Title

Neural substrates of the emotion-word and emotional counting Stroop tasks in healthy and clinical populations: a meta-analysis of functional brain imaging studies

Abbreviated title

Meta-analysis of the emotional Stroop task in healthy and clinical populations

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Abstract

The emotional Stroop task (EST) is among the most influential paradigms used to probe attention-related or cognitive control-related emotional processing in healthy subjects and clinical populations. The neuropsychological mechanism underlying the emotional Stroop effect has attracted extensive and long-lasting attention in both cognitive and clinical psychology and neuroscience; however, a precise characterization of the neural substrates underlying the EST in healthy and clinical populations remains elusive. Here, we implemented a coordinate-based meta-analysis covering functional imaging studies that employed the emotion-word or emotional counting Stroop paradigms to determine the underlying neural networks in healthy subjects and the trans-diagnostic alterations across clinical populations. Forty-six publications were identified that reported relevant contrasts (negative > neutral; positive > neutral) for healthy or clinical populations as well as for hyper- or hypo-activation of patients compared to controls. We demonstrate consistent involvement of the vlPFC and dmPFC in healthy subjects and consistent involvement of the vlPFC in patients. We further identify a trans-diagnostic pattern of hyper-activation in the prefrontal and parietal regions. These findings underscore the critical roles of cognitive control processes in the EST and implicate trans-diagnostic cognitive control deficits. Unlike the current models that emphasize the roles of the amygdala and rACC, our findings implicate novel mechanisms underlying the EST for both healthy and clinical populations.

Key words: emotion-word Stroop, emotional counting Stroop, activation likelihood estimation, fMRI, meta-analysis, cognitive control, emotional regulation

Introduction

The emotional Stroop effect and its clinical significance

Flexible goal-directed behaviors require the ability to control or inhibit task-irrelevant, often emotional information that interferes with ongoing task performance. The cognitive control of emotion required to maintain ongoing task demands has been extensively examined using the emotional Stroop task (EST) (MacLeod, 1991; Williams et al., 1996). In the most popular emotion-word version of the EST, participants are required to name the color of emotional or neutral words, while ignoring the semantic meaning of the words. Typically, the response times for naming the colors of emotional words are longer than those for neutral words, an effect referred to as "emotional Stroop effect" (Williams et al., 1996). Notably, the emotional words in the EST do not possess a semantic conflict with the target color (De Ruiter and Brosschot, 1994; Williams et al., 1996); rather, the response delay is induced by the emotional relevance of the word.

The robustness of the emotional Stroop effect promoted clinical interest in this effect. Gotlib and McCann (1984) were among the first to demonstrate that patients with depression show a stronger interference effect in response to depression-related words than healthy individuals do. This finding inspired several subsequent studies, which demonstrated, for example, that the interference in the EST in response to depression-relevant words is directly related to the severity of depressive symptoms (Epp et al., 2012). A large body of research has additionally demonstrated emotional interference in other mental disorders, particularly in anxiety and substance use disorders. For instance, interference in color-naming performance has been identified across different types of anxiety disorders (Bar-Haim et al., 2007; Williams et al., 1996), such that patients with spider phobia exhibit slower naming of spider-related words (Watts et al., 1986), whereas patients with social phobia exhibit slower naming of speech-related words (Becker et al., 2001; Lundh and Öst, 1996). Notably, the pronounced interference of anxiety-related words normalized with successful treatment, suggesting that

the interference in the EST represents a treatment-sensitive marker (Lavy et al., 1993; Mattia et al., 1993; Watts et al., 1986). Finally, robust interference effects have been demonstrated in patients with substance use disorders, who typically exhibit a strong color-naming interference for substance-related words, an effect that has been directly related to craving and drug seeking (Cox et al., 2006; Field et al., 2009; Hester et al., 2006). Together, these findings in clinical populations indicate that the emotional Stroop effect might reflect a reliable marker that is strongly related to the current psychopathological symptom load of the patient.

Psychological and computational models of the emotional Stroop effect

Despite the well-documented clinical significance, the neurocognitive mechanisms underlying the emotional Stroop effect remain a matter of debate (Algom et al., 2004; Dalgleish, 2005; McKenna and Sharma, 2004). Generally, the previous overarching frameworks attributed the interference effect to two underlying cognitive mechanisms: (i) fast and automatic attentional allocation to emotional stimuli (i.e., the 'automatic-attention' hypothesis) (Wentura et al., 2000; Williams et al., 1996; Williams et al., 1988); or (ii) strategic monitoring of salient information following exposure to emotional stimuli, which results in a reduction in the cognitive control required for the ongoing task (i.e., the 'strategic-monitoring' hypothesis) (Harley, 1996; Phaf and Kan, 2007; Todd et al., 2012; Wells and Matthews, 2014).

The automatic-attention hypothesis is in line with ample evidence indicating that emotional stimuli, presumably due to their motivational significance, capture attentional resources at early processing stages in a fast and automatic manner (Lang et al., 1990; LeDoux, 1998; Öhman and Mineka, 2001; Vuilleumier et al., 2001). For instance, even subliminally presented emotional stimuli interfere with color naming, as has been shown by studies employing backward masking procedures (MacLeod and Hagan, 1992; MacLeod and Rutherford, 1992; Putman et al., 2004). However, more recent studies examining interference effects at the level of single trials revealed a between-trial effect in the EST (Bertels and Kolinsky, 2016; Kunde and Mauer, 2008; McKenna, 1986; McKenna and Sharma, 2004;

Waters et al., 2005; Waters et al., 2003), which is hard to reconcile with a simple automatic-attentional bias for emotional stimuli. McKenna and Sharma (2004) demonstrated that both emotional and neutral words with a preceding emotional word showed prolonged color-naming reaction times, whereas no interference was observed for either emotional or neutral words following a neutral word. To account for the between-trial effects, the strategic-monitoring hypothesis suggests that the interference in the EST is based on a mechanism implicated in strategic reductions in the cognitive control of the ongoing task to monitor salient (emotional) information in the environment (Wells and Matthews, 2014; Wyble et al., 2008). That is, strategic reductions in cognitive control operate slower than the ongoing task performance, preferentially disrupting one's performance on subsequent trials (McKenna and Sharma, 2004).

These theoretical frameworks have increasingly been elaborated using computational modeling in combination with brain imaging approaches (Stolicyn et al., 2017; Wyble et al., 2005; Wyble et al., 2008). The 'conditioned task-set competition' (CTC) model emphasizes that conditioned responses to emotional words and associated neural mechanisms contribute to the interference effect in the EST (Stolicyn et al., 2017). Specifically, emotional words as conditioned stimuli evoke automatic conditioned responses (e.g., escape behavior) that compete with color-naming performance. On the neural level, conditioned stimuli and the associated behavioral responses critically rely on the amygdala and its inhibition of task-related representations in the dorsolateral prefrontal cortex (dlPFC) (Stolicyn et al., 2017). In contrast, the 'adaptive attentional control' (AAC) model proposes that the degree of task involvement is adaptively regulated by emotional information (Wyble et al., 2005; Wyble et al., 2008). In particular, emotional salience detected by the rostral anterior cingulate cortex (rACC) triggers a transient suppression of task-related attentional processing in the dorsal ACC (dACC) and dlPFC. Such a mechanism would facilitate the detection of salient and potentially behaviorally-relevant information in the environment (Wyble et al., 2008).

These models do not only propose neuropsychological mechanisms underlying the EST but also provide a framework to understand the exaggerated interference effects observed in clinical populations. For instance, according to the CTC model, the exaggerated interference in clinical populations can be attributed to a hyperactive amygdala (Stolicyn et al., 2017), whereas the AAC model attributes the exaggerated interference to a hyperactive rACC (Wyble et al., 2008). Although a meta-analysis does not allow direct assessment of the different hypotheses of these models, we will discuss our findings in light of the aforementioned theoretical frameworks.

Human brain imaging investigation of the emotional Stroop effect

Past decades have witnessed an increased interest in unveiling the neural underpinnings of the emotional Stroop effect in healthy subjects and the neural alterations that underlie exaggerated interference in the EST in clinical populations (Buhle et al., 2010). In line with the CTC model, emotional words in the EST elicited stronger amygdala responses than neutral words did (Henckens et al., 2012; Isenberg et al., 1999; Mohanty et al., 2005), and patients with anxiety or depression exhibited hyper-reactivity of the amygdala to task-irrelevant emotional words (Engels et al., 2010; Lagopoulos and Malhi, 2007). However, prior findings of the role of the amygdala in the EST have generally been equivocal, and many other studies have failed to replicate the enhanced activation of the amygdala in response to emotional words relative to neutral ones (Bremner et al., 2004; Compton et al., 2003; Dresler et al., 2012; George et al., 1997; Veroude et al., 2013).

Prior neuroimaging research also provided some support for the AAC model, such that the rACC has frequently been found to be engaged by the EST (George et al., 1994; Mitterschiffthaler et al., 2008; Mohanty et al., 2007; Shin et al., 2001; Whalen et al., 1998). Indeed, the EST has been employed as a robust approach to challenge rACC functioning in both healthy and clinical populations (Shin et al., 2001; Whalen et al., 1998; Wingenfeld et al., 2009). As predicted by the ACC model, enhanced rACC activation in response to emotional

distractors was associated with a concomitant decrease in dACC activity (Mohanty et al., 2007; Rahm et al., 2013). Furthermore, several studies have demonstrated hyper-activations of the rACC or exaggerated rACC-amygdala functional connectivity in patients with anxiety or depression relative to healthy controls (Britton et al., 2009). Similarly, activation of the rACC has been found to be positively correlated with response latencies for negative items in depressive people (Mitterschiffthaler et al., 2008). Together, these findings lend support to the AAC model, suggesting a pivotal role of the rACC in emotional processing and the generation of emotional responses that interfere with task-relevant processing. Of note, however, many other studies have emphasized the regulatory role of the rACC in down-regulating emotional interference signals in the amygdala (Egner et al., 2007; Etkin et al., 2006; Freed et al., 2009; Mayberg et al., 1997; Witthöft et al., 2013).

Finally, both models predict deactivation of cognitive control regions (e.g., dlPFC) in response to emotional distractors. In contrast to these predictions, however, the activity of prefrontal and parietal regions has been consistently observed during the EST (Dresler et al., 2012; Herrington et al., 2005; Lagopoulos and Malhi, 2007; Wingenfeld et al., 2009). These findings complement the notion of the engagement of a cognitive control network during tasks that target emotion-cognition interactions (Cromheeke and Mueller, 2014), including cognitive regulation of negative affect (Ochsner and Gross, 2005; Ochsner et al., 2012) and interference induced by emotional stimuli (Song et al., 2017; Xu et al., 2016). Therefore, it is conceivable that the presentation of emotional distractors induces enhanced regulatory engagement of cognitive control regions, rather than reduces activity in these regions. In summary, although recent neuroimaging studies have provided preliminary support for both the CTC and AAC models, the evidence is far from conclusive.

