

# **The overlapping neurobiology of induced and pathological anxiety: a meta-analysis of functional neural activation**

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## ABSTRACT

**Objective:** Anxiety can be an *adaptive* response to unpredictable threats, while *pathological* anxiety disorders occur when symptoms adversely impact daily life. Whether or not adaptive and pathological anxiety share mechanisms remains unknown, but if they do, induced (adaptive) anxiety could be used as an intermediate translational model of pathological anxiety to improve drug-development pipelines. Meta-analyses of functional neuroimaging studies of induced and pathological anxiety were therefore compared. **Methods:** A systematic search was conducted in June 2019 on the PUBMED database for whole-brain functional magnetic resonance imaging articles. Eligible articles contrasted anxious patients to controls, or an unpredictable-threat condition to a safe condition in healthy participants. Five anxiety disorders were included: post-traumatic stress disorder, social anxiety disorder, generalized anxiety disorder, panic disorder, and specific phobia. 3433 records were identified, 181 met criteria and the largest subset of task type was emotional (N=138). Seed-based d-mapping software was used for all analyses. **Results:** Induced anxiety (n=693 participants) and pathological anxiety (n=2554 patients and 2348 controls) both showed increased activation in the bilateral insula (xyz=44,14,-14 and xyz=-38,20,-8;k=2102 and k=1305 respectively) and cingulate cortex/medial prefrontal cortex (xyz=-12,-8,68;k=2217). When split by disorders, specific phobia appeared the most, and generalized anxiety disorder the least, similar to induced anxiety. **Conclusions:** This meta-analysis indicates a consistent pattern of activation across induced and pathological anxiety, supporting the proposition that some neurobiological mechanisms overlap and that the former may be used as a model for the latter. Induced anxiety might, nevertheless, be a better model for some anxiety disorders than others.

## INTRODUCTION

Anxiety disorders are the most prevalent mental health condition (1) with a lifetime prevalence of 17% (2), resulting in significant individual and social impairment (1) and a considerable overall burden of disease, ranked 9<sup>th</sup> cause of years lived with disability in the world in 2015 (3). Response rates to existing treatments usually range between 40 and 60% (4) which leaves a large number of people with debilitating symptoms and/or high probability of relapse (5).

Development of new treatments for symptoms of anxiety has, however, stagnated for several decades (6), partly due to the difficulty of establishing robust translational links between models of fear and anxiety in rodents and clinical anxiety in humans. It has recently been argued, therefore, that models of anxiety [as defined by aversive anticipation and apprehension of perceived potential, but unpredictable, threats] in healthy humans, could help us bridge this gap and facilitate therapeutic progress (7).

More precisely, using *the same* techniques to induce anxiety in healthy individuals and animal models should enable us to both better understand the neurobiological basis of anxiety, as well as provide an intermediate route to screen the efficacy of candidate interventions prior to full clinical trial (8). This experimental approach is possible because, perhaps unique amongst psychiatric symptoms, anxiety is *also* an adaptive behavior with a benefit to survival. Anxiety enhances vigilance to threat and primes defense mechanisms (9) which allows the individual to react faster in dangerous situations. It occurs naturally in every individual, when walking down a dark alley at night for instance. This adaptive anxiety can be reliably induced in healthy individuals in the lab by exposing them to unpredictable threat of rare electrical shocks. This approach is a well-validated (10), reliable both for self-report and task performance (11) and, critically, it is also fully translational – a close paradigm is used in animal models (12). A growing body of literature shows that, in addition to clear increases in subjective and physiological reports of anxiety (13), threat of shock results in cognitive and psychophysiological changes mirroring pathological anxiety (14–16).

Induced and pathological anxiety therefore overlap at the level of symptoms, as both promote functions and states that promote harm avoidance. What remains insufficiently explored, however, is the extent to which underlying neurobiological mechanisms overlap, or whether ostensibly similar symptoms are driven by dissociable underlying mechanisms. Critically, for the ‘experimental psychopathology’ (7) approach to be valid then the assumption that induced anxiety evokes (at least some of) the same neurobiological mechanisms as pathological anxiety must be met, particularly on emotion-related paradigms where the literature suggests they lead to similar changes in cognitive performance (14).

Induced anxiety via unpredictable threat paradigms has been shown to involve brain regions involved in emotional processing, decision-making and reward circuitry, such as the amygdala, anterior cingulate cortex (ACC), medial prefrontal cortex (PFC), bed nucleus of the stria terminalis (BNST), insula or striatum (17–21) but this has not been systematically meta-analyzed. Meta-analyses and systematic reviews exploring the neural circuitry of different anxiety disorders suggest that many of the same regions have been implicated (22–25), however differences across disorders have not been investigated in the past decade (26,27). Meta-analyses of fear conditioning studies, a related experimental model that focuses on predictable (rather than unpredictable) threats, also reported resulting hyperactivation in dorsal ACC and bilateral anterior insula (28,29) and one meta-analysis that came out whilst this paper was in submission investigated shared neural correlates across mood and anxiety disorders (30). What is lacking, however, is an up-to-date systematic meta-analysis directly assessing the extent to which the neural activation in anxiety disorders in emotion-related paradigms overlaps with that evoked by unpredictable threat-induced anxiety (as opposed to fear conditioning) in the general population. That is, in other words, a systematic assessment of the neurobiological links between induced and pathological anxiety; and a quantitative assessment of the ‘experimental psychopathology’ approach to anxiety.

This meta-analysis therefore aims to 1) investigate the common functional neural activity pattern across induced anxiety studies, 2) update disorder-specific maps for 5 anxiety disorders and examine commonalities across all pathological anxiety brain activity in emotion-related paradigms, 3) compare

neural patterns of induced anxiety to pathological disorder(s). We used a coordinate-based meta-analytic (CBMA) whole-brain approach (31) to test the broad prediction that activation patterns overlap across induced and pathological anxiety. The CBMA approach has important strengths over conventional activation likelihood meta-analyses as it uses the effect sizes and enables investigation of voxelwise publication bias (32). Unthresholded groups maps were also collected where possible to ensure that the results were as precise as possible. In addition, we explored overlap of our induced anxiety results with a recent fear-conditioning meta-analysis.

## **METHODS**

### **Literature search and inclusion**

A systematic search was conducted on the PUBMED database (all studies published before 11<sup>th</sup> of June 2019, including studies in press) for fMRI whole-brain BOLD activity papers reporting anxious or depressed patients vs. controls contrasts, or an unpredictable threat vs. safe conditions contrasts (flowchart in Fig. S1, see Supplementary methods for full details).

181 publications were identified comprising 2911 anxious patients and 2685 controls. To improve consistency across the paradigms used for the patient vs. controls contrasts, articles were then split into broad tasks categories: emotion (exposure to phobic [e.g., spider images], traumatic [e.g., combat films], socioemotional [e.g., faces] or general strongly aversive stimuli [e.g., loud noises]), attention (sensory detection and focus, Go/NoGo), decision (strategic planning and calculus, monetary decision-making) and memory (working memory encoding and retrieval, learning tasks). The main patients vs. controls analyses were focused on the emotion category (138 articles) which includes 2554 patients and 2348 controls (of which 27 were a depressed (but not anxious) control sample and the rest healthy) because it was the largest paradigm subset. 693 participants undergoing induction of anxiety were included. Post-traumatic stress disorder (PTSD) articles using traumatized controls (22 articles, 325 patients, 353 controls) were not included in the main analysis. Unthresholded maps for 17 of the 138 included articles were obtained

(one included induced anxiety study provided unthresholded maps but did not report coordinates (15 subjects)). See Tables S1 for a full description of the samples and included articles.

