Neural Correlates of Emotional Processing in Bipolar Disorder, Major Depressive Disorder and Schizophrenia: A Neuroimaging Meta-Analysis

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Word Count: 10385

Master Thesis

Specialization: Neuropsychology

Maastricht University

26 August 2025

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Abstract

Objective: Emotional processing is fundamental to human cognition and social interaction, yet it is frequently disrupted in psychiatric disorders such as schizophrenia, bipolar disorder, and major depressive disorder (MDD). This thesis conducted a coordinate-based meta-analysis to identify both shared and disorder-specific neural alterations underlying emotional processing across these conditions.

Methods: A systematic literature search identified 119 eligible fMRI studies, including 32 on bipolar disorder, 52 on depression, and 35 on schizophrenia. Activation Likelihood Estimation (ALE) was applied to determine consistent brain activation patterns, with separate meta-analyses performed for each disorder and pooled analyses used to assess transdiagnostic convergence. Conjunction and contrast analyses further clarified overlapping and distinct neural correlates.

Results: Results revealed robust convergence in the amygdala, prefrontal, and limbic regions across disorders, confirming their central role in emotional dysfunction. The pooled transdiagnostic analysis revealed consistent activation differences in the right amygdala and the left inferior frontal junction (IFJ). Follow-up analyses suggested that disorder-specific heterogeneity and task-related variability may obscure consistent effects in some groups.

Conclusion: These findings provide evidence for both common and distinct neural mechanisms of emotional processing deficits, highlighting the amygdala as a transdiagnostic hub while also emphasizing disorder-specific patterns. The results offer valuable insights for tailoring clinical interventions aimed at improving emotion regulation in psychiatric populations.

Introduction

Emotional Processing

Emotions are physiological responses to external and/or internal stimuli that lead us to action (Krause et al., 2021). Since emotions influence various mental processes (e.g., memory, decision-making) and observable behaviors (e.g., helping behaviour, drug use), the ability to recognize and regulate emotions is vital and a fundamental aspect of mental health (Kret & Ploeger, 2015).

Emotional processing is a fundamental component of daily functioning, influencing perception, social interaction, and decision-making. It is essential for adaptive functioning and mental health. Disruptions in emotional processing are associated with multiple forms of psychopathology. Emotional processing encompasses interrelated mechanisms such as the somatic and psychological experience of emotions, verbal or behavioral expression, and cognitive evaluation of the goals, significance, and consequences of emotional experiences (Pascual-Leone, 2017). Models of emotional processes consist of several key components, including (i) recognizing and focusing on information that may trigger emotional responses, (ii) experiencing emotional arousal in reaction to such stimuli, and (iii) regulating that arousal through various mechanisms (Townsend et al., 2012).

Emotion processing engages multiple large-scale neural networks, integrating both cortical and subcortical regions, to facilitate the recognition of a stimulus's emotional significance, as well as the generation and regulation of affective states and emotional behaviors. It involves the detection and evaluation of salient stimuli as well as the regulation of one’s emotional (affective) response to these stimuli (Phillips, 2006). Emotion processing can thus be passed along the different dimensions of emotion perception, experience, expression, recognition and regulation.

Neural Correlates of Emotional Processing

Early models of emotional processing emphasized the limbic system as the core neural substrate for emotion (MacLean, 1949; LeDoux, 1998). The limbic system typically includes the amygdala, hippocampus, hypothalamus, anterior cingulate cortex (ACC), insula, and parts of the prefrontal cortex (Rolls et al. 2015), although its exact boundaries and functions have long been debated. While the term "limbic system" remains in use, more recent research has shown that emotion processing is not confined to this system alone but rather involves distributed and dynamic networks spanning both cortical and subcortical regions, with the limbic structures acting as key integrative hubs (Pessoa, 2008; Lindquist et al., 2012).

Among these, the amygdala has consistently been identified as playing a central role in the detection and appraisal of emotionally salient stimuli, particularly negative emotions such as fear and sadness. Neuroimaging studies have repeatedly shown that the amygdala is robustly activated during the processing of emotional facial expressions (Breiter et al., 1996; Blair et al., 1999). Beyond mere detection, the amygdala is believed to coordinate affective responses by interacting with cortical regions involved in attention, perception, and regulation, including the prefrontal cortex and anterior cingulate cortex (Pessoa & Adolphs, 2010, 2011). Mesulam (2000) further proposed that the amygdala and hippocampus serve as key nodes within broader emotion-related networks, with overlapping connectivity patterns converging particularly in the ACC—a region associated with both emotional evaluation and regulation.

Emotion processing relies on the dynamic interaction of multiple brain systems, rather than isolated regions. According to widely accepted models and meta-analytic findings (e.g., Phillips et al., 2003), two partially overlapping but functionally distinct neural systems are implicated: a ventral system, involved in the automatic identification and generation of

emotional responses, and a dorsal system, responsible for the voluntary regulation and evaluation of these responses. The ventral system includes structures such as the amygdala, insula, ventral striatum, and orbitofrontal cortex, which process the salience, valence, and reward value of emotional stimuli. These regions support rapid, bottom-up processing and are particularly active in response to emotionally arousing or personally relevant cues.

In contrast, the dorsal system—including the dorsolateral prefrontal cortex (DLPFC), dorsal anterior cingulate cortex (dACC), and parietal cortices—is involved in top-down cognitive processes such as attention to emotion, conscious appraisal, and regulatory control over affective responses. These systems interact through reciprocal connections, allowing for modulation of emotional reactivity based on context, goals, or cognitive strategy. The anterior cingulate cortex acts as a key interface between these systems, integrating emotional and cognitive information and guiding adaptive behavioural responses.

Emotional Processing in Psychiatric Disorders

Emotional difficulties in mental disorders may be associated with factors such as emotional intensity, duration, frequency, type, awareness, regulation goals, and regulation strategies (Gross & Jazaieri, 2014). Dysregulated emotional responses can lead to pathological mood states often seen in psychiatric disorders like schizophrenia, bipolar disorder and depression. These three disorders are particularly relevant for the study of emotional processing because they are each marked by prominent affective symptoms, such as blunted affect or anhedonia in schizophrenia, mood instability in bipolar disorder, and persistent negative affect in depression. Moreover, despite their clinical differences, they share overlapping features of emotional dysfunction, making them ideal candidates for examining both shared and disorder-specific neural mechanisms.

Emotional Processing in Bipolar Disorder. Bipolar Disorder is a chronic and severe psychiatric illness characterized by recurrent episodes of mania or hypomania and depression, often interspersed with periods of euthymia (normal mood). These mood episodes are accompanied by significant changes in cognition, behaviour, and emotional reactivity. Emotional dysfunction is a central feature of the disorder and is present across mood states, including during euthymia (Phillips et al., 2008). Many individuals with psychiatric disorders struggle to identify, understand and describe both their own emotions and those of others. This is characterized by impairments in emotional awareness and is associated with altered anterior cingulate and mediofrontal activity during emotional stimulus processing (Berthoz et al., 2002; Van der Velde et al., 2013).

At the behavioural level, individuals with bipolar disorder often experience affective instability, difficulty in emotional perception, and impairments in emotional labelling and regulation (Killgore et al., 2008). These deficits may emerge early in the course of the illness and can persist independently of mood state. Emotional dysregulation can contribute to pathological mood swings and is associated with poorer functional outcomes.

Functional imaging studies on mania are limited due to the difficulty of scanning manic patients. Existing research primarily focuses on frontal–amygdala activity using emotion-based paradigms (Hariri et al., 2000). Studies investigating the manic state have consistently reported hyperactivation of the amygdala, particularly in the left hemisphere, in response to emotional stimuli such as facial expressions or affective scenes (Taylor et al., 2000; Strakowski et al., 2011). Strakowski et al. (2011) found that manic individuals exhibited increased left amygdala activation regardless of emotional valence, while right amygdala hypoactivation was specific to negative emotional stimuli. Notably, converging evidence suggests reduced activity in prefrontal regions, including the vlPFC and dlPFC,

which may reflect impaired top-down regulation of limbic responses (Rubinsztein et al., 2001; Van der Schot et al., 2010).

Findings in bipolar depression are more heterogeneous. During the depressed state, studies in bipolar participants shows more variability in activation differences compared to mania. Some studies report increased subcortical activity (e.g., in the basal ganglia, thalamus, hypothalamus, and left amygdala) in response to emotional stimuli (Malhi et al., 2004), while others find frontal hypoactivation rather than limbic hyperactivation (Van der Schot et al., 2010). Decreased frontal lobe activity, particularly in the vlPFC, is a more consistent finding across studies of bipolar depression.

During euthymia, findings suggest that limbic hyperactivation—especially in the amygdala—can persist even in the absence of overt emotional stimuli, pointing to trait-like abnormalities in emotional reactivity (Hassel et al., 2008). However, frontal hypoactivation, especially in the vlPFC, also remains a common feature during euthymic states, supporting the idea of persistent deficits in regulatory control (Van der Schot et al., 2010).

Comparing mood states, manic individuals exhibit greater amygdala and fusiform gyrus activation than bipolar depressed patients, particularly during implicit emotion tasks (Chen et al., 2006). However, a study by Chen et al. (2010) reports less amygdala activation in mania compared to euthymia. Additionally, manic patients tend to show reduced frontal activity, including in the vlPFC and dlPFC (Van der Schot et al., 2010), while bipolar depression is associated with amygdala hyperactivation to sad faces and weaker amygdala–vlPFC connectivity during emotional processing (Versace et al., 2010).

Emotional Processing in Major Depressive Disorder (MDD). MDD is a highly prevalent, persistent, and clinically significant mood disorder characterized by persistent low mood, anhedonia, cognitive impairment, and emotional dysregulation. It is associated with

substantial impairments in social functioning, occupational performance, and quality of life (WHO, 2017). Depressed individuals exhibit reduced sensitivity in recognizing emotional facial expressions and demonstrate a negative response bias, often perceiving neutral faces as sad and happy faces as neutral (Persad & Polivy, 1993; Elliot et al., 2011). This bias may stem from impaired top-down regulation by the dorsolateral prefrontal cortex (dlPFC) resulting in excessive activation of the hippocampus during depressive episodes (Persad & Polivy, 1993; Ketter et al., 2003). Furthermore, it has been reported that depressed patients consistently exhibit heightened amygdala activity in response to masked, subliminally presented negative stimuli, along with reduced activity to positive stimuli when compared to healthy individuals (Klug et al., 2024). In facial affect recognition tasks, most studies found greater amygdala activation in depressed patients compared to healthy controls, with some studies also reporting increased activity in the basal ganglia. (Frodl et al., 2009).

Greater depression severity has been linked to heightened amygdala activation during passive viewing (Lee et al., 2007) and emotion regulation tasks (Erk et al., 2010), indicating atypical amygdala responses to affective stimuli in MDD. Studies using personally relevant stimuli report increased amygdala and striatal activation during acute depression (Kessler et al., 2011). In parallel, prefrontal–limbic connectivity, robust in healthy individuals, is weakened in depression but improves with antidepressant treatment (Anand et al., 2005a, 2005b, 2007; Johnstone et al., 2007; Chen et al., 2008), underscoring disrupted amygdala–prefrontal regulation circuitry. Greater symptom severity has also been tied to broader cognitive and emotional impairments (Salthouse, 1996) and specific deficits in facial emotion recognition (Sullivan & Ruffman, 2004).

However, there are various inconsistencies in findings across both individual studies and meta-analyses in depression. This variability may stem from methodological issues in previous analyses or differences in their scope. Some studies have examined general

emotional or cognitive processing (Phan et al., 2022), while others have specifically focused on negative, sad, or positive emotions (Groenewold et al. 2013; Kauschke et al., 2019). Additionally, some analyses have combined cognitive and affective processing, which could contribute to the observed discrepancies (Mueller et al., 2017).

Emotional Processing in Schizophrenia. Schizophrenia (SCZ) has traditionally been viewed as a psychiatric illness with prominent clinical features of psychosis or positive symptoms, negative symptoms, and cognitive dysfunction (Kahn et al., 2015). Over the past two decades, extensive research has explored the ability of individuals with schizophrenia to recognize emotional expressions in others. Studies, including those by Archer et al. (1994), have found that schizophrenia patients exhibit deficits in both facial and vocal affect recognition.

Research examining the link between affect recognition and specific symptoms in schizophrenia has produced mixed results. Many studies suggest a connection between facial affect recognition and negative symptoms like alogia (Gaebel & Wölwer, 1992; Kohler et al., 2000). Some findings indicate that deficits in both affect, and identity recognition are associated with negative symptoms, such as anergia (Lukoff et al., 1986; Mueser et al., 1996) and the overall severity of negative symptoms (Baudouin et al., 2002). Additionally, some studies have identified a link between affect recognition deficits and positive symptoms, including bizarre behaviour (Schneider et al., 1995), thought disorder (Kohler et al., 2000), and overall positive symptom severity (Lewis & Garver, 1995).

The processing of positive and negative expressions in schizophrenia appears to be altered in distinct ways. Studies have reported abnormal activation in limbic and paralimbic regions during facial emotion perception (Gur et al., 2002, Habel et al. 2007), though findings have not always been consistent. Schizophrenia (SCZ) is associated with marked impairments

in emotional processing, which affect the recognition, experience, and expression of emotions. These difficulties are thought to stem from dysfunction in fronto-limbic and paralimbic networks—systems involved in evaluating emotionally salient stimuli and regulating affective responses (Kohler et al., 2010; Anticevic et al., 2012). The processing of both positive and negative emotions appears to be altered in distinct ways, with numerous studies reporting abnormal activation in regions such as the amygdala, hippocampus, anterior cingulate cortex (ACC), and prefrontal cortex during facial emotion perception tasks (Gur et al., 2002; Habel et al., 2007; Hall et al., 2008; Anticevic et al., 2012).

For example, the amygdala, a key structure in emotion salience detection, frequently shows blunted or inconsistent activation in response to emotional faces in individuals with schizophrenia (Gur et al., 2002; Hall et al., 2008; Taylor et al., 2012). Meanwhile, prefrontal regions, particularly the dorsolateral prefrontal cortex (DLPFC) and ventromedial prefrontal cortex (VMPFC), show reduced engagement during emotional appraisal and regulation tasks (Barch et al., 2003; Szabó et al., 2017), suggesting impaired top-down modulation of limbic responses. These dysfunctions may contribute to the emotional blunting and poor social cognition frequently observed in patients. Although several studies, including those by Lepage et al. (2011) and Habel et al. (2007), have reported hyperactivation in some prefrontal and posterior cortical regions during emotion processing, findings across the literature remain mixed, with variability in task paradigms, sample characteristics, and medication status likely contributing to these inconsistencies.

Current Study

While there is already substantial, though heterogeneous, evidence of neural aberrations in all three disorders, inconsistencies in findings may stem from variations in experimental designs and differences in sample characteristics, such as specific diagnoses, subtypes, and symptom severity. Additionally, methodological issues in previous analyses

and differences in study focus contribute to this heterogeneity. Some studies have examined general emotional or cognitive processing, while others have specifically investigated negative, sad, or positive emotions, or combined cognitive and affective processing. Meta-analysis can help address these limitations by identifying consistent activation patterns across studies. To enhance the specificity of our findings, we will code detailed information on experimental paradigms, diagnostic criteria, and symptom profiles, allowing for sub-analyses that focus on aspects of emotion processing. Furthermore, while individual fMRI studies have compared schizophrenia with bipolar disorder or bipolar disorder with depression (Delvecchio et al., 2012; Lawrence et al., 2004; Surguladze et al., 2005; Trotti et al., 2021), no meta-analysis has systematically examined shared and distinct neural alterations in emotion processing across all three disorders.

The aim of the present study is to assess the neural correlates of emotional processing in Bipolar Disorder, Depression, and Schizophrenia by quantitatively integrating the literature using neuroimaging meta-analysis. The meta-analytic approach was chosen, because single studies are often limited by small samples and methodological variability (Button et al., 2013; Carp, 2012), whereas meta-analyses increase reliability by pooling larger, more diverse datasets. Coordinate-based ALE meta-analyses (Eickhoff et al., 2009; Bzdok et al., 2013; Turkeltaub et al., 2002; Turkeltaub et al., 2012) will be used to integrate results from neuroimaging studies investigating emotional processing. Separate meta-analyses were conducted for each disorder alone and further a transdiagnostic meta-analysis was performed to reveal shared aberrancies across disorders. To establish a reliable reference point for interpreting between-group differences, the present study first examined within-group activation patterns in healthy controls.

