In search of convergent regional brain abnormality in cognitive emotion regulation: a transdiagnostic neuroimaging meta-analysis

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Abstract

Cognitive emotion regulation (i.e., reappraisal) impairment is a key feature in a wide variety of mental disorders, suggesting common nature of disruption across psychiatric diagnoses. However, the extent of potential shared neurobiological disturbances related to reappraisal impairment is incompletely understood. This study, therefore, aimed to identify neurobiological substrates of disturbed reappraisal using a transdiagnostic approach by performing a quantitative coordinate-based meta-analysis (CBMA) on reappraisal tasks across various mental disorders. Following the best-practice guidelines for neuroimaging meta-analysis, we systematically searched PubMed, ScienceDirect and Web of Science databases for whole-brain neuroimaging studies published through February 2020 that compared brain activation in patients with mental disorders and matched healthy controls during a reappraisal task. Out of 1608 publications, we retrieved 32 publications with 1240 unique individuals, providing sufficient power for conducting CBMA using activation likelihood estimation (ALE) method. The reported peak coordinates for the patient/control difference in selected articles were extracted and several ALE analyses were performed to identify spatial convergence. Surprisingly, neither the pooled ALE analysis nor additional analyses restricting to in-/decreased contrasts and more homogeneous grouping of coordinates (i.e., regulation direction, stimulus valence and disorder category) provided significant convergent findings. This CBMA indicates a lack of convergent regional abnormality related to reappraisal across various mental disorders. This might be due to the complex nature of reappraisal, heterogeneous clinical populations or methodological flexibility including preprocessing and analytical methods.

Introduction

Throughout our daily lives, we are continuously exposed to a wide range of emotionally arousing situations. People do not just passively respond to affective situations, but usually attempt to modulate their subjective emotional experiences. Emotion regulation (ER) is regarded as a bundle of automatic (implicit) or effortful (explicit) processes, which modify the occurrence, intensity and duration of an emotional response (Etkin, et al., 2015; Gross and John, 2003; Phillips, et al., 2008). ER effectiveness is basically related to individuals' emotion awareness, their short- and long-term goals, and their flexibility in selection and implementation of various ER strategies (Gross and Jazaieri, 2014). Cognitive reappraisal (also called 'reappraisal') is among the most effective cognitive ER strategies, which is frequently used for the up/down-regulation of negative and positive affect, in a way we believe would promote the attainment of our goals and needs. Reappraisal is a form of cognitive change that involves reconceptualization of an affective situation and/or its consequences in order to modify its undesired emotional impact. (Etkin, et al., 2015; Kim, et al., 2015; Ochsner, et al., 2012). We apply self-focused reappraisal or "distancing", when we take the perspective of a detached third-person. Alternatively, situation-focused reappraisal requires to re-interpret the meaning, cause and consequences of an emotional event (McRae, et al., 2012a; Ochsner and Gross, 2008).

Successful implementation of reappraisal strategy has been shown to be related to decreased arousal and subjective distress, propitious interpersonal functioning, physical and mental well-being and higher quality of life (De Castella, et al., 2013; Gross, et al., 2019; McRae and Gross, 2020; McRae, et al., 2012b). On the other hand, poor reappraisal performance has been demonstrated to have negative effects on mental health and is associated with development, maintenance and lower treatment response of various psychiatric disorders (Aldao, et al., 2010; Jazaieri, et al., 2013; Silvers, et al., 2014). In addition, improving the ER skills increases the effectiveness of psychotherapy (Berking, et al., 2008). These findings indicate that abnormal reappraisal implementation could be considered as a cross-cutting feature of psychopathology across various mental illnesses (Beauchaine and Cicchetti, 2019; Cludius, et al., 2020).

In behavioral experiments, successful reappraisal has been conceptualized as the deliberate change in the self-reported impact (arousal or valance) of an emotional stimulus according to the aim of the given study and the lack of such a change has been defined as the failure in reappraisal. In a typical task, participants are presented with a series of evocative (positive or negative) stimuli such as emotional scenes, facial reactions, memory recalls or distressing events and are instructed to either naturally respond to them or apply the reappraisal strategy to up- or down-

regulate their emotional response. Participants are generally instructed to use the reappraisal by implementing one of the two principal tactics i.e., reinterpretation or distancing, or they are allowed to choose between them. Subjective rating comes after each condition to estimate the reappraisal effect.

Neuroimaging studies use a similar paradigm to investigate the neural substrates of reappraisal processes in healthy (see e.g., Mulej Bratec, et al., 2015; Mulej Bratec, et al., 2017; Xie, et al., 2016) and clinical populations (see e.g., Larabi, et al., 2018; Heller, et al., 2009). Successful reappraisal in healthy individuals has been shown to depend on the proper interactions between limbic regions, responsible for ‘bottom-up’ emotional responses and frontoparietal regions, responsible for ‘top-down’ cognitive control of emotions (Buhle, et al., 2014; Kohn, et al., 2014; Langner, et al., 2018; Morawetz, et al., 2020). However, findings from clinical studies have yielded heterogeneous and often conflicting results (Hofmann, et al., 2012; Zilverstand, et al., 2017).

In the context of these inconsistencies, coordinate-based meta-analysis (CBMA) approaches such as activation likelihood estimation (ALE) provide the possibility to integrate convergent results quantitatively (Eickhoff, et al., 2012) to reveal the common neural substrates underlying psychiatric disorders (Tahmasian, et al., 2019). In particular, the ALE approach allows for the study of large amounts of functional imaging data in a spatially unbiased manner (Eickhoff, et al., 2012).

Previous meta-analyses of emotion processing (McTeague, et al., 2020) and cognitive control tasks (McTeague, et al., 2017) found a transdiagnostic pattern of aberrant activation in the limbic and frontoparietal regions across various psychiatric disorders. Considering these parallel regional disturbances in reappraisal related processes, it is tempting to expect a common pattern of neural disruption underlying reappraisal. Therefore, a CBMA study that exclusively focuses on reappraisal in clinical populations may help to elucidate the nature of impairments. Here, providing a quantitative consolidation of previous neuroimaging works using ALE method, we aimed to identify a transdiagnostic regional abnormality in reappraisal performance by comparing the brain activation in patients with different psychiatric disorders and healthy individuals while doing a reappraisal task.

