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Review article

Explicit emotional memory biases in mood disorders: A systematic review

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ABSTRACT

Research suggests that major depressive disorder (MDD) and bipolar disorder (BD) are both associated with unique emotional memory (EM) biases. To better elucidate the EM phenotypes of these disorders, we systematically reviewed the literature on non-autobiographical explicit EM biases in individuals with MDD and BD compared to healthy controls. The following databases were searched: Cochrane, Embase, HAPI, LILACs, Medline, PsycInfo and Web of Science. Grey literature and hand searches were also performed. Fourteen studies met full eligibility criteria. Eleven studies included data from an MDD sample (10 during acute depression, 1 during euthymia) and 3 studies included data from a BD sample (2 during acute mood episodes, 1 during euthymia). Only 3 of the studies in acute depression revealed a negative explicit EM bias. One study in MDD during euthymia revealed an EM deficit for negative stimuli. One of the two studies in BD (type I; BD-I) during an acute mood episode revealed a positive explicit EM bias, while the other showed no bias. One study in BD during euthymia showed an EM deficit for negative stimuli. Overall, this review concludes that current empirical evidence does not readily support the existence of an explicit EM bias in MDD during acute depression. The identification and implications of potential moderating factors on explicit EM performance in MDD and BD during both illness stages are discussed.

1. Introduction

Major depressive disorder (MDD) and bipolar disorder (BD) are chronic, disabling conditions characterized by abnormal changes in mood state (American Psychiatric Association, 2013; Goldstein, 2010). MDD and BD together affect approximately 300 million people worldwide (American Psychiatric Association, 2013; Goldstein, 2010; World Health Organization, 2017). MDD is defined as a disorder consisting of recurrent major depressive episodes, which may include extended periods of low mood and/or loss of pleasure or interest in daily activities, changes in appetite, sleep disturbances, changes in psychomotor activity, feelings of worthlessness, decreased cognitive abilities and/or suicidal ideation (American Psychiatric Association, 2013). BD is associated with both major depressive episodes and either mania (type I BD; BD-I) or hypomania (type II BD; BD-II; American Psychiatric Association, 2013; Goldstein, 2010). Mania is characterized by heightened or irritable mood, with increased self-esteem, decreased sleep, racing thoughts, increased distractibility, increased participation in goal-directed activities and/or increased participation in potentially harmful activities (American Psychiatric Association, 2013; Goldstein, 2010). Hypomania is characterized by the same symptoms as mania, but the symptoms are typically less severe and, by definition, the hypomanic episodes do not cause marked functional impairment (American Psychiatric Association, 2013).

Disturbances in several cognitive domains have been associated with MDD and BD, including changes in attention, memory, planning, and verbal fluency (Bora and Pantelis, 2015; Marvel and Paradiso, 2004; Rock et al., 2013). A specific cognitive feature suggested to be associated with both disorders is the presence of an emotional memory (EM) bias. The conceptualization of an EM bias in existing literature is

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ambiguous. In this systematic review, we operationally define an EM bias as the tendency for individuals with MDD and BD to *more* accurately remember information of a particular valence (i.e., positive or negative) compared against healthy controls (HCs; LaBar and Cabeza, 2006; LaBar and Phelps, 1998). On the other hand, the tendency for individuals with MDD and BD to *less* accurately remember information of a particular valence (i.e., positive or negative) compared against HCs is characterized here as an EM deficit. In this way, EM performance in HCs represents the behavioural phenotype expected under normal physiological conditions and therefore represents an estimate of baseline performance to which performance in the clinical groups can be compared. Previous studies have consistently shown that HCs display more accurate memory for both emotionally-positive and emotionally-negative information, compared to emotionally-neutral information (Asl et al., 2015; Cahill and McGaugh, 1995; Flaisch et al., 2016; Williams et al., 2015). This may be explained by the observation that emotional salience is more likely to signify an event that is relevant to survival (Cahill and McGaugh, 1995; Flaisch et al., 2016). It is thus conceivable that enhanced memory for emotional stimuli (positive and negative) is an evolved adaptation to promote human survival (Hamann, 2001). Cognitive neuroscience research has implicated the amygdala as a key player in the formation of emotional memories (LaBar and Cabeza, 2006; LaBar and Phelps, 1998; LeDoux, 1993). It is widely believed that, upon exposure to emotionally-arousing stimuli, increased activation of the amygdala leads to the modulation of visual cortex, prefrontal cortex, and hippocampal activity through the recruitment of stress hormones (i.e., norepinephrine) and corticosteroids (Hamann, 2001; Kensinger and Corkin, 2004; LaBar and Cabeza, 2006; Turkileri and Sakaki, 2017). This modulatory interaction between the amygdala and other brain regions critical for memory formation consequently results in more efficient memory encoding and retrieval for emotional information. Despite this, research supports the existence of unique EM biases in individuals diagnosed with a mood disorder, such as MDD and BD (Leppänen, 2006). Therefore, there may be a malfunction in this evolved adaptation in mood disorders that is responsible for manifesting unique EM biases, or the lack thereof (Durisko et al., 2016; Wakefield, 1992).

Considerable research has explored the existence of a mood-congruent negative EM bias in depression (Matt et al., 1992; Watkins et al., 1992; Wittekind et al., 2014). A mood-congruent bias refers to the more accurate memory retrieval of information consistent with one's current emotional state (Moritz et al., 2005). Several theories have hypothesized that depressed individuals show a proclivity towards remembering negative emotional stimuli (Beck, 1979; Bower, 1981; Matt et al., 1992; Williams et al., 1988). For example, the cognitive model of depression, which has been informed by decades of clinical, cognitive, biological and evolutionary research (e.g., Beck, 1974, Bylsma et al., 2008; Beck and Bredemeier, 2016), posits that individuals suffering from depression may experience a systemic negative cognitive bias across all levels of information processing, including emotional reactivity and memory (Beck and Bredemeier, 2016). When exposed to a stressor, cognitive structures, called *schemas*, within which beliefs are set, become differentially activated depending on the stimulus' intensity. In depression, a triad of unique maladaptive cognitive schemas exist, collectively termed the cognitive triad, that may lead to negative beliefs about the self, world and future in response to a negative stimulus (Beck, 1974, 2008; Beck and Bredemeier, 2016). These schemas, in turn, may enforce negative information processing biases and reinforce the symptoms of depression through negative subjective appraisals. For instance, negative subjective appraisals can lead to: (1) automatic negative thoughts, which are responsible for the cognitive symptoms of MDD; and/or (2) activation of the autonomic nervous system and immune system (also mediated by the neurotransmission of serotonin and dopamine), which results in the "sickness behaviours" observed in MDD (i.e., anhedonia, loss of energy, etc.; Beck and Bredemeier, 2016). In MDD, and conceivably in mood disorders in general (see Panchal et al., 2019), this cognitive mechanism is hypothesized to underly symptoms of an acute mood episode and promote biased interpretations of the self and environment. As such, the phenomenon of EM may play a clinically-relevant role in the manifestation and maintenance of the symptoms associated with MDD and BD. Consistent with this model, Matt et al. (1992) reported that acutely depressed individuals display a mood-congruent negative EM bias. However, recent evidence has challenged the notion that such a mood-congruent bias exists in MDD (Bylsma et al., 2008; Cheng et al., 2015). Furthermore, while research into cognitive deficits in BD has increased in recent years (see, for example, Lima et al., 2017), questions remain about the existence of a mood-congruent EM bias in BD.

To enhance our understanding of the cognitive deficits associated with mood disorders, the current paper systematically reviewed the literature to investigate the existence of a non-autobiographical explicit EM bias in individuals with MDD and BD. Considering the similar clinical and cognitive aspects of these mood disorders (Cuellar et al., 2005), researchers have identified a necessity for a comprehensive joint evaluation of the cognitive phenotypes associated with MDD and BD (Bearden et al., 2006; MacQueen and Memedovich, 2017). The current investigation involved the assessment of incidental, rather than intentional, emotional memories. Incidental memories refer to "memories that are acquired without intention" (i.e., without attention, effort or resources; Glisky, 2011) and represent a more prominent phenotype in daily functioning (Kontaxopoulou et al., 2017). Patterns of incidental EM formation thus inform a more naturalistic understanding of EM biases in mood disorders by highlighting cognitive processes that function independently from one's conscious attention. Explicit EM was chosen as the focus for the current review because it refers to the classification of EM that involves conscious recollection of information (Hine and Tsushima, 2018). Moreover, research investigating explicit EM involves experimental procedures with highly controlled stimulus sets and memory tasks (i.e., compared to other sub-domains of memory that involve the retrieval of personal experiences or self-generated material; Matt et al., 1992).[2] We classified EM differences in MDD and BD according to: (1) individuals experiencing a current mood episode compared to HCs; and (2) euthymic individuals compared to HCs.