Hypotheses

1. All three disorders will show abnormalities in the amygdala which is a key emotion-processing region, compared to healthy controls (Delvecchio et al., 2012; Li et al., 2010; Mueller et al., 2017)
2. While some brain abnormalities will be shared across disorders, each disorder will also have unique patterns of dysfunction (Whalley et al., 2009; Phillips et al., 2006)
3. Bipolar disorder will show inconsistent brain activity in regions outside the amygdala depending on the differences in mood states (Chen et al., 2010; Malhi et al., 2004; Van de Schot et al., 2010)
4. Depression will be associated with consistent differences in activity in emotion-related regions (e.g., amygdala), particularly for individuals who are more severely affected (Dannlowski et al., 2009; Klug et al., 2024; Erk et al., 2010) as approximated by the presence of antidepressant medication use in the included samples.
5. Schizophrenia will be primarily associated with consistent differences in activity in regions such as the amygdala and inferior frontal gyrus (Li et al., 2010; Gur et al., 2002; Kohler and Martin, 2006)

Methodology

Selection of Studies

Neuroimaging experiments using functional magnetic resonance imaging (fMRI) published from January 1, 1998, to December 31, 2024, were searched in the databases Web of Knowledge (http://apps.webofknowledge.com), PubMed (https://pubmed.ncbi.nlm.nih.gov), and Google Scholar (http://scholar.google.de). A systematic search approach was used, involving the following search strings: (1) title: "schizophrenia" OR "psychosis" OR "bipolar disorder" OR "euthymia" OR "mania" OR "depression" OR "major depressive disorder"; (2) title/abstract: "emotion" OR "emotional processing" OR "emotion regulation" OR "affective processing" OR "emotion identification" OR "emotion perception" OR "emotional experience" OR "emotional domain"; (3) abstract: "fMRI" OR "functional magnetic resonance imaging" OR "neuroimaging" OR "neural correlates" OR "brain activation".

When the initial search approach was saturated, more specific emotion-related task terms were included to capture the full range of relevant paradigms: for example, "passive viewing," "explicit emotion perception," "facial emotion recognition," "implicit emotion perception," "emotion labelling," and "affective go/no-go." In addition to the database searches, reference lists of relevant articles and recent ALE studies were manually screened to identify any studies that may have been missed.

Inclusion and Exclusion Criteria

General and Sample-Related Criteria

Included studies had to meet several criteria to ensure methodological rigor and comparability. Only peer-reviewed fMRI studies that acquired full-brain coverage and analysed the whole brain, reporting the results as coordinates in standard reference space, i.e.,

Montreal Neurological Institute (MNI) or Talairach (Talairach, & Tournoux, 1988) space, were included. Consequently, region-of-interest (ROI) studies and any studies that failed to report coordinates were excluded. The analysis focused on task-based fMRI studies that specifically investigated emotional processing in psychiatric populations in comparison with a healthy control sample. All included studies examined implicit or explicit emotional processing within the emotional domain. Examples of such tasks included those that used emotional stimuli with tasks like recognizing facial expressions, gender discrimination tasks, emotional Stroop task or regulating emotional responses to stimuli.

While ROI-based analyses were excluded, studies were included if between-group contrasts were implicitly masked with the positive task effect derived from whole-brain analyses (e.g., in controls or across all participants). In contrast, studies using anatomical masks on small regions like the amygdala without a full-brain analysis were excluded. Further, results from connectivity analyses, and those reporting correlations and interactions with other variables (e.g. group x performance interaction, correlation with reaction time) were not considered. Only studies investigating adult psychiatric patients (>18 years) diagnosed with schizophrenia, bipolar disorder, or depression, as well as the respective healthy control groups were included.

Selection Criteria for Experimental Design and Contrasts

The study included experiments that reported between-group effects in emotion processing comparing individuals with psychiatric diagnoses (schizophrenia or depression or bipolar disorder) to healthy controls. Additionally, the within group effects for controls were also selected to examine the general task network recruited by the respective task. If no within-group effect for the control group but only the main task effect across groups was reported, the latter was included. For the purposes of this study, a task was considered

emotional if it involved exposing participants to emotionally valenced visual or auditory stimuli. The study focused on contrasts that target emotion processing, including comparisons between emotional and neutral stimuli, as well as emotion vs. baseline conditions.

However, comparisons between two emotional conditions (e.g., happy > fearful) and contrasts where the emotional component was factored out within the contrast itself (e.g., Emotional No-go > Emotional Go) were excluded, as they do not isolate general emotional processing effects. Experiments that reported only correlations or interactions with other variables (e.g., group × performance interactions or correlations with clinical measures) were excluded, as these reflect either effects unrelated to the core task or highly specific findings that limit generalizability. Further, if different studies reported results based on the same participant groups, only one of the studies was included to avoid dependence of effects.

Tasks were included where emotional processing was investigated explicitly, i.e. the emotion was task relevant (where participants actively process emotional content), or implicitly, i.e. task irrelevant (where emotional content is present but not the primary focus). For the purposes of distinguishing between different types of explicit and implicit emotional processing, explicit emotional processing experiments were classified into the following emotional components:

- **Emotion identification** – participants explicitly label or categorize emotions (e.g. Facial Affect Recognition Task)
- **Explicit emotion perception** - participants assess or match emotional stimuli without necessarily labelling them (e.g. Perceived Emotion Intensity)
- **Emotion experience** - Participants view emotional stimuli and report their subjective feelings (e.g. Mood Induction Task)

- **Explicit emotion regulation** - Participants are instructed to actively regulate their emotions using cognitive strategies (e.g. regulate vs look, dampen vs observe tasks)
- **Explicit passive viewing** - Participants view emotional stimuli with instructions to simply observe, without making any judgment or response (e.g. passive viewing of emotional or neutral images from the IAPS)

The studies focusing on implicit emotion processing were labelled as "implicit emotion perception", where participants processed emotional stimuli without being explicitly instructed to focus on the emotional content; emotion is processed automatically as part of another task (e.g. Gender Discrimination Task, Emotional Stroop Task)

Additionally, studies reporting reward processing, anticipation paradigms, conditioning, and pain perception were excluded from the study. Reward processing and anticipation tasks were excluded because they primarily engage motivational and decision-making circuits, focusing on the anticipation or receipt of reward rather than emotional perception or experience (Knutson & Greer, 2008). Further, Reward-processing and reward-anticipation paradigms chiefly engage the meso-corticolimbic motivational loop rather than the perceptual networks that decode emotional cues (Oldham et al., 2018; Haber et al., 2010). Further, conditioning paradigms were excluded as they focus on learning mechanisms rather than emotional processing. Classical fear-conditioning experiments centre on associative learning in the amygdala–hippocampal circuit (Deng et al., 2024). Lastly, pain-perception paradigms activate the so-called "pain matrix"—anterior insula, mid-anterior cingulate, and related nociceptive hubs, whose primary function is processing somatic discomfort, not emotion recognition (Xiang et al., 2018).

Studies examining emotional regulation were excluded if they contrasted two emotional conditions (e.g., reappraisal vs. emotional viewing), as these focus primarily on

regulatory processes rather than emotional processing itself. However, studies were included if they contrasted reappraisal with a non-emotional baseline (e.g., reappraisal vs. neutral viewing), as these also include general emotional processing and not purely emotion regulation. Studies examining the effects of pharmacological or psychological treatments were included only if they reported activations at baseline. Studies focusing exclusively on treatment-by-group interactions and post-treatment activations were excluded. The included studies (per disorder) and their clinical specifications are listed in Tables 1-3.

Activation Likelihood Estimation (ALE) Meta-Analysis

This study employed coordinate-based meta-analysis (CBMA) techniques to systematically examine neural correlates of emotional processing in schizophrenia, bipolar disorder, and depression. All meta-analyses were conducted using the revised version of the ALE algorithm for coordinate-based meta-analysis of neuroimaging results (Eickhoff et al., 2009, 2012; Turkeltaub et al., 2002, 2012) implemented using in-house Python scripts. The key idea behind ALE was to identify areas of spatial convergence of reported coordinates for differences between patients and control participants during emotional processing tasks.

Using this method, coordinates are considered as the center of a 3D Gaussian probability distribution instead of a single focal point. This is done to account for the spatial uncertainty of neuroimaging measurements. The width of the Gaussian distribution is influenced by empirical data, namely by the variance between subjects and between templates. As the number of subjects increases, the Gaussian curve becomes tighter and taller, reflecting greater reliability due to a larger sample size (Eickhoff et al., 2009). The probabilities of the distribution of all foci in a specific experiment are aggregated for each voxel, resulting in a modeled activation (MA) map (Turkeltaub et al., 2002). Then, an ALE map is computed by aggregating across all individual MA maps. These scores reflect the correspondence of results between experiments at each brain location. The ALE scores are

then compared to a null distribution created by an analytic procedure called non-linear histogram integration. The resulting statistics need to be corrected for multiple comparisons, which is achieved through voxel- and cluster-level family wiser error rate correction. These corrections are based on Monte-Carlo simulations, which allow the estimation of a corrected null distribution (Frahm et al., 2025).

Resulting nonparametric p values were thresholded at a cluster-level family-wise-error-corrected p value of 0.05 (cluster-forming threshold at voxel-level $p<0.001$). To ensure sufficient statistical power and to avoid results being driven by a small number of studies, meta-analyses were only conducted when at least 17 experiments were available for a given contrast (Eickhoff et al., 2016; Müller et al., 2018). Additionally, anatomical labelling of significant clusters was performed using the SPM Anatomy Toolbox, allowing for precise assignment of peak coordinates to probabilistic cytoarchitectonic maps.

Meta-Analyses Calculated

Within-Group Analyses: Main Task Effect

To identify the core emotion-processing network consistently recruited by the task, a meta-analysis of the main task effects within the healthy controls was conducted for each of the three datasets. This included all studies reporting emotion versus neutral/baseline contrasts in healthy control groups alone. However, if within-group effects for the healthy controls were not reported separately, contrasts across all participants (patients and controls combined) were included instead. The goal was to isolate brain regions reliably activated during emotional processing tasks, independent of diagnostic status.

Between-Group Analyses

The datasets were pooled across coordinates for between-group activation in patients and control participants for each disorder to identify transdiagnostic patterns of “aberrant

activation," thereby pinpointing the most reliable hubs of dysfunction. No separate analysis was performed for activation coordinates associated with hyper-activation (patients > controls) and hypo-activation (patients < controls). This decision was made because the direction of group differences in fMRI studies is often difficult to interpret consistently, as it depends on the specific analytical approach used. For example, some studies calculate simple task-versus-baseline contrasts and then compare groups, while others use group × condition interactions. Variability in how control conditions are modelled can unpredictably influence the observed direction of activation differences. Therefore, pooling across both types of contrasts provides a more stable and comprehensive summary of aberrant activation during emotional processing, regardless of whether patients show increased or decreased activation relative to controls (Müller et al., 2018). The following analyses were conducted:

1. **Disorder-specific contrasts**: Separated by disorder-specific contrasts, between-group activation in each patient group relative to its matched controls (bipolar vs. controls, depression vs. controls, schizophrenia vs. controls).
2. **Pooled between-group analysis**: A pooled meta-analysis was also conducted, combining coordinates from all three diagnostic groups to identify transdiagnostic patterns of aberrant activation across all three disorders to identify common neural patterns associated with emotional processing abnormalities.

Subgroup and Valence-Specific Analyses

Follow-up ALE analyses were also conducted to reduce heterogeneity and to explore more specific effects related to demographic characteristics, medication status, diagnosis, and task- or stimulus-related factors (e.g., explicit vs. implicit emotion tasks). These subgroup analyses aimed to clarify whether sample or task features were associated with distinct patterns of convergence. Additionally, separate between-group analyses were conducted for

each stimulus valence category (negative, positive, emotional), as well as analyses against neutral stimuli and, separately, against baseline conditions. These analyses were only carried out when a minimum of 17 experiments was available to maintain sufficient statistical power and reliability.

Individual Experiment Contributions

Each significant cluster identified in the ALE analysis was further examined to determine the contribution of individual experiments. This involved assessing the influence of each individual experiment on the overall ALE value within each cluster. Specifically, the proportion each experiment contributed to the overall ALE value within a cluster was calculated to assess its influence on the observed convergence. For every cluster and each included experiment, the summed ALE value across all voxels in the cluster was calculated twice: once using the full set of experiments, and once with the experiment in question removed. If the total ALE value within the cluster decreased after removing a given experiment, that experiment was considered to have contributed to the convergence observed in that cluster. This method allowed for a detailed assessment of which studies were driving the meta-analytic effects.

Results

A total of 119 studies were included in this meta-analysis, comprising 32 studies on bipolar disorder, 52 studies on depression, and 35 studies on schizophrenia. The detailed screening process is illustrated in the PRISMA flow diagram in Figure A1-A3, and a summary of all meta-analyses conducted is provided in Figure A4. Within-group analyses, capturing patterns of brain activation during emotion processing within each diagnostic group's control participants, included 20 experiments each for bipolar disorder, depression, and schizophrenia. Between-group analyses, combining both hyper- and hypo-activation contrasts, included 28 experiments for bipolar disorder, 48 experiments for depression, and 34 experiments for schizophrenia. Additionally, a pooled between-group analysis combining activation contrasts across all three disorders comprised 108 experiments. Additional meta-analyses were only conducted when the number of available studies was sufficient to support a statistically meaningful and interpretable analysis.

Emotional Task Composition

Figure 1 illustrates the distribution of emotional task components reported across included studies for bipolar disorder, major depressive disorder (MDD), and schizophrenia. Across all three disorders, emotion identification tasks accounted for the largest proportion of studies (bipolar: 16; MDD: 19; schizophrenia: 17), reflecting a strong emphasis on recognition, matching and labelling of emotional stimuli. Implicit emotion perception was also prominently represented, particularly in MDD (n = 18) and schizophrenia (n = 11), and to a lesser extent in bipolar disorder (n = 9). Notably, the type of tasks used to assess implicit emotion perception varied across diagnostic groups: studies on bipolar disorder predominantly employed emotional Stroop tasks, while in MDD and schizophrenia, gender discrimination tasks using emotional faces were more commonly applied. Explicit emotion regulation tasks appeared most frequently in studies on MDD (n = 4), with fewer instances in

bipolar disorder (n = 1) and schizophrenia (n = 1). Passive viewing and emotion perception tasks showed variable representation across disorders, while tasks targeting emotion experience were the least common in all groups. Overall, the chart highlights that while certain emotional processes, such as identification and implicit perception, are commonly studied across disorders, the focus and distribution of task types differ slightly by diagnosis, reflecting both theoretical emphasis and methodological choices in the literature.

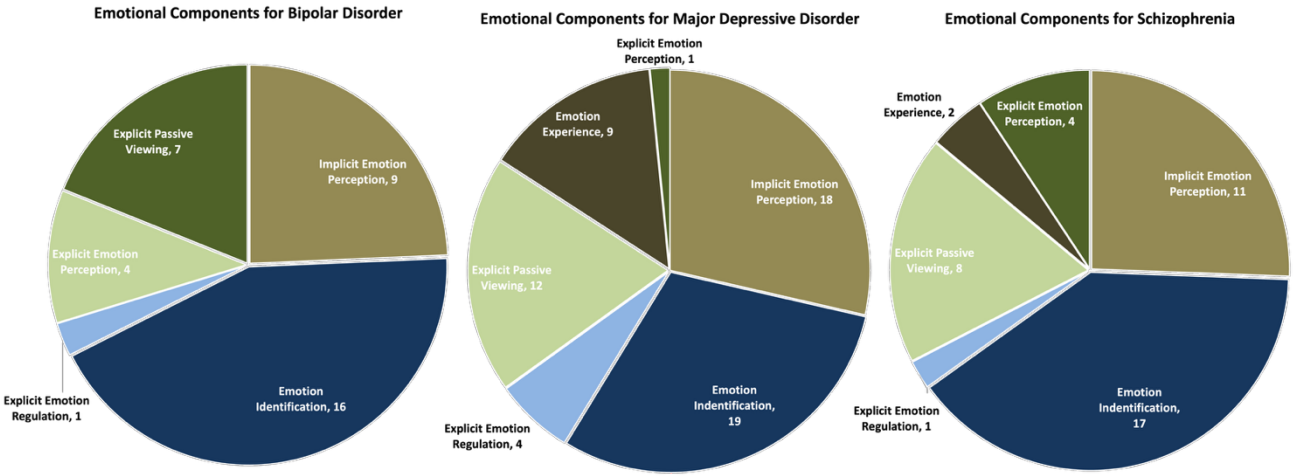


Figure 1. *Distribution of key components in emotion processing across Bipolar Disorder, Major Depressive Disorder and Schizophrenia. Each pie chart shows the relative proportions of explicit and implicit emotional processes reported across studies for each disorder.*

Within-Group Analyses

Bipolar Disorder. The within-group ALE meta-analysis (n = 20) of healthy controls included in studies investigating bipolar disorder revealed two clusters of significant convergence during emotional processing (cluster-level $p < .05$, family-wise error-corrected; voxel-level $p < .001$). As shown in Figure 2A and Table 4, convergence was observed in the left amygdala. The second cluster of significant convergence across studies was in the left lateral occipital cortex (inferior division).

Major Depressive Disorder. The within-group ALE meta-analysis of healthy controls drawn from studies on major depressive disorder (MDD) (n = 20) identified multiple

regions of significant convergence during emotional processing (cluster-level $p < .05$, FWE-corrected; voxel-level $p < .001$).

As shown in Table 5 and Figure 2B, robust convergence was observed in right fusiform gyrus (area FG4) and the left fusiform gyrus (area FG4). Additionally, significant convergence was observed in bilateral amygdala. Lastly, a smaller but reliable cluster of convergence was detected in the medial prefrontal cortex, including the left frontal pole and paracingulate gyrus, and the left frontal pole and superior frontal gyrus.