Aim of the present study

The current theoretical models aimed to describe the neural mechanisms of emotional interference in healthy subjects and the neural basis of disruptions in this domain in clinical

populations. However, these models are mainly based on qualitative and selective reviews of the literature, which are subject to biased conclusions (Borenstein et al., 2009). A meta-analytic approach may promote a more objective characterization of the neural systems underlying the emotional Stroop effects. Furthermore, the precise neural network underlying the exaggerated emotional Stroop effect observed across psychiatric patient populations remains to be determined. The current study aimed to address these issues by conducting a coordinate-based meta-analysis on the functional neuroimaging EST literature, which allows to quantitatively examining convergence across studies. Using this meta-analytic strategy, the present study aimed to (i) characterize the neural networks that underlie the EST in healthy subjects and to (ii) characterize common neural alterations that underlie the exaggerated EST interference observed in clinical populations. In particular, we determined (i) the neural systems that are consistently involved in the EST among healthy volunteers as well as across different clinical populations, and (ii) the neural systems that consistently exhibit hyper- or hypo-activation in patients relative to healthy controls.

Materials and Methods

Literature search and selection

In the initial step, a systematic online database search was performed in accordance with the PRISMA-guidelines (Shamseer et al., 2015). The search was performed during July 2017 and included the PubMed, ISI Web of Science and Google Scholar databases using the combination of relevant search terms (e.g., ['emotional distractor' OR 'affective distractor' OR 'emotional Stroop' OR 'affective Stroop' OR 'implicit emotional processing' OR 'implicit affective processing'] AND ['fMRI' OR 'magnetic resonance imaging' OR 'PET' OR ' positron emission tomography' OR 'neuroimaging']). In addition, we explored several other sources, including (1) the BrainMap database (http://brainmap.org); (2) the bibliography and citation

indices of the preselected articles; (3) the reference list of relevant reviews (Carretié, 2014; Levin et al., 2007; Mohanty and Sussman, 2013; Sussman et al., 2016); and (4) direct searches of the names of frequently occurring authors. The determined studies were further assessed according to the following criteria (**Figure 1**): first, subjects performed a color-word emotional Stroop task (George et al., 1994) or an emotional counting Stroop task (Whalen et al., 1998). The emotional counting Stroop paradigm was developed to minimize head movement during fMRI scanning by requiring a motor response instead of a spoken response. In this task, participants were instructed to report the number of presented words by pressing buttons, regardless of each word's meaning (Whalen et al., 1998; Whalen et al., 2006). These variants of the EST represent the prevailing EST paradigms employed in cognitive and clinical psychology (Phaf and Kan, 2007; Williams et al., 1996). The semantic meanings of the emotional words (distractors) used in these tasks do not directly conflict with the participants' responses to printed color (target); instead, disruptions in performance are associated with the emotional significance of the words (Algom et al., 2004; Dalgleish, 2005). That is, emotional conflict in these traditional EST paradigms does not reflect response conflict, but rather reflects conflict in the sense that emotional distractors divert processing resources away from the ongoing task demands (Krug and Carter, 2010). In contrast, more recent versions of the EST include the simultaneous presentation of congruent (e.g., the word "HAPPY" displayed on a smiling face) or incongruent information (e.g., the word "HAPPY" displayed on an angry face) (e.g., Etkin et al., 2006) or use a priming procedure with emotional material before administration of the cognitive Stroop paradigm (e.g., Hart et al., 2010). Thus, these tasks inherently involve response conflict between task-relevant and task-irrelevant information. Accordingly, we excluded these variants of the EST that involve a direct cognitive or emotional conflict (e.g., Etkin et al., 2006; Hart et al., 2010; Melcher et al., 2011). Recent meta-analyses of these latter EST tasks are provided in Song et al. (2017) and Xu et al. (2016). Second, we restricted the meta-analysis to studies that employed the fMRI or

PET imaging modality. Third, the studies reported whole-brain general-linear-model-based results (rather than region of interest [ROI] analyses). Fourth, the results were derived from a general linear model based on either a binary contrast or parametric analyses. Finally, activations were presented in standardized stereotaxic space (Talairach or Montreal Neurological Institute, MNI). Note that for studies reporting Talairach coordinates, a conversion to the MNI coordinates was employed, as implemented in the GingerALE software with Brett's mni2tal algorithm (http://imaging.mrc-cbu.cam.ac.uk/imaging/MniTalairach). Filtering the search results according to the inclusion/exclusion criteria yielded a total of 46 published fMRI or PET articles (**Table 1**). It should be noted that a potential overlap of the subject samples in the selected publications cannot be ruled out.

< Insert Figure 1 & Table 1 here >

Main activation likelihood estimation (ALE) approach

A coordinate-based meta-analysis of reported fMRI studies was conducted, employing the ALE algorithm (in-house MATLAB scripts) (Eickhoff et al., 2017; Eickhoff et al., 2009). The ALE algorithm determines the convergence of foci reported from different functional (e.g., blood-oxygen-level dependent contrast imaging) or structural (e.g., voxel-based morphometry) neuroimaging studies with published foci in either the Talairach or MNI space (Laird et al., 2005; Turkeltaub et al., 2002). The ALE algorithm interprets reported foci as spatial probability distributions, whose widths are based on empirical estimates of the spatial uncertainty due to the between-subject and between-template variability of the neuroimaging data (Eickhoff et al., 2009). The ALE algorithm weights the between-subject variability based on the number of subjects analyzed in the studies, modeling larger sample sizes with smaller Gaussian distributions and, thus, presupposing more reliable approximations of the 'true' activation observed in larger sample sizes (Eickhoff et al., 2009).

The union of the individual modulated activation maps first created from the maximum probability associated with any one focus (always the closest one) for each voxel (Turkeltaub et al., 2012) is then calculated to obtain an ALE map across studies. This ALE map is assessed against a null-distribution of random spatial associations between studies using a non-linear histogram integration algorithm (Eickhoff et al., 2012; Turkeltaub et al., 2012). In addition, the average non-linear contribution of each experiment for each cluster was calculated from the fraction of the ALE values at the cluster with and without the respective experiment (Eickhoff et al., 2016). Based on the calculated contribution, we employed an additional two criteria to select significant clusters: (1) the contributions to one cluster were from at least two experiments to prevent the findings from being driven by the results from a single study; and (2) the average contribution of the most dominant experiment (MDE) did not exceed 50%, and the average contribution of the two most dominant experiments (2MDE) did not exceed 80% (Eickhoff et al., 2016).

Applying the ALE algorithm, the reported coordinates of the brain areas associated with the emotional Stroop effect were converged across different experiments. Specifically, the neural signatures of the emotional Stroop were converged using the following meta-analytic strategies: (i) the emotional Stroop effect in healthy subjects (i.e., negative words > neutral words; positive words > neutral words; 45 contrasts, 337 foci, and 997 subjects); (ii) emotional Stroop effect in clinical populations (i.e., negative/concern-related words > neutral words; positive words > neutral words; 20 contrasts, 211 foci, and 392 subjects**, Table 2**); (iii) hyper-activity in patients relative to controls (37 contrasts, 220 foci, 1182 subjects**, Table 2**); (iv) hypo-activation in patients relative to controls (18 contrasts, 83 foci, and 509 subjects**, Table 2**); and (v) aberrant activation in patients relative to controls that pooled across the coordinates of hyper- and hypo-activation in patients (55 contrasts, 303 foci, and 1691 subjects), including data from the patient samples listed in the abovementioned analyses of hyper-activity and hypo-activity (for a similar approach see also McTeague et al., 2017).

< Insert Table 2 here >

Validation analysis

We implemented additional analyses to validate the findings derived from the conventional the ALE meta-analysis approach. First, we implemented a leave-one-experiment-out (LOEO) analysis for each of ALE meta-analyses to ensure that the main meta-analytic results were not driven by the coordinates from a single contrast. In each fold, one contrast was excluded and the ALE meta-analysis was conducted on the remaining N-1 contrasts. Subsequently, we conducted a conjunction analysis on the ALE results of all folds to identify the brain regions that were robustly engaged by the EST. As such, the identified brain regions were present in *all* folds of the LOEO analysis. These analyses were employed to validate our main ALE meta-analytic findings.

Second, several of the identified studies were conducted in populations with subclinical disorders (e.g., high-anxiety individuals) (e.g., Canli et al., 2004; Witthöft et al., 2013) or in high-risk populations (e.g., first-degree relatives of depressive patients) (Canli et al., 2005; Kaiser et al., 2015; Mannie et al., 2008; Sebastian et al., 2010). Therefore, we implemented additional meta-analyses to identify the correspondence across clinical and subclinical/vulnerable populations by synthesizing the neuroimaging findings from different lines of clinical/subclinical research (**Table S1**).

Third, previous meta-analyses have indicated that the emotional Stroop effect can be induced by both negative and positive words among both healthy and clinical populations (Epp et al., 2012; Pool et al., 2016). Therefore, our main meta-analyses focused on the contrasts between emotional words (both negative and positive) versus neutral words, excluding the contrasts between negative and positive words. In a final validation analysis, we focused specifically on the contrasts related to negative or concern-related words (i.e.,

negative/concern-related words > neutral words; negative/concern-related words > positive words), excluding the contrasts associated with positive words (**Table S2**).

All maps were thresholded using a cluster-level family-wise error (cFWE) correction ($P < 0.05$) with a cluster-forming threshold of $P < 0.001$ using 10,000 permutations for correcting multiple comparisons. Please note that all of the current analyses were implemented using key functions of the most recent version of GingerALE (version 2.3.6), which uses valid multiple-comparison corrections (Eickhoff et al., 2017).

Results

Main ALE meta-analyses

In healthy volunteers, consistent maxima were identified in the left ventrolateral prefrontal cortex (vlPFC), the dorso-medial prefrontal cortex (dmPFC) and the middle occipital gyrus (**Figure 2 & Table 3**). Sixteen out of 45 contrasts contributed to the cluster in the left vlPFC (MDE = 10.77%; 2MDE = 21.26%). Eleven contrasts contributed to the cluster in the dmPFC (MDE = 18.58%; 2MDE = 33.47%). Five contrasts contributed to the cluster in the middle occipital gyrus (MDE = 25.51%; 2MDE = 49.3%) (**Table S3**).