Methods and Materials

The current study was pre-registered at the International Prospective Register of Systematic Reviews (PROSPERO, CRD42019119121) and the search strategy was based on the Preferred Reporting Items for Systematic Reviews and Meta-Analyses statement (Moher, et al., 2009).

Following the most recent best-practice guidelines for neuroimaging meta-analyses (Müller, et al., 2018; Tahmasian, et al., 2019), we performed ALE meta-analyses on existing reappraisal neuroimaging studies that looked for the regulating disturbances in psychiatric patients versus healthy individuals by contrasting the brain activations during the two conditions of reappraisal implementation and natural responding.

Search strategies and selection criteria

The literature search was conducted in February 2020 through PubMed, ScienceDirect and Web of Science databases with the following search terms: (cognitive OR volitional OR voluntary OR effortful) AND (emotion OR affect) AND (regulat* OR reappraisal OR reinterpretation OR distancing) AND (fMRI OR "functional MRI" OR "functional magnetic resonance imaging" OR PET OR "positron emission tomography") AND (patient OR disorder). Additional publications were identified by reference tracking from reviews/meta-analyses. The resulting pool consisted of 1608 records, assessed by two authors independently (T.K. and Z.S.). Eligible studies were selected in two steps: Firstly, non-English language publications, case-reports, letter to editors, reviews/meta-analyses, and structural or task-free imaging studies were excluded by screening the abstracts. Secondly, full-texts of all remaining studies were screened carefully and studies that met each of the following criteria were included:

- fMRI/PET studies that compared the reappraisal task between patients suffering from any kind of mental disorders and healthy subjects,
- if significant brain activation results were reported for the contrast of interest (reappraisal vs. natural responding),
- if "whole-brain" analyses were performed and coordinates from the peak of task-based activations were reported in Montreal Neurological Institute (MNI) (Evans, et al., 1993) or Talairach (Talairach, 1988) spaces,
- if standardized diagnostic criteria (DSM-IV-TR, or DSM-5, or ICD-10) for patient recruitment was used,
- if adult subjects were recruited (range age between 18 and 60),
- if at least seven subjects were in each group.

Data extraction

For all included studies, we reported sample sizes, demographic data of participants (age, gender), clinical characteristics of patients (diagnosis, diagnostic tool, symptom severity,

medication status, comorbidities) and experimental setup (stimulus arousal, valence, regulation strategy and direction, imaging modality, scanner type, analysis software package, statistical analysis criteria). In addition, the peak coordinates of between-group experiments for the reappraisal vs. natural responding contrast were extracted (Table 1). Here, “study” reflects an individual publication and “experiment” indicates a set of coordinates belong to a particular analysis or contrast of interest (i.e., patients vs. controls group comparison) in a given study. Subsequently, the experiments were coded as “increased” when the brain activation during the reappraisal condition was higher in patients than healthy controls (patients > controls) or “decreased” when it was lower in patients than controls (patients < controls). The peak coordinates reported in Talairach space were converted into MNI space to set all the coordinates in the same reference space (Lancaster, et al., 2007). To avoid convergence over the analyses performed on the same/overlapping samples (reported within or between studies), we merged the coordinates from multiple experiments (e.g., in-/decreased) pertaining to the studies with the same/overlapping samples, making sure each study contributes once per analysis (Turkeltaub, et al., 2012).

Activation likelihood estimation

A revised version of activation likelihood estimation (ALE) (Eickhoff, et al., 2012) was used to test whether the activation foci reported in peak coordinates significantly clustered into specific spatial locations, rather than randomly distributed across the whole brain. The ALE analyses were performed in three steps: First, spatial 3D Gaussian probability distributions were modelled around the peak coordinates of activated foci from experiments of interest. The width of the aforementioned probability determines the spatial uncertainty associated with variations in sampling effects, data processing and data analysis. Since the foci of contrasts with smaller sample size have a smaller effect on the modeled uncertainty, it was adjusted for the number of subjects in the smaller group. Then, “modeled activation” maps of all foci of each experiment were pooled into an ALE activation map by computing their overlap across the experiments (Eickhoff, et al., 2012; Turkeltaub, et al., 2012). Finally, the ALE maps were assessed against a null-distribution map to enable the random-effects inference by using non-linear histogram integration (Eickhoff, et al., 2012; Turkeltaub, et al., 2012). As suggested previously, the above-chance clustering activation foci were assessed by setting the threshold at $p < 0.05$ family-wise error at the cluster level (cFWE) to correct for multiple comparisons and avoid spurious findings (Eickhoff, et al., 2017; Müller, et al., 2018).

To identify transdiagnostic patterns of ER-related abnormalities, multiple ALE analyses were conducted. For the primary analysis, we assessed convergence across all reported effects by pooling all experiments together. Then, we analyzed in-/decreased activations separately, in order to test the direction of aberrant activity. To further investigate the nature of ER dysfunctions, we performed complementary analyses on more homogeneous groups of experiments by categorizing data regarding regulation direction (down- or up-regulation), stimulus valence (negative or positive), disorder category (mood, anxiety, mood and anxiety or others) and reappraisal tactic (reinterpretation or distancing or reinterpretation/distancing) with considering at least 17 experiments in each category to achieve 80% power for statistical analysis (Eickhoff, et al., 2016). As a result, only the regulation direction (down-regulation), stimulus valence (negative) and disorder category (mood and anxiety) had enough experiments for further valid analyses (Table 2).