Schizophrenia. The within-group ALE meta-analysis (n = 20) of healthy controls from schizophrenia studies revealed a broad pattern of consistent convergence across multiple regions implicated in emotion processing (cluster-level $p < .05$, FWE-corrected; voxel-level $p < .001$). As shown in Figure 2C, and Table 6, convergence was found in the right amygdala as well as frontal regions, particularly in the right inferior frontal junction, middle frontal gyrus, and precentral gyrus. Furthermore, a significant cluster was also found in the left inferior frontal junction (area 44). Another cluster of convergence was found in the left insula extending into orbitofrontal cortex (area Id7). Robust convergence was also found in visual and visual-associative regions, particularly in the right lateral occipital cortex (inferior division), assigned to area hOc4la. In addition, convergence in the right lingual gyrus and intracalcarine cortex was observed (hOc1 [V1]). Further visual involvement was indicated by significant convergence in the right fusiform gyrus (area FG4). Additional clusters were in the left occipital pole and intracalcarine cortex and in the left lateral occipital cortex.

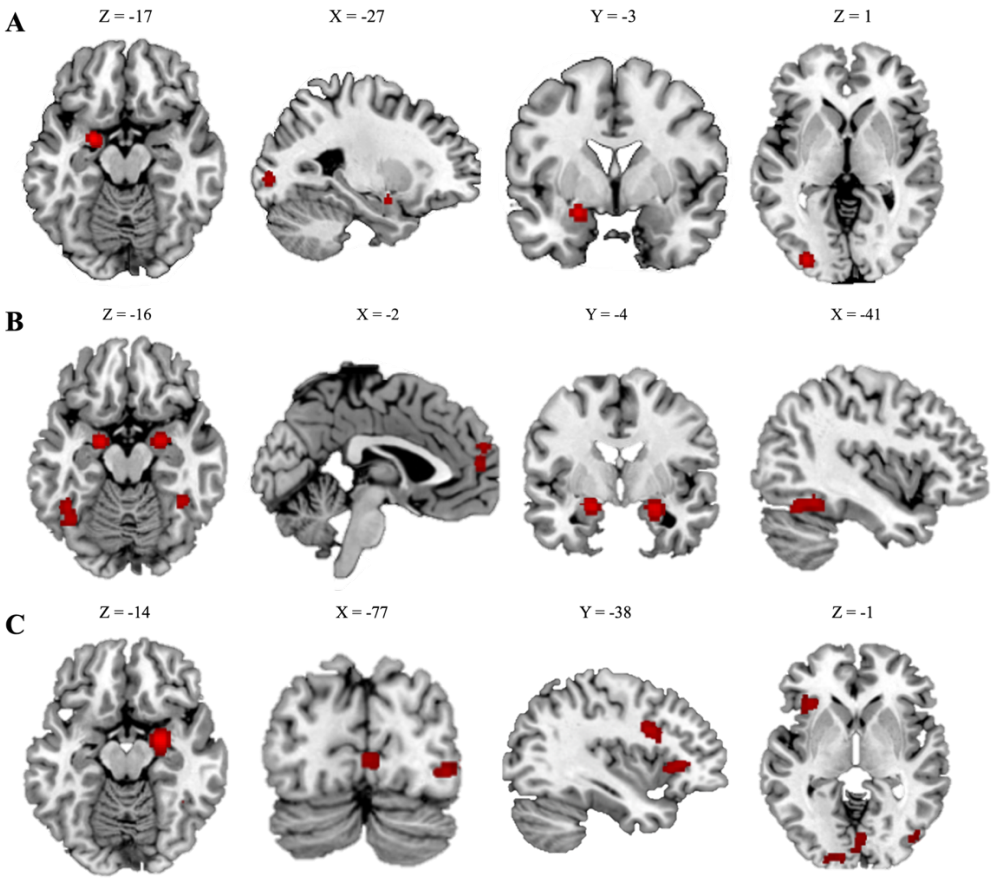


Figure 2. *Brain regions showing significant convergence of activity for emotional processing for healthy controls within the experiments investigating (A) bipolar disorder, (B) major depressive disorder, and (C) schizophrenia (cluster-level $p < .05$, family-wise error- corrected for multiple comparison, cluster forming threshold $p < .001$ at voxel level).*

Between-Group Analyses

Bipolar Disorder. The between-group ALE meta-analysis (n = 28) comparing patients with bipolar disorder to healthy controls did not reveal any clusters of significant convergence in differences in brain activity during emotional processing tasks, after correction for multiple comparisons (cluster-level $p < .05$, FWE-corrected). Notably, a separate meta-analysis restricted to medicated patients (n = 23) and a focused analyses conducted for negatively valenced stimuli (n = 19) contrasted against neutral or baseline condition did not yield any significant results.

Major Depressive Disorder. The between-group ALE meta-analysis (n = 48) comparing patients with major depressive disorder (MDD) to healthy controls did not yield any significant clusters of convergence when all emotional processing tasks were considered together. A follow-up analysis was conducted specifically for studies involving explicit emotional processing tasks (n = 32).

As shown in Figure 3A and Table 7, the focused analysis revealed a cluster of convergent activation differences (cluster-level $p < .05$, FWE-corrected; voxel-level $p < .001$) located in the right lingual gyrus, extending into the right intracalcarine cortex. Most contributing studies (71.4%) reported hypoactivation in this region in patients relative to controls, suggesting that reduced activation primarily drove the observed effect. A closer look at the contributing experiments revealed that a large proportion (44%) employed emotional identification tasks; however, this pattern was not exclusive, and no other study characteristic (e.g., medication status, unmedicated samples) consistently accounted for convergence in this cluster. Notably, a separate meta-analysis restricted to medicated patients (n = 26) conducted as specified in the hypotheses did not yield any significant results. Additionally, focused analyses conducted for negatively (n = 39) and positively (n = 21) valenced stimuli, contrasted against neutral or baseline condition did not yield any significant results.

Schizophrenia. The between-group ALE meta-analysis (n = 34) comparing patients with schizophrenia to healthy controls revealed a significant cluster of convergent activation during emotional processing (cluster-level $p < .05$, FWE-corrected; voxel-level $p < .001$) within right amygdala (Figure 3B and Table 8). Among the experiments contributing to the observed convergence, 80.31% reported hypo-activation in patients compared to healthy controls.

To explore the potential influence of stimulus type, follow-up between-group ALE analyses were also conducted. First, all trials featuring any emotionally valenced stimuli (positive, negative, or mixed) were contrasted against neutral stimuli. Second, a focused analysis considered only negatively valenced stimuli, contrasted against neutral or baseline condition. These analyses revealed the same right amygdala cluster as found for the general emotional processing analysis, indicating that the observed aberrant activity within the amygdala in patients was largely driven by negative emotional stimuli, rather than being uniformly present across all valence types. This interpretation is supported by the contribution analysis of the general emotional processing analysis, where most studies contributing to this cluster used negatively valenced stimuli.

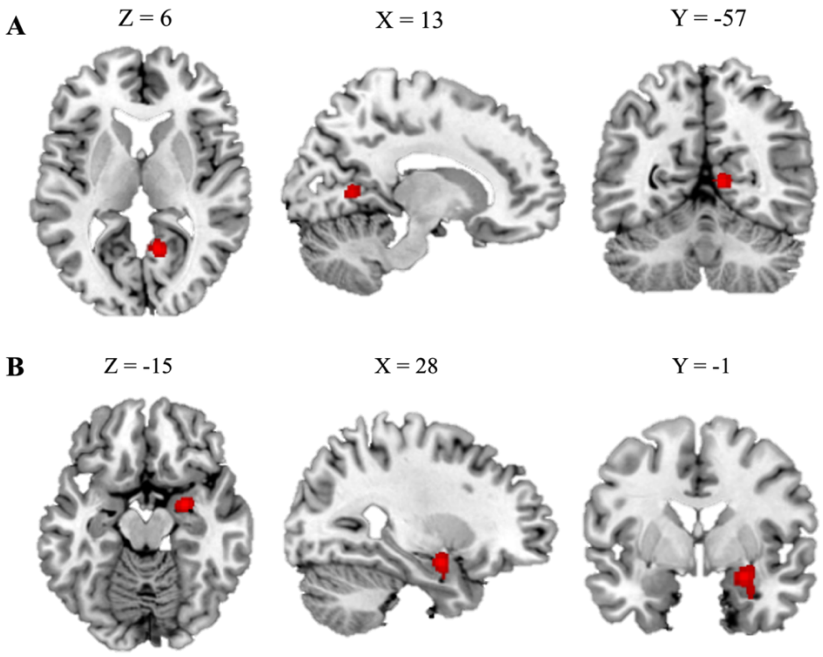


Figure 7. *Brain regions showing significant convergent differences in activity for emotional processing for (A) between-group analyses for explicit emotional processing in major depressive disorder, and (B) between-group analyses for schizophrenia, compared to healthy controls (cluster-level $p < .05$, family-wise error- corrected for multiple comparison, cluster forming threshold $p < .001$ at voxel level). Follow-up analyses focusing specifically on emotional vs. neutral and negative vs. baseline/neutral contrasts revealed the same regions.*

Pooled Analyses Across Between-Group Effects

The pooled between-group ALE meta-analysis (n = 108), which combined the between-group analyses for bipolar disorder, major depressive disorder (MDD), and

schizophrenia, identified two significant clusters of convergent activation during emotional processing (cluster-level $p < .05$, FWE-corrected; voxel-level $p < .001$). As shown in Figure 4 and Table 9, the first and most robust cluster was in the right amygdala and the second cluster of consistent convergence was in the left inferior frontal junction (IFJ).

As seen in Table 10, the contributing studies were examined by diagnosis, emotional task component, and direction of activation difference. For Cluster 1 (right amygdala), the largest proportion of contributing studies originated from schizophrenia samples (56.84%), followed by bipolar disorder (22.59%) and major depressive disorder (20.57%). Across all three diagnostic groups, the most common emotional components associated with this cluster were explicit passive viewing, and implicit emotion perception. The directionality of activation differences varied, with some studies showing greater activation in controls than patients (e.g., Control > Patient), others showing the opposite pattern (Patient > Control), and several reporting both hyper- and hypo-activation across regions.

Cluster 2 (left inferior frontal junction) demonstrated a more balanced distribution of contributions across diagnoses, with 38.62% from bipolar disorder, 33.22% from depression, and 28.15% from schizophrenia. Emotional tasks contributing to this cluster were mostly emotion identification tasks. Notably, most studies associated with this cluster reported greater activation in healthy controls than in patients.

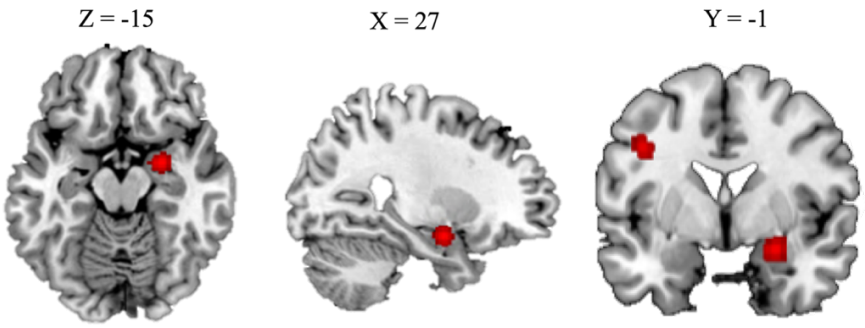


Figure 4. *Brain regions showing significant convergent differences in activity for emotional processing across bipolar, depression and schizophrenia compared to their healthy controls (cluster-level p < .05, family-wise error- corrected for multiple comparison, cluster forming threshold p < .001 at voxel level).*

Discussion

The aim of the present study was to assess the neural correlates of emotional processing in bipolar disorder, depression, and schizophrenia using coordinate-based ALE meta-analyses. Seven main meta-analyses were executed, comprising both within-group and between-group contrasts, and several supplementary meta-analyses were conducted that were stratified by task type, stimulus valence (e.g. focusing on positive, negative, emotional stimuli), and control condition (neutral stimuli or baseline).

The results revealed several significant findings. Within-group analyses for control participants across disorders showed robust activation in fronto-limbic regions, including the amygdala, prefrontal cortex, and occipital areas. Between-group analyses in MDD revealed that the attenuated response was centred on the primary visual cortex (lingual gyrus/intracalcarine cortex), whereas in schizophrenia it was confined to the right amygdala, a key limbic hub for affective salience. Pooled analyses across disorders were additionally performed to identify transdiagnostic patterns of dysfunction. The analysis revealed consistent activation differences in two regions across all three disorders: the right amygdala and the left inferior frontal junction (IFJ). In the following sections, results will be discussed separately for each region of convergence and diagnostic group.

Within-Group Analyses

The within-group ALE meta-analyses revealed a consistent engagement of the core emotion-processing network within healthy control groups drawn from studies on bipolar disorder, major depressive disorder (MDD), and schizophrenia. Notably, the amygdala showed robust convergence across all three control groups—supporting amygdala plays central role in emotion perception and salience detection (Santos et al., 2011). This finding underscores the amygdala as a key hub in affective processing, with its reliable recruitment

across datasets suggesting a fundamental role in detecting and appraising emotional significance, independent of diagnostic context.

While the amygdala showed consistent activation, convergence in other regions such as the fusiform gyrus, occipital cortices, and inferior frontal cortex was less uniform, appearing only in some of the datasets. These variations most likely reflect methodological and stimulus-related influences, including task type, stimulus modality, and the demographic composition of the included control groups, rather than fundamental neural differences. For instance, fusiform convergence was more frequently observed in datasets where tasks involved face perception or recognition. The fusiform gyrus is strongly implicated in face-selective processing and emotional expression recognition (Haxby et al., 2000; Vuilleumier & Pourtois, 2007), suggesting that its recruitment in some but not all analyses mirror the relative prevalence of face-based stimuli in the underlying studies. Similarly, convergence in lateral occipital regions was more prominent in studies employing emotional Stroop or word-based tasks. Given the established association of the lateral occipital cortex with visual word and object recognition (Grill-Spector et al., 2001), this pattern may reflect a bias towards left-hemisphere visual engagement during verbal or symbolic emotional processing.

Convergence in inferior frontal and prefrontal regions was likewise uneven across the datasets. These regions are commonly linked to higher-order cognitive control, response selection, and the explicit evaluation of emotional stimuli (Ochsner et al., 2012). Their presence in only some analyses likely reflects the task demands of explicit emotion identification or labelling, which place greater reliance on top-down regulatory processes. By contrast, studies employing implicit or passive viewing paradigms may have recruited subcortical and occipital regions more strongly, resulting in reduced frontal convergence.

An additional point of variation across the datasets concerned hemispheric lateralization of the amygdala. Controls associated with bipolar disorder studies demonstrated a greater likelihood of left-lateralized convergence, those linked to schizophrenia studies showed right-lateralized convergence, and MDD-related controls exhibited bilateral engagement. Rather than representing meaningful neurobiological differences between groups of healthy individuals, this lateralization pattern most plausibly reflects the types of emotional tasks that predominated within each diagnostic literature. For example, bipolar disorder studies frequently employed explicit emotion identification or Stroop-like paradigms, which are known to preferentially recruit the left amygdala due to its stronger involvement in sustained, verbal appraisal of emotion (Baas et al., 2004). In contrast, schizophrenia-related studies more often used implicit emotion perception or passive viewing tasks, conditions that preferentially engage the right amygdala in rapid, automatic responses to dynamic or visually salient stimuli (Öhman, 2005). Studies linked to MDD employed a more balanced mixture of explicit and implicit paradigms, which may explain the bilateral convergence observed in these controls. Thus, even within healthy populations, systematic task biases within the diagnostic literatures appear to shape observed patterns of lateralization.

These findings highlight the importance of task demands, stimulus properties, and sample characteristics in shaping within-group results. Variability across regions such as the fusiform gyrus, occipital cortex, and inferior frontal areas reflects methodological influences, while patterns of amygdala lateralization show how task type systematically alters neural recruitment. Across all groups, however, the amygdala consistently converged as a core hub for emotional salience detection. Task distributions (Figure 1) suggest that different paradigms selectively recruit the left amygdala for sustained, verbal appraisal and the right for rapid, visually salient processing (Baas et al., 2004; Morris et al., 1998; Öhman, 2005;

Sergerie et al., 2008), with these hemispheric differences likely reflecting task and stimulus biases in the diagnostic literatures.

Between-Group Analyses

Bipolar Disorder. The between-group ALE meta-analysis comparing patients with bipolar disorder to healthy controls did not reveal any significant clusters of convergence. This non-significant finding is important as it suggests that, at the group level, there are no robust differences in emotional processing between patients with bipolar disorder and healthy individuals. While the number of experiments included (n = 28) exceeded the standard minimum often recommended for ALE analyses (Eickhoff et al., 2016), this may still have been insufficient to overcome the disorder's internal variability and to capture subtle, state-dependent effects.

Another explanation may be heterogeneity in the bipolar population, including medication effects, sub-type, age, and comorbidities. Many studies included medicated patients and those with Axis-I comorbidities, adding diagnostic variability. Bipolar disorder is also marked by fluctuating mood states (mania, depression, euthymia) (Silva et al., 2013) and varying symptom severity (Townsend & Altshuler, 2012). Since most included studies did not control for mood state, differences across subgroups likely masked convergence, as each engages distinct neural circuits during emotion processing (Hulvershorn et al., 2012). Many studies were conducted during euthymia, when aberrant activation is known to attenuate compared to manic or depressive episodes (Hulvershorn et al., 2012; Wessa & Linke, 2009). Although it was hypothesised that bipolar disorder would show inconsistent brain activity across states, a state-specific analysis was not feasible due to insufficient data.