### **SDM meta-analysis procedure**

Activation and deactivation coordinates, as well as the t-threshold and t-values, were collected from each article for the contrast of interest and entered into the SDM-PSI (32) software (version 6.12).

See Supplementary methods for full details, but briefly, for each article group, coordinate-based maps were reconstituted and preprocessed with default parameters (FWHM 20mm, gray matter mask). This led to the following analyses: 1) a meta-analysis of all induced anxiety articles, 2) a meta-analysis of all pathological anxiety articles using emotional tasks, 3) convergence analysis of induced anxiety vs. pathological anxiety, 4) separate meta-analyses of the PTSD, social anxiety disorder, generalized anxiety disorder, panic disorder and specific phobia diagnostic groups of pathological anxiety, and 5) separate convergence analyses of induced anxiety vs. each of the 5 main diagnostic groups.

Publication bias was assessed for each cluster with the Egger's test implemented in SDM using, for each cluster, the mean effect size from each study. See also Supplementary materials for exploratory analyses of the non-emotion tasks in pathological anxiety and of PTSD patients with traumatized controls.

An exploratory similarity analysis between our induced anxiety results and a recent fear-conditioning meta-analysis (28) was also conducted via the Neurovault comparison tool in the similarity search (chosen map: CS+ vs. CS-, pseudo Z scores). Regional correlations were calculated from a brain-masked, 4mm transformation of the original images.

## RESULTS

Findings from 138 papers and 5595 participants are presented here. See Table S2 in Supplement for full list of included papers. All collected coordinates and t-value files are available online at <https://osf.io/9s32h/>. All unthresholded whole-brain activation and convergence maps reported below are available online at <https://neurovault.org/collections/6012/>.

### **Pathological anxiety-associated brain activity**

Anxious patients across disorders (N=2554) vs controls (N=2348) demonstrated increased activation bilaterally in a cluster encompassing the middle and superior temporal gyri, insula and inferior frontal gyrus (IFG), the left part extending to the amygdala, parahippocampal gyrus, hippocampus, bilateral lingual and fusiform gyri and thalamus ( $z=5.413$  and  $z=6.156$  for left and right clusters respectively). Increased activation was also found in the anterior and mid-cingulate and superior medial frontal gyrus ( $z=3.951$ ). Other clusters of increased activation include left middle occipital, left postcentral gyrus, bilateral caudate, bilateral calcarine fissure, bilateral precuneus, right supramarginal, bilateral superior parietal and superior occipital gyri, right parahippocampal gyrus, left middle frontal gyrus, and supplemental motor area. No clusters of reduced activation were significant. No significant publication bias was revealed by the Egger's test for any peak, including the left (bias=0.34,  $p=0.519$ ) and right (bias=0.46,  $p=0.355$ ) superior temporal gyrus (STG)/insula/IFG clusters and the cingulate/medial frontal clusters (bias=0.25, bias=0.14 and bias = 0.05,  $p=0.666$ ,  $p=0.780$  and  $p=0.927$  respectively) (see Figure 1A and Table 1 for full information). Upon specific examination, bilateral peri-aqueductal grey (PAG) increased activation was also found.

*[Fig. 1]*

*[Table 1]*

*Diagnostic group analyses*

Breaking down analyses into diagnostic groups (Figure 2A), specific phobia (414 patients) shows the three increased activation clusters in cingulate and bilateral IFG/insula. Panic disorder (263 patients) and PTSD (436 patients) also show more activation in bilateral insula/STG but no activation or deactivation in the mid- and anterior cingulate cortex. Social anxiety disorder (805 patients) shows activation in the right insula/IFG/STG and left amygdala but no activation or deactivation in the cingulate as well. In contrast to the other disorders, generalized anxiety disorder (233 patients) shows *deactivation* in the cingulate cortex and in bilateral insula. See Table S3 Supplement for full disorder-specific peak information. No significant publication bias was revealed by the Egger's test for any peak. PAG increased activation was found bilaterally in specific phobia and in the left hemisphere in panic disorder.

[Fig. 2]

### **Induced anxiety-associated brain activity**

Across participants (N=693), induced anxiety in threat vs. safe conditions demonstrated greater activation in the cingulate and medial frontal cortices ( $z = 6.415$ ), and bilaterally in the inferior frontal gyrus (IFG)/anterior insula/Rolandic operculum ( $z = 5.183$ ,  $z = 5.067$  for right and left clusters respectively). Other areas of increased activation include bilateral supramarginal, right superior temporal (STG), right middle frontal, and right precentral gyri. Reduced activation was found in bilateral parahippocampal gyrus, fusiform and lingual gyri, as well as in bilateral calcarine fissure, bilateral inferior temporal, middle temporal, inferior occipital bilateral, left postcentral and left orbital medial frontal gyri. The Egger's test for publication bias was not significant for any clusters, including the cingulate/medial frontal (bias=1.5,  $p=0.11$ ) the left (bias=1.3,  $p=0.20$ ) and right (bias=1.91,  $p=0.15$ ) IFG/anterior insula clusters (See Figure 1B, Table 1 for full information). Upon specific examination, BNST and PAG increased activation was also found. Restricting the analysis to induction of anxiety via threat-of-shock did not affect primary outcome (see Table S4 for details).

### **Comparison between induced and pathological anxiety**



When comparing all pathological anxiety with induced anxiety (Figure 1C) we see convergence for increased activation in bilateral insula/IFG and in ACC/mid-cingulate cortex (MCC)/superior medial frontal cortex. These clusters were also present, both for activity and convergence, in the complementary FWHM 10mm analysis. Convergence was also found in bilateral PAG activation. Excluding articles reporting any medicated patients or articles using a youth patient sample did not affect primary outcomes (see Table S5).

#### *Diagnostic group analyses*

When compared with induced anxiety (Figure 2B), specific phobia shows convergence for increased activation in cingulate/medial prefrontal and in bilateral insula/IFG/putamen/STG pole. Panic disorder shows convergence for bilateral insula/IFG hyperactivation whereas PTSD is only convergent with induced anxiety for increased activation in insula/IFG opercular, but not for IFG triangular or orbital. Social anxiety disorder converges in the right insula/IFG orbital and triangular. Generalized anxiety disorder shows very limited overlap with induced anxiety. These findings are illustrated in Figure 2B. See Table S6 in Supplement for full pairwise convergence peaks. All clusters mentioned above were also present in the FWHM 10mm analysis for convergence with induced anxiety, although the left insula contribution to activity in panic disorder was absent. Specific phobia also converged with induced anxiety for bilateral BNST and PAG activation.

#### **Overlap of induced anxiety with fear-conditioning**

Induced anxiety showed a whole-brain Pearson correlation coefficient of  $r=0.66$  with a fear-conditioning meta-analysis. Regional correlations were  $r=0.76$  for the putamen,  $r=0.75$  for the insula,  $r=0.73$  for the frontal lobe,  $r=0.65$  for the parietal lobe,  $r=0.57$  for the caudate and  $r=0.54$  for the thalamus.

## **DISCUSSION**

Consistent with the hypothesis that induced anxiety may be an experimental psychopathological model of anxiety disorders, induced and pathological anxiety show overlapping neurobiological activations.

Specifically, induced anxiety and pathological anxiety both converged in increased activation in the cingulate cortex/medial PFC, bilateral insula/IFG and PAG. However, there were also some important dissociations, especially when pathological anxiety was broken down into component disorders, perhaps suggesting that induced anxiety overall might be a closest model for specific phobia and furthest from generalized anxiety disorder.