Results

A pool of 1608 records was screened and 107 full-text papers assessed for eligibility. Subsequently, 75 studies were excluded for the following reasons: lacking either healthy controls or group comparison analyses, restricting samples to adolescents or older adults, not analyzing the whole brain, reporting non-significant group comparisons, and including individuals at a high-risk of mental illnesses or with subclinical diagnosis (Table S1). Finally, a total of 32 publications were included in our meta-analysis (Figure 1), with three overlapping samples that were reported in multiple publications, one sample was used in three papers (Larabi, et al., 2018; van der Meer, et al., 2014; Zhang, et al., 2020), and two samples each used in a pair of papers (Goldin, et al., 2009a; Heller, et al., 2009; Johnstone, et al., 2007; Ziv, et al., 2013). As mentioned earlier, we rigorously avoided convergence over analyses performed on the same samples both within and across papers. Accordingly, we merged all studies with overlapping samples that resulted in 28 independent samples. Demographical, clinical and experimental characteristics of the included papers are shown in Table 1. Overall, we conducted several separate ALE meta-analyses. Neither our primary, nor complementary ALE meta-analyses yielded significant results for the following domains: (i) Task [reappraisal: 28 experiments ($p = .418$), reappraisal-decreased: 21 experiments ($p = .570$) and reappraisal-increased: 20 experiments ($p = .832$)]; (ii) Regulation direction [down-regulation: 27 experiments ($p = .859$)]; (iii) Stimulus valence [negative: 28 experiments ($p = .930$)]; (iv) Disorder category [mood and anxiety: 20 ($p = .076$)]. Of note,

repeating all analyses with a more liberal statistical threshold (i.e., threshold-free cluster enhancement, TFCE) demonstrated no significant convergence as well (Table 2). Figure 2 displays the sporadic distribution of foci based on the reappraisal experiments across literature.

Discussion

This study aimed to answer the ongoing dispute i.e., whether there is a convergent regional brain abnormality during reappraisal performance across various mental disorders. Following the best-practice guidelines for neuroimaging meta-analysis and using a strict statistical approach for multiple comparison correction, we did not find any consistent findings in our main ALE-based meta-analysis and sub-analyses, despite a comparably high homogeneity of the applied tasks (i.e., reappraisal). Of note, some previous inquiries on the neural alterations of various neuropsychiatric disorders did not reveal spatial convergence either (Giehl, et al., 2019; Muller, et al., 2017; Nickl-Jockschat, et al., 2015; Samea, et al., 2019; Tahmasian, et al., 2018a). This variance, if not completely attributable to the methodological divergence in neuroimaging and/or heterogeneity in clinical populations (Tahmasian, et al., 2018b), suggests that underlying domains of emotion and cognition are differently impaired across mental disorders. Importantly, distinct psychopathology of impaired reappraisal across different clinical populations was previously highlighted (D'Agostino, et al., 2017; Taylor and Liberzon, 2007). We discuss further this heterogeneity in the next section.

The potential reasons of divergent findings

i. Distinct pathophysiology of impaired reappraisal

Theoretically, dysfunctional ER may take place in different stages including the identification of regulation necessity, selection of regulatory strategy, implementation of selected strategy, and stopping/switching of implemented process (Fernandez, et al., 2016; Gross, et al., 2019). Indeed, clinical conditions are characterized by abnormalities in one or more regulatory stages, and may not involve disruptions in common brain regions. For example, depression is involved with overestimation of mood-congruent stimuli, as apparent in differential amygdala activity (Zilverstand, et al., 2017) and conversely, a helplessness to ignite a regulatory action (Sheppes, et al., 2015). Bipolar patients overvalue the hedonic benefits of manic states and are not usually convinced to down-regulate the positive affect (Fernandez, et al., 2016). Moreover, anxiety is associated with attentional biases toward threat stimuli as reflected in differential activity of

parietal lobule (Zilverstand, et al., 2017) and consequently, an amplified representation of regulation urgency (Gross and Jazaieri, 2014). Exaggerated sense of regulation necessity and decreased flexibility in strategy selection are mainly observed in post-traumatic stress disorder (PTSD) and obsessive-compulsive disorder (OCD) (Taylor and Liberzon, 2007). Additionally, patients with social anxiety disorder (SAD) may be uncertain about the effectiveness of reappraisal implementation due to insufficient self-efficacy, which probably results in premature stopping of regulatory effort (Sheppes, et al., 2015). Borderline personality disorder is also associated with monitoring deficits of impulsive strategy switching (Gross, et al., 2019). Furthermore, failing to stop the maladaptive strategies (e.g., rumination) may affect the implementation of adaptive ones (e.g., reappraisal) in depression and anxiety disorders (Dryman and Heimberg, 2018). All these examples show that differences in clinical populations regarding the awareness of emotions, beliefs about controllability of emotions, tendency to regulate emotions and availability of regulatory resources (Kim, et al., 2015) are critical factors influencing the successful reappraisal performance, and probably, the pertinent neural engagement.

ii. Experimental flexibility

Absence of convergent regional abnormality due to reappraisal impairment can be further explained by the taxonomy of experimental designs in neuroimaging studies of reappraisal. Although we only included studies which used the prototypical reappraisal paradigm, some experimental factors and underlying cognitive functions could well affect the neural basis of cognitive reappraisal. Attentional engagement is a relevant example which can be modulated with interrelated exogenous e.g., arousal and valence (Sussman, et al., 2013) and endogenous factors e.g., needs, goals and motivations (Ochsner and Gross, 2005). Remarkably, reappraisal of high-arousal stimuli involves greater cognitive demands (Ortner, et al., 2016), as appeared in differential prefrontal recruitment (Silvers, et al., 2015). Unfortunately, the majority of our included studies have not reported the mean arousal of presented stimuli (63%). Relatedly, various negative stimuli (e.g., sad, disgust, and fearful) are reported to reflect similar arousals, but involve different emotion processing networks (Fusar-Poli, et al., 2009) and thereby, they probably engage distinct regulatory circuitries. Particularly, disorder-relevant stimuli are expected to be more motivationally salient than irrelevant ones. For example, reappraisal of social threats in SAD individuals involves excessive neural disturbances compared with physical threats (Goldin, et al., 2009b). Again, most of the included studies have not reported disorder-relevancy of presented stimuli (81%). Importantly, high burdens of down-regulating salient stimuli may be facilitated with a concurrent distraction strategy (Sheppes, et al., 2014), which activates the broader regulatory networks. Unfortunately, most of the included studies have not measured attentional engagement

during reappraisal using pupil dilation (94%), which cannot provide enough evidence regarding the substantial variance on regulatory brain circuits caused by attention deployment. Thus, at least a part of non-replicable results may stem from uncontrolled moderating factors related to external heterogeneous stimuli and/or internal diverse representations.