Even within "emotional processing tasks," there can be significant differences in the specific paradigms used (e.g., passive viewing, emotion identification, emotion regulation,

different valences of stimuli). This variability could contribute to inconsistent findings (Townsend & Altshuler, 2012). Thus, the results may reflect that the current imaging literature is so heterogeneous that no generalised effects may be found. Unfortunately, there are too little studies in the field to allow for more specific analyses, such as separating the studies by mood state or task type. Furthermore, the small sample sizes in some experiments might have also made results inconsistent.

However, previous meta-analyses have reported hyperactivation in individuals with bipolar disorder in regions such as the hippocampus, temporal cortex, and amygdala (Mesbah et al., 2023). Similarly, Ahmed et al. (2023) found increased activation in subcortical and limbic areas, including the caudate, amygdala, and parahippocampal gyrus, when comparing patients with BD to healthy controls. One possible explanation for the discrepancy with the current study lies in the differences in inclusion criteria. While the present meta-analysis focused exclusively on emotion processing tasks, earlier reviews have included a broader range of paradigms such as emotion regulation, reward anticipation, or cognitive-affective tasks which engage overlapping but distinct neural circuits (Phillips et al., 2008; Townsend & Altshuler, 2012; Delvecchio et al., 2012). This suggests that the current meta-analytic approach, while robust for identifying consistent patterns, may not be sensitive enough to capture the dynamic nature of brain activity in bipolar disorder (Müller et al., 2018; Kober & Wager, 2010).

Major Depressive Disorder. The analysis for between-group differences in MDD did not yield a significant result which is in line with findings from previous meta-analyses by Müller et al., (2017), which similarly reported a lack of robust convergence in emotional processing networks. This stands in contrast to expectations based on the broader neuroimaging literature, which frequently suggests consistent emotion-related abnormalities in MDD. Given the relatively large number of included experiments, this absence of

significant convergence is notable and suggests a high degree of heterogeneity across the available studies.

To reduce potential effects of task-related heterogeneity, a follow-up analysis was conducted including only studies that employed explicit emotional processing tasks in the between-group comparison. The lack of significant findings in the general pooling of all emotional tasks could be attributed to the inclusion of implicit tasks, which are less likely to engage early visual areas. Explicit tasks require conscious attention and processing of emotional content while implicit tasks often rely on automatic processing of emotional stimuli (Cohen et al., 2016; Flaisch et al., 2015), which may not require the same level of engagement from early visual areas as explicit tasks do. Although the general analysis involved a larger number of experiments, the inclusion of diverse paradigms may have diluted convergent effects that are specific to explicit processing. Thus, isolating task types in the follow-up analysis likely enhanced sensitivity to functionally specific group differences.

The between-group analysis for MDD revealed a reliable cluster of convergent activation differences in the right lingual gyrus extending into the intracalcarine cortex (hOc1, V1), indicating aberrant neural activation during explicit emotional processing. This effect was mainly driven by contrasts where controls showed greater activation than patients, suggesting reduced engagement of early visual areas in MDD during effortful emotion tasks. As primary visual regions (Rosenthal et al., 2016), their hypoactivation points to diminished visual processing of emotional stimuli. Supporting this, Li and Wang (2021) found decreased right lingual gyrus activation in MDD, and several task-based studies report reduced engagement of early and mid-level visual cortices during explicit face tasks (Fan et al., 2023; Han et al., 2016). However, the fact that the only convergent finding was in the visual cortex rather than in regions typically emphasized as central to emotional dysfunction in depression (e.g., amygdala, prefrontal cortex) (Sheline et al., 2001; Drevets et al., 2000; Phillips et al.,

2003) further underlines the limited and somewhat unexpected nature of consistent effects in this dataset.

Importantly, a separate analysis focusing only on medicated MDD patients, as specified in the hypotheses, also revealed no significant convergence. This suggests that medication status alone cannot explain the lack of robust and consistent findings, further supporting the view that variability in task paradigms, analytic approaches, and clinical subgroups substantially contributes to the absence of stronger convergence in MDD.

These findings could imply that depressed individuals are less engaged with or less effectively process the visual features of emotional stimuli at a fundamental level, even when explicitly instructed to do so. This is in line with theories of negative cognitive bias in depression (Beck, 1963; Rnic et al., 2016), which posit that individuals with MDD have impaired processing of positive emotional stimuli and heightened processing of negative stimuli (Lee et al., 2007). In the case of explicit tasks, this would translate to increased attention and processing of negative stimuli, as well as decreased attention and processing of positive stimuli.

Schizophrenia. The between-group analysis for schizophrenia revealed a significant cluster of convergent activation differences in the right amygdala, indicating consistent aberrancies in neural activation during emotion processing tasks. This finding was consistent across analyses against neutral and when focussing on negatively valenced stimuli only. A review of the contributing studies indicated that this convergence was primarily driven by contrasts where controls showed greater activation than patients (80.31%) and only 19.69% studies reported greater activation in patients compared to controls.

The right amygdala plays a significant role in threat detection and emotional processing, particularly for negative emotions like fear (Whalen et al., 1998; Fox et al.,

2015). Previous meta-analyses (Li et al., 2010; Delvecchio et al., 2013) support the findings as they reported amygdala hypo-activation in schizophrenia patients compared to controls during emotional recognition tasks. However, some meta-analyses also report hyper-activation in schizophrenia patients compared to controls (Holt et al., 2006; Mier et al. 2010). Hall et al. (2008) found that individuals with schizophrenia showed reduced amygdala activity when viewing fearful faces, but only when comparing it to their brain activity when viewing neutral faces. Another meta-analyses by Anticevic and colleagues (Anticevic et al., 2012) also reported decreased amygdala activity in schizophrenia patients for negative emotions, specifically when compared to neutral emotions. This suggests that the current findings are in line with the literature.

The current findings suggest that individuals with schizophrenia show reduced engagement of the right amygdala during emotional tasks compared to healthy individuals. This could be suggestive of a reduced or blunted initial emotional response in schizophrenia (Fahim et al., 2005; Lepage et al., 2011). The right amygdala is associated with the early, automatic evaluation of emotional stimuli (Dannlowski et al., 2007), particularly in response to negatively valenced items.

However, in the study by Pankow and colleagues (2013), it was reported that schizophrenia patients revealed stronger right amygdala activation during negative and attenuated response during positive affective picture processing compared to healthy controls. Another study by Morris et al. (2009) reported that amygdala hyperactivity can also be detected when people with schizophrenia fail to appropriately recognize fear faces. This finding is consistent with the results of the present study, despite a small proportion of patients exhibiting hyperactivation compared to healthy controls. It's possible that while some individual studies show hyper-activation, the overall trend across a larger body of literature points to hypo-activation, considering a broader range of emotional stimuli.

Pooled Analyses Across Between-Group Effects

The pooled analysis across all experiments of aberrant activation across all three disorders i.e. bipolar disorder, major depressive disorder (MDD), and schizophrenia (108 experiments), identified two significant clusters of convergent activation differences: the right amygdala and the left inferior frontal junction (IFJ). These results highlight the presence of shared neural alterations in emotional processing across these major psychiatric disorders, supporting the idea that, despite diagnostic boundaries, common brain mechanisms may underlie emotional dysfunction (Goodkind et al., 2015; McTeague et al., 2020).

Right Amygdala. The most robust convergence (170 voxels) was found in the right amygdala across all three disorders suggesting a transdiagnostic neural correlate of emotional processing dysfunction. The findings point toward a specific impairment in the right amygdala's function in emotional salience detection, rather than simply highlighting its general role in emotion processing. More than half of the contributing contrasts stemmed from schizophrenia samples (56.8 %), with smaller—but still meaningful—contributions from bipolar disorder (22.6 %) and MDD (20.6 %).

The emergence of a robust right amygdala cluster in the pooled between-group analysis—despite its absence in the disorder-specific contrasts for bipolar disorder and MDD—suggests that this region may reflect a shared transdiagnostic vulnerability that is not consistently detectable within each individual diagnostic group. Notably, the right amygdala showed significant convergence of aberrant activation in the schizophrenia, which also mostly contributed (56.84%) to the cluster in the pooled analysis. This could imply that the pooled result may be primarily driven by consistent hypoactivation observed in schizophrenia, while smaller and more heterogeneous effects in bipolar disorder and MDD diluted the statistical significance in those groups individually.

One plausible explanation is that affective salience detection, a function heavily reliant on the right amygdala (Attar et al., 2010), may be consistently impaired in schizophrenia and subtly altered in bipolar disorder and MDD, with the latter effects only reaching statistical significance when data are pooled across disorders. Emotional misinterpretation, heightened threat sensitivity, or flattened affect are not exclusive symptoms of schizophrenia and may also appear in bipolar disorder and depression depending on clinical phase (e.g., mania vs. euthymia) or subtype (Muhtadie & Johnson, 2015). Thus, depression and bipolar disorder may represent more heterogeneous conditions in this context, with subgroups or phases that are not uniformly associated with impairments in salience processing—potentially diluting convergence in group-level analyses. As such, shared emotional symptoms even if inconsistently represented across studies can lead to a significant transdiagnostic signal when pooled. This interpretation is consistent with transdiagnostic meta-analytic findings (e.g., McTeague et al., 2020) that identify the amygdala as a core region of aberrant emotion processing across a broad range of psychiatric conditions. Thus, the absence of right amygdala convergence in bipolar and MDD alone may not reflect true lack of dysfunction, but rather variability in sampling, task design, or symptom expression that becomes smoothed out at the transdiagnostic level.

Task-wise, 51.85% of the studies contributing to the right amygdala cluster involved implicit emotional perception or explicit passive viewing tasks, with 29.62% derived solely from implicit emotional perception tasks. This suggests that the right amygdala shows consistent aberrant activity in automatic, unconscious emotional processing across all three disorders (Gainotti, 2012). Studies have consistently shown that the right amygdala is more responsive to masked or implicit emotional stimuli, especially fearful faces (Morris et al., 1998). Studies by Ethofer et al. (2009) and Mitchell et al. (2007) confirm that amygdala activation is stronger during implicit paradigms than during explicit emotion labelling.

Although both hyperactivation and hypoactivation studies contributed to the cluster, the direction of effects was not evenly distributed. As discussed above, schizophrenia contributed primarily with hypoactivation, while bipolar disorder contributed mainly with hyperactivation. MDD showed a mixture of both directions, consistent with prior findings that report amygdala hyperreactivity to negative stimuli (e.g., sad or fearful faces) particularly during acute depressive episodes, and hypo-reactivity to positive stimuli, which may persist even during remitted or less symptomatic phases (Siegle et al., 2002; Stuhrmann et al., 2011). This suggests that amygdala activation patterns in MDD are influenced both by symptom phase and stimulus valence.

Left Inferior Frontal Junction (IFJ). The second significant cluster of convergence was identified in the left inferior frontal junction (IFJ), a region situated at the intersection of the inferior frontal sulcus and the precentral sulcus (Derrfuss et al., 2012). The IFJ is associated with top-down cognitive control, particularly in contexts requiring goal-directed attention, working memory, and emotional regulation (Tamber-Rosenau et al., 2018). This cluster showed a balanced contribution, with bipolar disorder accounting for 38.62%, MDD for 33.22%, and schizophrenia for 28.15% of the contributing studies.

Most tasks contributing to this cluster involved emotion identification (63.5%). The IFJ, situated at the intersection of emotional and cognitive control systems, is central to interference resolution, task switching, and attentional modulation (Brass et al., 2005; Derrfuss et al., 2005). Its role in emotion identification is evident from studies showing increased IFJ activity during explicit emotion labelling or judgment (Gorno-Tempini et al., 2001; Wegrzyn et al., 2017). Such tasks recruit the IFJ more strongly than passive paradigms because they require verbal coding and executive control (Sundermann & Pfleiderer, 2012). As most contributing experiments reported hypoactivation in patients relative to controls, the

findings suggest that aberrant IFJ activation reflects shared difficulties in recognizing and labelling emotions, pointing to disrupted higher-order appraisal processes across disorders.

In the analysis, the emotional identification component was among the most frequent contributors to the IFJ cluster, especially in bipolar and MDD studies. This supports the interpretation that IFJ hypoactivation reflects a shared difficulty in applying top-down control during emotion evaluation—potentially undermining emotion regulation strategies such as reappraisal. Indeed, IFJ involvement in cognitive control over affective processing has been observed across several disorders (Cromheeke & Mueller, 2014) and is often impaired in mood (Qin et al.2022; Roberts et al., 2013; Rolls et al., 2020), and psychotic conditions (Li et al., 2012).

Comparison to Previous Meta-Analyses

The present pooled ALE analysis reports two significant clusters of convergence—right amygdala and left IFJ—whereas earlier coordinate-based reviews of emotional processing in psychiatric illness have reported somewhat broader or different patterns of convergence. There is a low number of studies reporting the pattern of emotional processing across these three disorders. The few meta-analyses that assessed emotional processing across disorders are not directly comparable to the present study, as they used different methodologies. For example, Boisvert et al. (2023) included cognitive control tasks in their analyses, which is not the case in the current study. Further, most existing studies only report pairwise comparisons between two disorders (e.g., bipolar vs. MDD), rather than systematically examining all three diagnostic groups (Trotti et al., 2021; Korgaonkar et al., 2019). In contrast, the present study conducted separate meta-analyses for each disorder, allowing for indirect comparison of shared and distinct neural alterations across all three groups.

Past coordinate-based meta-analyses already highlighted amygdala dysfunction in bipolar disorder and major depression (Delvecchio et al., 2012) as well as in schizophrenia (Li et al., 2010). The pooled analysis corroborated that the right amygdala is a shared locus of alteration; however, examination of experiment contributions revealed that bipolar studies tended to report hyperactivation, schizophrenia studies predominantly reported hypoactivation, and depression studies showed a mixture of both hyper- and hypoactivation patterns. This mirrors the pattern reported by Anticevic et al. (2012), who observed blunted amygdala engagement when schizophrenia patients processed negative facial expressions, but heightened responses in several bipolar samples (Altshuler et al., 2005). Thus, the present findings regarding the right amygdala are broadly consistent with earlier meta-analytic studies that reported amygdala involvement in emotional processing across psychiatric disorders (e.g., Phillips et al., 2003; Goodkind et al., 2015), although some previous analyses identified bilateral or left-lateralized effects.

Several prior meta-analyses have consistently identified the anterior cingulate cortex (ACC) and ventromedial prefrontal cortex (vmPFC) as key regions of dysfunction, particularly in depression (Groenewold et al., 2013) and across mood disorder populations (Phillips et al., 2008). In contrast, the current findings highlight the right amygdala and left inferior frontal junction (IFJ) as loci of transdiagnostic convergence. The IFJ is often associated with executive functions, such as task switching and cognitive control (Brass et al., 2005). This apparent discrepancy likely stems from differences in the emotional task paradigms examined. While earlier studies focused on explicit emotion regulation or self-referential tasks, which are known to recruit the ACC and vmPFC (Wagner et al., 2015), the current meta-analysis included a larger proportion of emotion identification tasks. These tasks explicitly require participants to recognize, label, or cognitively appraise emotions, a process that consistently activates the IFJ (Derrfuss et al., 2004; Wegrzyn et al., 2017). The observed

IFJ hypoactivation across disorders in the present study may therefore reflect a shared disruption in this critical component of emotional cognition.

Limitations and Strengths

This meta-analysis has several limitations. Firstly, although efforts were made to standardize the inclusion criteria, inherent variability persists across studies. This includes differences in specific task paradigms (e.g., explicit vs. implicit, different emotional valences), stimulus types, scanner parameters, patient characteristics (e.g., symptom severity, chronicity, comorbidity), and medication status. While such heterogeneity is inherent in clinical neuroimaging research, it poses a particular challenge for comparing findings across diagnostic groups. For example, the presence of convergent findings in one disorder but not another may reflect not only true disorder-specific neural differences, but also disparities in the number of studies available, the consistency of task designs, or the characteristics of the sampled populations.

In some disorders, convergence may emerge due to a higher concentration of studies using similar paradigms or recruiting more homogeneous samples. In contrast, findings may appear more variable or entirely absent in other disorders simply due to greater methodological and clinical heterogeneity. Furthermore, several studies included a small sample size, which may reduce statistical power and limit the generalizability of specific effects. This brings a large degree of heterogeneity to the data, which could mask or distort group effects. This decreases the statistical power of the analyses and increases the risk of false negatives. Moreover, the issue of missing data arises in meta-analyses, as not all studies report the necessary statistics or provide supplementary information.

Like all meta-analyses, this study is susceptible to publication bias, where studies with significant or novel findings are more likely to be published than those with null results. This

can lead to an overestimation of effect sizes or a skewed representation of the data. Even though attempts were made to conduct sub-analyses stratified by task type and stimulus valence, many of these did not meet the minimum study threshold of 17 experiments. for between-group analyses.

Despite its limitations, this meta-analysis offers several strengths that enhance the robustness and generalizability of its findings. Firstly, the use of ALE meta-analysis provides a quantitative and objective method for identifying consistent patterns of brain activation across multiple studies. This approach overcomes the limitations of qualitative reviews by statistically assessing the convergence of reported foci, thereby increasing the reliability of the identified neural correlates. Additionally, the use of strict inclusion criteria ensures a basic level of methodological consistency across studies, such as full-brain coverage and standard coordinate reporting. This enhances the validity of the findings and reduces the risk of bias or confounding effects arising from methodological differences.