2. Methods

Complete methodology for the current systematic review was registered in PROSPERO (Bogie et al., 2017), an international prospective register of systematic reviews.

2.1. Search strategy

The current review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher et al., 2009). A literature search was performed to retrieve peer-reviewed, primary studies with a population diagnosed with MDD or BD.

A research librarian (D.S.) constructed the search strategy. The librarian combined subject headings and keywords to build a literature search comprised of three concepts: 'emotional memory bias', 'major depressive disorder' and 'bipolar disorder'. 'Emotional memory' is not an established term in the indexing of health literature. Consequently, the current search strategy was necessarily and purposely broad in scope to accommodate for variations in terminology related to the concept of EM (see Supplementary File 1). The search strategy is also available on PROSPERO (Bogie et al., 2017).

The following databases were searched on December 10, 2018: Cochrane, OVID Medline, OVID Embase, OVID PsycInfo, OVID Health

[2] The reader is directed to Gaddy and Ingram (2014) and Köhler et al. (2015) for a review of implicit and autobiographical memory in MDD, respectively.

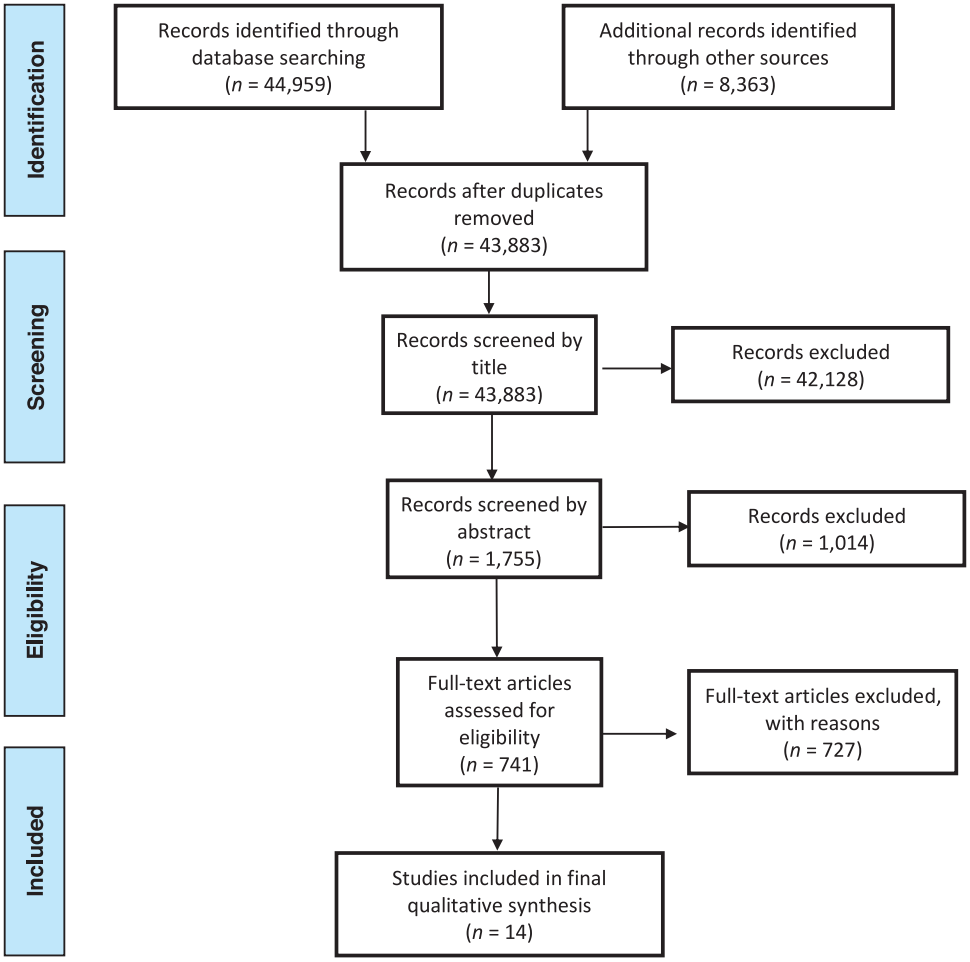

Fig. 1. PRISMA flow diagram summarizing the results of the screening process (Moher et al., 2009).

and Psychosocial Instruments (HAPI), LILACs and Web of Science. A search for grey literature and hand searches were also performed. While previous reviews/meta-analyses exist that investigate emotional information processing phenotypes in depression or BD *in general* (e.g., Leppänen, 2006; Matt et al., 1992; Robinson et al., 2006; Vöhringer et al., 2013), no systematic review has focused on non-autobiographical explicit EM biases in MDD and BD *specifically*. Therefore, no geographic, language, or date restrictions were imposed in the current review. The search strategy was peer-reviewed by a second librarian. The reference management software, RefWorks (2018), was used to organize retrieved literature from all searches and to detect duplicate citations.

2.2. Identification of eligible studies

Sources were eligible if they included original data from subjects diagnosed with MDD or BD, aged 18 to 66, compared against a HC group. To be eligible, diagnoses of MDD or BD must have been confirmed using standardized, validated diagnostic assessment tools that adhered to either the Diagnostic and Statistical Manual of Mental Disorders' (DSM; American Psychiatric Association, 2013) or the International Classification of Diseases' (ICD; World Health Organization, 2018) criteria for MDD and BD. Studies were eligible if they measured non-autobiographical explicit EM performance using any type of non-autobiographical explicit EM task that included experimentally-controlled positive, neutral and/or negative stimuli. Studies including data from mixed populations were eligible if the data were reported separately for MDD and BD samples.

Studies were ineligible if they included subjects with MDD or BD with a co-morbid diagnosis of delusional disorder, schizoaffective disorder, schizophrenia, and/or current alcohol or substance use disorder. Studies were excluded if the study design involved an intervention before the assessment of EM, or if subjects were privy to the memory task.

One author (D.S.) performed database and grey literature searches and two authors (B.J.M.B., M.R.P.) independently performed hand searches. Two authors (B.J.M.B., M.R.P.) independently reviewed all titles and abstracts for pre-defined eligibility criteria. Disagreement was resolved through discussion. Potentially eligible studies were read in full independently by two authors (B.J.M.B., M.R.P.) to confirm eligibility. Disagreement was resolved through discussion.

2.3. Data extraction

Data on study methodology, sample composition and study findings were extracted from eligible studies and recorded in a data extraction spreadsheet independently by two authors (B.J.M.B., M.R.P.). Discrepancies were resolved through discussion.

2.4. Quality assessment of eligible studies

The quality of eligible studies was assessed using a revised form of the Effective Public Health Practice Project's (EPHPP) quality assessment tool for quantitative studies (Effective Public Health Practice Project, 1998). This validated tool includes six components: selection bias; design; confounders; blinding; data collection methods; and withdrawals and dropouts. The revised EPHPP tool used here

disregarded sections B and D (Q1) because they were irrelevant to the types of studies included in the current review. Each component was assigned a rating of weak, moderate or strong. All component ratings contributed to a global rating. The procedure for converting the component ratings into a global rating has been reported elsewhere (Hall et al., 2017). Two authors (B.J.M.B., M.R.P.) independently assessed the quality of each eligible study. Disagreement was resolved through discussion.

3. Results

Given the necessary breadth of our search strategy, our searches yielded 43,883 titles. Following the PRISMA guidelines (Moher et al., 2009), title screening excluded 42,128 articles and abstract screening excluded a further 1,014 articles. The remaining 741 articles underwent full text review. Of these, 14 studies met full eligibility criteria. The remaining 723 articles were excluded primarily because of insufficient diagnostic procedures (e.g., the use of inappropriate diagnostic tools, not assessing co-morbid diagnoses) and exclusionary methodological designs (e.g., interventional studies that imposed an intervention before the assessment of EM). Fig. 1 summarizes the results of each step in the screening process.