< Insert Figure 2 & Table 3 here >

In the clinical populations, consistent maxima were found in two clusters in the left vlPFC (**Figure 3 & Table 3**). Nine out of 20 contrasts contributed to the first cluster in the vlPFC (MDE = 19.88%; 2MDE = 39.53%). Eight contrasts contributed to the second cluster in the vlPFC (MDE = 21.14%; 2MDE = 38.21%) (**Table S4**).

< Insert Figure 3 here >

Examining the contrasts of hyper-activation in patients relative to controls demonstrated consistent maxima in the left vlPFC, right dorso-lateral PFC (dlPFC), inferior parietal lobule (IPL), bilateral dmPFC, and posterior cingulate cortex (PCC) (**Figure 4 & Table 3**). Eleven

out of 37 contrasts contributed to the cluster in the dmPFC (MDE = 16.00%; 2MDE = 32.00%). Seven contrasts contributed to the cluster in the right IPL (MDE = 19.57%; 2MDE = 39.14%). Five contrasts contributed to two clusters in the right dlPFC (cluster 1: MDE = 27.41%; 2MDE = 54.82%; cluster 2: MDE = 24.62%; 2MDE = 49.24%). Five contrasts contributed to the cluster in the PCC (MDE = 24.13%; 2MDE = 48.26%). Four contrasts contributed to the cluster in the left vlPFC (MDE = 25.89%; 2MDE = 51.78%) (**Table S5**).

< Insert Figure 4 here >

No significant consistent maxima were identified for the contrasts of hypo-activation in patients relative to healthy controls.

Examining the contrasts of aberrant activation in the patients compared to the controls, consistent maxima were revealed in the right dlPFC, IPL, bilateral dmPFC, dorsal anterior cingulate cortex (dACC), and PCC (**Figure S1 & Table 4**). Twelve out of 55 contrasts contributed to the cluster in the dmPFC (MDE = 15.33%; 2MDE = 30.66%). Eight contrasts contributed to the cluster in the right IPL (MDE = 20.33%; 2MDE = 40.66%). Seven contrasts contributed to the cluster in the right dlPFC (MDE = 26.34%; 2MDE = 52.68%). Seven contrasts contributed to the cluster in the dACC (MDE = 23.62%; 2MDE = 47.24%). Five contrasts contributed to the cluster in the PCC (MDE = 23.71%; 2MDE = 47.42%) (**Table S6**).

Validation analyses: the LOEO analysis

In healthy volunteers, consistent maxima in the left vlPFC, dmPFC and middle occipital gyrus were identified in all folds of the LOEO analysis (**Figure 5a & Table 4**).

In the patients, consistent maxima in the left vlPFC were identified in all folds of the LOEO analysis (**Figure 5b & Table 4**).

< Insert Figure 5 & Table 4 here >

With respect to the contrasts of hyper-activation in patients relative to controls, consistent maxima in the left vlPFC, right dlPFC, IPL, bilateral dmPFC, and PCC were found in all folds of the LOEO analysis (**Figure 5c & Table 4**).

No significant consistent maxima were identified for the contrasts of hypo-activation related contrasts.

With respect to the aberrant activation in patients compared to controls, consistent maxima in the left vlPFC, right dlPFC, IPL, bilateral dmPFC, dACC and PCC were revealed in all folds of the LOEO analysis (**Figure S2 & Table 4**).

Validation analyses: combining clinical and subclinical groups

Combining the neuroimaging findings from both clinical and subclinical/vulnerable populations revealed consistent maxima in the bilateral vlPFC (**Figure S3a, Table S7 & Table S8**).

In regard to the hyper-activation related contrasts, consistent maxima were identified in the right dlPFC, IPL, bilateral dmPFC and dACC (**Figure S3b, Table S7 & Table S9**). No significant consistent maxima were identified for the contrasts of hypo-activation related contrasts.

With respect to the contrasts of the aberrant activation of patients compared to controls, consistent maxima were identified in the right IPL, bilateral mPFC and dACC (**Figure S3c, Table S7 & Table S10**).

Validation analyses: contrasts associated with negative words

Focusing on the contrasts related to negative or concern-related words, consistent maxima were found in the following brain regions among healthy volunteers: the left vlPFC and the dmPFC (**Figure 6a, Table 5 & Table S11**). For the clinical populations, consistent maxima were identified in the left vlPFC (**Figure 6b, Table 5 & Table S12**).

With respect to the hyper-activation related contrasts, consistent maxima were revealed in the right dlPFC, bilateral dmPFC and PCC (**Figure 6c, Table 5 & Table S13**). No

significant consistent maxima were identified for the contrasts of hypo-activation related contrasts.

Regarding the contrasts of aberrant activation in patients compared to controls, consistent maxima were revealed in the left amygdala, right dlPFC, bilateral dACC and PCC (**Figure 6d, Table 5 & Table S14**).

Taken together, the validation analyses confirmed the robustness of the findings obtained from the main ALE meta-analyses.

< Insert Figure 6 & Table 5 here >

Discussion

Using a coordinate-based approach, the present meta-analysis employed a quantitative approach to delineate the neural underpinnings of two popular EST paradigms: emotion-word and emotional counting Stroop (Cox et al., 2006; Phaf and Kan, 2007; Williams et al., 1988). Specifically, we aimed to determine the brain regions that are consistently engaged in the EST in healthy individuals and to determine the brain regions that underlie the consistently observed exaggerated interference during the EST in clinical populations. Our results demonstrated a convergent involvement of brain regions critically engaged in cognitive control and emotion regulation (Duncan, 2010; Duncan and Owen, 2000; Ochsner and Gross, 2005, 2008). Specifically, consistent involvement of the vlPFC and dmPFC was identified in healthy controls whereas patients most consistently recruited the vlPFC. Furthermore, we identified a trans-diagnostic pattern of hyper-activation in multiple cognitive control regions, including the vlPFC, dmPFC, dlPFC, and parietal cortex. Critically, our main findings remained robust across several validation approaches, including eliminating the effect of a single contrast, combining both clinical and subclinical/vulnerable populations, and focusing on contrasts particularly related to negatively valenced words.

Implications for the current models of the emotional Stroop effect

In contrast to the predictions of both the AAC and CTC models, the present analysis revealed a robust recruitment of cognitive control regions, particularly the dmPFC, vlPFC, dlPFC and the parietal cortex during the EST. These cognitive control regions have previously been shown to be engaged in processing across diverse cognitive domains, ranging from selective attention to working memory and response selection (Duncan, 2010, 2013; Duncan and Owen, 2000). In particular, the dmPFC has been implicated in the monitoring of ongoing performance and in the adaptive control of attention (Botvinick et al., 2001; Bush et al., 2000), suggesting that this region may be involved in monitoring of different response tendencies and in signaling the need for regulation. Activity of the vlPFC has been associated with inhibitory control of prepotent responses and information from semantic memory (Aron et al., 2004; Swick et al., 2008; Thompson-Schill et al., 1998; Whitney et al., 2010), implicating a role of inhibitory control of the emotional distractors to maintain task performance. Finally, the dlPFC and the parietal cortex have a well-established role in directing attention based on current goals and task demands (Curtis and D'Esposito, 2003; Fan et al., 2002; Posner and Rothbart, 2007). Accordingly, they may be responsible for directing attention to task-relevant stimulus features (e.g., color) and for maintaining current task sets in the EST context. Overall, prefrontal and parietal regions may implement different mechanisms to facilitate cognitive control and suppress interferences by emotional distractors to maintain performance.

Evidence from previous research examining the EST has provided further support for the regulatory role of the cognitive control network. First, examinations of both the emotional and classical Stroop effect in the same sample demonstrated that these regions were recruited across both tasks (Compton et al., 2003; Kaiser et al., 2015; Mincic, 2010; Rahm et al., 2014). Likewise, Davis et al. (2005) identified a population of neurons in the human dmPFC/dACC that respond to both cognitive and emotional Stroop tasks. Moreover, an intervention study revealed that behavioral performance was improved for both emotional and cognitive Stroop

tasks after treatment of the dmPFC/dACC (To et al., 2017). Second, the dmPFC, vlPFC, and dlPFC have been shown to exhibit increased negative functional coupling with the amygdala during the presentation of task-irrelevant emotional words (Britton et al., 2009; Henckens et al., 2012; Price et al., 2011). Third, neural activity of the dmPFC, vlPFC and dlPFC was found to correlate negatively with the magnitude of the emotional Stroop effect at the behavioral level (Mincic, 2010; Price et al., 2011).

Together, the current findings indicate that cognitive control regions, such as the dmPFC, vlPFC and dlPFC may regulate the influence of emotional material on ongoing task demands. In line with our findings, several recent meta-analytic studies reported a consistent involvement of cognitive control regions in task paradigms probing emotion-cognition interactions (Cromheeke and Mueller, 2014; Song et al., 2017; Xu et al., 2016). Together, these findings do not lend support to the notion that emotional distractors induce a reduction in the cognitive control of ongoing task demands, as proposed by the ACC and CTC models (Stolicyn et al., 2017; Wyble et al., 2008). Furthermore, the current work did not reveal a consistent involvement of the amygdala or the rACC as predicted by these models. Given the critical engagement of these regions in emotion processing, the lack of meta-analytic support for their consistent contribution to the EST is counterintuitive and surprising.

Several potential explanations for the unexpected findings are conceivable. First, the absence of consistent amygdala engagement is in line with recent meta-analytic findings suggesting that the amygdala is not consistently involved in other variants of the EST (Song et al., 2017; Xu et al., 2016). However, another meta-analysis reported consistent involvement of the amygdala during emotion cognition interactions (Cromheeke and Mueller, 2014). Discrepant findings between the present meta-analysis and Cromheeke & Mueller's study may be related to the disparity in the original studies covered, including differences in stimulus material (verbal vs. pictorial) or experimental tasks (traditional EST vs. a variety of cognitive control tasks in the context of emotion), as well as different contrasts of interest

(main effect of emotion vs. interaction between cognitive control and emotion) used in the meta-analyses. For instance, it is conceivable that the verbal stimuli commonly used in the traditional EST paradigms are less arousing than pictorial stimuli and their potential to engage the amygdala when presented as task-irrelevant stimuli in the EST is limited (see also Phaf and Kan, 2007). Accordingly, the lack of amygdala engagement in the present study may specifically be related to the use of verbal emotional stimuli in the emotion-word and emotional counting Stroop tasks rather than being generalizable to emotion-cognition interactions overall.