Further, the systematic and extensive search across major neuroimaging databases ensured a broad and representative sample of the existing literature. Furthermore, the pooling of all three disorders in the between-group analyses allows for an exploration of transdiagnostic patterns of emotional processing dysfunction. This transdiagnostic approach provides valuable insights into common underlying mechanisms and disorder-specific pathways, which is crucial for developing more targeted interventions. By pooling data from 134 studies, the meta-analysis benefits from a significantly larger sample size than individual fMRI studies. This increased statistical power enhances the ability to detect subtle but consistent effects. Finally, the analysis of individual experiment contributions to significant clusters provides transparency and allows for a deeper understanding of which studies and experimental conditions primarily drive the observed effects.

Future Recommendations

Based on the findings and limitations of this meta-analysis, several recommendations for future research emerge. Future fMRI studies on emotional processing in psychiatric disorders would benefit from greater standardization of experimental paradigms. Developing consensus on core tasks and stimuli would facilitate more comparable meta-analyses. Additionally, including more detailed information on participant characteristics, scanner parameters, and specific analyses performed would reduce the issue of missing data and enhance the transparency of the findings, making follow-up analyses easier. Furthermore, given the non-significant between-group findings for bipolar disorder, future meta-analyses should prioritize sub-analyses stratified by mood state (manic, depressive, euthymic). However, such efforts will only become feasible once a larger number of fMRI studies focused on individual mood states have been published, as the current literature lacks sufficient subtype-specific data to support meaningful comparisons.

Beyond activation differences, future meta-analyses should explore alterations in functional and structural connectivity within emotional processing networks. This could provide insights into how brain regions communicate in psychiatric disorders, and whether this differs from healthy populations. Furthermore, future studies should investigate functional connectivity or network-level alterations, which are increasingly recognized as crucial aspects of psychiatric disorders. Additionally, most studies focus on cross-sectional samples, but to understand the dynamic nature of emotional processing deficits, it is important to conduct more longitudinal fMRI studies. Future meta-analyses could aim to integrate clinical and behavioural measures (e.g., symptom severity, specific emotional regulation strategies, social functioning) with neuroimaging data. This would allow for a more nuanced understanding of the relationship between brain dysfunction and real-world impairments. Ultimately, more single studies are necessary to thoroughly investigate

emotional processing in each disorder. The inclusion of more studies in the future would enable more powerful meta-analyses and increase the reliability of the findings.

Conclusion

The present study provides a comprehensive overview of the neural correlates of emotional processing in healthy controls for bipolar disorder, major depressive disorder, and schizophrenia. The within-group analyses demonstrate that healthy participants recruited as control groups in clinical research consistently engage core emotion- and visual-processing regions. However, the exact brain regions activated varied depending on which diagnostic group the controls were matched to, suggesting that sample characteristics and study designs significantly shape the observed activation patterns. This underscores that the neural correlates of emotion processing are not only robust but also sensitive to context-specific methodological factors, such as task type, stimulus characteristics, and clinical sampling.

The between-group analyses revealed disorder-specific aberrancies in activity: in the right lingual gyrus/intracalcarine cortex for major depressive disorder during explicit tasks, and in the right amygdala for schizophrenia. Furthermore, the pooled transdiagnostic analysis identified the right amygdala and left inferior frontal junction as common regions of dysfunction across all three disorders, supporting the hypothesis of both shared and distinct neural mechanisms. The findings highlight the importance of the right amygdala in emotional processing across disorders and suggest that alterations in top-down regulation from the IFJ may contribute difficulties in emotional processing in patients. These results highlight the intricate and varied characteristics of difficulties in processing emotions within the context of mental disorders.

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Disclosure of the use of Generative AI in the writing process

During the preparation of this work, I used ChatGPT and Yomu to refine my English phrasing, aid in refining complex statements to simpler statements, for brainstorming and aid in correcting grammatical errors. After using these tools, I reviewed and edited the content as needed and I take full responsibility for the content of the publication.

Appendix: Figures and Tables

Additional information on meta-analytic workflow and sample characteristics

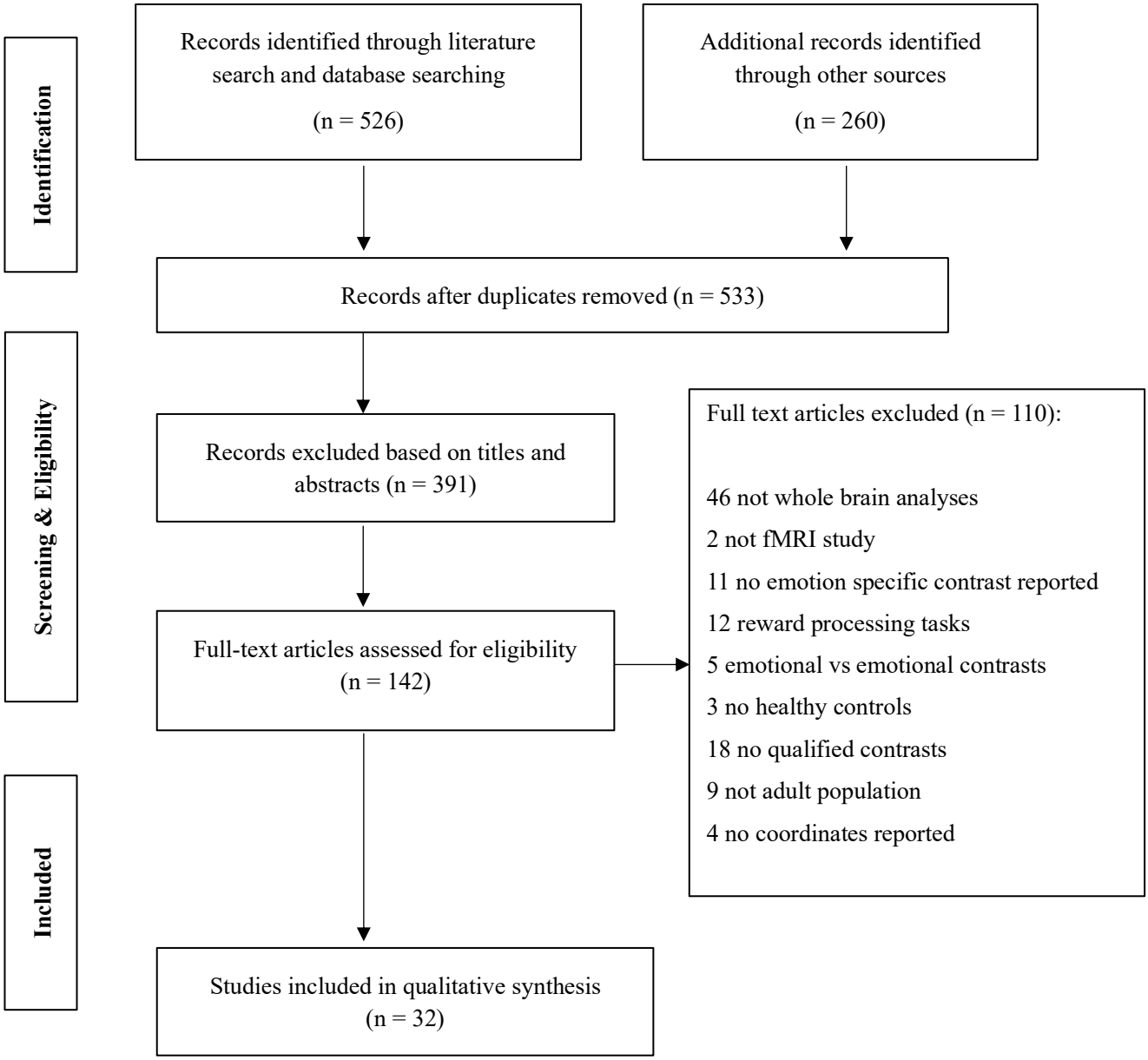


Figure A1. *PRISMA flow diagram for screening process and different steps conducted for bipolar disorder.*

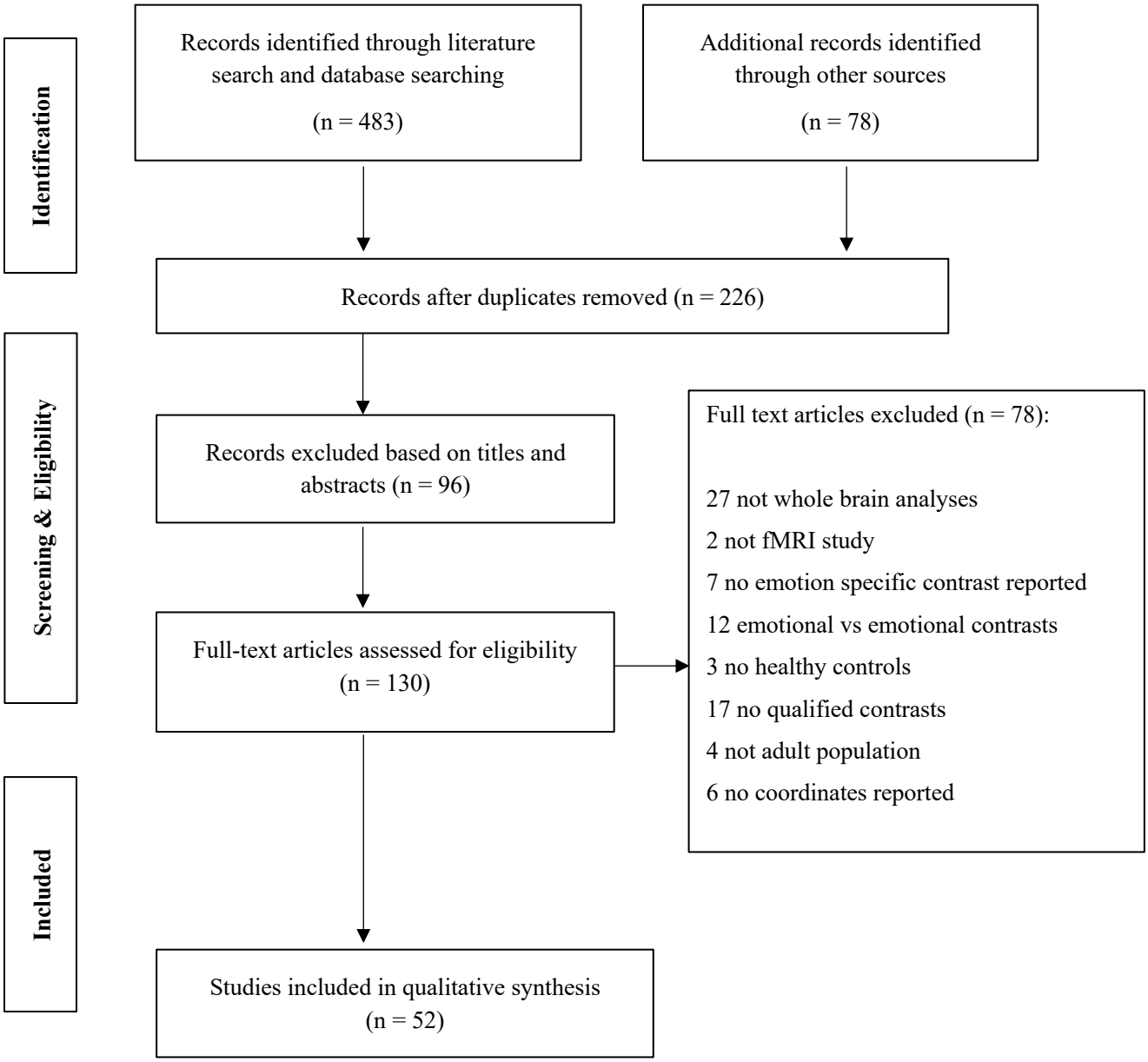


Figure A2. *PRISMA flow diagram for screening process and different steps conducted for major depressive disorder.*

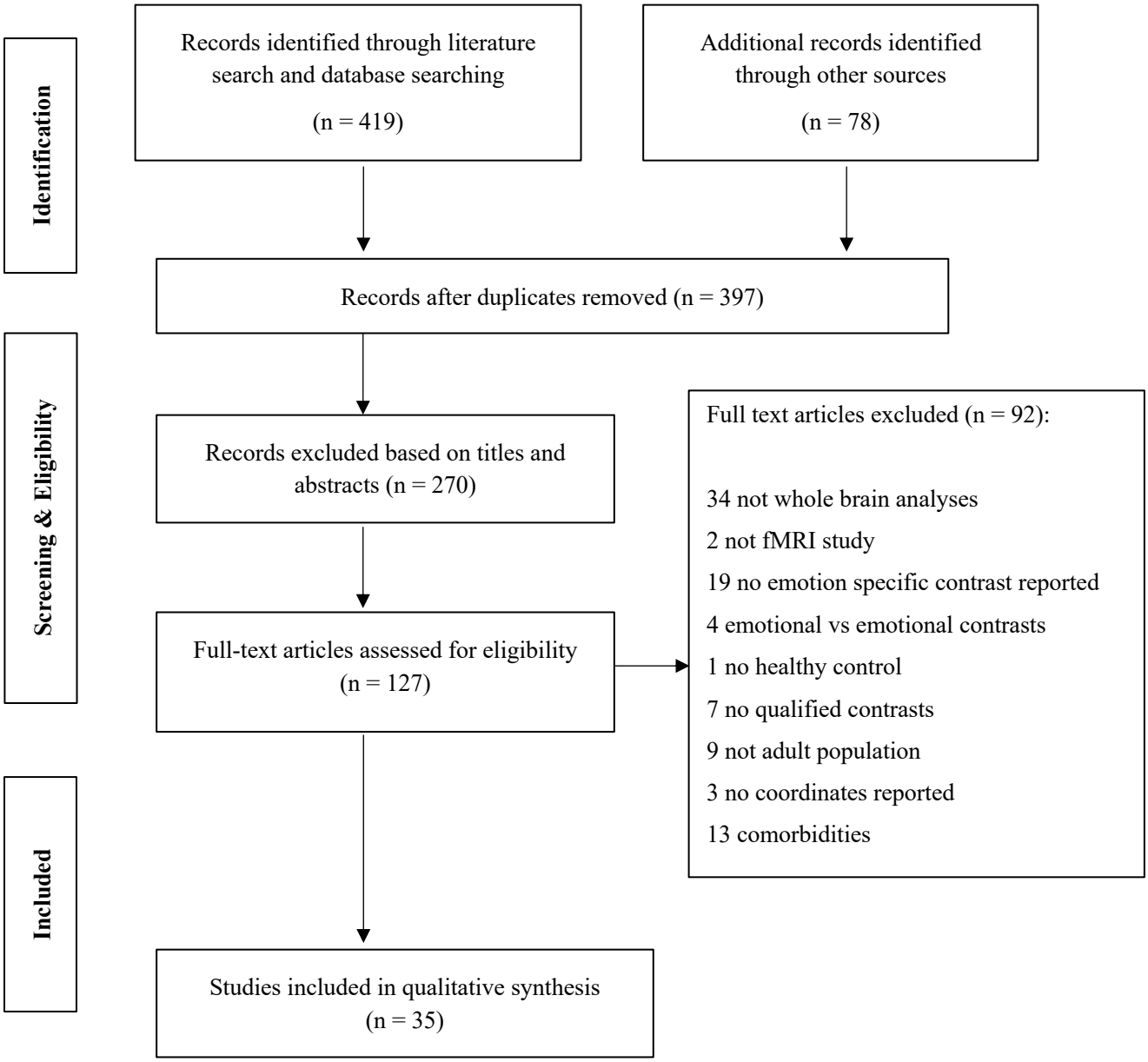


Figure A3. *PRISMA flow diagram for screening process and different steps conducted schizophrenia.*