Of the 14 eligible articles, 11 included an MDD sample and 3 included a BD sample (see Table 1 for a summary of the included studies). Seven of the included studies received a strong global quality rating (Baños et al., 2001; Hamilton and Gotlib, 2008; Kauer-Sant'Anna et al., 2008; Olsen et al., 2015; Ridout et al., 2009; Whalley et al., 2009; Williams et al., 2015), five received a moderate rating (Delgado and Chaves, 2013; Denny and Hunt, 1992; Ellwart et al., 2003; Gotlib et al., 2004; Liu et al., 2012) and two received a weak rating (Rinck and Becker, 2005; Serfaty et al., 2002) according to the EPHPP quality assessment tool. The eligible studies assessed explicit EM in samples of: (1) MDD, acutely depressed (Baños et al., 2001; Denny and Hunt, 1992; Ellwart et al., 2003; Gotlib et al., 2004; Hamilton and Gotlib, 2008; Liu et al., 2012; Olsen et al., 2015; Ridout et al., 2009; Rinck and Becker, 2005; Serfaty et al., 2002); (2) MDD, euthymic (Williams et al., 2015); (3) BD, acute mood episode (Delgado and Chaves, 2013; Whalley et al., 2009); and (4) BD, euthymic (Kauer-Sant'Anna et al., 2008). Findings from the included studies are organized below according to the included sample's episode status and the type of explicit EM paradigm used (i.e., free recall versus recognition tasks). Previous research has identified partly distinct *and* partly shared neural correlates underlying explicit recall and explicit recognition memory processes, making both paradigms appropriate at assessing explicit EM (Buckner and Koutstaal, 1998; Cabeza and Nyberg; 2000). For the purposes of this review, stimuli originally defined as “happy” were re-categorized as positive and stimuli originally defined as “depression-related” or “sad” were re-categorized as negative; analyses of any additional valences other than positive, neutral and negative are not discussed (e.g., panic-related (Baños et al., 2001), physically- and socially-threating (Gotlib et al., 2004), social phobia-related (Rinck and Becker, 2005) stimuli). These stimuli were largely included to assess explicit EM in other groups included in these studies (i.e., panic disorder (Baños et al., 2001) and social phobia (Gotlib et al., 2004; Rinck and Becker, 2005) groups). Methodological heterogeneity across the included studies precluded meta-analysis. For example, there was significant heterogeneity across included studies regarding the study design, use of encoding task, type of stimuli, valences of stimuli, delay period, memory task and the nature of the dependent variable (i.e., percent correct, memory sensitivity, normalized memory scores). Therefore, given this diversity, the authors did not find it meaningful or appropriate to conduct a meta-analysis (see also Hasselbalch et al. (2011) for similar reasoning regarding the decision to perform a meta-analysis).

3.1. Explicit EM in MDD during acute depression

A total of 10 studies assessed explicit EM in currently depressed individuals with MDD (Baños et al., 2001; Denny and Hunt, 1992; Ellwart et al., 2003; Gotlib et al., 2004; Hamilton and Gotlib, 2008; Liu et al., 2012; Olsen et al., 2015; Ridout et al., 2009; Rinck and Becker, 2005; Serfaty et al., 2002). Of these 10 studies, 7 studies (Baños et al., 2001; Denny and Hunt, 1992; Ellwart et al., 2003; Gotlib et al., 2004; Liu et al., 2012; Rinck and Becker, 2005; Serfaty et al., 2002) used a free recall paradigm and 5 studies (Hamilton and Gotlib, 2008; Liu et al., 2012; Olsen et al., 2015; Ridout et al., 2009; Serfaty et al., 2002) used a recognition memory paradigm. Results within this section are reported separately according to recall and recognition paradigms to account for variations in study protocol. It should be noted that 3 of the included studies in this section used exclusively female subjects (Denny and Hunt, 1992; Olsen et al., 2015; Rinck and Becker, 2005). Most of the included studies with an acutely depressed MDD sample recruited subjects from specialized outpatient clinics (Baños et al., 2001; Gotlib et al., 2004; Hamilton and Gotlib, 2008; Liu et al., 2012; Olsen et al., 2015; Ridout et al., 2009; Serfaty et al., 2002), while the remaining studies recruited subjects from inpatient units (Denny and Hunt, 1992; Ellwart et al., 2003), or community samples (Olsen et al., 2015; Rinck and Becker, 2005).

Only 2 of the 7 studies investigating explicit EM using a free recall paradigm found an explicit EM bias in MDD (Gotlib et al., 2004; Rinck and Becker, 2005), a finding that does not support an explicit EM bias during acute MDD (it is noted, however, that Denny and Hunt (1992) showed that, within the MDD group, negative stimuli were recalled significantly more accurately than positive stimuli). These 2 studies found that the MDD group recalled more negative stimuli than HCs. Three of the remaining 7 studies found an EM deficit in the MDD group compared to HCs. For example, Denny and Hunt (1992) found that the MDD group recalled significantly fewer positive stimuli than HCs; Serfaty et al. (2002) found that the MDD group recalled significantly fewer positive and negative stimuli than HCs; and Liu et al. (2012) found that the MDD group performed significantly worse than HCs on all but one valence category (the negative low-arousal category, where there was no between-group difference). While these results may reflect an EM deficit *specifically*, it is possible that these results reflect a rather *general* memory impairment in MDD. For example, Denny and Hunt (1992) and Serfaty et al. (2002) both showed that the MDD groups had worse overall memory compared to HCs, and Liu et al. (2012) showed that the MDD group had significantly worse memory for neutral stimuli compared to HCs, which is indicative of a general memory impairment.

Of the 5 studies (Hamilton and Gotlib, 2008; Liu et al., 2012; Olsen et al., 2015; Ridout et al., 2009; Serfaty et al., 2002) that used a recognition memory paradigm, only the study by Hamilton and Gotlib (2008) demonstrated a negative EM bias in MDD. Furthermore, the study by Ridout et al. (2009) found a general EM impairment in the MDD group compared to HCs; however, this did not translate into a valence-specific explicit EM bias or deficit. The remaining studies reported no differences between groups on the explicit EM recognition task.

In summary, neither the free recall nor the recognition memory findings readily support the existence of an explicit EM bias in acutely depressed MDD subjects. The inconsistent findings across included studies assessing explicit EM in acutely depressed MDD subjects may, in part, be explained by important moderating factors (discussed below).

3.2. Explicit EM in MDD during euthymia

Only 1 study assessed explicit EM in a euthymic MDD sample (Williams et al., 2015). Subjects were pregnant and non-pregnant women with a history of MDD compared against pregnant and non-pregnant HCs. This study employed a recognition memory paradigm

Table 1
Summary of included articles.