Second, and alternatively, the absence of the amygdala may be explained by a rapid down-regulation of amygdala reactivity via the cognitive control network (e.g., the prefrontal and parietal regions discussed above) (Buhle et al., 2014; Goldin et al., 2008; Kim and Hamann, 2007; Kohn et al., 2014; Ochsner et al., 2012). Preliminary evidence in line with this explanation indicated that the amygdala, among other emotional processing regions, exhibited deactivation during the EST (Compton et al., 2003; Han et al., 2010; Kaiser et al., 2015; Price et al., 2011). Likewise, a previous meta-analysis that covered emotion regulation studies indicated that downregulation of emotion was consistently accompanied by increased activity in prefrontal control regions and concomitantly decreased amygdala activity (Buhle et al., 2014). These findings align with the functional interplay between cognitive control regions and the amygdala (Britton et al., 2009; Henckens et al., 2012; Price et al., 2011). Specifically, the amygdala has exhibited negative functional coupling with prefrontal regions during the EST (Henckens et al., 2012; Price et al., 2011), and the functional connectivity between the dlPFC and the amygdala has been found to correlate negatively with the magnitude of the emotional Stroop effect (Freed et al., 2009).

With respect to the rACC, a substantial number of the original studies covered in the present meta-analysis reported EST-associated activity in the rACC (Table S15). However, the label rACC has been used to label activity in various regions of the ACC, including

Broadmann areas 24, 25, 32 and 33 (see also Bush et al., 2000; Mohanty et al., 2007). Thus, the label "rACC" referred to heterogeneous ACC regions in previous studies (Fig. S4), and this regional heterogeneity may have resulted in the lack of consistent findings in a coordinate-based meta-analysis.

Alternatively, the absence of consistent rACC engagement might be attributed to the prevailing use of block design in prior neuroimaging studies (40 of 46 included studies employed a block design). Specifically, the rACC is thought to implement implicit (or 'model-free') emotion regulation, which refers to the cognitive control of emotion according to the experience-dependent alternation in need of such control (Etkin et al., 2015). This type of cognitive control is specifically employed when emotional significance needs to be regulated for ongoing cognitive challenges (Braunstein et al., 2016), as in the EST (e.g., Blair et al., 2007; Buhle et al., 2010; Etkin et al., 2006; Luo et al., 2007; Whalen et al., 1998). However, the block design allows the participants to predict the emotional significance during later stages of the experiment, arguably leading to a shift to explicit (or 'model-based') cognitive control, which primarily engages prefrontal and parietal regions rather than the rACC (Etkin et al., 2015). Indeed, Whalen et al. (1998) demonstrated an involvement of the rACC during initial but not later blocks of an EST task. However, this account is very tentative, since the limited number of studies employing event-related design did not allow for a reliable meta-analysis and direct comparison between different designs (Müller et al., 2017).

In summary, the current findings differ in predictions of the current theoretical models in two critical aspects. First, we identified consistent activation of prefrontal and parietal regions during the EST. These findings, however, align with previous observations of the engagement of cognitive control regions in domains of emotion-cognition interactions. Second, we did not demonstrate consistent engagement of the amygdala or the rACC, as would be predicted by the AAC and CTC models. Potential interpretations are provided, but they are still tentative and await further investigation.

Trans-diagnostic pattern of hyper-activation and clinical significance

With regard to the second aim of the present work we determined hyper-activation in patient populations relative to controls in cognitive control regions. Complementing the current findings, there is rich evidence showing exaggerated emotional Stroop interference at the behavioral level across a variety of patient populations (Bar-Haim et al., 2007; Cox et al., 2006; Epp et al., 2012; Field et al., 2009; Williams et al., 1996). Moreover, evidence from recent brain imaging studies has suggested a common neurobiological substrate across psychopathological disorders (Goodkind et al., 2015; McTeague et al., 2017; Sha et al., 2017; Sprooten et al., 2017). For instance, McTeague et al. (2017) identified a common neural circuit (e.g., dlPFC) disruption across psychiatric diagnoses and cognitive control tasks that parallels the currently observed cognitive control network. These findings together implicate a general dysfunction in the neural circuit consisting of prefrontal and parietal regions, which may traverse both clinical populations and task domains. Hyper-activation in the prefrontal and parietal circuits may reflect an over-engagement of the cognitive control networks to modulate the impact of emotional distractors among patients (Dresler et al., 2012; Kaiser et al., 2015; Passarotti et al., 2010; Pavuluri et al., 2010; Sadeh et al., 2011; Simons et al., 2016). In particular, the exaggerated recruitment of cognitive control regions may represent an unsuccessful attempt to compensate disrupted functional couplings between control systems and emotion processing regions (Britton et al., 2009; Sadeh et al., 2011; Szekely et al., 2017; Zimmermann et al., 2017).

Thus, the current findings demonstrate a common neural mechanism emphasizing cognitive control regions for a transdiagnostic understanding of clinical disorders. In other words, our results implicate common cognitive control deficits in the etiology and symptoms of a variety of clinical disorders. This is different from the current models emphasizing hyperactive amygdala or rACC. Nevertheless, it should be noted that the current work included a relatively small number of studies and involved primarily anxiety and depression

disorders. Furthermore, applications of the current findings to treatment settings could be explored—for instance, exploring whether increasing cognitive control capabilities, perhaps via cognitive training, is effective in treatment on clinical symptoms (Keshavan et al., 2014; Siegle et al., 2007). Lastly, the current findings may also suggest new ways of understanding prior demonstrations of exaggerated interference in clinical populations. That is, a number of clinical studies have employed the EST to probe automatic attentional bias to emotional stimuli (e.g., Williams et al., 1996), whereas our findings suggest that cognitive control processes play a key role in this task, presumably via regulating or inhibiting processing of emotional distractors.

Limitations

Several limitations of the current work should be noted. First, the ALE coordinate-based meta-analysis employed in the current study only assesses the convergence of reported peak coordinates. Therefore, our approach provides no information on the effect sizes of the activation and does not allow for assessing the potential publication bias with funnel plots (Radua and Mataix-Cols, 2012). Relatedly, it is possible that the existing reports of peak coordinates are biased toward reporting matches to existing publications. Future meta-analyses are needed to address this issue by implementing an image-based approach with unthresholded statistical maps. In this regard, future meta-analyses will be benefit from an increased availability of experimental results in publicly accessible databases (e.g., Poldrack and Gorgolewski, 2017). Second, the limited number of studies did not allow us to examine neural substrates of the emotional Stroop effect for separate clinical populations. Therefore, generalization to other psychiatric disorders, such as psychotic and bipolar disorders and autism, should await further investigation. Likewise, we were able to detect only effects that are shared across diagnoses, and the current data does not exclude the presence of potential diagnostic-specific effects. Finally, due to the fact that most of the previous neuroimaging

studies on the EST have employed a block design, the current work did not distinguish the neural mechanisms underlying fast and slow effects on the EST, which, nevertheless, have been extensively debated in the cognitive and clinical psychology literature.

Conclusion

In conclusion, the current findings underscored the involvement of a cognitive control neural circuit in the emotion-word and emotional counting Stroop paradigms, consisting of the vlPFC, dlPFC, dmPFC and parietal cortex. These regions are presumably responsible for the cognitive control of emotion processing required to maintain behavioral performance in the EST. Moreover, the current work identified a trans-diagnostic pattern of hyper-activation in these regions among patient populations relative to healthy controls, which could be attributed to deficits associated with cognitive control of emotional distractors. Unlike current models that emphasize the roles of amygdala and rACC, our findings implicate novel mechanisms underlying the EST in both healthy and clinical populations.

Acknowledgments:

This work was supported by the National Natural Science Foundation of China under Grant agreement No. 81401398 (to T.C.), No. 91632117 (to B.B.), and No. 31500920 (to C.F.), the National Postdoctoral Program for Innovative Talents under grant agreement No. BX201600019 (to C.F.), and the China Postdoctoral Science Foundation under grant agreement No. 2017M610055 (to C.F.) and 2013M530401 (to T.C.).

Conflict of Interest:

The authors are unaware of any conflicts of interest, financial or otherwise.

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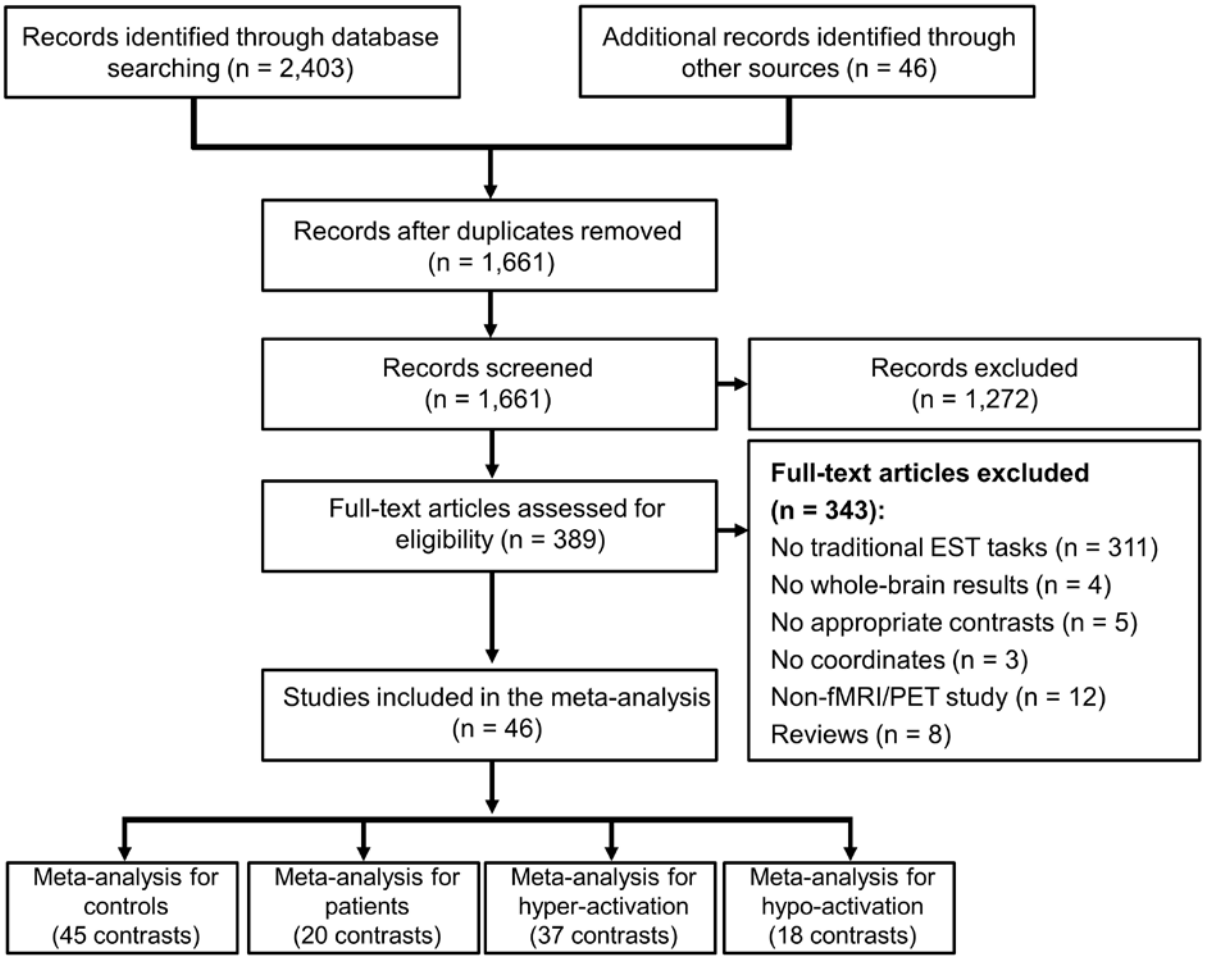
965 **Captions to Figures and Tables**