### **Induced anxiety as a model for pathological anxiety**

The first thing to note is that induced anxiety evokes ACC, MCC, medial PFC and insula activation, as well as activation in the BNST and PAG. The insula and cingulate regions have been argued to form part of a ‘fear-conditioning’ circuitry (28) and/or a ‘salience’ network (33) which drives interoception in particular (34). In fact, Neurovault similarity analysis reveals that our induced anxiety map shows reasonably high ( $r \sim 0.7$ ) correlation with a recent meta-analysis investigating Pavlovian fear-conditioning neural correlates (28). The overlapping regions perhaps therefore reflect a shared circuitry that responds to the threats common to anxiety- and fear-conditioning, with the non-overlapping circuits perhaps being specific to the spatial/temporal predictability of these threats. MCC electroencephalographic activity has also been reported to play a key role in adapting behaviour to uncertainty and to be modulated by anxiety (35). It is therefore possible that these regions contribute to circuitry which (in the case of the cingulate) detect salient environmental stimuli and then promote behavioural avoidance of threats (via connections to motor cortex), or (in the case of the insula) detect salient internal change that require some kind of homeostatic response (e.g. heart rate increases). The overall effect being to reduce the negative impact of potential harms (perhaps in concert as part of a putative ‘salience’ network).

Critically, the same insula and cingulate activations are seen across pooled anxiety disorders in our data, as well as in older meta-analyses (26,36–38). They may therefore play the same role in pathological anxiety disorders – promoting avoidance responses to salient negative stimuli. Indeed, insula and midcingulate response is thought to be a promising predictor of psychotherapy response (39), which suggests that this circuitry is also important for clinical response (which is largely defined as a reduced avoidance/response to threats).

Thus, induced anxiety holds promise as an intermediate translational model of anxiety disorders (7). In other words, promising new candidate medications might be shown to first modify the effects of threat of shock in animal models (i.e., subjecting animal to unpredictable shocks) (12), and then the effects of threat of shock in healthy humans, before being rolled out in a full-scale clinical trial in anxiety disorders. This would provide greater confidence that the candidate medication targets relevant symptoms and mechanisms and therefore improve the (currently very poor) hit rate of psychiatric drug development (4,5). This is important because it has been suggested that fear and anxiety in humans can and should both be conceptually segregated across two systems with separate but interacting circuitry: the behavioral and physiological response on the one hand, and the conscious feeling and state on the other hand (40). Conceptually, induction of anxiety via unpredictable threat spans *both* systems in humans: a conscious but diffuse feeling of anxiety as well as avoidance and physiological defensive arousal. The overlapping activity we observe may therefore be involved in both of these facets, but future work (ideally with identical cognitive tasks across both induced and pathological anxiety) is needed to truly disentangle these important distinctions. At the same time, if very similar manipulations can be used in animal models, it (to a certain extent) circumvents the problem that it is not possible to measure subjective feeling/states in animal models. In other words, consistent translational manipulations provide a more direct bridge from animal models to human clinical work as well as a means of eventually reconciling disparate anxiety-related systems (40).

### **Specificity across disorders**

However, it is important to recognize that although similarities were found with induced anxiety when all the pathological anxiety studies were pooled together, some differences became apparent when studies were split by disorder. These results may be confounded by biases in sample sizes and/or cognitive tasks (see limitations) thus we must refrain from excessive interpretation, but specific phobia was revealed to be most similar to induced anxiety, showing significant increased activation convergence in the cingulate cortex/medial PFC and bilateral insula/IFG as well as BNST and PAG. PTSD and panic disorder converged with induced anxiety for increased activation in bilateral insula but not for cingulate hyperactivation. Finally, both social anxiety disorder and generalized anxiety disorder had a more complex pattern, the former only converging in the right insula and the latter actually failing to show convergence for bilateral insula and cingulate hyperactivation. As such, it may also be that induced anxiety is a better model for some sub-types of pathological anxiety than others.

Overall, the disorder-specific findings might indicate that pathological anxiety mechanisms are diverse and that we should not always assume similarities across disorders. Indeed, with the detection power allowed by the current literature, induction of anxiety – mainly by threat of unpredictable shock – appears to be a very good model for specific phobia, relatively good for panic disorder, PTSD and possibly social anxiety disorder, and less relevant for generalized anxiety disorder at the functional activation level. However, future direct comparison with the exact same tasks and the same power in all groups is needed to be confident in this prediction.

### **Limitations**

To our best knowledge, this is the first meta-analysis to investigate functional activity in anxiety induced via unpredictable threat paradigms and compare it with up-to-date meta-analyzed functional activity of pathological anxiety disorders. However, it is important to recognize several limitations.

Firstly, in order to collect sufficient samples in each group of eligible articles, we did not exclude any articles based on male/female ratio, age, as well as potential medication, individual comorbidities and

clinical severity – all of which could potentially confound the functional correlates of anxiety in patients or in healthy controls.

Secondly, splitting by anxiety type and disorders (and restricting to emotional tasks) resulted in varied group sizes, leading to differences in power between diagnostic-specific meta-analyses as well as an overabundance of some task types in some disorders (e.g. symptom provocation in specific phobia). Similarly, at a more global level, the tasks within the induced anxiety sample are more consistent than the diverse emotion tasks in the pathological anxiety sample. Ultimately, this likely makes all interpretation of the differences between groups less solid than the observed common/shared effects.

Thirdly, although we did restrict analyses to broad task categories, we did not filter our systematic analysis by precise task, because we wanted to examine as many aspects of anxiety as the body of literature allowed. As a result, the tasks used in eligible articles are somewhat diverse. For example, the criteria of our broad emotion task category were: exposure to threatening or strongly aversive (electrical shocks, loud noises, etc), phobic (images of spider, sounds of dental care, etc), traumatic (combat-related movies, etc) or socioemotional (faces or words with or without emotion, etc) stimuli. Since all induced anxiety papers included strongly aversive threat, they all qualified by definition into this category.

Coincidentally, all the eligible specific phobia papers used phobic stimuli in their specific tasks and as a result also qualified for the emotion category. Hence, one explanation for the consistency between specific phobia and induced anxiety may be that the symptom provocation tasks used (i.e. those designed to provoke symptoms using shocks or phobic stimuli) were most similar across these studies. In a broader sense, our inclusive approach where we pool articles across different diagnoses and paradigms will inevitably lead to biases that limit inference. In an ideal world, we would restrict analyses to single tasks, but this would severely limit our detection power. As it stands, our inference is perhaps stronger for the conjunction analyses (where we are seeing similarities *in spite of* confounds) rather than our difference analyses (where discrepancies might simply be driven by confounds).

Fourthly, it is worth noting that we restricted our meta-analysis to articles reporting a whole-brain analysis. Unfortunately, for a small number of articles, it was unclear as to whether the analysis was carried out with homogenous thresholding across the whole brain or whether some regions of interest were singled out (again, we would recommend increased clarity in reporting of future research). Notably, some key structures, the amygdala in particular, as well as the BNST and PAG, often do not emerge in whole-brain analyses, most of which have a comparatively large cluster threshold. Thus, our results for the most part did not reflect amygdala activation or deactivation, which are often reported in region-of-interest analyses only.