Study	Groups (Sample Size, % Female, Mean Age)	Memory Task(s)	Stimuli and Valences	Summary of Findings
Baños et al. (2001)	MDD, acute depression (n = 20, 75%, age range: 20–50) HC (n = 20, 80%, age range: 20–50) (Panic disorder sample also included)	Graphemic, semantic, and self-referent encoding tasks; completed two distractor tasks and an IM task; completed free recall explicit EM task	Words; positive, neutral, depression-related (i.e., negative)	No significant between-group differences on EM; significantly more depression-related words were recalled within each group ($p < 0.001$); significantly better EM for neutral words within both MDD and HC groups following self-referent encoding ($p < 0.001$)
Delgado and Chaves (2013)	BD-I psychotic, manic episode (n = 19, 63.2%, 37.3) BD-I nonpsychotic, manic episode (n = 12, 50%, 47.0) HC (n = 27, 74.1%, 39.2)	Word span task: word lists of the three valence categories were consecutively presented; immediate recall paradigm	Words; positive, neutral and negative	Significant between-group difference for the recall of positive words ($p = 0.042$); BD-I nonpsychotic > BD-I psychotic > HCs
Denny and Hunt (1992)	MDD, acute depression (n = 16, 100%, 29.2) HC (n = 16, 100%, 24.0)	Self-referent encoding task; immediately completed IM and explicit EM tasks in counterbalanced order (explicit EM task was free recall)	Words; positive, negative	The MDD group recalled significantly fewer words overall ($p < 0.001$); the MDD group recalled significantly fewer positive words than HCs ($p < 0.001$); the MDD group recalled significantly more negative than positive words ($p < 0.001$); HCs recalled significantly more positive than negative words ($p < 0.01$)
Ellwart et al. (2003)	MDD, acute depression (n = 36, 77.8%, 42.1) HC (n = 36, 72.2%, 42.5)	Self-referent encoding task; immediately completed IM and explicit EM tasks in counterbalanced order (explicit EM task was free recall)	Words; positive, neutral, negative	No significant between-group differences; Both MDD and HC groups recalled significantly more positive words ($p < 0.05$)
Gotlib et al. (2004)	MDD, acute depression (n = 88, 70.7%, 34.5) HC (n = 55, 75.0%, 33.6) (Social phobia sample also included)	Self-referent encoding task; three-minute delay/filler task; completed EM free recall task	Words; positive, depression-related (i.e., negative)	The MDD group recalled significantly more depression-related words than HCs ($p < 0.05$); both MDD and HC groups recalled significantly more positive words ($p < 0.01$)
Hamilton and Gotlib (2008)	MDD, acute depression (n = 14, 57%, 36.5) HC (n = 12, 50%, 31.4)	Participants rated the emotional intensity and affective valence of 210 images (70 in each valence category) while in an fMRI scanner; returned one week later for incidental recognition EM task	IAPS images; positive, neutral, negative	The MDD group had significantly greater memory sensitivity for negative images than HCs ($p < 0.05$); HCs recalled significantly more positive than negative images ($p < 0.05$)
Kauer-Sant'Anna et al. (2008)	Emotional and neutral conditions; BD groups comprised types I and II Euthymic BD-Emotional (n = 10, 83%, 45.7) Euthymic BD-Neutral (n = 10, 67%, 43.2) HC-Emotional (n = 10, 75%, 42.4) HC—Neutral (n = 10, 75%, 43.5)	Participants viewed an 11-slide slideshow; conditions differed in slides 5–8 (phase 2); immediate emotional intensity rating; one-week delay; incidental recognition EM task (multiple-choice test)	Narrated slide show; emotional (negative) and neutral conditions	The BD group displayed significantly worse memory performance overall compared to HCs ($p = 0.002$); the BD group displayed significantly worse memory for the negative material compared to HCs ($p = 0.01$); no significant difference between BD-Emotional and BD-Neutral groups on negative EM performance; the HC-Emotional group had significantly greater memory performance than the HC—Neutral group for the negative information ($p = 0.008$)
Liu et al. (2012)	MDD, acute depression (n = 71, 47.9%, 27.3) HC (n = 61, 49.2%, 26.1)	Participants rated each word on a pleasant/arousal scale; no delay/filler task; immediate EM tasks (free recall and recognition)	Words; positive high-arousal, positive low-arousal, neutral, negative high-arousal, negative low-arousal	The MDD group recalled significantly fewer positive high- and low-arousal, neutral, and negative high-arousal words than HCs (all $ps < 0.001$); both groups recalled significantly more high-arousal than low-arousal words ($p < 0.05$); HCs recalled significantly more positive high-arousal than negative low-arousal words ($p < 0.001$); no significant differences for the recognition task
Olsen et al. (2015)	MDD, acute depression (n = 18, 100%, 35.9, age range: 18–55) HC (n = 33, 100%, 38.4, age range: 18–58) (Schizophrenia sample also included)	Participants rated the emotional intensity of 90 images (30 in each valence category); returned one day later for incidental recognition EM test	IAPS images; positive, neutral, negative	No significant between-group differences; all groups recognized negative images more accurately than positive images ($p < 0.05$)
Ridout et al. (2009)	MDD, acute depression (n = 16, 68.8%, 43.7) HC (n = 18, 77.8%, 39.3)	Participants completed a gender identification task for 30 images of human faces; five-minute filled delay; completed an incidental recognition EM task	Images of human faces; happy (i.e., positive), neutral, sad (i.e., negative)	The MDD group performed significant worse on the EM task overall compared to HCs ($p < 0.001$); no significant difference between the MDD and HC groups on EM performance
Rinck and Becker (2005)	MDD, acute depression (n = 27, 100%, 23.5) HC (n = 55, 100%, 21.4) (Social phobia sample also included)	Encoding tasks included searching for words one-by-one in a word matrix and self-referent encoding; average of one day between matrix and self-referent tasks;	Words; positive, neutral, depression-related (i.e., negative)	The MDD group recalled significantly more depression-related words than the HC group ($F = 5.15$); the MDD group recalled significantly

(*continued on next page*)

Table 1 (*continued*)

Study	Groups (Sample Size, % Female, Mean Age)	Memory Task(s)	Stimuli and Valences	Summary of Findings
		additional five-minute delay/filler task after self-referent encoding; completed IM and explicit EM (free recall) tasks in counterbalanced order, separated by a five-minute distracter task		more depression-related words than positive words, and significantly more positive than neutral words ($F = 4.91$); HCs recalled significantly more positive words than any other valence ($F = 5.39$)
Serfaty et al. (2002)	MDD, acute depression ($n = 15$, 33.3%, 41.7) HC ($n = 15$, 46.7%, 33.3)	Self-referent encoding task; no delay/filler task; immediate EM task (free recall), five-minute delay, and recognition task	Words; positive, neutral, negative	The MDD group recalled significantly fewer positive ($p < 0.002$) and negative ($p < 0.001$) words than HCs; both groups recalled significantly more positive than negative words ($p < 0.002$ for HCs, $p < 0.02$ for MDD); no significant between-group differences for recognition EM; both groups recognized significantly more positive words than negative words ($p < 0.002$)
Whalley et al. (2009)	BD (included euthymic, acute manic, and acute depressed, with and without psychotic features; $n = 14$, 35.7%, 41.5) HC ($n = 14$, 28.6%, 31.4) (Schizophrenia sample also included)	Participants rated the emotional intensity of 72 images (36 in each valence category) while in an fMRI scanner; the incidental recognition EM task occurred immediately after the scan	IAPS images; positive, neutral	No significant between-group differences; both MDD and HC groups recognized significantly more positive than neutral images ($p = 0.01$)
Williams et al. (2015)	Pregnant and non-pregnant conditions. Euthymic MDD-Pregnant ($n = 14$, 100%, 31.0) Euthymic MDD-Non-pregnant ($n = 13$, 100%, 27.0) HC-Pregnant ($n = 30$, 100%, 29.0) HC–Non-pregnant ($n = 20$, 100%, 23.0)	Participants rated the emotional intensity of 144 images (48 in each valence category); one-week delay; completed an incidental recognition memory test	IAPS images; positive, neutral, negative	Pregnancy status did not significantly affect memory performance; participants with a history of MDD had significantly worse recognition memory for negative images compared to HCs ($p = 0.01$); there was no significant difference in EM performance between pregnant women with and without a history of MDD

Note: The words "recalled" and "recognized" were carefully chosen in the final column of this table to denote findings from free recall and recognition memory paradigms, respectively.
BD, bipolar disorder; EM, emotional memory; fMRI, functional magnetic resonance imaging; HC, healthy control; IAPS, International Affective Picture System (Lang et al., 1997); IM, implicit memory; MDD, major depressive disorder.

using stimuli from the International Affective Picture System (IAPS; Lang et al., 1997). The euthymic MDD females were recruited from specialized outpatient clinical services and community services. This study found that euthymic women with a history of MDD showed significantly worse memory for negative images (i.e., a negative EM deficit) compared to euthymic women without a history of MDD. There were no group differences for positive or neutral images, and pregnancy had no effect on these results.

3.3. Explicit EM in BD during an acute mood episode

One study compared explicit EM in acutely manic BD-I individuals, with and without psychotic features, against HCs (Delgado and Chaves, 2013). This study employed a verbal episodic memory test using a word span task with an immediate free recall paradigm. All patients in this study were recruited from a psychiatric inpatient unit. This study showed significant group differences on the recall of positive words: the BD-I nonpsychotic group had greater accuracy than the BD-I psychotic group, who in turn had greater accuracy than the HC group on their recall of positive words. Therefore, this study suggests a possible positive EM bias in acute mania.

Another study combined BD-I individuals with acute depression, acute mania and euthymic individuals (more than half of the BD sample was in an acute mood episode), compared to HCs (Whalley et al., 2009). This study employed a recognition memory paradigm using stimuli from the IAPS (Lang et al., 1997). Patients were recruited from inpatient and outpatient clinics. While both the BD and HC groups recognized significantly more positive images than neutral images (negative images were not included), there was no between-group difference on explicit EM performance.