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967 **Figure 1. Flow chart of the study selection process for the meta-analysis.** ES, emotional Stroop task; fMRI,
968 functional magnetic resonance imaging; PET, positron emission tomography.
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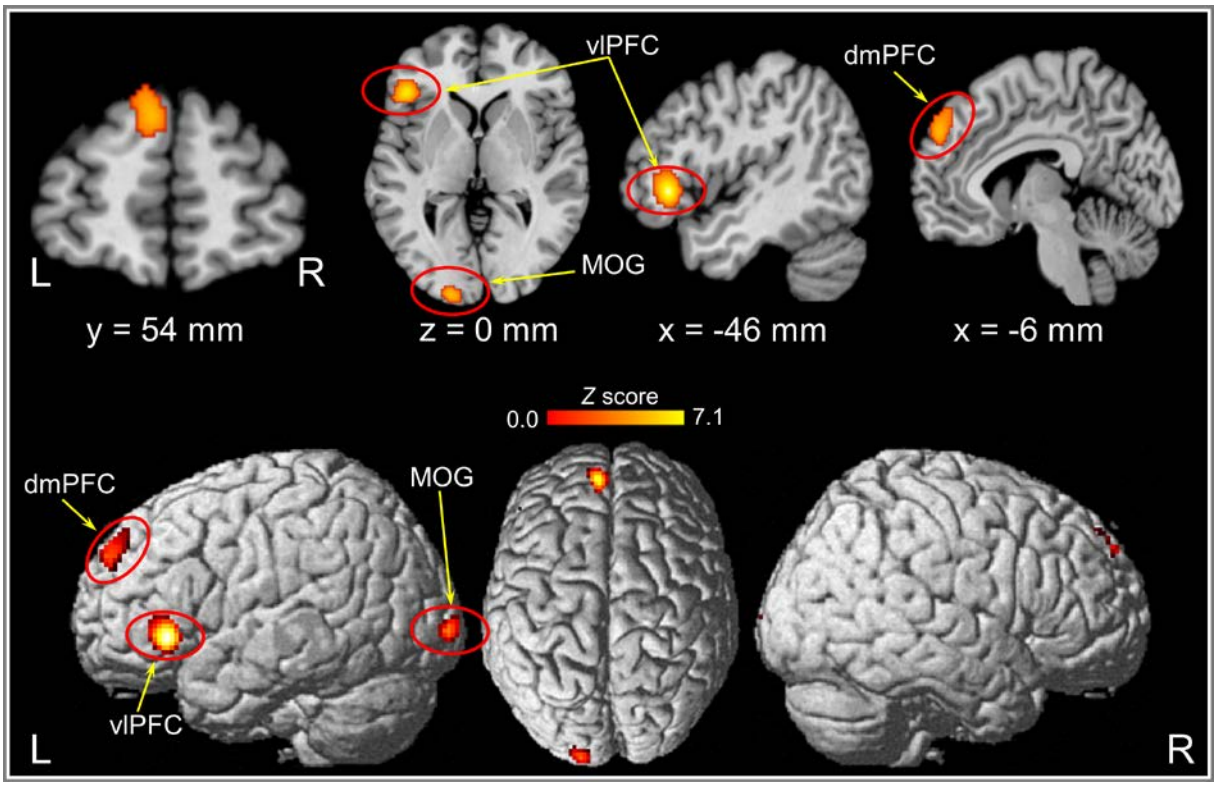

Figure 2. Significant clusters from the main meta-analysis of emotional Stroop task for healthy volunteers (cluster-level family-wise error correction ($P < 0.05$) with a cluster-forming threshold of $P < 0.001$ using 10,000 permutations). Consistent maxima were found in the bilateral dmPFC, left vlPFC and MOG. L, left; R, right; dmPFC, dorsomedial prefrotnal cortex; vlPFC, ventrolateral prefrontal cortex; MOG, middle occipital gyrus.

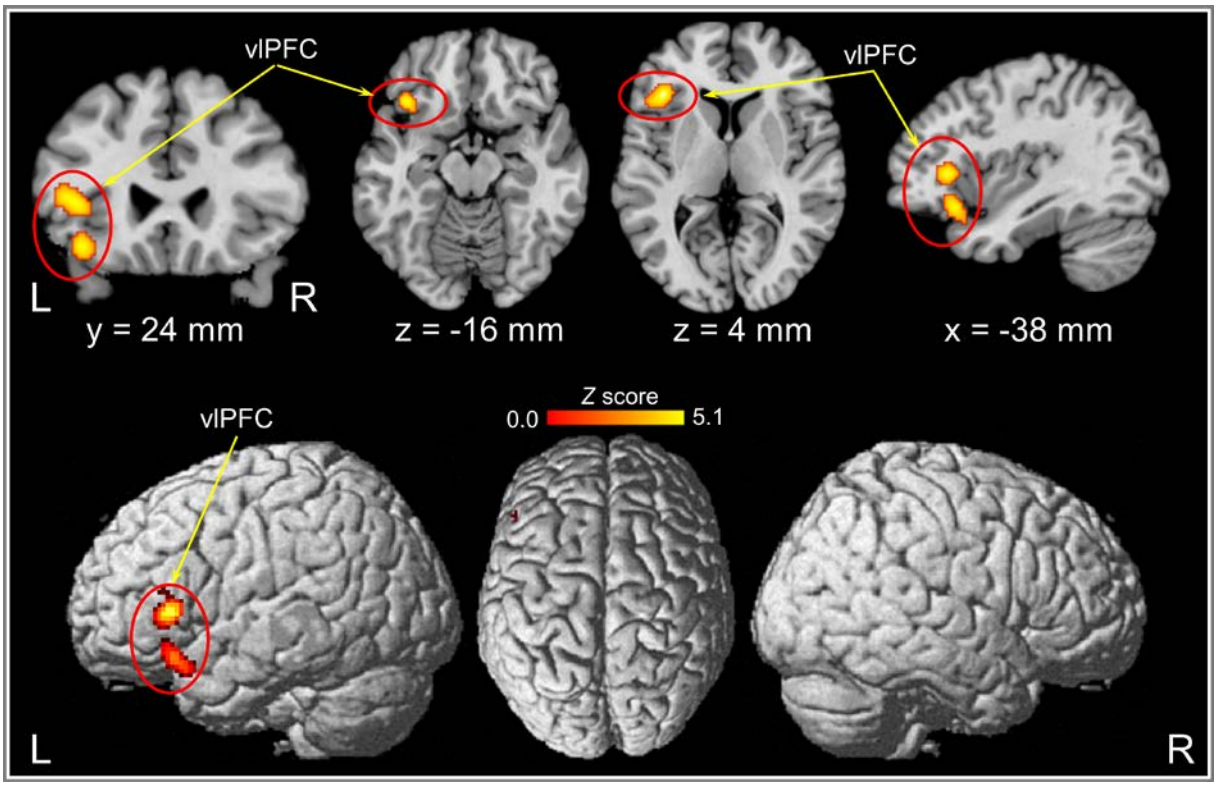

Figure 3. Significant clusters from the main meta-analysis of emotional Stroop task for patient populations (cluster-level family-wise error correction (*P* < 0.05) with a cluster-forming threshold of *P* < 0.001 using 10,000 permutations). Consistent maxima were found in the left vlPFC. L, left; R, right; vlPFC, ventrolateral prefrontal cortex.

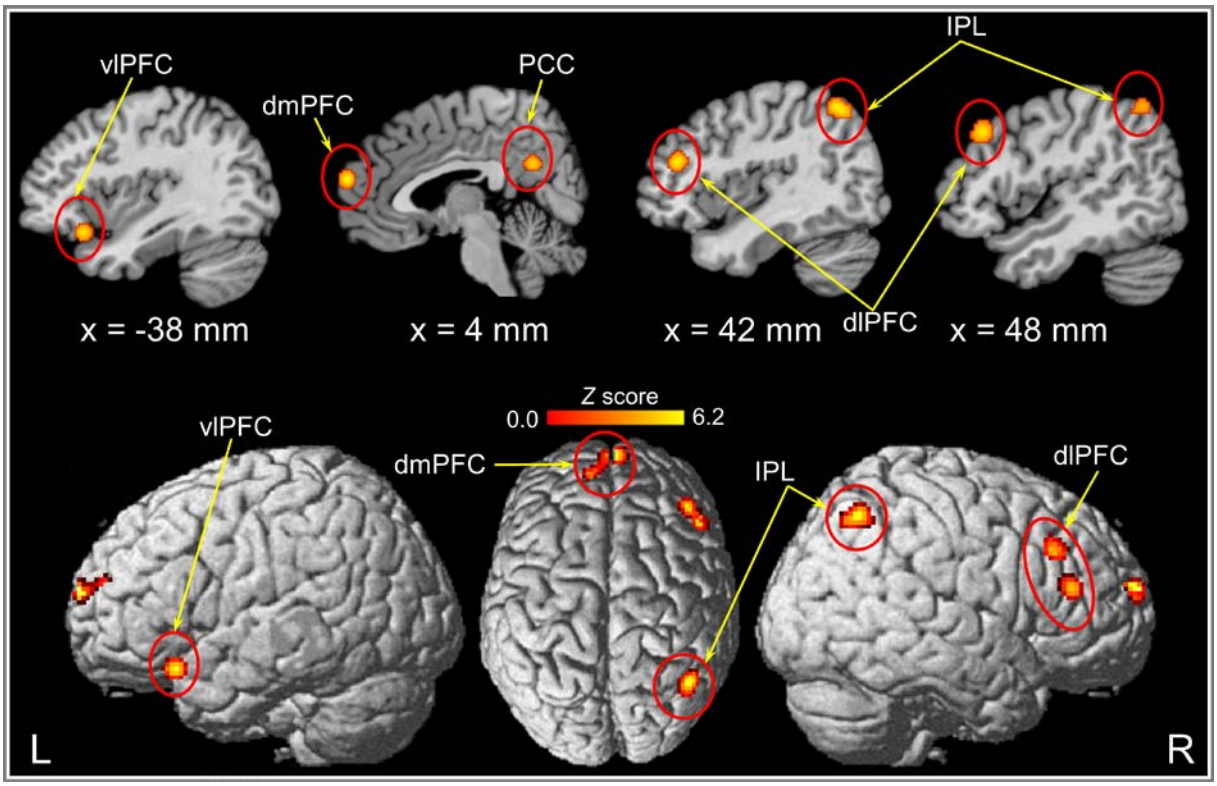

Figure 4. Significant clusters from the main meta-analysis of emotional Stroop task for hyper-activation (patients > controls) (cluster-level family-wise error correction (*P* < 0.05) with a cluster-forming threshold of *P* < 0.001 using 10,000 permutations).. Consistent maxima were found in the bilateral dmPFC, PCC, left vlPFC, right dlPFC and IPL. L, left; R, right; dmPFC, dorsomedial prefrotnal cortex; PCC, posterior cingulate cortex; vlPFC, ventrolateral prefrontal cortex; dlPFC, dorsolateral prefrontal cortex; IPL, inferior parietal lobule.