iii. Heterogeneous clinical populations

Clinical discrepancy of patients can be another important confounder causing divergent neuroimaging results. For instance, 31% of our included studies were medicated (only 9% lifetime drug-naïve). Meanwhile, the modulatory role of psychotropic medications on the emotional brain circuitries have been widely accepted (Roiser and Sahakian, 2013). Moreover, 19% of our studies were single-sex investigations. Again, gender difference entails neural differences in emotional reactivity and regulation (McRae, et al., 2008; Whittle, et al., 2011), including interactions between diagnosis and biological sex. Heterogeneous clinical entities of disorders should be also accounted for. As an example, MDD (25% of our included studies) is diagnosed by considering the presence of at least five of nine symptoms. Hence, it may present itself in a number of variants with different somatic/emotional/cognitive clinical states and a variety of pertinent neural disturbances (Rive, et al., 2013). Additionally, our included samples of a particular diagnosis (e.g., depression) had wide range of symptom severity and clinical status (e.g., acute, chronic, remitted), which clearly affects the quality/quantity of reappraisal-related brain activation (Dixon, et al., 2020; Stephanou, et al., 2017). Thus, such heterogeneity in clinical/demographical characteristics of patients may importantly contribute to the inconsistent finding in neural correlates of impaired reappraisal.

iv. Flexible methodology

Methodological flexibility in neuroimaging studies (e.g., image acquisition, preprocessing, software and analysis pipeline) (Bowring, et al., 2019; Kharabian Masouleh, et al., 2019) could also explain our null findings. In a very recent neuroimaging study, 70 independent teams analyzed the same dataset and interestingly, even no two teams performed identical analysis workflows. This analytical flexibility in neuroimaging had noticeable effects on the reported results and their interpretation (Botvinik-Nezer, et al., 2020). As a relevant example in our work, 16% of included studies have not applied any correction for multiple comparisons, which might produce non-replicable spurious findings. Moreover, the remaining 84% studies have not followed similar methodological approaches [78% of them used family-wise error (FWE) and 6% false-discovery rate (FDR)]. It is worthy to mention that FWE is statistically more conservative than FDR (Bennett, et al., 2009; Han and Glenn, 2018). Moreover, the included FWE-corrected results have not been

similarly calculated [59% with Monte Carlo simulation (MCS) and 19% with random field theory (RFT)]. The RFT seems to have less FWE rates than MCS (Nichols, 2012). Additionally, the MCS studies have not been analyzed alike [40% AFNI (34% version < 2017, 6% version > 2017 and 19% non-AFNI software)]. Indeed, a 15-year-old bug in the AFNI's MCS has been inflated false-positive rates, which was fixed later in 2017 (Cox, et al., 2017). Put together, the observed heterogeneity in reappraisal literature might be due to methodological diversity.

v. Questionable research practices and publication bias

Another explanation could be that the anticipated effect might not be so stringent, as hypothesized. As a matter of fact, scales are usually tipped in favor of publishing positive results. This provides the wrong motivations to manipulate the analytical parameters in order to obtain the significant relations, which sometimes leads to spurious findings. Thus, at least in some cases, the “faithful” reappraisal disturbances in clinical populations could have resulted from a biased overestimation. For instance, there is some evidences that MDD patients are able to efficiently use reappraisal, when they “explicitly trained to do so” (Ebneabbasi, 2020; Liu and Thompson, 2017). Several neuroimaging studies also reported the intact neural underpinning of reappraisal in MDD (Davis, et al., 2018; Doré, et al., 2018; Loeffler, et al., 2019; Rubin-Falcone, et al., 2020; Rubin-Falcone, et al., 2018). Importantly, when non-significant results are published, they are not usually entered in ALE meta-analyses. Indeed, insensitivity of ALE to non-significant results helps to increase the publication bias (Acar, et al., 2018). Notably, we excluded 22 eligible studies due to non-significant group comparisons results.

Collation with previous meta-analyses

Following the best-practice guideline for neuroimaging meta-analysis (Müller, et al., 2018; Tahmasian, et al., 2019) and rigorous methodological approach, our null result is expected to reflect a representation of the existing reappraisal studies across psychiatric disorders and should not be attributable to a lack of statistical power or statistical issues. Strikingly, we had some methodological differences with two previous ER meta-analyses on mood and anxiety disorders (Pico-Perez, et al., 2017) and various anxiety disorders (Wang, et al., 2018). Firstly, those meta-analyses used effect size signed differential mapping (ES-SDM), which is statistically more lenient than ALE. A previous simulation study showed that an uncorrected threshold of $p = .005$ and $SDM-Z > 1$ adequately controlled the probability of detecting an effect by chance (Radua, et al., 2012). However, this is an informal control of the false positive rate and may be too conservative or too liberal in other datasets. Secondly, here, we included 32 studies, comparing to substantially smaller number of studies in previous meta-analyses i.e., eight studies (Wang, et al., 2018) or 13

studies (Pico-Perez, et al., 2017). Of note, each of those meta-analyses included two non-significant studies. Thus, significant convergence might be driven by only few experiments. Thirdly, we applied stringent exclusion criteria compared with previous work that entered two non-eligible studies i.e., one non-whole-brain and one adolescence (Pico-Perez, et al., 2017). Fourthly, our search was not restricted to mood and anxiety disorders, and thereby, additional relevant disorders were covered.