3.4. Explicit EM in BD during euthymia

Only 1 study assessed explicit EM in euthymic individuals in a group of BD-I and BD-II subjects (Kauer-Sant'Anna et al., 2008). This study employed a recognition memory paradigm using a narrated slideshow with emotional (negative) and neutral content. The euthymic BD subjects were recruited from a specialized outpatient clinic. The euthymic BD subjects showed significantly worse memory for the emotionally-negative content compared to HCs (i.e., a negative EM deficit); however, these researchers also showed that the BD group demonstrated a general EM impairment compared to HCs. Thus, at present, it is unclear whether these findings represent a general or valence-specific EM deficit for negative information in BD during euthymia.

4. Discussion

The current systematic review assessed explicit EM in MDD and BD during acute mood episodes and euthymia. The main finding is that the results from the included studies do not readily support the existence of an explicit EM bias in acutely depressed individuals with MDD. In BD, given the low number of included studies, we can only show preliminary evidence for a potential positive EM bias in acute mania (no studies in the current review investigated explicit EM in BD during acute depression); however, future research is needed to replicate this preliminary finding. Similarly, the current review shows preliminary evidence that euthymic individuals with a history of MDD, BD-I, or BD-II do not display an explicit EM bias; instead, preliminary findings suggest the potential for an explicit EM *deficit* for negative stimuli in MDD, BD-I and BD-II during euthymia.

The majority of studies that assessed explicit EM in acutely depressed MDD subjects did not show an explicit EM bias in MDD compared against HCs. Careful consideration of the methodological nuances revealed a striking similarity across the 7 studies that did not find an explicit EM bias: the delay period between the encoding and memory retrieval tasks was very short, ranging from minutes (Baños et al., 2001; Denny and Hunt, 1992; Ellwart et al., 2003; Liu et al., 2012; Ridout et al., 2009; Serfaty et al., 2002) to one day (Olsen et al., 2015). On the other hand, the studies that demonstrated an explicit EM bias in MDD used delay periods ranging from one day (Gotlib et al., 2004; Rinck and Becker, 2005) to one week (Hamilton and Gotlib, 2008), which suggests that less than a day might be too short of a delay to detect potential explicit EM biases in MDD. Indeed, previous research has argued that the beneficial effects of emotionally-arousing stimuli on the mechanisms of EM formation are greater with a longer delay between encoding and memory retrieval tasks (LaBar and Cabeza, 2006). This observation raises the question of what the shortest delay period between encoding and memory retrieval tasks is for an explicit EM bias to be reliably detected. Moreover, is there an interplay between memory paradigm and delay period? The 5 studies (Baños et al., 2001; Denny and Hunt, 1992; Ellwart et al., 2003; Liu et al., 2012; Serfaty et al., 2002) that employed a free recall paradigm and found negative results all included a particularly short delay period (minutes), while the 2 studies that found an explicit EM bias (Gotlib et al., 2004; Rinck and Becker, 2005) both included a longer delay period (one day). Similarly, the 4 studies that used a recognition paradigm and found negative results again used a short delay period of minutes (Liu et al., 2012; Ridout et al., 2009; Serfaty et al., 2002) to one day (Olsen et al., 2015), while the one study reporting an explicit EM bias used a delay period of one week (Hamilton and Gotlib, 2008). Hamilton and Gotlib (2008) imposed a one-week delay between encoding and recognition tasks and identified a negative EM bias in MDD during acute depression. Olsen et al. (2015) used a similar methodology to Hamilton and Gotlib (2008); however, these researchers imposed a one-day delay period and subsequently did not find an explicit EM bias. Again, this observation suggests that a longer delay period may be necessary for detecting an explicit EM bias in MDD. This requirement likely reflects the importance of the time-dependent process of consolidation on the strengthening of the memory trace (Hamann, 2001). Indeed, research suggests that, in response to an emotionally-arousing stimulus, the modulatory activity of the amygdala—through the activation of glucocorticoid receptors and the activity of norepinephrine—leads to glutamatergic synaptic plasticity in the hippocampus (i.e., a time-dependent process; LaBar and Cabeza, 2006; Phelps, 2004). This, in turn, results in enhanced consolidation of the memory trace and, by extension, more accurate memory performance for emotional compared to unemotional stimuli (LaBar and Cabeza, 2006). A longer delay period between the encoding and memory retrieval tasks thus allows more time for the process of glutamatergic synaptic plasticity to translate into enhanced (detectable) EM performance (LaBar and Cabeza, 2006; Phelps, 2004). Therefore, in future studies, we recommend that the minimum delay periods used to investigate an explicit EM bias using free recall and recognition memory paradigms should be at least one day and one week, respectively.

It is also important to consider the potential effects of stimulus type and encoding procedure on explicit EM performance. Five of the 7 studies reporting negative results in an acutely depressed MDD sample used word stimuli (Baños et al., 2001; Denny and Hunt, 1992; Ellwart et al., 2003; Liu et al., 2012; Serfaty et al., 2002), while the remaining studies used human faces (Ridout et al., 2009) and IAPS images (Olsen et al., 2015). The 3 studies showing an explicit EM bias in MDD used words (Gotlib et al., 2004; Rinck and Becker, 2005) and IAPS images (Hamilton and Gotlib, 2008); thus, stimulus type alone does not appear to influence explicit EM performance in this population.

Could the lack of an explicit EM bias in acutely depressed MDD subjects be explained by self-referent encoding effects? Evidence suggests that personally-relevant stimuli can profoundly affect memory performance (Abraham, 2013; Zupan et al., 2017). Of the 10 studies including an acutely depressed MDD sample, 6 studies (Baños et al., 2001; Denny and Hunt, 1992; Ellwart et al., 2003; Gotlib et al., 2004; Rinck and Becker, 2005; Serfaty et al., 2002) involved using self-referential encoding (i.e., an encoding strategy where information is

processed/encoded with reference to the self; Durbin et al., 2017; Zupan et al., 2017). Two of these studies identified a negative EM bias (Gotlib et al., 2004; Rinck and Becker, 2005), while one study (Denny and Hunt, 1992) found that the MDD group performed worse than HCs in the remembering of positive information, and another study (Serfaty et al., 2002) found that the MDD group performed worse than HCs in the remembering of positive and negative information. Future research is therefore needed to better elucidate the influence of personally-relevant stimuli and self-referential encoding on explicit EM performance in individuals with MDD.

The lack of an identified explicit EM bias in acutely depressed MDD subjects may also be understood by considering variability in clinical factors across studies that may have differentially influenced explicit EM performance, such as symptom severity, length of illness, number of previous mood episodes and medication status. While the present review imposed strict eligibility criteria to control for as many confounding clinical factors as possible (i.e., the use of standardized diagnostic interviews, exclusion of current alcohol and substance use disorders), the impact of the abovementioned variables on explicit EM cannot be discounted. Depressive symptom severity does not seem to influence the overall results of our systematic review since the studies that found an explicit EM bias included subjects with moderately severe depression (Hamilton and Gotlib, 2008) and very severe depression (Gotlib et al., 2004), while the studies reporting no EM bias included subjects from mild/moderately severe (Liu et al., 2012; Olsen et al., 2015) to severe (Baños et al., 2001; Ridout et al., 2009; Serfaty et al., 2002) depression. However, the influence of illness duration, the number of previous depressive episodes and medication status might also be important factors to consider when assessing explicit EM. Unfortunately, only half of the included studies assessing explicit EM in currently depressed MDD subjects reported information on medication status (Ellwart et al., 2003; Hamilton and Gotlib, 2008; Olsen et al., 2015; Ridout et al., 2009; Serfaty et al., 2002), only 2 studies reported average illness length (Hamilton and Gotlib, 2008; Liu et al., 2012), and no studies reported the number of previous depressive episodes.

Two studies assessed explicit EM in BD subjects during an acute mood episode. Delgado and Chaves (2013) identified a positive EM bias and Whalley et al. (2009) found no explicit EM bias. It should be noted, however, that the study by Whalley et al. combined acutely depressed, acutely manic and subjects in euthymia into a single BD sample. This is a methodological problem since the grouping of subjects with different mood episode statuses into a single BD group likely confounded the results. For example, given the profound effect of mood state on memory performance (Bower, 1981; Matt et al., 1992), combining participants with different mood states (i.e., mania, hypomania, depression, euthymia) neglects the unique cognitive features of the independent mood states, leading to the potentially inaccurate conclusion of no explicit EM bias. Still, this study reported a bias within the BD group towards remembering positive stimuli with greater accuracy. Given the sample composition of Whalley et al.'s study, and considering that both included studies on BD during an acute mood episode only included BD-I subjects, future research is necessary to differentiate the explicit EM phenotypes of individuals with BD-I from BD-II during an acute depressive or (hypo)manic episode. Nevertheless, these findings provide preliminary support for a potential positive explicit EM bias in BD-I during an acute mood episode.