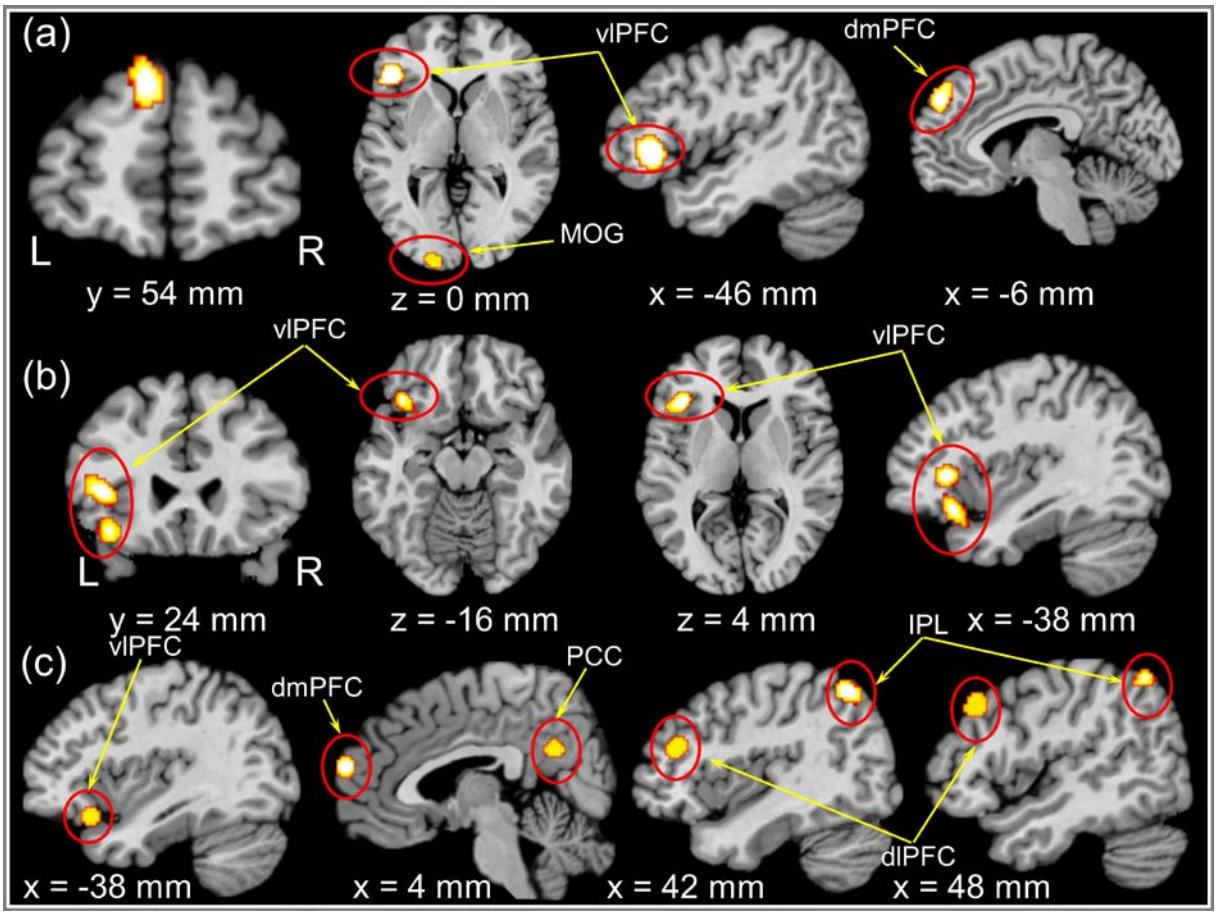

Figure 5. Significant clusters identified in all folds of the leave-one-experiment-out analysis (LOEO). (a). Consistent maxima in the left vlPFC, dmPFC and MOG were identified for healthy volunteers. **(b).** Consistent maxima in the left vlPFC were identified for patients. **(c).** Consistent maxima in the bilateral dmPFC, PCC, left vlPFC, right dlPFC and IPL were found for hyper-activation of patients relative to healthy controls. L, left; R, right; vlPFC, ventrolateral prefrontal cortex; dmPFC, dorsomedial prefrontal cortex; MOG, middle occipital gyrus; PCC, posterior cingulate cortex; dlPFC, dorsolateral prefrontal cortex; IPL, inferior parietal lobule.

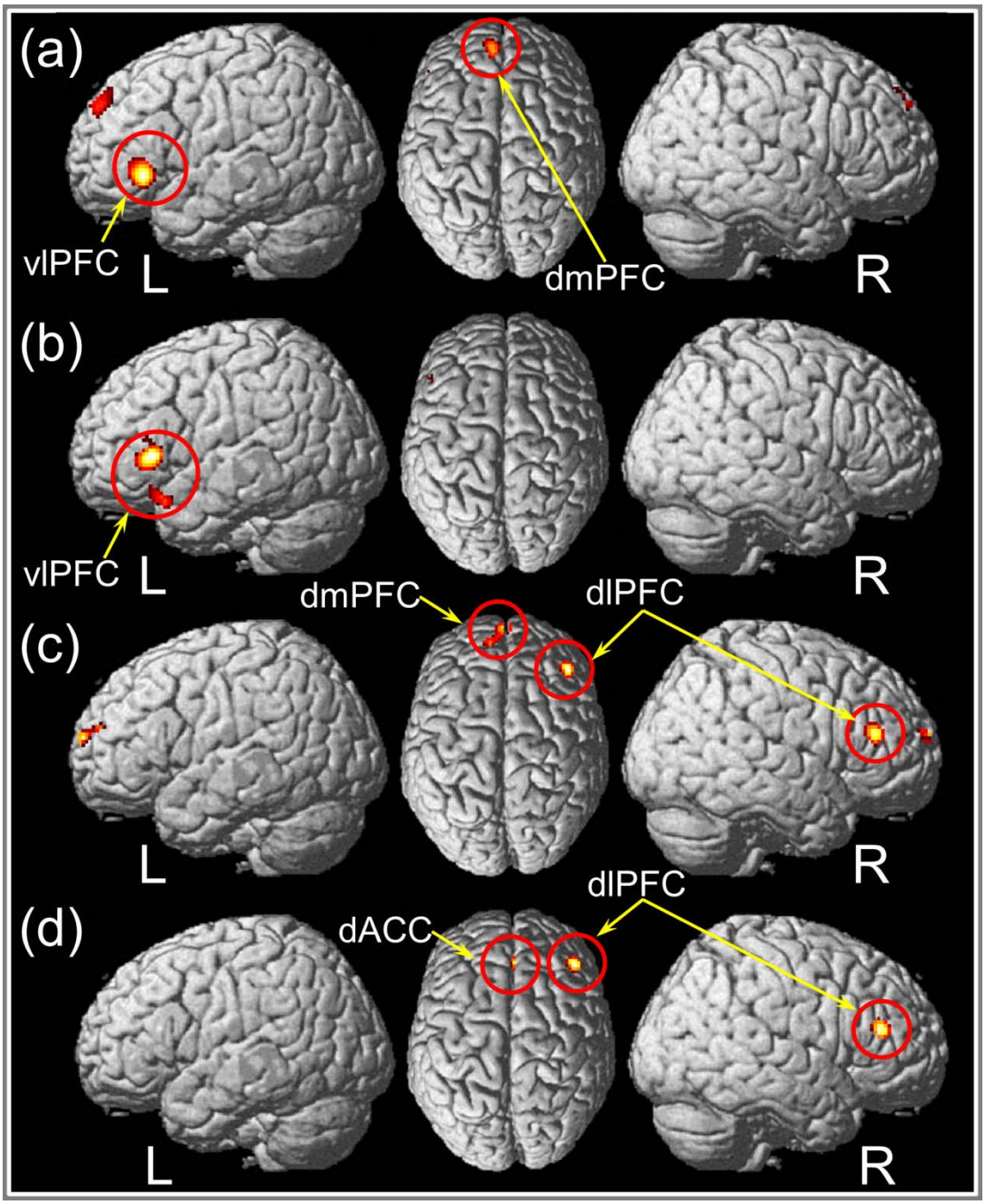

Figure 6. Significant clusters from the meta-analysis of emotional Stroop task focusing contrasts associated with negative words. (a). Consistent maxima were found in the bilateral and left vlPFC for healthy controls. **(b).** Consistent maxima were found in the left vlPFC for clinical patients. **(c).** Consistent maxima were found in the bilateral dmPFC, PCC, and dlPFC for hyper-activation. **(d).** Consistent maxima were found in the left amygdala, right dlPFC, bilateral dACC, and PCC for aberrant activation**.** All maps were FWE-corrected at cluster-level ($P < 0.05$) with a cluster-forming threshold of $P < 0.001$ using 10,000 permutations). L, left; R, right; vlPFC, ventrolateral prefrontal cortex; dmPFC, dorsomedial prefrontal cortex; dlPFC, dorsolateral prefrontal cortex; dACC, dorsal anterior cingulate cortex; PCC, posterior cingulate cortex.

