In a study entitled "Effect of ketamine on task-based functional magnetic resonance imaging findings in major depressive disorder: a mini-review", eight articles were included.[6] Most of these studies suggested that ketamine affects brain activity especially in the anterior cingulate cortex, the dorsolateral prefrontal cortex and the amygdala. Correlations were noted between changes in brain activity and reduced depression symptoms. These correlations especially involved the prefrontal cortex, the anterior cingulate cortex and cortico-cerebellar circuits. Limitations of this review included the small number of studies and the lack of longitudinal data. This research in general has been limited by the researchers' selection of different regions of the brain for the fMRIs they have conducted.

In a paper entitled "Combining transcranial magnetic stimulation with antidepressants in major depressive disorder: a systematic review and meta-analysis", the results are given in the title.[7] The ten randomized controlled trials that were included (N=654 participants) showed greater short-term efficacy for the groups that received transcutaneous magnetic stimulation combined with antidepressants versus the no treatment control groups. These data are confounded, however, by the stimulation and the antidepressants being combined.

Surprisingly, a similar systematic review and meta-analysis suggested that fewer sessions of repetitive transcranial stimulation (10 or fewer sessions) had a greater effect on reducing depression symptoms than those studies that involved 11-20 sessions.[8] The "fewer sessions' effect" is possibly related to the "peaking" of the treatment effect on earlier trials.

A systematic review and meta-analysis were conducted on data from deep brain stimulation including 7 randomized controlled trials (N =198) and 8 open-labeled trials (researchers and participants not blinded to group assignment) (N= 77) (Reddy et al, 2024). This surgical procedure that implants electrodes into the brain to deliver electrical impulses was related to a 47% improvement in long-term depression scale scores. This improvement was noted after approximately 20-23 months of stimulation. Long-term (12 to 60 months) remission occurred for 35% of the samples. An interpretation for these effects of deep brain stimulation was not given, but the effects are likely related to the stimulation/activation of several brain regions and their interconnections.

The effects of bright light therapy on those with major depressive disorder were also explored in a systematic review and meta-analysis of randomized controlled trials (N= 15 trials, 883 patients).[9] Trials that lasted 2 weeks or less or those with 60 minutes or more of daily exposure were associated with greater therapeutic effectiveness. These results again suggest that the intensity of the therapy rather than the duration of the protocol is the critical variable. Bright light therapy has been noted to stimulate melatonin that effectively improves sleep patterns which may have been a mediator for the relationship between bright light therapy and decreased depression.

Long-term cognitive behavior therapy (CBT) has also been effective in the management of major depression. In a systematic review and meta-analysis of 23 randomized controlled trials (N=5877 participants), CBT significantly reduced depression symptoms as well as anxiety symptoms.[10] That both depression and anxiety symptoms were reduced was not surprising given that depression and anxiety symptoms are often comorbid. Meta-regression analysis suggested that the number of CBT sessions contributed to the effectiveness of CBT. Unfortunately, most of the studies came from Europe which limits the generalizability of the data, and very few comparisons were made between different types of CBT and different CBT components.

In a systematic review and meta-analysis on **yoga** for major depression disorder, 24 studies were included.[11] Yoga practice had a statistically significant short-term effect on depression severity when compared to a passive control group but not when compared to an active control group. That result was not surprising given that several forms of exercise including yoga have similar effects.[3] However, reemission rates were greater for the yoga group versus both the passive and active control groups possibly because the yoga participants continued that practice following the treatment period.

Generalized anxiety disorder

Generalized anxiety disorder has been defined as severe, ongoing anxiety that interferes with daily activities. It impacts approximately 40 million people 18 years-old and older and causes distressing and fearful feelings. The symptoms including constant worry, restlessness and difficulty concentrating are like those of depression possibly because depression and anxiety are frequently comorbid. Not unlike the literature on depression, the current literature on generalized anxiety disorder has been focused on potential underlying biological mechanisms and therapeutic interventions.

Potential underlying biological mechanisms

A few potential underlying biological mechanisms for emotional disorders have appeared in this current literature. These include low antioxidants, proinflammatory cytokines and grey matter alterations. Although these have been considered potential underlying biological mechanisms, they may also result from depression as in reciprocal variables. As these are cross-sectional data, directionality/causality cannot be determined (Table 2).