4.1. Strengths and limitations

This is the first article to comprehensively and systematically review the literature on explicit EM biases in MDD and BD. While previous reviews and meta-analyses have attempted to identify cognitive dysfunction in MDD and BD *in general* (Robinson et al., 2006; Rock et al., 2013; Vöhringer et al., 2013), none have investigated explicit EM *specifically*. Moreover, this review presents and discuses several potential moderating variables that may influence explicit EM performance across MDD and BD during both illness stages. This discussion may inspire several future research foci in the investigation of explicit EM in MDD and BD across illness stages. Despite these strengths, some limitations must be considered. First, there were considerable differences in the sample sizes of included studies. Second, there was an unequal balance of studies including MDD and BD samples (11 versus 3, respectively). As a result, less synthesis was possible in the BD results. Third, only 1 study in each population included a euthymic sample, highlighting the need for more investigation into potential interepisodic explicit EM biases in MDD and BD. Fourth, several studies identified a general memory impairment in the clinical groups compared to the HC group (e.g., Denny and Hunt, 1992; Kauer-Sant'Anna et al., 2008; Liu et al., 2012; Ridout et al., 2009). The effect of general memory impairment on valence-specific explicit EM performance is unknown and thus may influence the detection of a valence-specific explicit EM bias and/or deficit. Considering these findings, further investigation aimed at identifying the influence of general memory impairment on valence-specific explicit EM performance is warranted. Finally, since no longitudinal studies were found, questions remain about the role that emotional cognitive patterns may play in the risk of relapse, or in the development of other co-morbid disorders.

4.2. Recommendations for future research

Given the neurobiological and neurocognitive differences observed between MDD and BD in previous research (Chiriţă et al., 2015; Fung et al., 2015; Harrison et al., 2018; MacQueen and Memedovich, 2017), it would be useful for future research to investigate explicit EM performance across MDD, BD and HC groups within a single experimental design. Such direct comparisons will help elucidate differences in explicit EM biases both between MDD and BD *and* between acute mood episodes and euthymia. Given that the most common reasons for excluding studies from the current review were the lack of a structured interview to ascertain psychiatric diagnoses and the use of an interventional methodology, future studies must attain greater methodological consistency when investigating explicit EM biases. To facilitate this, Table 2 presents considerations of specific methodological approaches to be used in future research. The use of consistent methodological designs in future research will provide the opportunity for more rigorous analysis (i.e., meta-analysis) across a larger number of studies.

5. Conclusions

The main conclusion of this systematic review is that current empirical evidence does not readily support the view that acutely depressed individuals with MDD display an explicit EM bias. The current review provides insights into explicit EM in mood disorders with implications for future research. This review highlights the potential importance of several moderating variables that may influence explicit EM performance, including clinical factors (e.g., illness duration, number of depressive episodes, symptom severity and medication status), stimulus type, encoding strategy and type of memory retrieval task (e.g., free recall versus recognition). Results of this review also suggest that an extended delay period between encoding and memory retrieval tasks may be particularly important to allow sufficient time for consolidation and detection of an existing explicit EM bias in individuals with mood disorders. Nevertheless, the current findings provide preliminary support against the existence of a mood-congruent explicit EM bias in MDD (however, future research should strive to disentangle the influence of negative stimulus types—e.g., depression-related/sad, anger, disgust, fear, etc.—on mood-congruent explicit EM performance in MDD. Our results also provide preliminary evidence for a potential positive explicit EM bias in acute mania. Finally, given that only one study employing a euthymic sample was included in each clinical population, future research must strive to investigate explicit EM performance in

Table 2
Recommendations for future research on explicit EM.

Procedures	Recommendation(s)
Sample Size	Include a sample size calculation.
Inclusion and Exclusion Criteria	Describe the age range (if applicable, stratify results for pediatric and geriatric samples). Define primary and acceptable co-morbid diagnoses; the following are diagnoses that should be the basic exclusion criteria for future EM research: schizophrenia, schizoaffective disorder, delusional disorder, current alcohol and substance use disorder. Current mood status should be explicit; if a euthymic group is included, then criteria for determining euthymia should be described. The use of a HC group matched to the clinical groups on age, sex, gender, IQ and years of education.
Diagnostic Procedures	The use of a validated diagnostic assessment tool that adheres to the most recent versions of the DSM (American Psychiatric Association, 2013) or ICD (World Health Organization, 2018) is crucial; e.g., the SCID (First et al., 2015). Moreover, the use of mood-specific assessments, such as the BDI (Beck et al., 1996), MADRS (Montgomery and Åsberg, 1979), HDRS (Hamilton, 1960), and/or YMRS (Young et al., 1978) should be included to assess current symptom severity.
Clinically-Relevant Variables	Description of length of illness, number of previous mood episodes, number of hospitalizations, past or current psychosis, suicide attempts, length of euthymia (if applicable) and medication status should all be included.
Encoding Task	Incidental encoding tasks could include a procedure wherein subjects rate the emotional intensity, emotional valence and/or personal relevance of each stimulus. Incidental encoding tasks prevent subjects from becoming aware of the future memory task, ensuring the outcome measure is baseline (i.e., naturalistic) memory performance and not learning or studying ability. Ratings of subjective valence and personal relevance may also be assessed following the EM task (if not included in the encoding task). EM performance may be stratified according to these phenomena.
Stimulus Type	The use of a normative collection of stimuli is important; e.g., from the IAPS (Lang et al., 1997). Personally-relevant stimuli should be used, when possible, to optimally activate underlying cognitive structures, or schemas (Beck, 2008). This may be accomplished by stratifying EM performance according to subjects' self-reported personal relevance ratings provided, for example, during the encoding task (see above recommendation) or after the EM task.
Stimulus Valances	All EM research should include an equal number of stimuli with positive, neutral and negative valences. Neutral stimuli must be included to rule out the possibility of a general (i.e., unemotional) memory impairment.
Delay Period	A minimum delay period of one day appears to be appropriate to detect true between-group differences in explicit EM free recall tasks. A minimum delay period of one week appears to be appropriate to detect true between-group differences in explicit EM recognition tasks. Future research investigating explicit EM performance using these minimum delay period recommendations will provide more data to either support or refute these hypotheses.
Memory Task	The memory task should be a surprise to all subjects. When employing a recognition task, this should involve presenting all stimuli from the incidental encoding task, plus additional distractor stimuli (an equal number in each valence category). During a recognition memory paradigm, subjects should be presented with a question to indicate whether the stimulus was recognized from encoding (i.e., via a yes/no or know-remember task, analyzed using receiver operating characteristics; see, for example, Yonelinas and Parks, 2007). Free recall should be used to assess recall ability.
Data Analysis and Presentation/Reporting	Data should be analyzed to determine: (1) general memory performance; and (2) explicit EM performance, overall and valance-specific. All descriptive statistics, including means and standard deviations, should be presented alongside any graphical representations of the results, if applicable. Moreover, effect sizes should be included to allow for direct comparisons between studies assessing explicit EM.

BDI, Beck Depression Inventory; DSM, Diagnostic and Statistical Manual of Mental Disorders; EM, emotional memory; HC, healthy control; HDRS, Hamilton Depression Rating Scale; IAPS; International Affective Picture System; ICD, International Classification of Diseases; MADRS, Montgomery-Åsberg Depression Rating Scale; SCID, Structured Clinical Interview for DSM Disorders; YMRS, Young Mania Rating Scale.

MDD and BD during euthymia to improve our understanding of the pattern of explicit EM across illness stages.