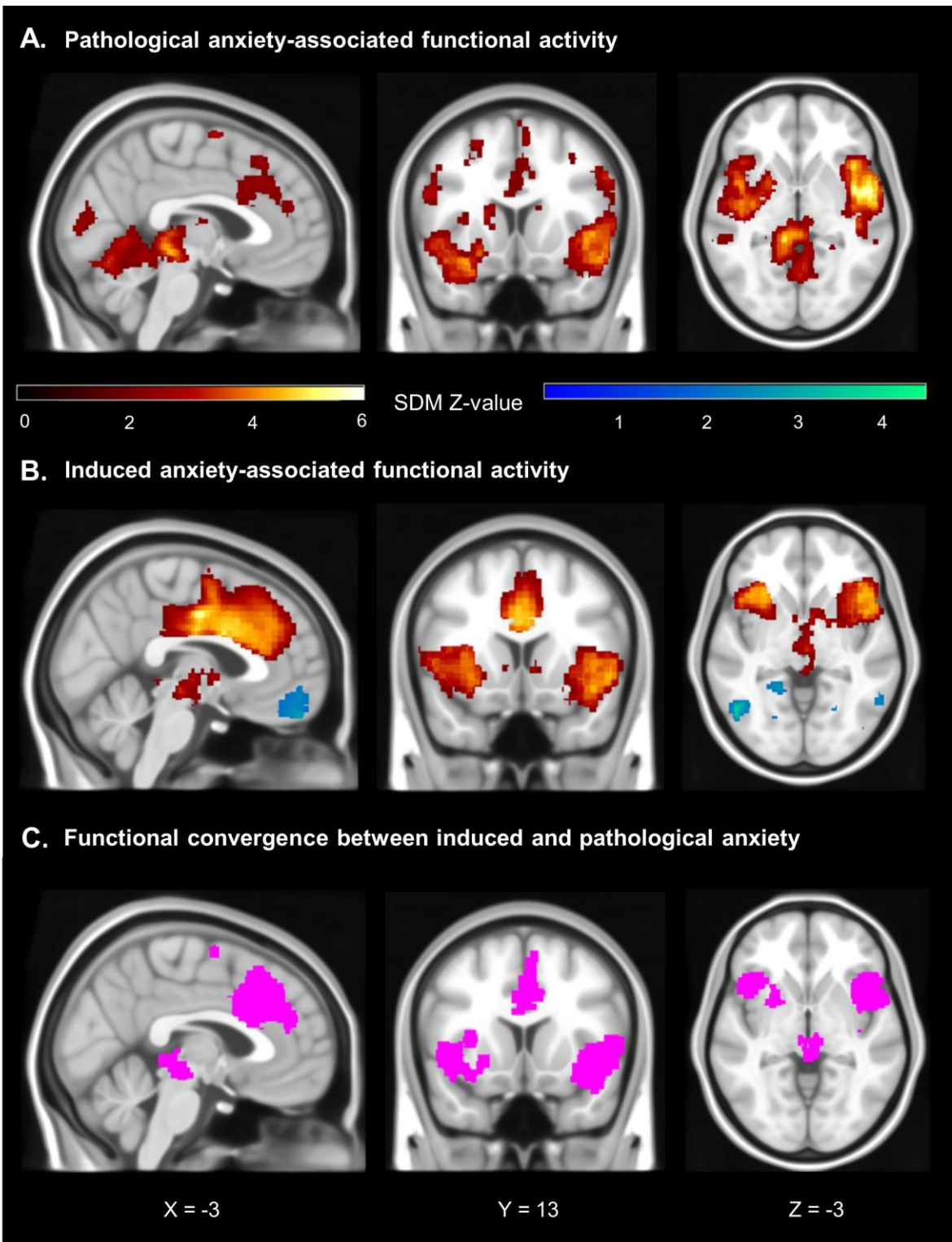
## **Conclusion**

This meta-analysis demonstrates that induced anxiety evokes activation of cingulate and insular regions in common with pathological anxiety, which (at least partially) validates the former as an intermediate translational model of the latter. Nevertheless, our findings also indicate functional differences between anxiety disorders, suggesting that induced anxiety might be a better model for some disorders than others.

## **AUTHOR CONTRIBUTIONS**

OJR designed the project and analysis strategy. AVC iteratively completed the search (by systematically screening articles and creating long eligible lists that were progressively narrowed), contacted researchers for raw data, and conducted the SDM analysis under the direct supervision of OJR. OJR and AVC wrote and edited the manuscript together.

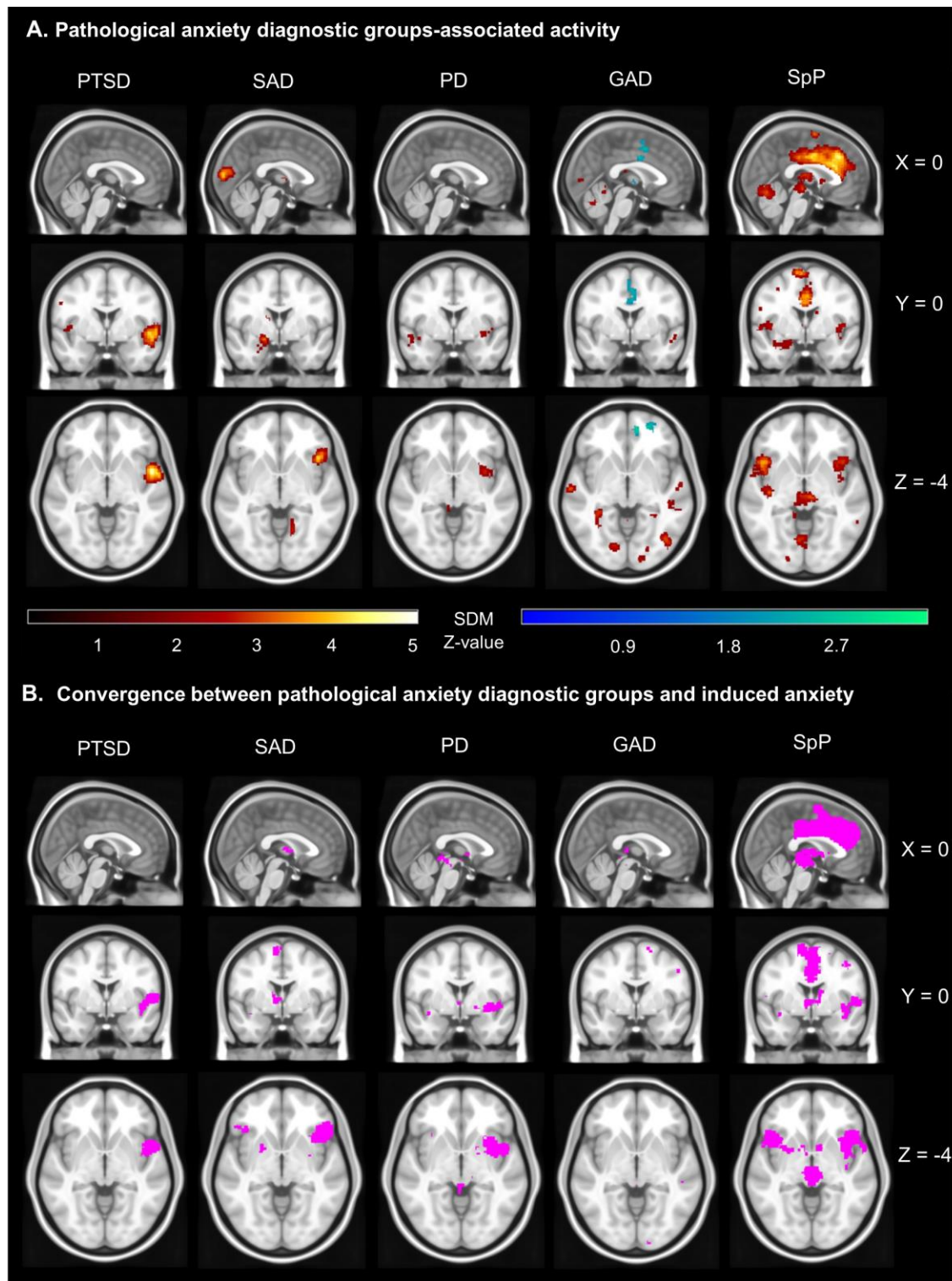
## FIGURES



**Figure 1: Functional activation and convergence for induced and pathological anxiety**



A- Brain regions differing significantly between threat vs. safe conditions in induced anxiety studies (693 participants). B- Brain regions differing significantly between 2554 anxious patients vs. 2348 controls across pathological anxiety studies. SDM Z-value of activation in red-yellow gradient, deactivation in blue-green. C- Convergence of brain regions between induced vs. pathological anxiety. Converging activation in purple.