Table 2 Generalized anxiety disorder potential underlying biological mechanisms and therapeutic interventions (and first authors)

Mechanisms	**First authors**
Low antioxidant intake	Rizk
Pro inflammatory cytokines	Mamum-Or-Rashid
Grey matter reductions	Ou
Interventions	
Benzodiazepines	Fernandez
Transcranial magnetic stimulation	Duan
Cognitive behavioral therapy	Lau, Liu
Acupuncture	Meira de Valle

Low antioxidant intake has been noted in a sample of adults with severe generalized anxiety disorder (GAD) (N=40).[12] When these

participants received daily antioxidants for six weeks, higher levels of antioxidants were associated with a significant decrease in anxiety symptoms. Eating a diverse range of colored fruits and vegetables can have similar therapeutic effects, although diet would be more difficult to empirically control than supplements.

Proinflammatory cytokines have been implicated as a potential underlying mechanism for generalized anxiety disorder (GAD). Specifically, the interleukins 17A and 23A were explored in a study on the pathophysiology and development of GAD.[13] In this sample (N=50 with GAD and 38 healthy controls), the severity of GAD was associated with the levels of the interleukins 17A and 23A. Elevated proinflammatory cytokines may also result from GAD as in reciprocal variables. Again, directionality cannot be determined from cross-sectional data.

Grey matter volume reductions have been related to the severity of generalized anxiety disorder. In a systematic review and meta-analysis of 11 studies, anxiety severity scores were significantly correlated with grey matter volume reductions in the limbic region including the right insula, lenticular nucleus, putamen and striatum,[14] brain regions that have been involved in emotional distress.

Therapeutic interventions

A few therapeutic interventions have reduced generalized anxiety symptoms. These include benzodiazepines, transcranial magnetic stimulation, cognitive behavior therapy and acupuncture.

Benzodiazepines have been effective in the treatment of GAD based on a systematic review and meta-analysis (N=56 studies, 7556 participants).[15] Benzodiazepines were more effective than placebo, but no differences were noted between the effects of different benzodiazepines. However, moderate heterogeneity and low inconsistency were observed in a treatment- tolerability network meta-analysis.

Transcranial magnetic stimulation was the most widely studied treatment for generalized anxiety disorder in a systematic review of randomized trials (N= 20 treatment arms, 405 participants).[16] The right dorsolateral prefrontal cortex was the most common treatment target for GAD in these trials. That was a surprising target given that data presented earlier suggested reduced grey matter volume in the limbic region not the prefrontal cortex.[14] Higher response and remission rates were noted for those adults receiving transcranial magnetic stimulation as compared to those engaging in active therapies like exercise, perhaps because the transcranial magnetic stimulation was a more controlled therapy with lower demands on the participants.

Cognitive behavioral therapy (CBT) has been effective for those with generalized anxiety disorder. In a cognitive behavioral therapy protocol of as few as four sessions (N= 72 participants), CBT was associated with moderate reductions in anxiety and large reductions in insomnia severity.[17] These effects were surprising given that this protocol included only 4 of the standard 8 CBT sessions. Post-treatment anxiety change was predicted by lower insomnia and rumination, younger age and lower baseline anxiety. Unfortunately, the relative contribution of these predictors was not determined by, for example, a regression analysis or structural equation modeling. In another meta-analysis on 52 randomized trials, group CBT was more effective than a waitlist control condition, as would be expected, and individual CBT was superior to remote CBT.[18]

Acupuncture treatment has also been effective for generalized anxiety disorder. This therapy was provided for participants two times per week for four weeks (N=10).[19] Heart rate variability increased, and anxiety symptoms decreased. Stimulating the pressure receptors under the skin, as in acupuncture, has been noted to increase heart rate variability (vagal activity) following other forms of pressure receptor stimulation, for example, massage therapy.[3] The authors suggested that larger randomized clinical trials, longer treatment duration and longer-term follow-ups are needed to support the results of this research.