A better understanding of the cognitive patterns of EM in mood disorders is important given that EM may influence mood state, symptom severity and/or psychosocial functioning. For example, it is hypothesized that there exists a bi-directional relationship between maladaptive cognitive schemas and biased information processing in individuals with depression (and, conceivably, with mania and hypomania; Beck and Bredemeier, 2016). These maladaptive schemas may also be responsible for predisposing individuals to the manifestation of the psychosomatic symptoms of MDD and BD in response to emotionally-arousing stressors (Beck and Bredemeier, 2016). Moreover, the degree of schematic activation may further influence the level of symptom severity. Treatment approaches that help patients control their cognitive response to internal and/or external emotional stressors (i.e., cognitive-behavioural therapy, emotion regulation therapy) may therefore prove effective at treating the clinical symptoms of an acute mood episode *and* the subclinical symptoms of euthymia. In this way, investigation of EM biases in MDD and BD represent an important research focus that may inform potential approaches to non-pharmacological treatments.

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Author contributions

B. Bogie and M. Persaud contributed equally to the conception and design of the study, data analysis, manuscript writing and critical editing.

D. Smith contributed to the design of the study, data acquisition and manuscript writing.

F. Kapczinski and B. Frey contributed to the conception and design of the study, manuscript writing and critical editing.

Supplementary materials

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References

Abraham, A., 2013. The world according to me: personal relevance and the medial prefrontal cortex. Front. Hum. Neurosci. 7, 341.

American Psychiatric Association, 2013. Diagnostic and Statistical Manual of Mental Disorders, fifth ed. American Psychiatric Publishing, Arlington, Virginia.

Asl, A.F., Ghanizadeh, A., Mollazade, J., Aflakseir, A., 2015. Differences of biased recall memory for emotional information among children and adolescents of mothers with MDD, children and adolescents with MDD, and normal controls. Psychiatry Res. 228 (2), 223–227.

Baños, R.M., Medina, P.M., Pascual, J., 2001. Explicit and implicit memory biases in depression and panic disorder. Behav. Res. Ther. 39 (1), 61–74.

Bearden, C.E., Glahn, D.C., Monkul, E.S., Barrett, J., Najt, P., Villarreal, V., Soares, J.C., 2006. Patterns of memory impairment in bipolar disorder and unipolar major depression. Psychiatry Res. 142 (2–3), 139–150.

Beck, A.T., 1974. The Development of Depression: A Cognitive Model. In: Friedman, R., Katz, M. (Eds.), Psychology of Depression: Contemporary Theory and Research. Winston, Washington, DC.

Beck, A.T., 1979. Cognitive Therapy of Depression. Guilford Press, New York.

Beck, A.T., 2008. The evolution of the cognitive model of depression and its neurobiological correlates. Am. J. Psychiatry 165 (8), 969–977.

Beck, A.T., Bredemeier, K., 2016. A unified model of depression: integrating clinical, cognitive, biological, and evolutionary perspectives. Clin. Psychol. Sci. 4 (4), 596–619.

Beck, A.T., Steer, R.A., Brown, G.K., 1996. Beck Depression Inventory-II 78. San Antonio, pp. 490–498.

Bogie, B., Persaud, M., Smith, D., 2017. Explicit Emotional Memory Biases in Major Depressive Disorder and Bipolar Disorder: A Systematic Review. PROSPERO. https://www.crd.york.ac.uk/prospero/display_record.php?RecordID=69909 (accessed 10 December 2018).

Bora, E., Pantelis, C., 2015. Meta-analysis of cognitive impairment in first-episode bipolar disorder: comparison with first-episode schizophrenia and healthy controls. Schizophr. Bull. 41 (5), 1095–1104.

Bower, G., 1981. Mood and memory. Am. Psychol. 36 (2), 129–148.

Buckner, R.L., Koutstaal, W., 1998. Functional neuroimaging studies of encoding, priming, and explicit memory retrieval. Proc. Natl. Acad. Sci. 95 (3), 891–898.

Bylsma, L.M., Morris, B.H., Rottenberg, J., 2008. A meta-analysis of emotional reactivity in major depressive disorder. Clin. Psychol. Rev. 28 (4), 676–691.

Cabeza, R., Nyberg, L., 2000. Imaging cognition II: an empirical review of 275 PET and fMRI studies. J. Cognit. Neurosci. 12 (1), 1–47.

Cahill, L., McGaugh, J., 1995. A novel demonstration of enhanced memory associated with emotional arousal. Conscious. Cognit 4 (4), 410–421.

Cheng, P., Preston, S.D., Jonides, J., Mohr, A.H., Thummala, T., Casement, M., Hsing, C., Deldin, P.J., 2015. Evidence against mood-congruent attentional bias in major depressive disorder. Psychiatry Res. 230 (2), 496–505.

Chiriță, A.L., Gheorman, V., Bondari, D., Rogoveanu, I., 2015. Current understanding of the neurobiology of major depressive disorder. Rom. J Morphol. Embryol. 56 (2 Suppl), 651–658.

Cuellar, A.K., Johnson, S.L., Winters, R., 2005. Distinctions between bipolar and unipolar depression. Clin. Psychol. Rev. 25 (3), 307–339.

Delgado, V.B., Chaves, M.L., 2013. Mood congruence phenomenon in acutely symptomatic mania bipolar I disorder patients with and without psychotic symptoms. Cognit. Neuropsychiatry 18 (6), 477–490.

Denny, E.B., Hunt, R.R., 1992. Affective valence and memory in depression: dissociation of recall and fragment completion. J. Abnorm. Psychol. 101 (3), 575.

Durbin, K.A., Mitchell, K.J., Johnson, M.K., 2017. Source memory that encoding was self-referential: the influence of stimulus characteristics. Memory 25 (9), 1191–1200.

Durisko, Z., Mulsant, B.H., McKenzie, K., Andrews, P.W., 2016. Using evolutionary theory to guide mental health research. Can. J. Psychiatry 61 (3), 159–165.

Effective Public Health Practice Project, 1998. Dictionary for the Effective Public Health Practice Project Quality Assessment Tool for Quantitative Studies. http://www.ephpp.ca/index.html(accessed 9 May 2017).

Ellwart, T., Rinck, M., Becker, E.S., 2003. Selective memory and memory deficits in depressed inpatients. Depress. Anxiety 17 (4), 197–206.

First, M.B., Williams, J.B.W., Karg, R.S., Spitzer, R.L., 2015. Structured Clinical Interview for DSM-5 Disorders. Clinician Version (SCID-5-CV). American Psychiatric Association, Arlington, Virginia.

Flaisch, T., Steinhauser, M., Schupp, H.T., 2016. Emotional aftereffects: when emotion impairs subsequent picture recognition. Emotion 16 (7), 987.

Fung, G., Deng, Y., Zhao, Q., Li, Z., Qu, M., Li, K., Zeng, Y.W., Jin, Z., Ma, Y.T., Yu, X., Wang, Z.R., 2015. Distinguishing bipolar and major depressive disorders by brain structural morphometry: a pilot study. BMC Psychiatry 15 (1), 298.

Gaddy, M.A., Ingram, R.E., 2014. A meta-analytic review of mood-congruent implicit memory in depressed mood. Clin. Psychol. Rev. 34 (5), 402–416.

Glisky, E.L., 2011. Incidental memory. In: Kreutzer, J.S., DeLuca, J., Caplan, B. (Eds.), Encyclopedia of Clinical Neuropsychology, Eds. Springer New York, New York, New York.

Goldstein, T.R., 2010. Bipolar Disorder. In: Weiner, I.B., Craighead, W.E. (Eds.), The Corsini Encyclopedia of Psychology.

Gotlib, I.H., Kasch, K.L., Traill, S., Joormann, J., Arnow, B.A., Johnson, S.L., 2004. Coherence and specificity of information-processing biases in depression and social phobia. J. Abnorm. Psychol. 113 (3), 386.

Hall, S.J., Ferguson, S.A., Turner, A.I., Robertson, S.J., Vincent, G.E., Aisbett, B., 2017. The effect of working on-call on stress physiology and sleep: a systematic review. Sleep Med. Rev. 33, 79–87.

Hamann, S., 2001. Cognitive and neural mechanisms of emotional memory. Trends Cognit. Sci. 5 (9), 394–400.

Hamilton, J.P., Gotlib, I.H., 2008. Neural substrates of increased memory sensitivity for negative stimuli in major depression. Biol. Psychiatry 63 (12), 1155–1162.

Hamilton, M., 1960. A rating scale for depression. J. Neurol. Neurosurg. Psychiatry 23, 56.

Harrison, P.J., Geddes, J.R., Tunbridge, E.M., 2018. The emerging neurobiology of bipolar disorder. Trends Neurosci. 41 (1), 18–30.