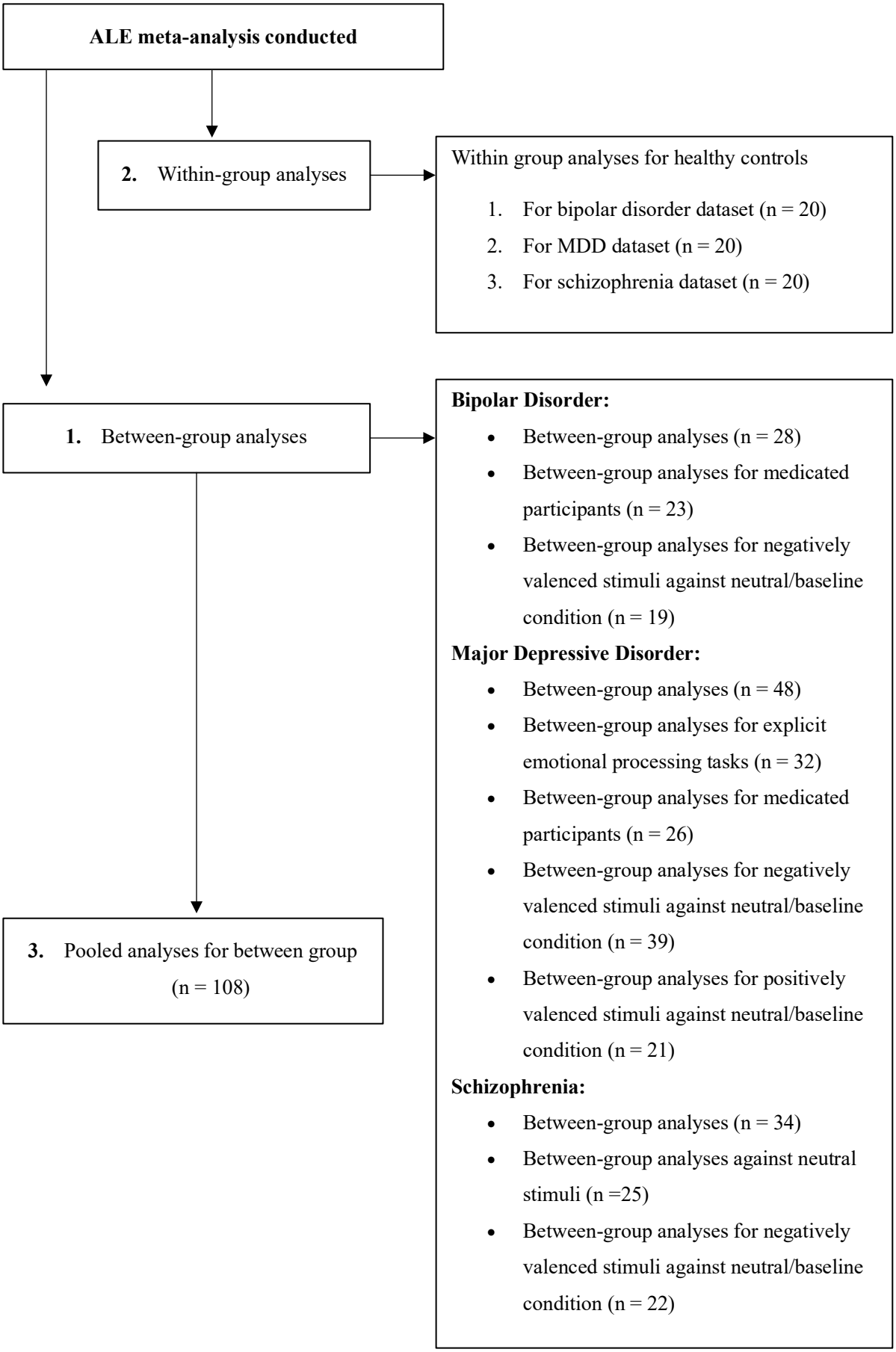


Figure A4. *Flowchart depicting the meta-analyses conducted*

Table 1. *Study sample characteristics for bipolar disorder (n=32)*

Publication	N (M: F)	Age (M±SD)	Mood State(s)	Medication Status	Design/Paradigm	Emotional Contrast	Emotional Component	Contrast(s) Used
Cerullo et al. 2014	BD = 25 (10:15) HC = 25 (7:18)	BD = 30 (8) HC = 26 (7)	Bipolar – I	Not medicated	Emotional oddball task	Negative vs neutral	Emotion identification	BD>HC, HC>BD
Chen et al. 2006	BD-M = 8 (8:0) BD-D = 8 (5:3) HC = 8 (2:6)	BD-M = 39 (13.4) BD-D = 41.8 (12.09) HC = 38.75 (12.5)	Manic Depressed	37 (100%)	Explicit and implicit facial affect recognition task	Negative vs neutral	Emotion identification	BD>HC
Deckerbach et al. 2008	BD = 9 (0:9) HC = 17 (0:17)	BD = 27.6 (2.8) HC = 25.6 (5.9)	Bipolar – I (Depressed)	9 (100%)	Emotional N-back task	Negative vs neutral	Explicit emotion perception	HC within, BD within
Dima et al. 2016	BD = 41 (20:21) HC = 46 (25:21)	BD = 44.3 (11.9) HC = 40.3 (13.2)	Euthymic BD-I	NA	Facial affect recognition task	Negative vs neutral	Emotion identification	HC>BD, main effect across group
Elliot et al. 2004	BD = 8 (4:4) HC = 11 (3:8)	BD = 33.5 HC = 37.6	Hypo/manic	7 (87.5%)	Word targets and distractors	Emotional vs neutral	Implicit emotion perception	BD>HC
Foland-Ross et al. 2012	BD = 24 (15:9) HC = 26 (15:11)	BD = 38.8 (12.8) HC = 37.9 (13.3)	Euthymic BD-I	16 (67%)	Labeling and matching emotional faces	Emotional vs neutral	Emotion identification	BD>HC
Han et al. 2018	BD = 10 (6:4) HC = 10 (6:4)	BD = 38.6 (9.4) HC = 31.8 (8)	Euthymic BD-I	10 (100%)	Emotional picture session	Negative vs baseline, positive vs baseline	Explicit passive viewing	BD>HC, HC within, BD within
Hulvershorn et al. 2012	BD-D = 30 (12:18) BD-E = 15 (5:10) BD-M = 30 (11:19) HC = 30 (11:19)	BD-D = 35 (11) BD-E = 31 (11) BD-M = 34 (11) HC = 32 (10)	BD– I and II (Depressed, euthymic, manic)	Not medicated	Matching emotional faces	Negative vs neutral	Emotion identification	BD>HC

Jogia et al. 2018	BD = 8 (5:3) HC = 15	BD = 42.1 (11.8) HC = 41.8 (10.9)	Bipolar – I	8 (100%)	Facial emotion recognition	Negative vs neutral	Emotion identification	BD>HC, HC>BD, HC within, BD within
Kanske et al. 2013	BD = 22 (8:14) HC = 22 (10:12)	BD = 39.4 (11.8) HC = 40.5 (11.8)	Euthymic BD-I	19 (86.3%)	Arithmetic task with emotional distractors	Emotional vs neutral	Implicit emotion perception	BD>HC, main effect across group
Keener et al. 2012	BD = 27 (10:17) HC = 27 (10:17)	BD = 31.7 (8.47) HC = 32.4 (6.52)	Euthymic BD-I	26 (96.3%)	Emotional face and identity morphs	Negative vs neutral, positive vs neutral, emotional vs neutral	Emotion identification Implicit emotion perception	BD>HC, main effect across group
Killgore et al. 2008	BD = 14 (11:3) HC = 13 (12:1)	BD = 28.1 (11.2) HC = 25.5 (4.7)	Bipolar – I	14 (100%)	Fearful face perception task	Negative vs baseline	Explicit passive viewing	BD>HC, HC>BD
Kim et al. 2009	BD = 14 (8:6) HC = 14 (8:6)	BD = 30.4 (5.9) HC = 27.5 (3.3)	Euthymic BD-I	14 (100%)	Perceived emotions and judgement task	Negative vs neutral, positive vs neutral, emotional vs neutral	Explicit emotion perception	BD>HC, HC>BD, HC within, BD within
Kjærstad et al. 2023	BD = 78 (27:51) HC = 56 (20:36)	BD = 30 (5) HC = 26 (7)	Bipolar – I and Bipolar – II	59 (76.6%)	Emotional regulation task	negative vs neutral	Explicit passive viewing	HC within
Lagopoulos et al. 2007	BD = 10 (0:10) HC = 10 (0:10)	BD = 31.29 (8.12) HC = 33.56 (6.73)	Euthymic BD-I	7 (70%)	Emotional Stroop task	Negative vs neutral	Implicit emotion perception	BD>HC, HC>BD, HC within, BD within
Lagopoulos et al. 2011	BD = 11 (0:11) HC = 11 (0:11)	BD = 33.3 (9.2) HC = 33.6 (6.7)	Euthymic BD-I	8 (72.7%)	Facial affect recognition task	Negative vs neutral	Emotion identification	BD>HC, HC>BD, HC within, BD within
Lennox et al. 2004	BD = 10 (8:2) HC = 12 (6:6)	BD = 37.3 (12.8) HC = 32.6 (10.7)	Hypo/manic	10 (100%)	Perceived emotional intensity	Negative vs neutral, positive vs neutral	Explicit emotion perception	BD>HC, HC>BD, HC within

Malhi et al. 2004	BD = 10 (0:10) HC = 10 (0:10)	BD = 34.3 (12) HC = 34 (13)	Bipolar – I (Depressed)	10 (100%)	Passive viewing emotional captioned images	Negative vs neutral, positive vs neutral	Explicit passive viewing	HC within, BD within
Malhi et al. 2005	BD = 12 (0:12) HC = 12 (0:12)	BD = 34.9 (9.1) HC = 33.7 (6.3)	Euthymic BD-I	8 (67%)	Emotional Stroop task	Emotional vs neutral	Implicit emotion perception	HC within, BD within
Malhi et al. 2007a	BD = 10 (0:10) HC = 10 (0:10)	BD = 32.4 (10.8) HC = 31.7 (11.9)	Euthymic BD-I	7 (70%)	Identifying target word	Negative vs neutral, positive vs neutral	Implicit emotion perception	HC>BD, HC within, BD within
Malhi et al. 2007b	BD = 10 (0:10) HC = 10 (0:10)	BD = 33.5 (8.7) HC = 32.4 (6.4)	Euthymic BD-I	7 (70%)	Facial affect recognition task	Negative vs neutral	Emotion identification	BD>HC, HC>BD, HC within, BD within
Marchand et al. 2011	BD = 16 (16:0) HC = 19 (19:0)	BD = 32.9 (7.5) HC = 33.7 (12.5)	Bipolar – II (Depressed)	Not medicated	Facial affect recognition task	Positive vs neutral	Emotion identification	HC>BD, HC within
Mitchell et al. 2004	BD = 11 (11:0) HC = 13 (13:0)	BD = 42.8 (1.8) HC = 32.2 (3.6)	Bipolar – I	9 (81.8%)	Emotional prosody	Emotional vs baseline	Implicit emotion perception	BD>HC, HC>BD, BD within
Morris et al. 2012	BD = 13 (5:8) HC = 15 (6:9)	BD = 41 (3) HC = 35 (2)	Bipolar – I	13 (100%)	Emotional regulation task	Negative vs neutral	Explicit emotion regulation	BD>HC, BD within, HC within
Perlman et al. 2012	BD-R = 31 (10:21) BD-D = 21 (2:19) HC = 25 (11:14)	BD-R = 32.6 (8.2) BD-D = 33.09 (8.38) HC = 31.75 (6.46)	Remitted BD Depressed BD	52 (100%)	Identify color of morphed faces	Emotional vs neutral	Implicit emotion perception	BD>HC
Rai et al. 2021	BD = 40 (16:24) HC = 41 (20:21)	BD = 39.2 (13.89) HC = 37.67 (13.76)	Euthymic BD-I	34 (85%)	Emotional regulation task	Negative vs neutral	Explicit passive viewing	BD>HC, HC>BD
Sepede et al. 2015	BD = 23 (9:14) HC = 24 (8:16)	BD = 35.2 (7.9) HC = 32.5 (6.2)	Euthymic BD-I	19 (82.6%)	Emotional target detection task	Negative vs baseline, negative vs neutral	Emotion identification	BD>HC, HC>BD, main effect across group

Surguladze et al. 2010	BD = 20 (9:11) HC = 20 (10:10)	BD = 42.7 (10.4) HC = 41.9 (11.6)	Bipolar – I	16 (80%)	Gender identification task	Negative vs baseline	Implicit emotion perception	BD within, HC within
Tesli et al. 2015	BD = 85 (33:52) HC = 121 (68:53)	BD = 34.8 (11.2) HC = 35 (8.8)	BD– I, BD-II, BD	85 (100%)	Matching emotional faces	Negative vs neutral, positive vs neutral, emotional vs neutral	Emotion identification	BD>HC, HC>BD
Vizueta et al. 2012	BD = 21 (11:10) HC = 21 (11:10)	BD = 38.4 (12.2) HC = 41.1 (10.9)	Bipolar – II (Depressed)	Not medicated	Emotional face matching task	Negative vs neutral	Emotion identification	HC>BD, HC within, BD within
Wessa et al. 2007	BD = 17 (10:7) HC = 17 (11:6)	BD = 44.94 (12.7) HC = 44.94 (11.36)	Euthymic BD-I and BD-II	15 (88.2%)	Emotional go/no go task	Emotional vs neutral	Emotion identification Explicit passive viewing	BD>HC
Whalley et al. 2009	BD = 14 (9:5) HC = 14 (10:4)	BD = 41.5 (10) HC = 35.4 (8.4)	Bipolar – I	14 (100%)	Perceived emotional intensity	Positive vs neutral	Explicit emotion perception	BD>HC

NA = not available

Table 2. *Study sample characteristics for major depressive disorder (n=52)*

Publication	N (M: F)	Age (M±SD)	Diagnosis	Medication Status	Design/Paradigm	Emotional Contrast	Emotional Component	Contrast(s) Used
Abler et al. 2007	MDD = 12 (0:12) HC = 12 (0:12)	MDD = 41.2 HC = 40.7	MDD	12 (100%)	Anticipation task	Negative vs neutral, positive vs neutral	Explicit passive viewing	MDD>HC
Admon et al. 2015	MDD = 33 (16:17) HC = 35 (19:16)	MDD = 47.4 (1.8) HC = 45.7 (2.7)	MDD	13 (39.3%)	Attention maintaining task	Negative vs baseline	Implicit emotion perception	MDD>HC
Alders et al. 2019	MDD = 48 (15:33) HC = 30 (8:22)	MDD = 34.7 (12.2) HC = 33.2 (9.8)	MDD	Not medicated	Facial affect recognition task	Emotional vs baseline	Emotion identification	HC>MDD
Arnone et al. 2012	MDD-C = 38 (13:25) MDD-R = 24 (3:21) HC = 54 (10:44)	MDD-C = 36.1 (8.8) MDD-R = 33.8 (10.7) HC = 32.4 (9.6)	MDD (current and remitted)	Not medicated	Gender identification task	Negative vs neutral, emotional vs neutral	Implicit emotion perception	HC>MDD, MDD>HC, main effect across group
Aust et al. 2013	MDD = 14 (5:9) HC = 14 (6:8)	MDD = 55.1 (11.3) HC = 54.9 (11.8)	Remitted	10 (71.4%)	Mood provocation task	Negative vs neutral, positive vs neutral	Emotion experience	MDD>HC, HC>MDD
Baeken et al. 2010	MDD = 12 (0:12) HC = 12 (0:12)	MDD = 36 (10.9) HC = 30.2 (8.1)	MDD	Not medicated	Passive view images	Negative vs neutral, positive vs neutral	Explicit passive viewing	HC within, MDD within
Beauregard et al. 1998	MDD = 7 (3:4) HC = 7 (3:4)	MDD = 42 HC = 45	MDD	2 (28.57%)	Passive view clips	Negative vs neutral	Explicit passive viewing	HC within, MDD within
Briceño et al. 2013	MDD = 24 (0:24) HC = 22 (0:22)	MDD = 37.8 (14.5) HC = 31.7 (14.4)	MDD	14 (58.3%)	Facial affect recognition task	Emotional vs neutral	Emotion identification	HC>MDD

Bürger et al. 2017	MDD = 36 (14:22) HC = 36 (17:19)	MDD = 40.72 (11.58) HC = 41.33 (6.05)	Unipolar	36 (100%)	Matching emotional faces	Negative vs neutral, positive vs neutral	Emotion identification	HC>MDD, MDD>HC
Canli et al. 2004	MDD = 15 (3:12) HC = 15 (3:12)	MDD = 35.1 HC = 30.7	MDD	7 (46.7%)	Judgement task	Negative vs neutral, positive vs neutral	Implicit emotion perception	HC>MDD, MDD>HC
Cerullo et al. 2014	MDD = 25 (10:15) HC = 25 (7:18)	MDD = 27 (7) HC = 26 (7)	MDD	Not medicated	Visual oddball paradigm	Negative vs neutral	Emotion identification	MDD>HC, HC>MDD
Chechko et al. 2013	MDD = 18 (5:13) HC = 18 (5:13)	MDD = 36.5 (10.8) HC = 36 (10.3)	MDD	12 (66.7%)	Facial affect recognition/ age identification task	Emotional vs baseline	Emotion identification Implicit emotion perception	HC>MDD, HC within, MDD within
Davis et al. 2018	MDD = 16 (0:16) HC = 20 (0:20)	MDD = 33.7 (10.6) HC = 32.8 (11.8)	MDD	Not medicated	Emotional regulation task	Negative vs neutral	Explicit emotion regulation	MDD>HC
De la Peña-Arteaga et al. 2021	MDD = 20 (6:14) HC = 19 (4:15)	MDD = 49.8 (13.2) HC = 35.8 (10.38)	MDD	Not medicated	Passive viewing images	Negative vs neutral	Explicit passive viewing	MDD>HC
Demenescu et al. 2011	MDD = 59 (20:39) HC = 56 (22:34)	MDD = 36.2 (10.7) HC = 39.7 (9.6)	MDD	14 (23.7%)	Gender identification task	Negative vs neutral, positive vs neutral, emotional vs neutral	Implicit emotion perception	MDD>HC, HC within, MDD within
Derntl et al. 2011	MDD = 15 (6:9) HC = 15 (6:9)	MDD = 34.1 (11.9) HC = 32.9 (10.9)	MDD	13 (86.6%)	Perception of facial expression	Negative vs neutral, positive vs neutral	Explicit emotion perception	MDD>HC
Dichter et al. 2009	MDD = 14 (7:7) HC = 15 (6:9)	MDD = 34.8 (14.3) HC = 30.8 (9.6)	MDD	Not medicated	Emotional oddball task	Negative vs neutral	Implicit emotion perception	HC>MDD, MDD>HC
Elliot et al. 2002	MDD = 10 (3:7) HC = 11 (3:8)	MDD = 42.2 (8.3) HC = 37.6 (9.7)	Unipolar recurrent	10 (100%)	Word targets and distractors	Emotional vs neutral	Emotion identification	HC>MDD, MDD>HC