**Figure 2: Functional activation and convergence with induced anxiety for each anxiety disorder**

A- Brain regions differing significantly between anxious patients vs. controls for each anxiety disorder (Post-traumatic stress disorder - PTSD: 436 patients vs. 411 controls; Social anxiety disorder - SAD: 805 vs. 741; Panic disorder - PD: 263 vs. 268; Generalized anxiety disorder - GAD: 233 vs. 218; Specific phobia - SpP: 414 vs. 373). SDM Z-value of activation in red-yellow gradient, deactivation in blue-green.

B- Convergence of brain regions between induced anxiety and each anxiety disorder. Converging activation in purple.

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**Table 1: Whole-brain meta-analysis of induced anxiety articles in threat vs. safe conditions, and of pathological anxiety articles across disorders**

$p \leq 0.005$ ,  $k \geq 10$ . Exploratory Egger's tests are reported for meta-analytic clusters. Egger's test not applicable to convergences, as those are pairwise comparisons of meta-analyses. ACC: anterior cingulate cortex; MCC: midcingulate cortex; MidFG: middle frontal gyrus; MedFG: Medial frontal gyrus; Orb. MedFG: orbital medial frontal gyrus; SMA: Supplementary motor area; STG: superior temporal gyrus; MTG: Middle temporal gyrus; ITG: inferior temporal gyrus; SPG: superior parietal gyrus; IPG: inferior parietal gyrus; SOG: superior occipital gyrus; MOG: middle occipital gyrus; IOG: inferior occipital gyrus

MNI coordinates	Voxels	Z value	Description	Egger's bias	Egger's p value
Pathological anxiety					
-36,6,-14	12836	5.413	L. Insula, IFG all, Putamen, Pallidum, Rolandic operculum, Precentral g., Postcentral g., MidFG, Heschl's g., STG pole, STG, MTG pole, MTG, Amygdala, Hippocampus, bilateral Parahippocampal g., bilateral Vermis 3-7, bilateral Cerebellum 3-6, Cerebellum 8, bilateral Lingual g., bilateral Fusiform g., bilateral thalamus	0.34	0.519
48,4,-14	5701	6.159	R. Insula, IFG all, STG pole, STG,	0.46	0.355



			MTG pole, MTG, Heschl's g., Rolandic operculum, ITG		
-10,-2,68	1203	3.951	Bilateral MCC, SMA, MedFG, ACC, paracentral lobule	0.25	0.666
-36,-74,30	565	3.589	L. MOG, IPG, SOG, SPG	-0.17	0.722
4,-90,12	333	3.362	Centre Calcarine, bilateral Cuneus	0.39	0.397
-26,22,40	280	3.149	L. MidFG, SFG	0.22	0.698
-16,12,14	202	2.975	L. Caudate, Thalamus	0.09	0.863
18,-56,50	180	2.667	R. SPG, Precuneus, IPG	0.09	0.859
20,-76,34	130	2.992	R. SOG, Cuneus	0.11	0.828
22,-16,-26	112	3.146	R. Parahippocampal g., Hippocampus	0.20	0.685
-14,-58,58	64	3.034	L. Precuneus, SPG	0.26	0.611
8,-66,18	65	2.574	R. Calcarine, Cuneus	0.01	0.992
14,10,18	41	2.536	R. Caudate	0.17	0.737
-44,-20,56	37	2.557	L. Postcentral g.	0.14	0.790
34,12,44	30	2.279	R. MidFG	0.03	0.948
58,-32,40	22	2.308	R Supramarginal g.	0.08	0.875
10,-2,66	21	2.433	R. SMA	0.05	0.916
12,-28,40	14	2.201	R. MCC	0.14	0.780
-8,6,38	10	2.114	L. MCC	0.05	0.927
Induced anxiety					
4,38,38	6538	6.415	ACC, MCC, Sup. MedFG	1.48	0.110
50,22,2	4537	5.183	R. Insula, IFG all, Rolandic operculum, STG pole	1.91	0.148
-30,18,-14	1811	5.067	L. Insula, IFG all, Putamen, Rolandic	1.30	0.199

			operculum		
60,-46,36	1373	4.458	R. Supramarginal g., STG, Angular g.	0.78	0.553
46,2,48	336	3.590	R. Precentral g., MidFG	1.53	0.161
-56,-44,28	303	3.354	L. Supramarginal	1.28	0.199
35,52,18	153	2.500	R. MidFG, SFG	2.65	0.222
-10,-34,-48	36	3.019	Possible cerebellum 9,10	0.30	0.778
-48,-62,-6	1251	-4.117	L. ITG, MTG, IOG	-0.32	0.752
-56,-22,46	636	-4.492	L. Postcentral g., IPG	-0.38	0.726
54,-60,-10	559	-4.102	R. ITG, IOG, MTG, MOG	-0.14	0.891
-8,52,-22	478	-3.267	L. Orb. MedFG	-0.11	0.932
42,-44,-16	188	-3.391	R. Fusiform g.	-0.25	0.826
-12,-64,12	185	-3.132	L. Calcarine, Lingual g.	-0.12	0.912
-20,-46,0	121	-3.063	L. Lingual g. Fusiform g.	-0.32	0.769
26,-62,-8,	47	-2.525	R. Fusiform g., Lingual g.	-0.34	0.744
-20,-70,-8	44	-2.697	L. Fusiform g., Lingual g.	-0.38	0.728
-32,-26,-20	42	-2.675	L. Fusiform g., Parahippocampal g.	-0.42	0.695
12,-70,16	41	-2.691	R. Calcarine	-0.09	0.940
-22,-16,-22	37	-3.052	L. Parahippocampal g., Hippocampus	-0.62	0.608
28,-24,-24	13	-2.448	R. Parahippocampal g., Fusiform g.	0.08	0.945
18,-78,14	12	-2.119	R. Calcarine	-0.21	0.842
Convergence					
-12,-8,68	2217		SMA, MCC, MedFG sup., ACC		
44,14,-14	2102		R. Insula, IFG all, STG pole, Rolandic operculum, STG, Putamen		

-38,20,-8	1305	L. Insula, IFG all, Putamen, Rolandic operculum, STG pole
-4,-22,-10	615	R. Thalamus, Vermis 3
50,2,44	183	R. Precentral g., MidFG
12,-26,40	43	R. MCC

## Supplementary Methods

### Literature search and inclusion

Database keywords for titles and abstracts included ‘major depression’, ‘major depressive’, ‘unipolar depression’, ‘traumatized’, ‘trauma’, ‘anxiety’, ‘anxious’, ‘phobia’, ‘phobic’, ‘phobics’, ‘post-traumatic stress disorder’, ‘posttraumatic stress disorder’, ‘panic’, ‘PTSD’ in conjunction with ‘fMRI’ or ‘functional magnetic resonance imaging’. Studies reporting null results for the whole-brain analysis were included.