1039 **Table 1. Summary of studies included for the main meta-analyses.**

Study	Subject Sample	N	Task	Contrast	No. of foci
			Healthy volunteers		
Brennan et al. (2015)	healthy volunteers	29	emotional counting Stroop task	negative words > neutral words	3
Britton et al. (2009)	healthy volunteers	12	emotional counting Stroop task	negative words > neutral words	8
Compton et al. (2003)	healthy volunteers	12	emotional color-word Stroop task	negative words > neutral words	12
Compton et al. (2003)	healthy volunteers	12	emotional color-word Stroop task	negative high arousing words > negative low arousing words	11
Compton et al. (2003)	healthy volunteers	12	emotional color-word Stroop task	positive high arousing words > positive low arousing words	1
Dresler et al. (2012)	healthy volunteers	23	emotional color-word Stroop task	negative words > neutral words	1
Engels et al. (2007)	healthy volunteers	18	emotional color-word Stroop task	negative words > neutral words	4
Evers et al. (2006)	healthy volunteers	19	emotional color-word Stroop task	negative words > neutral words	1
Evers et al. (2006)	healthy volunteers	19	emotional color-word Stroop task	positive words > neutral words	1
Freed et al. (2009)	healthy volunteers	20	emotional color-word Stroop task	negative words > neutral words	12
Freed et al. (2009)	healthy volunteers	20	emotional color-word Stroop task	negative words > neutral words	13
George et al. (1994)	healthy volunteers	21	emotional color-word Stroop task	negative words > neutral words	5
George et al. (1994)	healthy volunteers	21	emotional color-word Stroop task	positive correlation with RT for negative words	13
Golm et al. (2016)	healthy volunteers	16	emotional color-word Stroop task	negative words > neutral words	4
Han et al. (2010)	healthy volunteers	20	emotional color-word Stroop task	negative words > neutral words	6

Han et al. (2010)	healthy volunteers	20	emotional color-word Stroop task	negative words > neutral words	12
Henckens et al. (2012)	healthy volunteers	65	emotional color-word Stroop task	negative words > neutral words	3
Isenberg et al. (1999)	healthy volunteers	6	emotional color-word Stroop task	negative words > neutral words	4
Kaiser et al. (2015)	healthy volunteers	92	emotional color-word Stroop task	negative words > neutral words	4
Lagopoulos & Malhi (2007)	healthy volunteers	10	emotional color-word Stroop task	negative words > neutral words	10
Malhi et al. (2005)	healthy volunteers	12	emotional color-word Stroop task	emotional words > neutral words	20
McCabe et al. (2009)	healthy volunteers	24	emotional counting Stroop task	positive words >baseline	3
McCabe et al. (2009)	healthy volunteers	24	emotional counting Stroop task	negative words >baseline	1
Mincic (2010)	healthy volunteers	30	emotional counting Stroop task	negative words > baseline	19
Mitterschiffthaler et al. (2008)	healthy volunteers	17	emotional color-word Stroop task	negative words > neutral words	1
Mohanty et al. (2005)	healthy volunteers	17	emotional color-word Stroop task	negative words > neutral words	10
Posner et al. (2011)	healthy volunteers	15	emotional counting Stroop task	positive words > neutral words	5
Posner et al. (2011)	healthy volunteers	15	emotional counting Stroop task	negative words > neutral words	2
Price et al. (2011)	healthy volunteers	12	emotional color-word Stroop task	negative words > neutral words	3
Price et al. (2011)	healthy volunteers	12	emotional color-word Stroop task	negative words > neutral words	2
Puetz et al. (2016)	healthy volunteers	19	emotional color-word Stroop task	negative words > neutral words	9
Rahm et al. (2013)	healthy volunteers	11	emotional counting Stroop task	negative words > neutral words	6
Rahm et al. (2013)	healthy volunteers	11	emotional counting Stroop task	negative words > neutral words	7

Rahm et al. (2013)	healthy volunteers	11	emotional counting Stroop task	negative words > neutral words	4
Sebastian et al. (2010)	healthy volunteers	35	emotional color-word Stroop task	negative words > neutral words	8
Shin et al. (2001)	healthy volunteers	8	emotional counting Stroop task	self-related negative words > self-unrelated negative words	12
Shin et al. (2001)	healthy volunteers	8	emotional counting Stroop task	self-related negative words > neutral words	11
Spieberg et al. (2012)	healthy volunteers	80	emotional color-word Stroop task	emotional words > neutral words	7
Szekely et al. (2017)	healthy volunteers	22	emotional color-word Stroop task	negative words > neutral words	1
Veroude et al. (2013)	healthy volunteers	74	emotional color-word Stroop task	negative words > neutral words	5
Whalen et al. (1998)	healthy volunteers	9	emotional counting Stroop task	negative words > neutral words	2
Wingenfeld et al. (2009)	healthy volunteers	20	emotional color-word Stroop task	negative words > neutral words	7
Wingenfeld et al. (2009)	healthy volunteers	20	emotional color-word Stroop task	negative words > neutral words	19
Witthöft et al. (2013)	healthy volunteers	12	emotional color-word Stroop task	negative words > neutral words	25
Witthöft et al. (2013)	healthy volunteers	12	emotional color-word Stroop task	negative words > neutral words	20
			Patient populations		
Bremner et al. (2004)	posttraumatic stress disorder	12	emotional color-word Stroop task	negative words > neutral words	3
Brennan et al. (2015)	obsessive-compulsive disorder	30	emotional counting Stroop task	disorder-related words > neutral words	14
Brennan et al. (2015)	obsessive-compulsive disorder	30	emotional counting Stroop task	negative words > neutral words	16
Britton et al. (2009)	animal phobias disorder	12	emotional counting Stroop task	disorder-related words > neutral words	19
Dieter et al. (2017)	intrenet gaming addicts	13	emotional color-word Stroop task	social anxious words > baseline	1

Dresler et al. (2012)	panic disorder	20	emotional color-word Stroop task	disorder-related words > neutral words	10
George et al. (1997)	depression disorder	11	emotional color-word Stroop task	negative words > neutral words	3
Golm et al. (2016)	tinnitus disorder	16	emotional color-word Stroop task	disorder-related words > neutral words	12
Kilts et al. (2014)	cocaine dependence disorder	42	emotional color-word Stroop task	disorder-related words > neutral words	15
Kilts et al. (2014)	cocaine dependence disorder	42	emotional color-word Stroop task	negative words > neutral words	4
Kilts et al. (2014)	cocaine dependence disorder	42	emotional color-word Stroop task	disorder-related words > neutral words, positive correlation with attentional bias	16
Lagopoulos et al. (2007)	euthymic bipolar disorder	10	emotional color-word Stroop task	negative words > baseline	8
Malhi et al. (2005)	euthymic bipolar disorder	12	emotional color-word Stroop task	emotional words > neutral words	4
Mitterschiffthaler et al. (2008)	major depressive disorder	17	emotional color-word Stroop task	disorder-related words > neutral words	6
Posner et al. (2011)	attention deficit hyperactivity disorder	15	emotional counting Stroop task	positive words > neutral words	2
Posner et al. (2011)	attention deficit hyperactivity disorder	15	emotional counting Stroop task	positive words > neutral words	1
Shin et al. (2001)	posttraumatic stress disorder	8	emotional counting Stroop task	disorder-related words >general negative words	28
Shin et al. (2001)	posttraumatic stress disorder	8	emotional counting Stroop task	disorder-related words > neutral words	15
Winter et al. (2015)	borderline personality disorder	19	emotional color-word Stroop task	positive words > neutral words	9
Winter et al. (2015)	borderline personality disorder	18	emotional color-word Stroop task	negative words > neutral words	25
Hyper-activation (patients > controls)					

Boehme et al. (2015)	social anxiety disorder	32	emotional color-word Stroop task	disorder-related words > neutral words, patients > controls	6
Bremner et al. (2004)	posttraumatic stress disorder	21	emotional color-word Stroop task	negative words > neutral words, patients > controls	2
Brennan et al. (2015)	obsessive-compulsive disorder	59	emotional counting Stroop task	disorder-related words > neutral words, patients > controls	17
Brennan et al. (2015)	obsessive-compulsive disorder	59	emotional counting Stroop task	negative words > neutral words, patients > controls	5
Britton et al. (2009)	animal phobias disorder	24	emotional counting Stroop task	disorder-related words > neutral words, patients > controls	3
Dresler et al. (2012)	panic disorder	43	emotional color-word Stroop task	disorder-related words > neutral words, patients > controls	1
Fu et al. (2015)	major depressive disorder	47	emotional color-word Stroop task	negative words > neutral words, patients > controls	3
Fu et al. (2015)	major depressive disorder	47	emotional color-word Stroop task	negative words > neutral words, patients untreated > patients treated	3
Golm et al. (2016)	tinnitus disorder	32	emotional color-word Stroop task	disorder-related words > neutral words, patients, high distress > controls	3
Golm et al. (2016)	tinnitus disorder	32	emotional color-word Stroop task	disorder-related words > neutral words, patients, high distress > low distress	8
Lagopoulos and Malhi (2007)	euthymic bipolar disorder	20	emotional color-word Stroop task	negative words > baseline, patients > controls	2
Mitterschiffthaler et al. (2008)	major depressive disorder	34	emotional color-word Stroop task	disorder-related words > neutral words, patients > controls	2
Passarotti et al. (2010)	pediatric bipolar disorder	31	emotional color-word Stroop task	negative words > neutral words, patients > controls	11
Passarotti et al. (2010)	pediatric bipolar disorder	31	emotional color-word Stroop task	positive words > neutral words, patients > controls	5
Passarotti et al. (2010)	attention deficit hyperactivity	29	emotional color-word Stroop task	negative words > neutral words, patients > controls	5

	disorder				
Passarotti et al. (2010)	attention deficit hyperactivity disorder	29	emotional color-word Stroop task	positive words > neutral words, patients > controls	5
Pavuluri et al. (2010)	pediatric bipolar disorder	17	emotional color-word Stroop task	negative words > neutral words, patients untreated > patients treated	12
Pavuluri et al. (2010)	pediatric bipolar disorder	17	emotional color-word Stroop task	positive words > neutral words, patients untreated > patients treated	4
Pavuluri et al. (2010)	pediatric bipolar disorder	31	emotional color-word Stroop task	negative words > neutral words, patients untreated > patients treated	5
Pavuluri et al. (2010)	pediatric bipolar disorder	31	emotional color-word Stroop task	positive words > neutral words, patients untreated > patients treated	3
Pavuluri et al. (2010)	pediatric bipolar disorder	31	emotional color-word Stroop task	negative words > neutral words, patients > controls	10
Pavuluri et al. (2010)	pediatric bipolar disorder	31	emotional color-word Stroop task	positive words > neutral words, patients > controls	5
Posner et al. (2011)	attention deficit hyperactivity disorder	30	emotional counting Stroop task	positive words > neutral words, patients > controls	1
Price et al. (2011)	generalized anxiety disorder	28	emotional color-word Stroop task	negative words > neutral words, patients > controls	4
Price et al. (2011)	generalized anxiety disorder	28	emotional color-word Stroop task	negative words > neutral words, patients > controls	1
Redgrave et al. (2008)	anorexia nervosa disorder	12	emotional color-word Stroop task	disorder-related words > neutral words, patients > controls	5
Shin et al. (2001)	posttraumatic stress disorder	16	emotional counting Stroop task	disorder-related words >general negative words, patients > controls	21
Taylor et al. (2016)	chronic musculoskeletal pain disorder	30	emotional counting Stroop task	disorder-related words > neutral words, patients > controls	20