Epstein et al. 2006	MDD = 10 (1:9) HC = 12 (5:7)	MDD = 35.6 HC = 32	MDD	Not medicated	Read emotional words	Negative vs neutral, positive vs neutral	Implicit emotion perception	HC>MDD, MDD>HC
Erk et al. 2010	MDD = 17 (11:8) HC = 17 (11:8)	MDD = 43.5 (10.9) HC = 43.9 (10.1)	MDD	16 (94.1%)	Passive viewing images	Negative vs neutral	Explicit passive viewing Emotion experience	HC within, MDD within
Farb et al. 2011	MDD = 16 (11:5) HC = 16 (11:5)	MDD = 43.9 (13.3) HC = 39.3 (15.7)	Remitted	16 (100%)	Mood provocation task	Negative vs neutral	Emotion experience	HC>MDD, MDD>HC, HC within, MDD within
Frodl et al. 2009	MDD = 12 (5:7) HC = 12 (5:7)	MDD = 43.3 (11.2) HC = 41.3 (11.7)	MDD	8 (66.67%)	Matching emotional faces/ gender identification task	Negative vs neutral	Emotion identification Implicit emotion perception	HC>MDD, MDD>HC
Fu et al. 2004	MDD = 19 (6:13) HC = 19 (8:11)	MDD = 43.2 (8.8) HC = 42.8 (6.7)	MDD	Not medicated	Gender identification task	Positive vs baseline	Implicit emotion perception	HC>MDD
Fu et al. 2007	MDD = 19 (6:13) HC = 19 (8:11)	MDD = 43.2 (8.8) HC = 42.8 (6.7)	MDD	Not medicated	Gender identification task	Negative vs baseline	Emotion identification	MDD>HC
Gotlib et al. 2005	MDD = 18 (5:13) HC = 18 (5:13)	MDD = 35.2 HC = 30.80	MDD	9 (50%)	Gender identification task	Negative vs neutral, positive vs neutral	Implicit emotion perception	HC>MDD, MDD>HC
Grimm et al. 2009	MDD = 19 (8:11) HC = 29 (8:21)	MDD = 40 HC = 35.2	MDD	Not medicated	Emotional judgement task/ passive view images	Emotional vs baseline	Emotion identification Explicit passive viewing	HC>MDD, MDD>HC
Groves et al. 2018	MDD = 16 (6:10) HC = 10 (6:4)	MDD = 35.4 (12.7) HC = 35.1 (12.1)	MDD or BD-II (Depressed)	Not medicated	Emotion categorization task	Negative vs neutral	Emotion identification	MDD>HC
Hooley et al. 2009	MDD = 11 (0:11) HC = 12 (0:12)	MDD = 24.45 (3) HC = 24.33 (2.4)	Remitted	5 (45.45%)	Auditory mood provocation task	Negative vs baseline, positive vs baseline	Emotion experience	HC>MDD, MDD>HC, HC within, MDD within

Huang et al. 2019	MDD = 55 (17:38) HC = 40 (15:25)	MDD = 66.3 (5.42) HC = 68.10 (5.3)	MDD (Late-diagnosis)	55 (100%)	Emotional Stroop task	Emotional vs neutral	Implicit emotion perception	MDD>HC, HC>MDD
Keedwell et al. 2005	MDD = 12 (4.8) HC = 12 (4:8)	MDD = 43 (9.8) HC = 36 (14.6)	MDD	11 (91.67%)	Mood provocation task	Negative vs neutral, positive vs neutral	Emotion experience	HC>MDD, MDD>HC
Kumari et al. 2003	MDD = 6 (0.6) HC = 6 (0:6)	MDD = 47 (3.59) HC = 44 (2.44)	MDD	6 (100%)	Viewing emotional picture-caption pair	Negative vs neutral, positive vs neutral	Explicit passive viewing Emotion experience	HC>MDD, MDD>HC, HC within, MDD within
Lisiecka et al. 2013	MDD = 50 (18:32) HC = 46 (22:24)	MDD = 42.7 (10.5) HC = 37.35 (13.14)	MDD	37 (74%)	Emotional valence recognition	Emotional vs baseline	Emotion identification	MDD>HC, HC>MDD
Mitterschiffthaler et al. 2003	MDD = 7 (0:7) HC = 7 (0:7)	MDD = 46.3 (8.1) HC = 48.3 (10.1)	MDD	7 (100%)	Attention maintaining task	Positive vs neutral	Implicit emotion perception	HC>MDD, MDD>HC, HC within, MDD within
Mitterschiffthaler et al. 2008	MDD = 17 (3:13) HC = 17 (3:14)	MDD = 39.3 (9.4) HC = 39.4 (9.2)	MDD	Not medicated	Emotional Stroop task	Negative vs neutral	Implicit emotion perception	MDD>HC, HC within, MDD within
Murrough et al. 2015	MDD = 18 (10:8) HC = 20 (11:9)	MDD = 38.1 (13.8) HC = 35 (8.9)	MDD	Not medicated	Mood provocation task	Positive vs neutral	Emotion experience	HC>MDD
Radke et al. 2018	MDD = 22 (13:9) HC = 22 (13:9)	MDD = 32.6 (10.9) HC = 34.5 (9.9)	MDD	15 (68.2%)	Passive viewing images	Negative vs baseline	Explicit passive viewing	HC>MDD
Rai et al. 2021	MDD = 39 (19:20) HC = 41 (20:21)	MDD = 36.7 (12.2) HC = 37.67 (13.76)	MDD	13 (33.3%)	Emotion regulation task/ passive viewing	Negative vs neutral	Explicit emotion regulation Explicit passive viewing	MDD>HC
Regenbogen et al. 2015	MDD = 24 (13:11) HC = 24 (13:11)	MDD = 36.42 (12) HC = 35.25 (9.8)	MDD	24 (100%)	Speech and facial emotion identification task	Emotional vs neutral	Emotion identification	HC>MDD, MDD>HC, HC within, MDD within

Ritchey et al. 2011	MDD = 22 (9:13) HC = 14 (5:9)	MDD = 36.1 (10.1) HC = 34.6 (6.9)	MDD	Not medicated	Mood provocation task	Emotional vs neutral	Emotion experience Emotion identification	HC>MDD
Rizvi et al. 2013	MDD = 21 (7:14) HC = 18 (6:12)	MDD = 38.9 (11.4) HC = 36.2 (10.3)	MDD	Not medicated	Judgement task	Negative vs neutral, positive vs neutral	Implicit emotion perception	MDD>HC
Scheuerecker et al. 2010	MDD = 13 (10:3) HC = 15 (10:5)	MDD = 37.9 (10.1) HC = 35.5 (10.9)	MDD	Not medicated	Emotional face matching/ gender identification task	Negative vs neutral	Emotion identification Implicit emotion perception	MDD>HC
Schlund et al. 2012	MDD = 10 (4:6) HC = 10 (5:5)	MDD = 46.7 (7.2) HC = 40 (10.2)	MDD/GAD	Not medicated	Facial affect and emotional statement recognition task	Negative vs baseline, positive vs baseline	Explicit emotion regulation Explicit passive viewing	MDD>HC, HC>MDD
Seidel et al. 2012	MDD = 15 (6:9) HC = 15 (6:9)	MDD = 34.13 (11.9) HC = 32.87 (10.9)	MDD	13 (86.7%)	Causal attribution task	Emotional vs baseline	Emotion identification	HC>MDD
Stephanou et al. 2017	MDD = 53 (22:31) HC = 64 (24:40)	MDD = 19.7 (2.7) HC = 19.03 (2.45)	MDD	Not medicated	Emotional regulation task	Negative vs neutral	Explicit emotion regulation	MDD>HC, HC>MDD, HC within, MDD within
Surguladze et al. 2005	MDD = 16 (10:6) HC = 14 (8:6)	MDD = 42.3 (8.4) HC = 35.1 (13.2)	MDD	NA	Gender identification task	Negative vs baseline, positive vs baseline	Implicit emotion perception	HC>MDD, MDD>HC
Surguladze et al. 2010	MDD = 9 (5:4) HC = 9 (5:4)	MDD = 42.8 (7.2) HC = 39.7 (14.6)	MDD	9 (100%)	Facial affect recognition task	Negative vs neutral	Emotion identification	HC>MDD, MDD>HC, HC within, MDD within
Townsend et al. 2010	MDD = 15 (9:6) HC = 15 (9:6)	MDD = 45.6 (11.2) HC = 44.8 (11.7)	MDD	Not medicated	Matching emotional faces	Negative vs neutral	Emotion identification	HC>MDD, HC within, MDD within

Trettin et al. 2022	MDD = 36 (15:21) HC = 30 (10:20)	MDD = 40.71 (12.7) HC = 39.47 (13.19)	MDD	36 (100%)	Judgement task	Negative vs neutral	Implicit emotion perception	HC within, MDD within
Van Kleef et al. 2022	MDD = 46 (10:36) HC = 24 (6:18)	MDD = 35.11 (11.32) HC = 36.67 (12.58)	Remitted	Not medicated	Passive viewing task	Negative vs baseline, positive vs baseline	Explicit passive viewing	HC>MDD
Wang et al. 2008	MDD = 19 (7:12) HC = 20 (7:13)	MDD = 39.3 (9) HC = 36.5 (10.5)	MDD	8 (57.9%)	Emotional oddball task	Neutral vs negative	Implicit emotion perception	HC within, MDD within
Wang et al. 2012	MDD = 18 (7:11) HC = 18 (7:11)	MDD = 31.61 (7.88) HC = 31.67 (6.82)	MDD	Not medicated	Mood provocation task	Negative vs baseline, positive vs baseline	Emotion experience	HC>MDD, MDD>HC
Zhong et al. 2011	MDD = 29 (13:16) HC = 31 (15:16)	MDD = 20.4 (1.82) HC = 20.8 (1.49)	MDD	Not medicated	Emotional face matching task	Negative vs neutral	Emotion identification	HC>MDD, MDD>HC, HC within, MDD within

NA = not available

Table 3. *Study sample characteristics for schizophrenia (n=35)*

Publication	N (M: F)	Age (M±SD)	Diagnosis	Medication Status	Design/Paradigm	Emotional Contrast	Emotional Component	Contrast(s) Used
Ciaramidaro et al. 2018	SCZ = 20 (14:6) HC = 25 (21:4)	SCZ = 24.7 (5.03) HC = 19.68 (3.45)	SCZ (acute episode of paranoid)	12 (60%)	Facial affect recognition task	Negative vs neutral	Emotion identification Implicit emotion perception	HC>SCZ, HC within, SCZ within
Derntl et al. 2012	SCZ = 15 (10:5) HC = 15 (10:5)	SCZ = 34.2 (9.1) HC = 30.4 (8.9)	SCZ	15 (100%)	Emotion recognition task Label emotion Perceived emotion	Emotional vs neutral, emotional vs baseline	Emotion identification Explicit emotion perception	HC>SCZ, main effect across group
Fakra et al. 2008	SCZ = 14 (9:5) HC = 14 (9:5)	SCZ = 37.29 (8.8) HC = 34.64 (5.96)	SCZ	14 (100%)	Matching and labeling emotional faces	Negative vs neutral	Emotion identification	SCZ>HC, HC>SCZ, HC within, SCZ within
Ferri et al. 2014	SCZ = 22 (14:8) HC = 22 (12:10)	SCZ = 27.45 (5.07) HC = 28 (3.77)	SCZ (first episode psychosis)	22 (100%)	Emotion action task	Negative vs neutral, positive vs neutral, negative vs baseline	Explicit passive viewing	HC>SCZ, HC within, SCZ within
Habel et al. 2010a	SCZ = 14 (14:0) HC = 14 (14:0)	SCZ = 37.14 (8.81) HC = 35.5 (9.31)	SCZ	12 (85.7%)	Olfactory stimulation n-back task	Negative vs neutral	Implicit emotion perception	SCZ>HC, HC>SCZ
Habel et al. 2010b	SCZ = 17 HC = 17	SCZ = 34.4 (8.8) HC = 34.2 (7.7)	SCZ	16 (94.1%)	Emotion identification task	Negative vs baseline, positive vs baseline	Emotion identification	SCZ>HC, HC>SCZ
Hall et al. 2008	SCZ = 19 (12:7) HC = 24 (16:8)	SCZ = 37.7 (8.4) HC = 35.1 (9.7)	SCZ	19 (100%)	Gender identification task	Negative vs neutral	Implicit emotion perception	HC>SCZ
Holt et al. 2011	SCZ = 14 (11:3) HC = 18 (14:4)	SCZ = 42.9 (11.9) HC = 43.7 (8.3)	SCZ	14 (100%)	Emotion identification task	Negative vs neutral, positive vs neutral	Emotion identification	HC>SCZ, HC within, SCZ within

Horan et al. 2014	SCZ = 23 (17:6) HC = 23 (16:7)	SCZ = 46.5 (11.1) HC = 46.7 (6.9)	SCZ	23 (100%)	Action imitation & observation task	Emotional vs neutral, emotional vs baseline	Explicit passive viewing	HC within, SCZ within
Jimenez et al. 2018	SCZ = 20 (13:7) HC = 16 (11:5)	SCZ = 48.3 (9.9) HC = 44.69 (11.1)	SCZ	18 (90%)	Emotion identification task	Emotional vs baseline	Emotion identification	HC>SCZ, HC within, SCZ within
Johnston et al. 2005	SCZ = 10 (8:2) HC = 10 (8:2)	SCZ = 30.6 (8.6) HC = 31.2 (7.9)	SCZ	10 (100%)	Facial affect recognition task	Emotional vs neutral	Emotion identification	HC>SCZ, HC within, SCZ within
Kang et al. 2009	SCZ-H = 14 (7:7) SCZ-N = 14 (7:7) HC = 28 (14:14)	SCZ-H = 29.5 (4.1) SCZ-N = 30.4 (3.7) HC =29.9 (2.9)	SCZ (hallucinator and non-hallucinator)	28 (100%)	Gender identification task	Negative vs neutral, positive vs neutral	Implicit emotion perception	SCZ>HC, HC>SCZ, HC within, SCZ within
Kim et al. 2015	SCZ = 15 (7:8) HC = 15 (7:8)	SCZ = 28.4 (8) HC = 29.7 (5.3)	SCZ	15 (100%)	Implicit memory task	Negative vs baseline	Implicit emotion perception	HC>SCZ, HC within, SCZ within
Knobloch et al. 2024	SCZ = 22 (17:5) HC = 22 (18:4)	SCZ = 34.4 (11.5) HC = 30.2 (10.9)	SCZ	22 (100%)	Affective sharing task	Emotional vs baseline	Explicit emotion perception/ emotion experience	HC>SCZ
Lakis & Mendrek 2013	SCZ = 37 (19:18) HC = 37 (19:18)	SCZ = 32.46 (7.66) HC = 31.81 (6.91)	SCZ	37 (100%)	Passive view images	Negative vs neutral, positive vs neutral	Explicit passive viewing	HC>SCZ
Lee et al. 2014	SCZ = 15 (9:6) HC = 16 (10:6)	SCZ = 36.7 (8.1) HC = 36.8 (6.3)	SCZ	15 (100%)	Facial expression task	Emotional vs neutral	Emotion identification	SCZ>HC, HC>SCZ
Leitman et al. 2011	SCZ = 23 (15:8) HC = 28 (22:6)	SCZ = 34.1 (10.7) HC = 33.6 (9.2)	SCZ	23 (100%)	Prosody recognition task	Emotional vs neutral	Emotion identification	SCZ>HC, HC>SCZ
Lepage et al. 2011	SCZ = 26 (15:11) HC = 26 (12:14)	SCZ = 31.8 (7.7) HC = 28.3 (5.6)	SCZ	22 (84.6%)	Gender identification task	Emotional vs neutral	Implicit emotion perception	HC>SCZ, main effect across group