Unpredictability of the threat relied on the time of delivery and/or the intensity of the stimulus, and nociceptive stimuli were not excluded provided they fit these criteria. Only potent aversive stimuli (ex: electrical shocks, thermal stimuli) were selected as unpredictable threats to avoid potential confusion with stimuli used in other included articles (ex: emotional faces). Classical fear-conditioning paradigms were excluded as they did not fit the unpredictability criteria.

Records were excluded if they were not experimental (and therefore did not include data to analyze), if they were not published in English (as we did not want to risk misunderstanding the reported information), if activation was only reported after treatment administration, and if they used a sample fully comorbid with any current medical or mental condition (other anxiety disorders and unipolar depression excepted), as both treatment and comorbidity could confound reported activations. Authors were contacted for unthresholded activation maps

Full-text articles were excluded if they did not report activation/deactivation coordinates (as they constituted the main analysis), or if thresholding was inhomogenous across the brain (Müller et al., 2018). Exceptions were made for the latter criteria if only *one* region of interest was thresholded differently (in all cases but one this was an amygdala region of interest— we assumed that it was too small to come up in most whole-brain analyses, hence the need for authors to threshold it differently), in which case articles were included but *only* the data using the whole-brain threshold was collected. Articles using fully duplicate samples were excluded where reported. Articles were also excluded if they reported incomplete post-hoc analyses for a whole-brain interaction analysis with more than two levels, as this meant it was impossible to determine which group/condition was driving effects. No additional judgments of study quality were made beyond the exclusions reported above. Authors were contacted for unthresholded activation maps.

For the PTSD category, only articles using healthy controls (and not traumatized controls) were used in the main analysis. Eligible PTSD articles with traumatized controls (22 for emotion tasks, 325 patients, 353 controls) were kept aside only to be compared with the PTSD healthy controls articles. See Table S7 for a whole-brain meta-analysis of PTSD patients vs. traumatized controls for emotion tasks.

Note that for each study we do not take the effect of task, but the effect of anxiety (either induced or pathological) on the task contrast of interest. Our analysis therefore makes the assumption that the average effect of anxiety across multiple tasks provides an estimate of the average impact of anxiety on emotional cognitive task processing *in general*. Notably, this does not mean, for example, that we are comparing the effect of specific tasks in patients to the overall effect of threat of shock. We are comparing the *average impact of patients vs. controls on tasks relative to the average impact of threat vs safe on tasks*. In an ideal world we would compare the exact same tasks across types of anxiety. However, as with any meta-analysis we need to trade precision (i.e. restricting it to the small number of identical tasks) and power (i.e., number of studies) against each other. Given that there simply aren’t enough studies to solely examine a single task type, we believe that this analysis provides the best *current* approximation of the consistencies between induced and pathological anxiety. If anything, averaging across tasks increases the noise, making it harder to find consistent activations, thus the observed overlap is likely biased towards the lower end of true overlap (which in turn urges caution in interpretation of activation *differences*).

### SDM meta-analysis procedure

Z-values, or uncorrected p-values were collected and converted to t-values via the SDM online converter. SDM-PSI also allows for absence of a statistical value, requiring only the direction of the analysis (activation or deactivation): this was used for articles reporting only corrected p-values, F-values not originating from a 2x2 design, or not reporting any statistical value. Atlas space (MNI, Talairach) for each article was also entered into SDM-PSI, which uses matrix transforms (Lancaster et al., 2007) to convert into consistent space. According to SDM recommendations, cluster-forming height thresholds were used instead of voxel thresholds for articles using cluster-

based statistics. A conservative value of  $p = 0.001$  (converted to  $t$ ) was used as threshold for articles in which there was no uncorrected threshold reported or if the threshold was unclear

Articles were split by anxiety type (7 groups: Induced, post-traumatic stress disorder [PTSD], social anxiety disorder, generalized anxiety disorder, panic disorder, specific phobia, transdiagnostic [articles contrasting anxious subjects pooled across two or more of the 5 previous anxiety disorders, with the possible addition of separation anxiety disorder for pediatric samples, with healthy controls]).

Large clusters were also checked in a complementary FWHM 10mm analysis conducted with all other parameters identical. Meta-analytic maps were computed with the SDM mean analysis via permutation tests (Albajes-Eizagirre et al., 2019), thresholded with peak height 1, voxelwise  $p \leq 0.0025$  (paired one-tailed tests, resulting in two-tailed  $p \leq 0.005$ ), uncorrected and clusterwise  $k \geq 10$  voxels as the classical balance between Type I and Type II errors (Lieberman & Cunningham, 2009). Pairwise convergence maps approximating significant commonalities and differences between two article groups were computed with the multimodal SDM tool by assuming error in  $p$ -values and adjusting to reduce false negative rates, and thresholded with a voxelwise uncorrected  $p \leq 0.0025$  (four-tailed) and clusterwise  $k \geq 10$  voxels, peak height 0.00025 (Radua et al., 2013).

Identified clusters were large and encompassed many areas. This meant automated approaches would miss relevant areas by focusing solely on peaks (or indeed erroneously identify out-of-cluster coordinates by triangulating between contributing clusters). As such, all clusters reported were manually confirmed using the AAL atlas visualized on the MRICron software. All contributing brain regions within each cluster were listed. Additionally, the PAG (peri-aqueductal gray) and BNST (bed nucleus of the stria terminalis) regions were visually examined using MNI coordinates ( $x = \pm 4$ ,  $y = -29 (\pm 5)$ ,  $z = -12 (\pm 7)$  (Linnman et al., 2012) and  $x = \pm 8$ ,  $y = 0$ ,  $z = 5$  (Buff et al., 2017) respectively).

It should be noted that effect sizes for non-significant results are unknown and conservatively assumed to be 0 by SDM-PSI unless unthresholded maps were used ( $N = 17$  of 138), making the Egger's test exploratory only.

## **Supplementary results**

### **Other tasks**

We investigated brain activation patterns across anxiety studies for attention, decision and memory tasks as well, although they included an order of magnitude less papers than emotion tasks.

Briefly, anxious patients ( $N = 396$ ) vs. controls ( $N = 324$ ) during attention tasks did not show any significant activation or deactivation. During decision tasks (222 patients vs. 218 controls), they showed reduced activation in bilateral middle frontal gyri and right putamen. During the memory tasks, anxious patients ( $N = 217$ ) compared to controls ( $N = 216$ ) showed increased activation in left hippocampus and right inferior frontal gyrus (IFG) opercular, but reduced activation in anterior cingulate cortex (ACC), midcingulate cortex (MCC) and left insula. (See Table S8 for additional clusters description and peak information).

### **PTSD controls vs trauma**

No convergence was found between studies contrasting PTSD patients with healthy controls and studies contrasting PTSD patients with non-PTSD traumatized controls for emotion tasks.

## **Supplementary discussion**

### **Induced anxiety as a model for pathological anxiety**

Our findings of bilateral BNST activation in induced anxiety is in line with recent literature, which reports that the BNST plays a key role in generating anxious feeling and is engaged when faced with uncertain threat, both in humans and rodents (Davis et al., 2010; LeDoux & Pine, 2016). Interestingly, consistent with older meta-analyses (Etkin & Wager, 2007), BNST activations were not consistently seen in pathological anxiety (although this may depend on medication status; see below), which suggests that this might be an activation that is specific to imminent

state anxiety, which may be less present at any given time in anxious patients than someone undergoing direct threat manipulation (Gungor & Paré, 2016). This is in contrast with the amygdala which was observed in the pathological – but not induced – anxiety group, perhaps indicating more of a role in dispositional anxiety (Hur et al., 2019). Animal work nevertheless demonstrates that uncertain threat response in the BNST usually travels through the amygdala (Gungor & Paré, 2016; Hur et al., 2019) so it may actually be that this dissociation is an artefact of confounds/power issues inherent in meta-analyses. Interestingly, of prior anxiety-linked subcortical regions it was the PAG that was consistent across both groups, perhaps indicating a more general role in anxiety/negative affect. These patterns should of course be treated with caution until replicated in well-controlled direct test across anxiety groups, but they do highlight interesting predictions for future research.