Hasselbalch, B.J., Knorr, U., Kessing, L.V., 2011. Cognitive impairment in the remitted state of unipolar depressive disorder: a systematic review. J. Affect. Disord. 134 (1–3), 20–31.

Hine, K., Tsushima, Y., 2018. Not explicit but implicit memory is influenced by individual perception style. PloS One 13 (1), e0191654.

Kauer-Sant'Anna, M., Yatham, L.N., Tramontina, J., Weyne, F., Cereser, K.M., Gazalle, F.K., Andreazza, A.C., Santin, A., Quevedo, J., Izquierdo, I., Kapczinski, F., 2008. Emotional memory in bipolar disorder. Br. J. Psychiatry Suppl. 192 (6), 458–463.

Kensinger, E.A., Corkin, S., 2004. Two routes to emotional memory: distinct neural processes for valence and arousal. Proc. Natl. Acad. Sci. 101 (9), 3310–3315.

Köhler, C.A., Carvalho, A.F., Alves, G.S., McIntyre, R.S., Hyphantis, T.N., Cammarota, M., 2015. Autobiographical memory disturbances in depression: a novel therapeutic target? Neural Plast. 2015, 759139.

Kontaxopoulou, D., Beratis, I.N., Fragkiadaki, S., Pavlou, D., Yannis, G., Economou, A., Papanicolaou, A.C., Papageorgiou, S.G., 2017. Incidental and intentional memory: their relation with attention and executive functions. Arch. Clin. Neuropsychol. 32 (5), 519–532.

LaBar, K., Phelps, E., 1998. Arousal-mediated memory consolidation: role of the medial temporal lobe in humans. Psychol. Sci. 9 (6), 490–493.

LaBar, K.S., Cabeza, R., 2006. Cognitive neuroscience of emotional memory. Nat. Neurosci. 7 (1), 54.

Lang, P.J., Bradley, M.M., Cuthbert, B.N., 1997. International Affective Picture System (IAPS): technical manual and affective ratings. NIMH Center for the Study of Emotion and Attention,1, 39–58.

LeDoux, J.E., 1993. Emotional memory systems in the brain. Behav. Brain Res. 58 (1–2), 69–79.

Leppänen, J.M., 2006. Emotional information processing in mood disorders: a review of behavioral and neuroimaging findings. Curr. Opin. Psychiatry 19 (1), 34–39.

Lima, I.M., Peckham, A.D., Johnson, S.L., 2017. Cognitive deficits in bipolar disorders: implications for emotion. Clin. Psychol. Rev. 59, 126–136.

Liu, W.H., Wang, L.Z., Zhao, S.H., Ning, Y., Chan, R.C.K., 2012. Anhedonia and emotional word memory in patients with depression. Psychiatry Res 200 (2–3), 361–367.

MacQueen, G.M., Memedovich, K.A., 2017. Cognitive dysfunction in major depression and bipolar disorder: assessment and treatment options. Psychiatry Clin. Neurosci. 71 (1), 18–27.

Marvel, C., Paradiso, S., 2004. Cognitive and neurological impairment in mood disorders. Psychiatr. Clin. North Am. 27 (1), 19–36.

Matt, G.E., Vazquez, C., Campbell, W.K., 1992. Mood-congruent recall of affectively toned stimuli: a meta-analytic review. Clin. Psychol. Rev. 12 (2), 227–255.

Moher, D., Liberati, A., Tetzlaff, J., Altman, D.G., The PRISMA Group, 2009. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement (reprinted from Annals of Internal Medicine). Phys. Ther. 89 (9), 873–880 (2009).

Montgomery, S.A., Åsberg, M., 1979. A new depression scale designed to be sensitive to change. Br. J. Psychiatry 134 (4), 382–389.

Moritz, S., Gläscher, J., Brassen, S., 2005. Investigation of mood-congruent false and true memory recognition in depression. Depress. Anxiety 21 (1), 9–17.

Olsen, E.K., Bjorkquist, O.A., Bodapati, A.S., Shankman, S.A., Herbener, E.S., 2015. Associations between trait anhedonia and emotional memory deficits in females with schizophrenia versus major depression. Psychiatry Res. 230 (2), 323–330.

Panchal, P., Kaltenboeck, A., Harmer, C.J., 2019. Cognitive emotional processing across mood disorders. CNS Spectr. 24 (1), 54–63.

Phelps, E.A., 2004. Human emotion and memory: interactions of the amygdala and hippocampal complex. Curr. Opin. Neurobiol. 14 (2), 198–202.

RefWorks, 2018. https://www.refworks.com/refworks2/default.aspx?r=authentication::init (accessed 10 December 2018).

Ridout, N., Dritschel, B., Matthews, K., McVicar, M., Reid, I.C., O'Carroll, R.E., 2009. Memory for emotional faces in major depression following judgement of physical facial characteristics at encoding. Cognit. Emot. 23 (4), 739–752.

Rinck, M., Becker, E.S., 2005. A comparison of attentional biases and memory biases in women with social phobia and major depression. J. Abnorm. Psychol. 114 (1), 62.

Robinson, L.J., Thompson, J.M., Gallagher, P., Goswami, U., Young, A.H., Ferrier, I.N., Moore, P.B., 2006. A meta-analysis of cognitive deficits in euthymic patients with bipolar disorder. J. Affect. Disord. 93 (1–3), 105–115.

Rock, P.L., Roiser, J.P., Riedel, W.J., Blackwell, A.D., 2013. Cognitive impairment in depression: a systematic review and meta-analysis. Psychol. Med. 44 (10), 2029–2040.

Serfaty, M.A., Bothwell, R., Marsh, R., Ashton, H., Blizard, R., Scott, J., 2002. Event-related potentials and cognitive processing of affectively toned words in depression. J. Psychophysiol. 16 (1), 56.

Turkileri, N., Sakaki, M., 2017. Neural mechanisms underlying the effects of emotional arousal on memory. Memory in a Social Context. Springer, Tokyo, pp. 43–55.

Vöhringer, P.A., Barroilhet, S., Amerio, A., Reale, M.L., Vergne, D., Alvear, K.P., Ghaemi,

S.N., 2013. Cognitive impairment in bipolar disorder and schizophrenia: a systematic review. Front. Psychiatry 4, 87.
Wakefield, J.C., 1992. The concept of mental disorder: on the boundary between biological facts and social values. Am. Psychol. 47 (3), 373.
Watkins, P.C., Mathews, A., Williamson, D.A., Fuller, R.D., 1992. Mood-congruent memory in depression: emotional priming or elaboration? J. Abnorm. Psychol. 101 (3), 581.
Whalley, H.C., McKirdy, J., Romaniuk, L., Sussmann, J., Johnstone, E.C., Wan, H.I., McIntosh, A.M., Lawrie, S.M., Hall, J., 2009. Functional imaging of emotional memory in bipolar disorder and schizophrenia. Bipolar Disord 11 (8), 840–856.
Williams, J., Watts, F., MacLeod, C., Mathews, A., 1988. Cognitive Psychology and Emotional Disorders, second ed. John Wiley & Sons, Oxford, England.
Williams, M.E., Becker, S., McKinnon, M.C., Wong, Q., Cudney, L.E., Steiner, M., Frey, B.N., 2015. Emotional memory in pregnant women at risk for postpartum depression. Psychiatry Res. 229 (3), 777–783.
Wittekind, C.E., Terfehr, K., Otte, C., Jelinek, L., Hinkelmann, K., Moritz, S., 2014. Mood-congruent memory in depression – the influence of personal relevance and emotional context. Psychiatry Res. 215 (3), 606–613.
World Health Organization, 2017. Depression and Other Common Mental disorders: Global health Estimates. World Health Organization, Geneva License: CC BY-NC-SA 3.0 IGO.
World Health Organization, 2018. International Classification of Disease, Eleventh Revision (ICD-11). World Health Organization, Geneva.
Yonelinas, A.P., Parks, C.M., 2007. Receiver operating characteristics (ROCs) in recognition memory: a review. Psychol. Bull. 133 (5), 800.
Young, R., Biggs, J., Ziegler, V., Meyer, D.A., 1978. A rating scale for mania: reliability, validity and sensitivity. Br. J. Psychiatry 133 (5), 429–435.
Zupan, Z., Žeželj, I., Andjelković, I., 2017. Memory bias in depression: effects of self-reference and age. J. Soc. Clin. Psychol. 36 (4), 300–315.