Li et al. 2012	SCZ = 12 (6:6) HC = 12 (6:6)	SCZ = 29.8 (9.24) HC = 29.25 (7.24)	SCZ	12 (100%)	Facial emotional valence identification	Negative vs neutral, positive vs neutral	Emotion identification	SCZ>HC, HC>SCZ
Lindner et al. 2014	SCZ = 36 (22:14) HC = 40 (27:13)	SCZ = 30.8 (7.9) HC = 29.5 (8.3)	SCZ	36 (100%)	Passive view images	Negative vs neutral	Explicit passive viewing	SCZ>HC, HC>SCZ
Luck et al. 2015	SCZ = 18 (16:2) HC = 20 (14:6)	SCZ = 23 (0.96) HC = 23.75 (0.66)	SCZ	15 (83.3%)	Emotional encoding	Emotional vs neutral, neutral vs emotional	Emotion identification	SCZ>HC, HC>SCZ, HC within
Mendrek et al. 2012	SCZ = 22 (0:22) HC = 25 (0:25)	SCZ = 32.86 (6.56) HC = 29.38 (9.27)	SCZ	22 (100%)	Person identification task	Negative vs neutral, positive vs neutral	Implicit emotion perception	SCZ>HC, HC>SCZ, HC within, SCZ within
Michalopoulou et al 2008	SCZ = 11 (9:2) HC = 9 (5:4)	SCZ = 35 (9) HC = 32 (6)	SCZ	11 (100%)	Gender identification task	Negative vs neutral	Implicit emotion perception	HC>SCZ, HC within, SCZ within
Mier et al. 2014	SCZ = 11 (7:4) HC = 16 (11:5)	SCZ = 32.45 (7.6) HC = 34.5 (6.47)	SCZ	11 (100%)	Facial affect recognition task	Emotional vs neutral	Emotion identification	SCZ>HC, HC>SCZ
Mitchell et al. 2004	SCZ = 12 (12:0) HC = 13 (13:0)	SCZ = 45.7 (2.7) HC = 32.2 (3.6)	SCZ	12 (100%)	Emotional prosody	Emotional vs baseline	Implicit emotion perception	SCZ>HC, HC>SCZ, SCZ within
Morris et al. 2012	SCZ = 12 (8:4) HC = 15 (6:9)	SCZ = 44 (3) HC = 35 (2)	SCZ	9 (75%)	Emotion regulation task	Negative vs neutral	Explicit emotion regulation	SCZ>HC, HC>SCZ
Park et al. 2009	SCZ = 15 HC = 16	NA	SCZ	15 (100%)	Attribution task	Negative vs neutral, positive vs neutral	Explicit emotion perception	SCZ>HC, HC>SCZ
Park et al. 2019	SCZ = 17 (9:8) HC = 17 (9:8)	SCZ = 31.1 (9) HC = 32.8 (8.6)	SCZ	17 (100%)	Face recognition task	Negative vs neutral	Emotion identification	SCZ>HC, HC>SCZ, HC within, SCZ within
Regenbogen et al. 2015	SCZ = 20 (13:7) HC = 24 (13:11)	SCZ = 37.3 (8.44) HC = 35.25 (9.8)	SCZ	20 (100%)	Speech and facial emotion identification task	Emotional vs neutral	Emotion identification	SCZ>HC, HC>SCZ, HC within, SCZ within

Shin et al. 2015	SCZ = 16 (16:0) HC = 15 (15:0)	SCZ = 32 (7.8) HC = 32 (8.1)	SCZ	16 (100%)	Gender identification task	Negative vs baseline	Implicit emotion perception	SCZ>HC
Spilka et al. 2015	SCZ = 28 (15:13) HC = 27 (13:14)	SCZ = 41.07 (11.15) HC = 40.7 (11.1)	SCZ	28 (100%)	Passive viewing facial emotions task	Negative vs scrambled, negative vs neutral	Explicit passive viewing	HC>SCZ, HC within, SCZ within
Szabó et al. 2017	SCZ = 19 (11:8) HC = 18 (12:6)	SCZ = 37.4 (8.4) HC = 37.6 (10.9)	SCZ	19 (100%)	Facial affect recognition task	Negative vs neutral	Emotion identification	HC>SCZ
Takahashi et al. 2004	SCZ = 15 (10:5) HC = 15 (9:6)	SCZ = 29 (6.9) HC = 29.1 (7.8)	SCZ	11 (73.3%)	Mood provocation task	Negative vs neutral	Emotion experience	HC>SCZ, HC within, SCZ within
Whalley et al. 2009	SCZ = 15 (11:4) HC = 14 (10:4)	SCZ = 38.4 (10.2) HC = 35.4 (8.4)	SCZ	15 (100%)	Mood provocation task	Positive vs baseline	Explicit emotion perception	HC>SCZ
Williams et al. 2004	SCZ = 27 (17:10) HC = 22 (14:8)	SCZ = 27.3 (9.6) HC = 27.2 (8.1)	SCZ	27 (100%)	Gender identification task	Negative vs neutral	Implicit emotion perception	HC>SCZ, HC within

NA = not available

Table 4. *Brain Regions Showing Significant Convergence of Activity in Emotional Processing for Healthy Controls within the Studies investigating Bipolar Disorder and the Studies Contributing to the Clusters of Convergence*

Cluster/Macroanatomical Structure	**Cytoarchitectonic Assignment**	**Cluster Size (voxel)**	**MNI Coordinates**			**Z_{max}**	**Studies Contributing**	**Contribution (%)**
			x	**y**	**z**			
L Lateral Occipital Cortex, inferior division	L area hOc4lp	112	-30	-90	+0	4.84	Dima et al. 2016 Lagopoulos et al. 2011 Surguladze et al. 2010 Marchand et al. 2011 Sepede et al. 2015 Han et al. 2018 Keener et al. 2012	26.82 20.81 20.43 18.76 12.48 0.48 0.19
L Amygdala	L amygdala (CM)	109	-22	-2	-16	5.69	Kjærstad et al. 2023 Dima et al. 2016 Vizueta et al. 2012 Keener et al. 2012 Lagopoulos et al. 2007 Malhi et al. 2007	26.81 25.14 24.73 19.63 2.25 1.40

Note. L = left hemisphere, R = right hemisphere. Zmax = maximum z-score of the local maxima

Table 5. *Brain Regions Showing Significant Convergence of Activity in Emotional Processing for Healthy Controls within the Studies investigating Major Depressive Disorder and the Studies Contributing to the Clusters of Convergence*

Cluster/Macroanatomical Structure	Cytoarchitectonic Assignment	Cluster Size (voxel)	MNI Coordinates			Z_{max}	Studies Contributing	Contribution (%)
			x	y	z			
R Fusiform Gyrus	R area FG4	174	+38	-48	-20	6.04	Demenescu et al. 2011	28.92
							Surguladze et al. 2010	23.13
							Chechko et al. 2013	19.16
							Farb et al. 2011	17.99
							Arnone et al. 2012	10.42
							Baeken et al. 2010	0.38
L Fusiform Gyrus	L area FG4	165	-40	-60	-18	4.5	Demenescu et al. 2011	21.71
							Stephanou et al. 2017	19.88
							Beauregard et al. 1998	14.39
							Erk et al. 2010	12.91
							Farb et al. 2011	11.51
			-42	-52	-16	4.33	Arnone et al. 2012	8.74
							Surguladze et al. 2010	5.43
							Chechko et al. 2013	5.42
R Amygdala	R amygdala (LB)	159	+24	-4	-16	5.24	Stephanou et al. 2017	21.37
							Townsend et al. 2010	19.86
							Arnone et al. 2012	15.90
							Farb et al. 2011	15.19
							Trettin et al. 2022	14.75
	-		+24	-14	-8	3.18	Erk et al. 2010	6.90
							Regenbogen et al. 2015	5.33
							Briceño et al. 2013	0.69

L Amygdala	L amygdala (MF)	136	-20	-6	-14	5.57	Demenescu et al. 2011 Arnone et al. 2012 Townsend et al. 2010 Trettin et al. 2022 Surguladze et al. 2010 Farb et al. 2011 Briceño et al. 2013	22.72 21.45 20.85 16.34 15.43 3.00 0.19
L Frontal Pole L Paracingulate Gyrus	L area p32	98	-4	+56	+12	3.96	Tozzi et al. 2017 Trettin et al. 2022 Hooley et al. 2009 Beauregard et al. 1998 Regenbogen et al. 2015 Surguladze et al. 2010	37.91 21.62 19.86 16.50 3.94 0.11
L Frontal Pole L Superior Frontal Gyrus	-		-2	+58	+24	3.96		

Note. L = left hemisphere, R = right hemisphere. Zmax = maximum z-score of the local maxima

Table 6. *Brain Regions Showing Significant Convergence of Activity in Emotional Processing for Healthy Controls within the Studies investigating Schizophrenia and the Studies Contributing to the Clusters of Convergence*

Cluster/Macroanatomical Structure	Cytoarchitectonic Assignment	Cluster Size (voxel)	MNI Coordinates			Z_{max}	Studies Contributing	Contribution (%)
			x	y	z			
R Amygdala	R amygdala (LB)	328	+24	-6	-16	6.9	Holt et al. 2011	12.71
							Park et al. 2019	12.24
							Ciaramidaro et al. 2018	10.45
							Lepage et al. 2011	9.61
							Williams et al. 2004	9.60
							Jimenez et al. 2018	8.04
							Johnston et al. 2005	7.57
							Michalopoulou et al. 2008	7.38
							Mendrek et al. 2012	6.97
							Fakra et al. 2008	6.96
							Regenbogen et al. 2015	4.74
							Takahashi et al. 2004	3.63
R Inferior Frontal Junction	-	298	+50	+6	+38	5.29	Horan et al. 2014	28.93
							Mendrek et al. 2012	16.85
	-		+48	+12	+32	4.58	Jimenez et al. 2018	10.55
							Fakra et al. 2008	9.86
	-		+46	+14	+26	4.32	Kang et al. 2009	9.48
							Ferri et al. 2014	9.22
	-		+46	+14	+18	3.57	Ciaramidaro et al. 2018	7.13
							Williams et al. 2004	4.24
	R area 44		+48	+12	+20	3.55	Park et al. 2019	2.64
							Johnston et al. 2005	1.09

L Insular Cortex L Frontal Orbital Cortex	L area Id7	159	-32	+24	+2	4.77	Mendrek et al. 2012 Lepage et al. 2011 Spilka et al. 2015 Johnston et al. (2005) Kang et al. 2009 Fakra et al. 2008 Kim et al. 2015 Derntl et al. 2012 Ciaramidaro et al. 2018	16.43 15.00 14.60 13.67 12.18 12.11 9.42 6.39 0.16
R Lateral Occipital Cortex, inferior division	R area hOc4la	145	+48 +42	-74 -80	-6 -4	4.93 4.5	Mendrek et al. 2012 Kim et al. 2015 Park et al. 2019 Regenbogen et al. 2015 Michalopoulou et al. 2008 Luck et al. 2015 Johnston et al. 2005 Jimenez et al. 2018 Takahashi et al. 2004	31.69 15.41 13.49 13.02 12.50 11.56 1.97 0.18 0.13
L Inferior Frontal Junction	L area 44	124	-38	+8	+28	4.55	Horan et al. 2014 Kim et al. 2015 Johnston et al. 2005 Ferri et al. 2014 Lepage et al. 2011 Mendrek et al. 2012 Leitman et al. 2011 Kang et al. 2009 Regenbogen et al. 2015	27.20 22.45 17.92 17.26 7.15 5.00 2.60 0.28 0.10

R Lingual Gyrus R Intracalcarine Cortex	R area hOc1 [V1]	112	+4 +2	-78 -88	+2 -2	4.53 3.73	Ciaramidaro et al. 2018 Spilka et al. 2015 Horan et al. 2014 Mendrek et al. 2012 Kang et al. 2009 Lepage et al. 2011 Takahashi et al. 2004	24.48 19.47 18.22 14.64 13.81 5.11 4.25
R Fusiform Gyrus	R area FG4	104	+40	-44	-22	4.39	Horan et al. 2014 Ciaramidaro et al. 2018 Johnston et al. 2005 Spilka et al. 2015 Park et al. 2019 Luck et al. 2015 Mendrek et al. 2012	23.38 19.66 18.19 14.33 10.32 9.64 4.49
L Occipital Pole L Intracalcarine Cortex	L area hOc1 [V1]		-10	-94	+0	3.99	Mendrek et al. 2012 Johnston et al. 2005 Horan et al. 2014	27.80 16.44 15.39
L Lateral Occipital Cortex	L area hOc3v [V3v]	97	-20	-96	-2	3.6	Takahashi et al. 2004 Ferri et al. 2014 Kang et al. 2009	14.94 10.44 10.18
L Lateral Occipital Cortex	L area hOc4lp		-24	-96	+8	3.46	Regenbogen et al. 2015 Holt et al. 2011	4.58 0.12

Note. L = left hemisphere, R = right hemisphere. Zmax = maximum z-score of the local maxima

Table 7. *Brain Regions Showing Convergent Significant Differences in Activity in Emotional Processing for Major Depressive Disorder Compared to Healthy Controls and the Studies Contributing to the Clusters of Convergence*

Cluster/Macroanatomical Structure	**Cytoarchitectonic Assignment**	**Cluster Size (voxel)**	**MNI Coordinates**			**Z_{max}**	**Studies Contributing**	**Contribution (%)**
			x	**y**	**z**			
R Lingual Gyrus R Intracalcarine Cortex	R area hOc1 [V1]	91	+12	-60	+6	4.88	Van Kleef et al. 2022 Townsend et al. 2010 Hooley et al. 2009 Schlund et al. 2012 Rai et al. 2021 Briceño et al. 2013 Derntl et al. 2011	32.04 25.59 22.45 17.51 1.23 1.06 0.002

Note. L = left hemisphere, R = right hemisphere. Zmax = maximum z-score of the local maxima

Table 8. *Brain Regions Showing Convergent Significant Differences in Activity in Emotional Processing for Schizophrenia Compared to Healthy Controls and the Studies Contributing to the Clusters of Convergence*

Cluster/Macroanatomical Structure	Cytoarchitectonic Assignment	Cluster Size (voxel)	MNI Coordinates			Z_{max}	Studies Contributing	Contribution (%)
			x	y	z			
R Amygdala	R amygdala (IF)	132	+26	-4	-16	5.04	Kang et al. 2009	18.68
							Takahashi et al. 2004	18.43
							Williams et al. 2004	16.78
							Michalopoulou et al. 2008	13.84
							Fakra et al. 2008	9.42
							Lakis & Mendrek, 2013	8.65
	R amygdala (LB)		+30	-2	-24	3.55	Whalley et al. 2009	7.08
							Lindner et al. 2014	5.58
							Park et al. 2009	1.01
							Lee et al. 2014	0.51

Note. L = left hemisphere, R = right hemisphere. Zmax = maximum z-score of the local maxima

Table 9. *Brain Regions Showing Convergent Significant Differences in Activity in Emotional Processing for Pooled Data Compared to Healthy Controls and the Studies Contributing to the Clusters of Convergence*

Cluster/Macroanatomical Structure	Cytoarchitectonic Assignment	Cluster Size (voxel)	MNI Coordinates			Z_{max}	Studies Contributing	Contribution (%)
			x	y	z			
R Amygdala	R amygdala (IF)	170	+26	-4	-16	6.03	Vizueta et al. 2012	11.31
							Takahashi et al. 2004	10.58
							Canli et al. 2004	10.41
							Kang et al. 2009	9.98
							Williams et al. 2004	9.49
							Michalopoulou et al. 2008	9.20
							Han et al. 2018	9.20
							Fakra et al. 2008	7.50
							Ritchey et al. 2011	7.41
							Lakis & Mendrek, 2013	3.67
							Whalley et al. 2009	3.27
							Rai et al. 2021	1.94
	R amygdala (LB)		+30	+0	-24	3.36	Lindner et al. 2014	1.77
							Chen et al. 2006	1.28
							Park et al. 2009	0.80
							Lee et al. 2014	0.56
							Grimm et al. 2009	0.51
							Perlman et al. 2012	0.38
							Briceño et al. 2013	0.24
							Lagopoulos et al. 2007	0.23
							Keener et al. 2012	0.15
							Kumari et al. 2003	0.14
							Elliot et al. 2002	0.13

							Arnone et al. 2012	13.21
							Cerullo et al. 2014	12.96
							Leitman et al. 2011	11.77
							Vizueta et al. 2012	11.01
							Chen et al. 2006	9.18
							Fakra et al. 2008	8.50
							Scheuerecker et al. 2010	6.86
							Holt et al. 2011	6.65
							Frodl et al. 2009	5.56
L IFJ	-	138	-42	-2	+36	5.33	Malhi et al. 2007	5.19
							Keedwell et al. 2005	3.26
							Rai et al. 2021	3.21
							Habel et al. 2010	1.87
							Schlund et al. 2012	0.48
							Li et al. 2012	0.37
							Huang et al. 2019	0.31
							Grimm et al. 2009	0.17
							Whalley et al. 2009	0.15
							Jogia et al. 2008	0.12

Note. L = left hemisphere, R = right hemisphere. Zmax = maximum z-score of the local maxima

Table 10. *Descriptive Characteristics of the Studies Contributing to the Clusters of Convergence in Emotional Processing for Pooled Data Compared to Healthy Controls*

	Diagnosis (n)	**Contribution (n%)**	**Emotional Component (n)**	**Hyper/Hypo Activation (n)**
Right Amygdala	Bipolar disorder (6)	22.59	Emotion identification (2) Explicit passive viewing (1) Implicit emotion perception (3)	Control > Patient (1) Patient > Control (4) Both (1)
	Major depressive disorder (7)	20.57	Emotion identification (3) Explicit passive viewing (3) Emotion experience (2) Implicit emotion perception (2)	Control > Patient (1) Patient > Control (2) Both (4)
	Schizophrenia (10)	56.84	Emotion identification (2) Explicit passive viewing (2) Explicit emotion perception (3) Implicit emotion perception (3)	Control > Patient (5) Both (5)
Left IFG	Bipolar disorder (6)	38.62	Emotion identification (4) Explicit emotion perception (1) Implicit emotion perception (1)	Control > Patient (3) Patient > Control (2) Both (1)
	Major depressive disorder (8)	33.22	Emotion identification (4) Explicit passive viewing (1) Emotion experience (1) Explicit emotion regulation (1) Implicit emotion perception (3)	Control > Patient (1) Patient > Control (2) Both (5)
	Schizophrenia (5)	28.15	Emotion identification (4) Implicit emotion perception (1)	Control > Patient (3) Both (2)

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