Panic disorder

Panic disorder is a type of anxiety disorder characterized by recurrent unexpected panic attacks that are sudden periods of intense fear. The symptoms may include palpitations, shaking, shortness of breath and sweating as well as worry or avoidance behaviors related to the attacks. Typically, there is no real danger or apparent cause. Panic disorder and panic disorder comorbid with other disorders are associated with increased risk of suicide. Current literature on panic disorder, like the other mood and anxiety disorders, has been focused on potential underlying biological mechanisms and therapeutic interventions.

Potential underlying biological mechanisms

Only a few potential underlying biological mechanisms have been the focus of studies in this literature. They are all brain-related mechanisms including low neuropeptides, brain-derived neurotrophic factor protein levels and functional connectivity (Table 3).

Table 3 Panic disorder potential underlying biological mechanisms and therapeutic interventions (and first authors)

Mechanisms	First authors
Low neuropeptides	Orum
Low brain-derived neurotrophic factor	Shafiee
Low functional connectivity	Wang
Interventions	
Escatalopram (antidepressant)	Zou
Web-based cognitive behavioral therapy (CBT)	Kayahan
Digital cognitive behavioral therapy	Jung
Cognitive behavioral therapy and acceptance and commitment therapy	Leon-Quismonda

Neuropeptides including leptin, orexin and ghrelin have been significantly lower in patients with panic disorder (N=32) versus healthy controls (N=32). These neuropeptides which are involved in several physiological functions including mood regulation, hormonal regulation, stress responses and neuromodulation have recently been incorporated into treatments for patients with panic disorder.

Brain-derived neurotrophic factor (BDNF) protein levels have also been significantly lower in patients with panic disorder compared to healthy controls based on a systematic review and meta-analysis of 12 studies.[20] This protein promotes the development of nerve cells and regulates synaptic plasticity, making it essential for learning, memory and mood regulation. BDNF also decreases long-term stress and inflammation. Fortunately, BDNF levels can be increased by physical activity and diet such as blueberries and green tea.

Functional connectivity is reduced within the default mode network (regions of the brain involved in emotion regulation) which negatively affects cognitive activities in those with panic disorder.[21] Based on fMRIs, this study (N=26 panic disorder patients and 25 healthy controls) suggested altered activity in several emotion-regulation-related brain regions including the ventromedial prefrontal cortex, striatum, amygdala, dorsomedial prefrontal cortex and cerebellum.

Interventions

Both antidepressants and behavioral therapies have been effective interventions for panic disorder. Those that have appeared in this literature include antidepressant, web-based and digital cognitive behavioral therapy.

Escitalopram (antidepressant) for 12 weeks has reduced cortisol, ACTH and related gene expression in patients with panic disorder (N=77). These three treatment effects were significantly correlated. Surprisingly, this study was not focused on the reduction of panic disorder symptoms, although reduced cortisol and ACTH would be correlated with a reduction in panic disorder symptoms.

Web-based cognitive behavioral therapy for patients with panic disorder (N=31) has been effective after as few as 4 weeks of therapy.[22] This cost-effective intervention was noted to reduce panic-related symptoms in this sample. However, the web-based cognitive behavioral therapy group was not compared to a control group or waitlist group.

Digital cognitive behavioral therapy (dCBT) groups have experienced a decrease in panic disorder symptoms based on a systematic review of 31 controlled trials of dCBT compared to active and passive control groups.[23] Interoceptive exposure (identifying feared sensations), inhibitory learning (reducing obsessional fears) and personalization (overcoming self-blame) were effective clinical components. However, many findings of these studies were confounded by therapist bias and attrition bias.

Cognitive behavioral therapy (CBT) and acceptance and commitment therapy (ACT) have been effective for adults with panic disorder. The participants (N=80) received 12 group sessions and were assessed post-treatment and 3-months later. CBT was more effective for older participants, men and those with shorter duration panic disorder. ACT was more effective for younger participants, women and those with longer duration panic disorder.

Post-traumatic stress disorder (PTSD)

Post-traumatic stress disorder involves difficulty regulating emotions following a traumatic event. In a bibliometric analysis of 100 PTSD studies, 88% of the studies originated from the U.S. and the most studied population was military veterans (28%).[24] Surprisingly, only 6-7% of the studies involved female victims of sexual or physical violence, traumatized children and adult survivors of childhood abuse given that the prevalence of PTSD is high for those groups. Ten clinical trials evaluated psychological interventions but only three studies appeared on pharmacotherapy (Table 4).