Limitations and recommendations for future studies

Our study has some limitations. First, the number of patients in the included studies differed substantially across the psychiatric groups (e.g., $n=196$ in MDD and $n=17$ in substance use disorder and gambling disorder). The disproportionate share of coordinates, therefore, may affect the sensitivity of results to have a bias towards the clinical characteristics of larger groups. Moreover, none of the particular patient groups reached the minimum number of experiments to obtain the sufficient power for ALE analysis, which forecloses further representing the pathologically related differences in brain activation. In fact, beside pooled (ALL experiments) and between-group (patient<control and patient>control) contrasts, there were sufficient experiments only for few groupings of experiment (e.g., mood and anxiety) (Table 2), which restricts our understanding of reappraisal related difficulties across different diagnoses in spite of the task specificity. These notable limitations necessitate further studies on different psychiatric patient groups by using larger sample sizes and standard methodology ideally through collaborations to ameliorate site-idiosyncrasies, as well as sharing data openly to allow replication and future integration. Furthermore, to differentiate a true lack of localized consistency from clinical/methodological divergence, we propose the following recommendations for future clinical-neuroimaging studies: 1) investigate the neural correlates of model-driven/stage-based regulatory dysfunctions in clinical populations; 2) design the reappraisal experiments considering the experimental moderating factors such as stimulus features (e.g., valence, arousal and relevancy to disorder); 3) clearly report clinical (e.g., comorbidity, medication, age/gender and symptom severity) as well as methodological (e.g., preprocessing, software and analysis pipeline) characteristics for replication feasibility; and, 4) utilize stringent statistical thresholds to minimize the potential non-replicable spurious results.

Conclusion

The present meta-analysis demonstrated that the existing literature on emotion dysregulation has not yielded consistent, localized and cross-cutting neural anomaly during reappraisal

performance. We highlighted the distinct psychopathology of impaired reappraisal across different clinical populations, as well as divergent experimental, clinical and methodological factors that could explain our null results. This study, along with other transdiagnostic researches (Bagherzadeh-Azbari, et al., 2019; Goodkind, et al., 2015; Janiri, et al., 2020; McTeague, et al., 2016; Opel, et al., 2020; Schwartz, et al., 2019; Shao, et al., 2012) emphasizes the identification of concordant transdiagnostic abnormalities using task-based and resting-state neuroimaging methods. Such a transdiagnostic approach is necessary to guide the future studies and to refine and improve the current classification and treatment of mental disorders (Fusar-Poli, et al., 2019). For upcoming researches, searching for the potential neural congruency underlying various aspects of disturbances in emotional processing and regulation is promising.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Figure 1. Study selection strategy flow chart

Figure 2. Distribution of the included peak coordinates in the current study. The represented foci reflect functional alterations related to reappraisal tasks in patients with various psychiatric disorders compared to healthy subjects.

Table 1. Characteristics of 32 included studies in the present meta-analysis.

Abbreviations: MADRS (Montgomery-Asberg Depression Rating Scale), BDI (Beck Depression Inventory), HAM-D (Hamilton Depression Rating Scale), PANASS (Positive and Negative Syndrome Scale), LSAS-SR (Liebowitz Social Anxiety Scale - Self-Report), PDS (Posttraumatic Diagnostic Scale), CAPS (Clinician-Administered PTSD Scale), YMRS (Young Mania Rating Scale), QIDS (Quick Inventory of Depressive Symptomatology), BSL (Borderline Symptom List), ALS (Affect Lability Scale), BAI (Beck Anxiety Inventory), QASIS (Overall Anxiety Severity and Impairment Scale), HADS (Hospital Anxiety and Depression Scale), DRSP (Daily Record of Severity of Problems), OCI-R (Obsessive Compulsive Inventory-Revised), UPPS (urgency, premeditation, perseverance and sensation seeking scale).

Study	Number (M: F)		Mean age (SD)		Diagnosis	Medication (n)	Comorbidity (n)	Symptom severity	ER tactic / direction	Stimulus	Arousal/valence	Statistical threshold	Software	MRI
	Patients	Controls	Patients	Controls										
Major Depressive Disorder (MDD)														
(Stephanou et al., 2017)	53 (22: 31)	64 (24: 40)	19.72 (2.68)	19.03 (2.45)	SCID-IV	No (within last 4 weeks)	No (lifetime)	MADRS [MDD = 32.80 (4.80); controls = 2.14 (2.99)]	Reinterpretation/ Down-regulation	IAPS (negative social scenes), EPS, online sources	NA	FDR	SPM 8	3 T
(Wang et al., 2017)	12 (5: 7)	15 (7: 8)	29.50 (8.46)	25.80 (5.89)	SCID-IV	No (within last two weeks)	No (current psychiatric and lifetime neurologic)	BDI [MDD = 26.17 (12.65); controls = 4.27 (4.23)]	Reinterpretation-Distancing/ Down- and up-regulation	IAPS (positive and negative images), other sources	NA	FWE (REST, AlphaSim)	SPM 8	3 T
(Radke et al., 2018)	22 (13: 9)	22 (13: 9)	32.6 (10.9)	34.5 (9.9)	SCID-IV	15	3	BDI [MDD = 13.8 (9.5); controls = 2.7 (3.4)]	Distancing/ Up-regulation	FACES (angry face)	NA	FWE	SPM 8	3 T
(Greening et al., 2014)	19 (6: 13)	19 (6: 13)	26.79 (11.4)	27.63 (11.0)	SCID-IV	9	7	BDI [MDD = 25.53 (10.4); controls = 1.6 (2.3)]	Reinterpretation/ Down-regulation	IAPS (sad and positive scenes)	Normative mean arousal sad = 5.08 (.62); mean arousal positive = 5.03 (.55)	FWE (AlphaSim)	AFNI 2012	3 T
(Smoski et al., 2013)	18 (4: 15)	19 (7: 12)	24.5 (5.4)	27.9 (6.3)	SCID-IV	5	No (current)	BDI [MDD = 2.9 (5.0); controls = 1.4 (2.4)]	Reinterpretation-Distancing/ Down-regulation	IAPS (sad pictures), other normed images	NA	FWE (AFNI, 3dClustSim)	FSL	3 T
(Heller et al., 2009)	27 (12: 15)	19 (9: 10)	31.48 (11.58)	31.84 (14.65)	DSM-IV	No (lifetime)	No (current psychiatric and lifetime bipolar/ anxiety)	HAMD [MDD = 20.6 (2.39); controls = 1.2 (1.6)]	Reinterpretation-Distancing/ Down- and up-regulation	IAPS (positive and negative pictures)	Normative mean valence negative = 2.95 (.87); mean arousal negative = 5.44 (.8); mean valence positive = 7.13 (.62); mean arousal	FWE (AlphaSim)	AFNI	3 T