It is worth noting that medication status may be an important factor modulating the role of the BNST in pathological anxiety as excluding medicated patients led to BNST activations in the pathological anxiety group (consistent with the induced anxiety group). It is important therefore to test the effects of medication on BNST activation in future studies.

Our findings indicate a globally satisfactory functional overlap between induced anxiety and anxiety disorders, although some differences were also found, including the absence of amygdala activation in induced anxiety. We however advise caution in overinterpreting apparent differences, particularly for the amygdala and other small regions, and elaborate further on this in the limitations section.

### **Specificity across disorders**

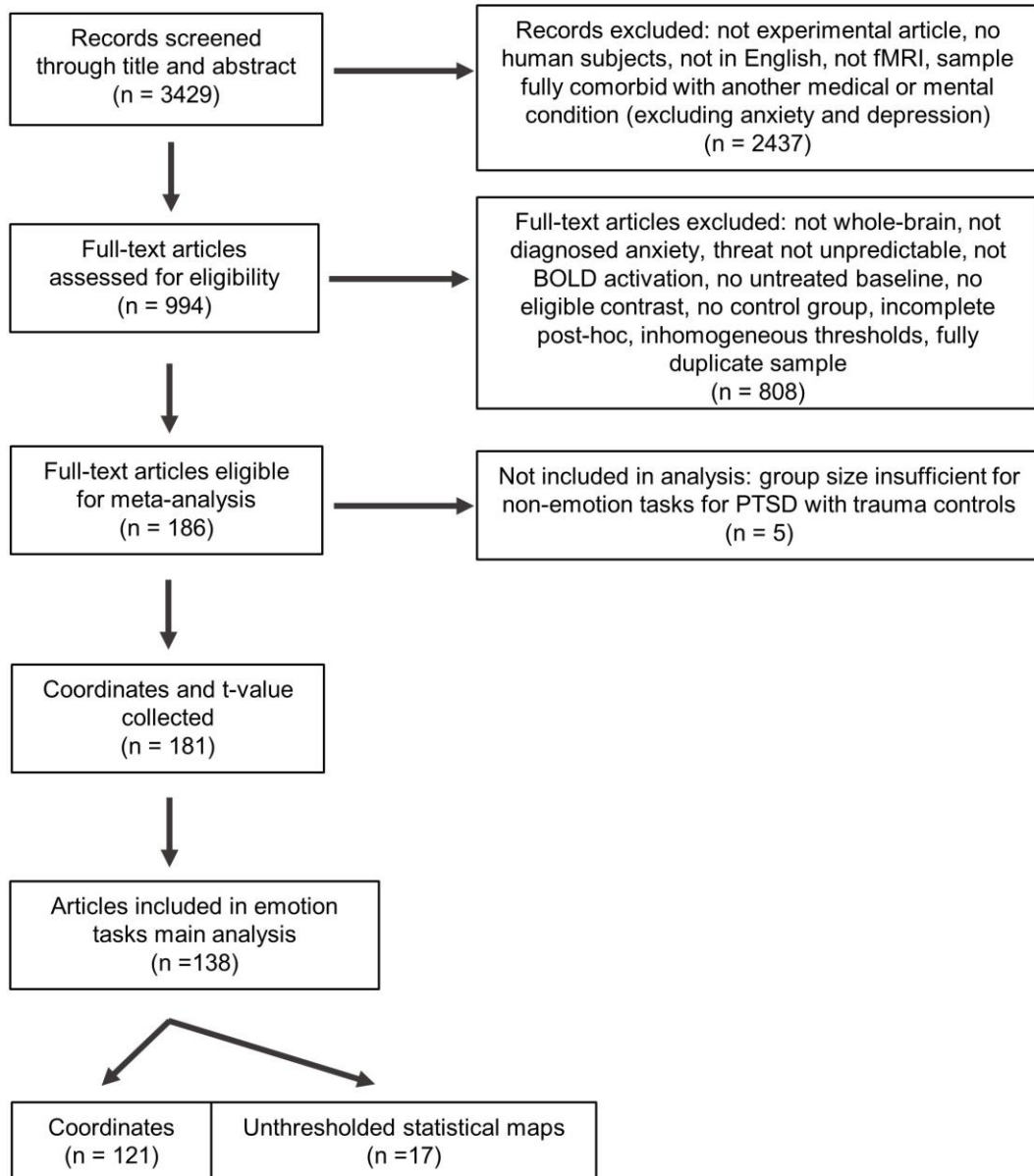
One reason for the apparent differences between diagnostic groups might be that the cingulate cortex is an architecturally complex structure, with anterior regions thought to be involved in social and emotional processing, while medial regions are involved in goal-directed behavior (Apps, 2018). Thus, increased activation in MCC and ACC may reflect an increase in goal-directed behavior in response to emotional stimuli, corresponding to the high state-anxiety following exposure to phobic stimuli in specific phobia patients. By contrast, *reduced* activation in generalized anxiety disorder could perhaps reflect a difficulty engaging in such behavior perhaps as illustrated by housebound avoidance behavior during more continuous trait-like anxiety. In other words, subtleties in symptoms may emerge from (or drive) activation patterns in adjacent medial prefrontal regions with dissociable roles.

The insula is also a complex brain structure receiving numerous sensory inputs and believed to play a key role in interoceptive perception and the integration of bodily sensations (Gogolla, 2017; Paulus & Stein, 2006). Our findings point to hyperactivation of the insula in specific phobia, panic disorder, PTSD and possibly social anxiety disorder, but potential deactivation in generalized anxiety disorder. This could be in line with the difference between defining symptoms of these disorders, some – like specific phobia or panic disorder – are more centred on bodily sensations, whereas generalized anxiety disorder is associated with general feelings of worry, often about external environmental factors. Thus, involvement of this region in anxiety disorders may be specific to symptoms involving interoception. Alternatively, since both cingulate and insula are also part of a hypothetical salience network as defined by Yeo (2011) (Yeo et al., 2011) deactivation in both regions in generalized anxiety disorder might be because it is characterized by more general apprehension and hypervigilance rather than targeted responses to specific salient (e.g. phobic) stimuli.

### **Limitations**

Finally, it is important to recognize some limitations with the meta-analytic approach specifically. SDM only allows one to prioritize maps that were corrected for multiple comparisons and does not filter studies by the p- or z-value threshold they use. There is also a potential publication bias towards activation in the patient vs. control contrast, as many eligible articles do not clearly state whether the opposite (controls vs. patients) contrast had null results meaning it was not possible to know if this evoked significant but unreported activations. The use of unthresholded maps in place of coordinates allows us to avoid those biases, but we were only able to collect 17 maps out of 138 articles. Unfortunately, even the raw data cannot fully circumvent the differences between MRI scanners and pre-processing strategies across all eligible studies. Nevertheless, the Egger's test indicated no significant publication bias for any of the clusters in the primary analyses of interest.

**Figure S1: Flowchart of article selection.**



N represents the number of articles.