Table 4 Post-traumatic stress disorder potential underlying biological mechanisms and therapeutic interventions (and first authors)

Mechanisms	First authors
Low functional connectivity	Bainter
Alteration of synaptic and myelin plasticity	Lopez-Lopez
Interventions	
Antidepressants	Guidetti
Transcranial magnetic stimulation	Vicheva
Targeted memory reactivation	der Heijden
Audio-visual based art and music therapy	Iyendo

Potential underlying biological mechanisms

Only two potential underlying biological mechanisms have been the focus of research in this current literature. They include reduced brain functional connectivity and alteration of synaptic and myelin plasticity.

Brain functional connectivity data based on a large community sample (N=569, 18-85 years- old) revealed a unique variability of connectivity for those with PTSD.[25] Reduced connectivity was noted in three networks including the default mode network, the central executive network and the salience network (made up of the anterior insula and dorsal anterior cingulate cortex responsible for detecting emotions).

Alteration of synaptic and myelin plasticity has occurred through electrophysiological and chemical variables affecting short and long-distance connections. As these authors suggest, "this remodeling of circuitry is crucial for the development of PTSD".

Interventions

Effective interventions for reducing PTSD include antidepressants, brain stimulation and alternative therapies. These include paroxetine, sertraline, transcranial magnetic stimulation, targeted memory reactivation, art and music therapy.

Antidepressants have modest efficacy in alleviating PTSD symptoms based on a systematic review and meta-analysis of 29 antidepressant-placebo randomized controlled comparison studies (N-4575).[26] These included randomized studies on paroxetine and sertraline.

Transcranial magnetic stimulation has reduced PTSD symptoms in 29% of 12 randomized controlled trials in a recent review.[27] Despite this reduction in symptoms, the research lacks a solid rationale for the cortical targets that have been selected for the transcranial magnetic stimulation. The intervention researchers have frequently targeted brain regions that were not reportedly affected by PTSD in fMRI studies.

Targeted memory reactivation enhances memory by presenting reminder cues during sleep, e.g. different sounds associated with different memories. The effects of this therapy were assessed by polysomnography. In this study, one group received the reactivation cues (N= 17) and the other group received sham stimulation (N=16). The group that received the reactivation cues experienced a reduction in PTSD symptoms, as if re-experiencing the different sounds with the memories was therapeutic. As the authors suggested, multi-night and follow-up studies are needed to further support the results of this study.

Audio-visual based art and music therapy effectively reduced PTSD symptoms in a sample of adolescents who were kidnapped (N=470).[28] The art therapy was more effective than the music therapy, likely because the students were more active during the art therapy versus the music therapy sessions.

Obsessive-Compulsive Disorder (OCD)

Obsessive-compulsive disorder is a mental health condition that affects 2-3% of people worldwide and it is characterized by distressing, intrusive thoughts (obsessions) and repetitive behaviors (compulsions) derived from elevated anxiety. Current literature on OCD has been focused on potential underlying biological mechanisms and therapeutic interventions (Table 5).

Potential underlying biological mechanisms

At least a few potential underlying biological mechanisms have been the focus of research in this current literature on OCD.

They include elevated inflammatory markers, decreased functional connectivity and cerebellar abnormalities.

Table 5 Obsessive-Compulsive disorder and its potential underlying biological mechanisms and interventions (and first authors)

Mechanisms	First authors
Elevated inflammatory markers and altered functional connectivity	Chen
Decreased functional connectivity	Yang
Cerebellar abnormalities	Yang
Interventions	
Antidepressants	Seyedmirzaei
Internet-based cognitive behavioral treatments	Polak
Deep transcranial magnetic stimulation	Li

Elevated inflammatory markers and altered functional connectivity have been noted in a sample of adults with moderate to severe OCD (N=55 with OCD and 54 healthy controls).[10] The elevated inflammatory markers included TNF-alpha, Il-2 and IL-6. fMRIs indicated that individuals with OCD exhibited atypical functional connectivity in the postcentral gyrus, angular gyrus and middle temporal gyrus, areas that are associated with sensory-motor processing, cognitive control and emotion regulation.