											positive = 5.28 (.58)			
(Sheline et al., 2009)	24 (12: 12)	21 (6: 15)	34 (9.4)	35 (7.3)	DSM-IV	No (within last 4 weeks)	No (lifetime)	HAMD [MDD = 21 (3.5); controls = 0 (.04)]	Reinterpretation- Distancing/ Down-regulation	IAPS (positive and negative pictures)	NA	NA	NA	3 T
(Johnstone et al., 2007)	21 (8: 13)	28 (7: 21)	33 (12)	28 (12)	DSM-IV	No (current)	No (current psychiatric and lifetime bipolar)	HAMD [MDD = 21 (2.5); controls = .5 (.6)]	Reinterpretation- Distancing/ Down- and up-regulation	IAPS (positive and negative pictures)	Normative mean valence negative = 2.95 (.87); mean arousal negative = 5.44 (.8); mean valence positive = 7.13 (.62); mean arousal positive = 5.28 (.58)	FWE (AlphaSim)	AFNI	3 T
Schizophrenia														
(Zhang et al., 2020)	16 (12: 4)	15 (10: 5)	31.75 (8.7)	33.60 (11.1)	DSM-IV and ICD-10	NA	No (current)	PANSS [schizophrenia = 26.69 (7.1)]	Reinterpretation/ Down-regulation	IAPS (negative pictures)	NA	FWE (AFNI 2018, 3dClustSim)	SPM 12	3 T
(Larabi et al., 2018)	30 (22: 8)	15 (10: 5)	35 (10.16)	33.6 (11.11)	DSM-IV and ICD-10	28	No (current)	PANSS [schizophrenia = 57.9 (14.71)]	Reinterpretation/ Down-regulation	IAPS (negative images)	Normative mean valence negative = 2.6; mean arousal negative = 5.7; mean valence neutral = 1.3; mean arousal neutral = 1.9	FWE	SPM 12	3 T
(van der Meer et al., 2014)	20 (14: 6)	20 (16: 4)	35.5 (11.7)	35.2 (10.8)	DSM-IV and ICD-10	20	NA	PANSS [schizophrenia = 29.9 (7.7)]	Reinterpretation/ Down-regulation	IAPS (negative images)	Normative mean valence negative = 2.6; mean arousal negative = 5.7; mean valence neutral = 1.3; mean arousal neutral = 1.9	FWE	SPM 5	3 T
(Morris et al., 2012)	12 (8: 4)	15 (6: 9)	44 (3)	35 (2)	DSM-IV	12	NA	PANSS [schizophrenia = 32 (2)]	Distancing/ Down- and up-regulation	IAPS (negative threat and suffering images)	NA	FWE	SPM 8	3T
Social Anxiety Disorder (SAD)														

(Ziv et al., 2013)	27 (15: 12)	27 (14: 13)	31.1 (7.6)	32.6 (9.5)	DSM-IV	No (current)	8	LSAS-SR [SAD = 99.3 (11.8); controls = 15.3 (9.1)]	Reinterpretation/ Down-regulation	Anger and contempt facial expressions	NA	FWE (AlphaSim)	AFNI	3 T
(Goldin et al., 2009a)	15 (6: 9)	17 (8: 9)	31.6 (9.7)	32.1 (9.3)	DSM-IV	No (current)	No (current psychiatric and lifetime neurologic)	LSAS-SR [SAD = 67.6 (21.1); controls = 29.3 (20.9)]	Distancing/ Down-regulation	Facial Action Coding System (angry and contempt facial expression), and physical threat scenes	NA	FWE (AlphaSim)	AFNI	3 T
(Goldin et al., 2009a)	27 (15: 12)	27 (15: 12)	32.1 (9.2)	32.2 (9.5)	DSM-IV	No (current)	6	LSAS-SR [SAD = 80.1 (16.8); controls = 15.7 (8.7)]	Reinterpretation/ Down-regulation	Written social situations	NA	FWE (AlphaSim)	AFNI	3 T
Post-traumatic Stress Disorder (PTSD)														
(Butler et al., 2018)	18 (18: 0)	27 (27: 0)	28.3 (6.4)	32.7 (5.9)	ICD-10	No (current)	No (lifetime axis-II)	PDS [PTSD = 36.28 (10.65); controls = 15.7 (8.7)]	Reinterpretation/ Down-regulation	Combat images	NA	FWE (AFNI 2016, 3dClustSim)	SPM 8	3 T
(Xiong et al., 2013)	20 (13: 7)	20 (14: 6)	32.92 (8.48)	31.53 (7.43)	SCID-IV	No (lifetime)	No (lifetime, except of past depression)	CAPS [PTSD = 52.33 (9.44); controls = 8.26 (9.31)]	Reinterpretation/ Down- and up-regulation	IAPS (negative images)	Normative mean valence negative = 2.17 (.34); mean arousal negative = 6.23 (.26); mean valence neutral = 5.12 (1.04); mean arousal neutral = 4.18 (.72)	FWE (REST, AlphaSim)	SPM 8	3 T
(New et al., 2009)	14 (0: 14)	14 (0: 14)	38.7 (11.2)	31.7 (10.3)	SCID-IV	No (lifetime)	No (lifetime, except of past depression)	CAPS [PTSD = 69.1 (17.6)]	Reinterpretation/ Down- and up-regulation	IAPS (negative images)	NA	FWE (REST, AlphaSim)	SPM 2	3 T
Bipolar Disorder (BD)														
(Zhang et al., 2020)	15 (6: 9)	15 (10: 5)	39.87 (12.5)	33.60 (11.1)	DSM-IV and ICD-10	NA	No (current)	YMRS [BD = 1.4 (1.5)] and QIDS [BD = 5.27 (5.4)]	Reinterpretation/ Down-regulation	IAPS (negative pictures)	NA	FWE (AFNI 2018, 3dClustSim)	SPM 12	3 T
(Townsend et al., 2013)	30 (19: 11)	26 (15: 11)	37.9 (12.6)	35.5 (12.4)	SCID-IV	21	No (current psychiatric and lifetime)	YMRS [BD = 1.7 (2.2)] and HAM-D [BD = 3.8 (1.9)]	Reinterpretation/ Down-regulation	IAPS (negative images)	Normative mean valence negative = 2.8; mean	NA	FSL	3 T