Taylor et al. (2016)	chronic musculoskeletal pain disorder	30	emotional counting Stroop task	disorder-related words > neutral words, patients > controls	13
Van den Heuvel et al. (2005)	obsessive-compulsive disorder	35	emotional color-word Stroop task	disorder-related words > neutral words, patients > controls	2
Van den Heuvel et al. (2005)	panic disorder	34	emotional color-word Stroop task	disorder-related words > neutral words, patients > controls	2
Weissman-Fogel et al. (2011)	temporomandibular disorder	34	emotional counting Stroop task	disorder-related words, patients > controls	11
Weissman-Fogel et al. (2011)	temporomandibular disorder	34	emotional counting Stroop task	disorder-related words > neutral words, patients > controls	7
Winter et al. (2015)	borderline personality disorder	38	emotional color-word Stroop task	positive words > neutral words, patients > controls	7
Winter et al. (2015)	borderline personality disorder	38	emotional color-word Stroop task	negative words > neutral words, patients > controls	1
Winter et al. (2015)	borderline personality disorder	37	emotional color-word Stroop task	negative words > neutral words, patients > controls	3
Zhang et al. (2011)	panic disorder	30	emotional counting Stroop task	negative words > neutral words, patients > controls	2
Hypo-activation (patients < controls)					
Arizmendi et al. (2016)	complicated grief	17	emotional counting Stroop task	negative words > neutral words, complicated grief < non-complicated grief	4
Bremner et al. (2004)	posttraumatic stress disorder	21	emotional color-word Stroop task	negative words > neutral words, patients < controls	6
Brennan et al. (2015)	obsessive-compulsive disorder	59	emotional counting Stroop task	negative words > neutral words, patients < controls	1
Britton et al. (2009)	animal phobias disorder	24	emotional counting Stroop task	disorder-related words > neutral words, patients < controls	2
Lagopoulos and Malhi (2007)	euthymic bipolar disorder	20	emotional color-word Stroop task	negative words > baseline, patients < controls	2
Malhi et al. (2005)	euthymic bipolar disorder	24	emotional color-word Stroop task	emotional words > baseline, patients < controls	13

Passarotti et al. (2010)	attention deficit hyperactivity disorder	29	emotional color-word Stroop task	negative words > neutral words, patients < controls	3
Passarotti et al. (2010)	attention deficit hyperactivity disorder	29	emotional color-word Stroop task	positive words > neutral words, patients < controls	2
Pavuluri et al. (2010)	pediatric bipolar disorder	31	emotional color-word Stroop task	negative words > neutral words, patients untreated < patients treated	1
Pavuluri et al. (2010)	pediatric bipolar disorder	31	emotional color-word Stroop task	positive words > neutral words, patients untreated < patients treated	1
Posner et al. (2011)	attention deficit hyperactivity disorder	30	emotional counting Stroop task	negative words > neutral words, patients < control	1
Price et al. (2011)	generalized anxiety disorder	28	emotional color-word Stroop task	negative words > neutral words, patients < controls	3
Price et al. (2011)	generalized anxiety disorder	28	emotional color-word Stroop task	negative words > neutral words, patients < controls	2
Redgrave et al. (2008)	anorexia nervosa disorder	12	emotional color-word Stroop task	disorder-related words > neutral words, patients < controls	5
Shin et al. (2001)	posttraumatic stress disorder	16	emotional counting Stroop task	disorder-related words >general negative words, patients < controls	12
Wingenfeld et al. (2009)	borderline personality disorder	40	emotional color-word Stroop task	negative words > neutral words, patients < controls	7
Wingenfeld et al. (2009)	borderline personality disorder	40	emotional color-word Stroop task	negative words > neutral words, patients < controls	13
Zhang et al. (2011)	panic disorder	30	emotional counting Stroop task	negative words > neutral words, patients < controls	5

Table 2. A summary of diagnostic information for conrasts related to clinical populations.

	Emotional Stroop effect			Hyper-activation			Hypo-activation		
	contrasts	foci	subjects	contrasts	foci	subjects	contrasts	foci	subjects
animal phobias	1	19	12	1	3	24	1	2	24
panic disorder	1	10	20	3	5	107	1	5	30
obsessive-compulsive disorder	2	30	60	3	24	153	1	1	59
posttraumatic stress disorder	3	46	28	2	23	37	2	18	37
generalized anxiety disorder	0	0	0	2	5	56	2	5	56
social anxiety disorder	0	0	0	1	6	32	0	0	0
depressive disorders	4	21	50	12	65	368	5	21	123
cocaine dependence	3	35	126	0	0	0	0	0	0
internet gaming addiction	1	1	13	0	0	0	0	0	0
borderline personality disorder	2	34	37	3	11	113	2	20	80
anorexia nervosa	0	0	0	1	5	12	1	5	12
chronic musculoskeletal pain	0	0	0	2	33	60	0	0	0
temporomandibular disorder	0	0	0	2	18	68	0	0	0
attention deficit hyperactivity disorder	2	3	30	3	11	88	3	6	88
tinnitus	1	12	16	2	11	64	0	0	0

Table 3. Significant clusters from the main meta-analysis of emotional Stroop task for healthy volunteers, patients, hyper-activation and aberrant activation.

Laterality	Brain Regions	BA	MNI Coordinates (mm)			peak Z score	Cluster Size (mm^3)
			x	y	z		
Healthy volunteers							
L	ventrolateral prefrontal cortex	47/45	-46	28	-4	7.0957	2512
L	middle occipital gyrus	18/17	-16	-100	0	5.119	776
L	dorsomedial prefrontal cortex	9/8/6	-6	54	34	5.0644	1424
Patients							
L	ventrolateral prefrontal cortex	47/38	-38	24	-16	4.9103	1256
L	ventrolateral prefrontal cortex	45/47/13	-40	26	4	5.1382	1576
Hyper-activation (patients VS controls)							
L	ventrolateral prefrontal cortex	47/38	-40	22	-18	6.1693	776
L/R	dorsomedial prefrontal cortex	10/9	4	64	18	5.7375	1976
R	dorsolateral prefrontal cortex	46/10	42	36	18	5.6116	776
L/R	posterior cingulate cortex	31/7/23	4	-58	28	4.845	912
R	dorsolateral prefrontal cortex	9	50	28	36	5.9378	744
R	inferior parietal lobule	40/7	44	-60	52	5.8008	1168
Aberrant activation							
L/R	dorsomedial prefrontal cortex	10	4	64	18	5.4525	1600
R	dorsolateral prefrontal cortex	46/10	42	36	18	5.3787	720
L/R	dorsal anterior cingulate cortex	32/9	4	36	24	4.8887	712
L/R	posterior cingulate cortex	31/7/23	4	-58	28	4.5811	688
R	inferior parietal lobule	40/7	44	-60	52	5.5139	944

P(FWE) < 0.05 at the cluster level with a cluster-forming threshold of $P < 0.001$ using 10,000 permutations.

Table 4. Significant clusters identified in all folds of the leave-one-experiment-out analysis for healthy volunteers, patients, hyper-activation and aberrant activation.

Laterality	Brain Regions	BA	MNI Coordinates (mm)			Cluster Size (mm^3)
			x	y	z	
Healthy volunteers						
L	ventrolateral prefrontal cortex	47/45	-44	26	-8	2656
L	middle occipital gyrus	18/17	-16	-102	-4	808
L	dorsomedial prefrontal cortex	9/8/6	-6	50	32	1552
Patients						
L	ventrolateral prefrontal cortex	47/38	-38	20	-20	1472
L	ventrolateral prefrontal cortex	45/47/13	-38	26	0	1752
Hyper-activation (patients VS controls)						
L	ventrolateral prefrontal cortex	47/38	-38	20	-22	784
L/R	dorsomedial prefrontal cortex	10/9	6	62	14	2064
R	dorsolateral prefrontal cortex	46/10	44	34	14	824
L/R	posterior cingulate cortex	31/7/23	2	-58	22	968
R	dorsolateral prefrontal cortex	9	52	26	32	752
R	inferior parietal lobule	40/7	44	-62	46	1224
Aberrant activation						
L	ventrolateral prefrontal cortex	47/38	-38	20	-22	712
L/R	dorsomedial prefrontal cortex	10/9	-4	58	20	1656
R	dorsolateral prefrontal cortex	46/10	46	34	14	760
L/R	dorsal anterior cingulate cortex	32/9	6	34	20	784
L/R	posterior cingulate cortex	31/7/23	4	-60	24	728
R	inferior parietal lobule	40/7	42	-66	48	1032

P(FWE) < 0.05 at the cluster level with a cluster-forming threshold of $P < 0.001$ using 10,000 permutations

1076 **Table 5. Significant clusters from the meta-analysis of emotional Stroop task focusing contrasts**
1077 **associated with negative words for healthy volunteers, patients, hyper-activation, and aberrant**
1078 **activation.**

Laterality	Brain Regions	BA	MNI Coordinates (mm)			peak Z score	Cluster Size (mm^3)
			x	y	z		
Healthy volunteers							
L	ventrolateral prefrontal cortex	47/45	-46	28	4	7.1215	2584
L	dorsomedial prefrontal cortex	9/8/6	-6	54	36	4.6955	1152
Patients							
L	ventrolateral prefrontal cortex	47/38	-38	22	-18	3.9643	752
L	ventrolateral prefrontal cortex	45/47/13	-40	26	4	5.8235	2112
Hyper-activation (patients VS controls)							
R	dorsolateral prefrontal cortex	46/10	42	36	18	5.7471	824
L/R	dorsomedial prefrontal cortex	10/9	-2	64	16	4.4724	1008
L/R	posterior cingulate cortex	31/7/23	4	-58	28	4.969	1048
Aberrant activation							
L	Amygdala		-24	-4	-24	4.5623	680
R	dorsolateral prefrontal cortex	46/10	42	36	18	5.4941	776
L/R	dorsal anterior cingulate cortex	32/9	4	36	24	4.9863	752
L/R	posterior cingulate cortex	31/7/23	4	-58	28	4.6877	744

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