Decreased functional connectivity was also observed between the left anterior thalamic radiation and the left dorsal lateral prefrontal cortex regions of the brain in patients with OCD based on fMRIs (N=52 patients with OCD and 39 healthy controls). This abnormal white matter structure and function were significantly correlated with OCD symptoms.

Cerebellar abnormalities have also been noted in OCD patients based on fMRIs. They also interact with the basal ganglia and the broader network of cortico-striato-thalamo-cortical circuits.

Interventions

Only a few interventions have been the focus of research on OCD. These include antidepressants, internet-based cognitive behavioral therapy and transcranial magnetic stimulation.

Antidepressants have altered brain structure and function in patients with OCD based on a systematic review of 13 neuroimaging studies.[29] fMRIs showed thalamic, amygdala and pituitary volume changes following antidepressant treatment. Changes were also noted in the ventral striatum, frontal and prefrontal cortex. As the authors suggested, these findings are limited by the small number of studies included in the review, by the heterogeneous samples and by the different antidepressants studied.

Internet-based cognitive behavioral treatments have been effective for individuals with OCD based on a systematic review and meta-analysis of 12 randomized controlled trials (N=1416).[30] Guided self-help versus unguided self-help trials were compared to active and passive control conditions. Guided self-help, not surprisingly, significantly reduced OCD symptoms.

Deep transcranial magnetic stimulation has been more effective than a sham control condition in decreasing scores on the Yale-Brown Obsessive-Compulsive Scale based on a meta-analysis of four randomized controlled trials (N=252).[17] This effect was, surprisingly, not only noted following the stimulation but also as late as a one-month follow-up assessment.

Methodological limitations of this literature

Several methodological limitations can be noted for this literature on emotional mood disorders including biased sampling and methods. Most of the samples were patients versus community members, limiting the generalizability of the findings. The participants also represented a broad age range within samples, obscuring any age-specific effects. The disorder and control groups were often different sample sizes. Several of the disorders were likely comorbid conditions, for example, depression and anxiety, which are often comorbid.[31]

The self-report data, as is typical for survey results, raises concerns about reliability of the findings. Different fMRI researchers focused on different brain regions, generally suggesting that many different regions were involved, as might be expected for the complex brain and its interactive regions. Given that the data were cross-sectional, not longitudinal, directionality or causality could not be determined. For example, the directionality of elevated cytokines in generalized anxiety disorder could not be determined. Most of the papers were systematic reviews and meta-analyses, making it difficult to critically evaluate the individual studies that comprised the reviews and meta-analyses.

Most of the studies were focused on mechanisms and interventions. The mechanisms and interventions were sometimes similar across the different disorders, for example, the low connectivity in the case of mechanisms and the transcranial magnetic stimulation and cognitive behavioral therapy in the case of interventions. The absence of waitlist control groups and comparisons between therapy and active control groups suggest that the intervention results were tenuous. Several of the studies were not randomized controlled trials introducing researcher and participant bias. The intervention studies often targeted brain regions that didn't appear as compromised brain regions in the mechanism studies, suggesting that the mechanism and intervention researchers were focused on different regions highlighting a disconnect between those types of research. In addition, several notably effective interventions did not appear in this current literature on mood disorders. These included eye movement desensitization and reprocessing (EMDR) that has been particularly effective for trauma, exposure and response prevention (ERP) that has been highly effective for obsessive compulsive disorder (OCD) and mindfulness based cognitive therapy (MBCT) that has been effective for depression. The omission of these notable intervention trials may relate to their significant coverage in earlier literature. However, their absence reduces the comprehensiveness of the current literature. Further, physiological and psychological interventions were not compared in this literature. These omissions are suggestive of priorities for future research.

The consistent methodological limitations in this literature limit the strength of any conclusions that are drawn. Despite these methodological limitations, this literature has highlighted the potential underlying biological mechanisms of the most common emotional mood disorders as well as effective interventions for reducing emotional disorder symptoms. The relative absence of studies on effects of emotional mood disorders and their risks/predictors highlights the importance of continuing research on emotional mood disorders.

Acknowledgments

None.

Conflicts of interest

The author declares there is no conflict of interest.

Funding

None.

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