							substance use/abuse)				arousal negative = 6.5			
(Morris et al., 2012)	13 (8: 5)	15 (6: 9)	41 (3)	35 (2)	DSM-IV	13	NA	NA	Distancing/ Down- regulation	IAPS (negative threat and suffering images)	NA	FWE	SPM 8	3 T
Borderline Personality Disorder (BPD)														
(van Zutphen et al., 2018)	55 (0: 55)	42 (0: 42)	30.88 (8.78)	28.33 (10.50)	SCID-IV	55	49	BPD checklist [BPD = 120.6 (26.92); controls = 50.73 (5.03)]	Reinterpretation/ Down- and up- regulation	IAPS (negative and positive), additional erotic pictures	NA	FWE	BrainVoyager	3 T
(Schulze et al., 2011)	15 (0: 15)	15 (0:15)	27.60 (7.85)	24.53 (2.85)	SCID-IV	NA	7	BSL [BPD = 183.87 (53.64); controls = 49.60 (16.04)]	Distancing/ Down- and up- regulation	IAPS (negative threat and suffering images)	NA	FWE	SPM 5	1.5 T
(Koenigsberg et al., 2009)	18 (8: 10)	16 (9: 9)	32.6 (10.4)	31.8 (7.7)	SCID-IV	No (within last 4 weeks)	No (lifetime)	ALS [BPD = 94.9 (23.7); controls = 20.3 (16.0)]	Distancing/ Down- regulation	IAPS (interpersonal situations)	Normative mean valence negative = 2.35; mean arousal negative = 5.9; mean valence neutral = 5.2; mean arousal neutral = 3.65	FWE (REST, AlphaSim)	SPM 2	3 T
Miscellaneous Anxiety Disorders														
(Blair et al., 2012)	53 (17: 36)	18 (8: 10)	33.73 (9.99)	33.4 (9.65)	SCID-IV	No (within last 6 months)	No (current)	BAI [miscellaneous = 10.93 (7.17); controls = 2.3 (2.02)]	Reinterpretation/ Down- regulation	IAPS (positive and negative images)	Normative mean valence negative = 3.08; mean arousal negative = 5.43; mean valence positive = 7.21; mean arousal positive = 5.15	FWE (AlphaSim)	AFNI	1.5 T
(Ball et al., 2013)	41 (9: 32)	22 (11: 11)	32 (9)	27 (9)	DSM-IV	No (within last 2 weeks)	17	QASIS [miscellaneous = 8.6 (3.3); controls = .8 (1.2)]	Reinterpretation- Distancing/ Down- regulation	IAPS (negative images)	NA	FWE (3dClustSim)	AFNI	3 T
(Campbell-Sills et al., 2011)	13 (2: 11)	13 (2: 11)	NA	NA	SCID-IV	No (lifetime)	12	NA	Reinterpretation/ Down- regulation	IAPS (negative images)	NA	FWE (AlphaSim)	AFNI	3 T
Panic Disorder (PD)														

(Reinecke et al., 2015)	18 (4: 14)	18 (4: 14)	36.5 (13.8)	32.3 (12.1)	SCID-IV	3	13	HADS-Anxiety [PD = 14.6 (4.1); controls = 2.0 (1.6)]	Reinterpretation/ Down-regulation	IAPS (accidents or funerals)	Normative mean valence negative = 2.8 (1.7); mean arousal negative = 6.0 (2.2)	NA	FSL	3 T
Pre-menstrual Dysphoric Disorder (PMDD)														
(Petersen et al., 2018)	18 (0: 18)	18 (0: 18)	29.2 (7.24)	25.4 (6.99)	SCID-IV	No (current)	No (lifetime except of unipolar mood disorders)	DRSP [PMDD = 3.53 (.63); controls = 1.01 (.05)]	Distancing/ Down-regulation	IAPS (negative images) and other ones	NA	NA	FSL	3 T
Obsession-Compulsion Disorder (OCD)														
(Thorsen et al., 2019)	43 (21: 22)	38 (18: 20)	37.58 (10)	39.05 (11.27)	SCID-IV	No (within last 4 weeks)	29	OCI-R [OCD = 24.67 (11.79); controls = 3.37 (4.71)]	Reinterpretation- Distancing/ Down-regulation	Fearful and OCD-related pictures	NA	NA	SPM 8	3 T
Substance Use Disorder (SUD)														
(Albein-Urios et al., 2014)	17 (16: 1)	18 (17: 1)	36.41 (5.99)	30.50 (4.64)	SCID-IV	NA	NO (current psychiatric and lifetime neurologic)	UPPS-Negative urgency [SUD = 33.17 (6.51); controls = 22.22 (5.1)]	Reinterpretation- Distancing/ Down-regulation	IAPS (negative images)	Normative mean valence negative = 3.51 (.86); mean arousal negative = 5.70 (0.6)	FDR	SPM 8	3 T
Gambling Disorder (GD)														
(Navas et al., 2017)	17 (16: 1)	21 (20: 1)	32.94 (7.77)	31 (4.6)	SCID-IV	NA	No (current psychiatric and lifetime neurologic)	UPPS-Negative urgency [GD = 29.18 (4.7); controls = 23.19 (5.46)]	Reinterpretation/ Down-regulation	IAPS (negative mutilation pictures)	Normative mean valence negative = 3.51 (.86); mean arousal negative = 5.70 (0.6)	FWE (REST, AlphaSim)	SPM 8	3 T

Table 2. Findings of conducted meta-analyses in patients compared to healthy subjects. Abbreviations: ALE (Activation Likelihood Estimation), TFCE (Threshold-Free Cluster Enhancement), cFWE (cluster-level Family-Wise Error). *Since at least 17 experiments provide sufficient power for statistical analysis, some sub-analyses were not performed.

Domain	ALE	Contrast	P-value		Number of experiments
			TFCE	cFWE	
Task	Reappraisal	All	.406	.418	28
	Reappraisal	Patients < Controls	.436	.570	21
	Reappraisal	Patients > Controls	.675	.832	20
Regulation direction	Down-regulation	All	.662	.859	27
	*Up-regulation	All	n/s	n/s	9 < 17
Stimulus valence	Negative	All	.615	.930	28
	*Positive	All	n/s	n/s	3 < 17
Disorder category	Mood and Anxiety	All	.120	.076	20
	*Mood	All	n/s	n/s	10 < 17
	*Anxiety	All	n/s	n/s	10 < 17
	*Others	All	n/s	n/s	8 < 17
Reappraisal tactic	*Reinterpretation	All	n/s	n/s	14 < 17
	*Distancing	All	n/s	n/s	7 < 17
	*Reinterpretation-Distancing	All	n/s	n/s	7 < 17

Table S1. List of excluded studies with reasons

No	Study	Reason of exclusion
1	(Kreifelts et al., 2017)	Non-significant results
2	(Fitzgerald et al., 2017a)	Non-significant results
3	(Corbalán et al., 2015)	Non-significant results
4	(Gaebler et al., 2014)	Non-significant results
5	(Dillon and Pizzagalli, 2013)	Non-significant results
6	(Rubin-Falcone et al., 2020)	Non-significant results
7	(Dixon et al., 2020)	Non-significant results
8	(Jansen et al., 2019b)	Non-significant results
9	(Jansen et al., 2019a)	Non-significant results
10	(Loeffler et al., 2019)	Non-significant results
11	(Chrysikou et al., 2019)	Non-significant results
12	Materna et al., 2019)	Non-significant results
13	(Davis et al., 2018)	Non-significant results
14	(Doré et al., 2018)	Non-significant results
15	(Rubin-Falcone et al., 2018)	Non-significant results
16	(Denny et al., 2015)	Non-significant results
17	(Wang et al., 2014)	Non-significant results
18	(Albein-Urios et al., 2013)	Non-significant results
19	(Fitzgerald et al., 2017b)	Non-significant results
20	(Schmitt et al., 2016)	Non-significant results
21	(Light et al., 2011)	Non-significant results
22	(Yip et al., 2018)	Non-significant results
23	(Hilland et al., 2018)	Lacking healthy controls
24	(Zhang et al., 2020)	Lacking healthy controls

25	(Contreras-Rodríguez et al., 2020)	Lacking healthy controls
26	(Fitzgerald et al., 2019)	Lacking healthy controls
27	(Schmitgen et al., 2019)	Lacking healthy controls
28	(Gorka et al., 2019)	Lacking healthy controls
29	(Klumpp et al., 2017b)	Lacking healthy controls
30	(Fonzo et al., 2017b)	Lacking healthy controls
31	(Fonzo et al., 2017a)	Lacking healthy controls
32	(Klumpp et al., 2017a)	Lacking healthy controls
33	(Reinecke et al., 2018)	Lacking healthy controls
34	(Klumpp et al., 2017c)	Lacking healthy controls
35	(Goldin et al., 2014)	Lacking healthy controls
36	(Reinecke et al., 2014)	Lacking healthy controls
37	(Brühl et al., 2013)	Lacking healthy controls
38	(Goldin et al., 2013a)	Lacking healthy controls
39	(Heller et al., 2013)	Lacking healthy controls
40	(Goldin et al., 2013b)	Lacking healthy controls
41	(Hermann et al., 2009)	Lacking healthy controls
42	(Kanske et al., 2015)	Non-whole-brain analysis
43	(Rabinak et al., 2014)	Non-whole-brain analysis
44	(Kanske et al., 2012)	Non-whole-brain analysis
45	(Lang et al., 2012)	Non-whole-brain analysis
46	(Erk et al., 2010)	Non-whole-brain analysis
47	(Douw et al., 2020)	Non-whole-brain analysis
48	(Klumpp et al., 2019)	Non-whole-brain analysis
49	(Jacob et al., 2019)	Non-whole-brain analysis
50	(Young et al., 2019)	Non-whole-brain analysis
51	(Zhang et al., 2018)	Non-whole-brain analysis
52	(Rive et al., 2015)	Non-whole-brain analysis

53	(De Wit et al., 2015)	Non-whole-brain analysis
54	(Ball et al., 2014)	Non-whole-brain analysis
55	(Hermann et al., 2013)	Non-whole-brain analysis
56	(Raschle et al., 2019)	Mean age < 18 or > 60
57	(Seidel et al., 2018b)	Mean age < 18 or > 60
58	(Seidel et al., 2018a)	Mean age < 18 or > 60
59	(LeWinn et al., 2018)	Mean age < 18 or > 60
60	(Cisler et al., 2016)	Mean age < 18 or > 60
61	(Murphy et al., 2016)	Mean age $\geq$ 18 or $\leq$ 60
62	(Belden et al., 2015)	Mean age < 18 or > 60
63	(Platt et al., 2015)	Mean age < 18 or > 60
64	(Karim et al., 2017)	Mean age < 18 or > 60
65	(Pitskel et al., 2014)	Mean age < 18 or > 60
66	(Andreescu et al., 2015)	Mean age < 18 or > 60
67	(Perlman et al., 2012)	Mean age < 18 or > 60
68	(Meluken et al., 2019)	High risk or subclinical diagnosis
69	(Simsek et al., 2017)	High risk or subclinical diagnosis
70	(Van Der Velde et al., 2015)	High risk or subclinical diagnosis
71	(Heissler et al., 2014)	High risk or subclinical diagnosis
72	(Felder et al., 2012)	High risk or subclinical diagnosis
73	(Modinos et al., 2010)	High risk or subclinical diagnosis
74	(Walsh et al., 2019)	Lacking group-comparison analysis
75	(Anand et al., 2019)	Lacking group-comparison analysis

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