**Table S1: Demographic sample description for all 181 articles meeting criteria**

	Induced anxiety	PTSD	PTSD HC	SAD	SAD HC	GAD	GAD HC	PD	PD HC	SpP	SpP HC	Tdiag	Tdiag HC	PTSD <sup>a</sup>	PTSD TC <sup>a</sup>
<b>Articles (studies)</b>	18 (19)	34		41		17		14		21 (23)		14		22	
<b>Sample</b>	693	576	539	830 <sup>b</sup>	766 <sup>c</sup>	289	260	310	323	414	373	492	424	325	353 <sup>d</sup>
<b>Mean Age</b>	25.15	32.48	31.38	28.70	28.83	34.79	35.01	33.04	32.32	25.53	22.66	21.13	21.58	34.88	35.13
<b>Mean Male %</b>	53.67	31.32	31.54	37.52	39.58	36.70	41.93	34.52	35.60	12.03	10.92	30.29	35.59	44.93	43.60

a: articles not using emotion tasks were not included in analysis because there were too few; b: including 33 comorbidly depressed SAD patients; c: including 27 depressed non-SAD controls; d: including 16 depressed non-PTSD patients; HC: healthy controls; TC: trauma-exposed controls; SAD: social anxiety disorder; GAD: generalized anxiety disorder; PD: panic disorder; SpP: specific phobia; Tdiag: transdiagnostic anxiety category, where each article reports two or more anxiety disorders in one pooled group of patient



**Table S2: Comprehensive list of the 181 articles meeting inclusion criteria**

<b>Eligible articles</b>	<b>Anxiety type</b>	<b>Task category</b>
Sakamoto et al. (2005)	PTSD	Emotion, Attention
Williams et al. (2006)	PTSD	Emotion
Strigo et al. (2010)	PTSD	Emotion
Zweerings et al. (2018)	PTSD	Emotion, Attention
Elman et al. (2018)	PTSD	Emotion
Dégeilh et al. (2017)	PTSD	Emotion
Brinkmann et al. (2017)	PTSD	Emotion
Aupperle et al. (2016)	PTSD	Attention
M. J. Kim et al. (2008)	PTSD	Emotion
Rabellino et al. (2016)	PTSD	Emotion
Werner et al. (2009)	PTSD	Memory
Moore et al. (2008)	PTSD	Attention
Simmons et al. (2008)	PTSD	Emotion
Xiong et al. (2013)	PTSD	Emotion
van Rooij et al. (2015)	PTSD	Emotion
Bryant et al. (2005)	PTSD	Attention
Thomaes et al. (2012)	PTSD	Emotion, Attention
Landré et al. (2012)	PTSD	Emotion, Memory
Fonzo et al. (2010)	PTSD	Emotion
Sailer et al. (2008)	PTSD	Decision
Cisler et al. (2015)	PTSD	Decision
Steiger et al. (2015)	PTSD	Emotion
van Rooij et al. (2014)	PTSD	Attention
Aupperle et al. (2012)	PTSD	Emotion
St Jacques et al. (2011)	PTSD	Emotion, Memory
Elman et al. (2009)	PTSD	Emotion
New et al. (2009)	PTSD, PTSD trauma	Emotion
Falconer et al. (2008)	PTSD	Attention
Astur et al. (2006)	PTSD	Memory
Mazza et al. (2012)	PTSD	Emotion
Zhang et al. (2013)	PTSD	Emotion, Memory
Garrett et al. (2019)	PTSD	Emotion
Steuwe et al. (2014)	PTSD	Emotion
Simmons et al. (2011)	PTSD	Emotion
Stevens et al. (2013)	PTSD trauma	Emotion
Naegeli et al. (2018)	PTSD trauma	Emotion
Ke et al. (2016)	PTSD trauma	Emotion
Mazza et al. (2013)	PTSD trauma	Emotion
Mazza et al. (2015)	PTSD trauma	Emotion
Mueller-Pfeiffer et al. (2013)	PTSD trauma	Emotion
Hou et al. (2007)	PTSD trauma	Emotion
Lanius et al. (2002)	PTSD trauma	Emotion
Shin et al. (2001)	PTSD trauma	Emotion
Whalley et al. (2009)	PTSD trauma	Emotion
Whalley et al. (2013)	PTSD trauma	Emotion
Cortese et al. (2018)	PTSD trauma	Emotion
Linnman et al. (2011)	PTSD trauma	Emotion
Rabinak et al. (2014)	PTSD trauma	Emotion
Simmons et al. (2013)	PTSD trauma	Emotion
Fani et al. (2012)	PTSD trauma	Emotion
<b>Eligible articles</b>	<b>Anxiety type</b>	<b>Task category</b>
Hayes et al. (2011)	PTSD trauma	Emotion
Lanius et al. (2007)	PTSD trauma	Emotion
Lanius et al. (2003)	PTSD trauma	Emotion
Yang et al. (2004)	PTSD trauma	Emotion

Fani et al. (2019)	PTSD trauma	Emotion
Patel et al. (2016)	PTSD trauma	Emotion
Stein et al. (2002)	Social anxiety disorder	Emotion
Månsson et al. (2016)	Social anxiety disorder	Emotion
Richey et al. (2014)	Social anxiety disorder	Emotion, Decision
Heeren et al. (2017)	Social anxiety disorder	Emotion, Decision
Kim et al. (2016)	Social anxiety disorder	Emotion
Richey et al. (2017)	Social anxiety disorder	Emotion, Decision
Frick et al. (2013)	Social anxiety disorder	Emotion
Prater et al. (2013)	Social anxiety disorder	Emotion
Giménez et al. (2012)	Social anxiety disorder	Emotion, Attention
Brown et al. (2019)	Social anxiety disorder	Emotion
Goldin et al. (2009a)	Social anxiety disorder	Emotion
Goldin et al. (2009b)	Social anxiety disorder	Emotion
Boehme et al. (2014)	Social anxiety disorder	Emotion
Gaebler et al. (2014)	Social anxiety disorder	Emotion
Ziv et al. (2013)	Social anxiety disorder	Emotion
Pujol et al. (2013)	Social anxiety disorder	Emotion, Memory
Schneier et al. (2011)	Social anxiety disorder	Emotion
Klumpp et al. (2012)	Social anxiety disorder	Emotion
Koric et al. (2012)	Social anxiety disorder	Decision
Blair et al. (2011a)	Social anxiety disorder	Emotion
Blair et al. (2011b)	Social anxiety disorder	Emotion
Brühl et al. (2011)	Social anxiety disorder	Emotion
Nakao et al. (2011)	Social anxiety disorder	Emotion
Klumpp et al. (2010)	Social anxiety disorder	Emotion
Sripada et al. (2009)	Social anxiety disorder	Emotion
Yoon et al. (2007)	Social anxiety disorder	Emotion
Yoon et al. (2016)	Social anxiety disorder	Memory
Sareen et al. (2007)	Social anxiety disorder	Attention, Memory
Phan et al. (2006)	Social anxiety disorder	Emotion
Amir et al. (2005)	Social anxiety disorder	Emotion
Evans et al. (2008)	Social anxiety disorder	Emotion
Heitmann et al. (2017)	Social anxiety disorder	Emotion
Heitmann et al. (2016)	Social anxiety disorder	Emotion
Blair et al. (2016)	Social anxiety disorder	Emotion
Binelli et al. (2016)	Social anxiety disorder	Emotion
Blair et al. (2010)	Social anxiety disorder	Emotion
Gentili et al. (2008)	Social anxiety disorder	Emotion
Schmidt et al. (2010)	Social anxiety disorder	Emotion
Stoddard et al. (2017)	Social anxiety disorder	Emotion
Waugh et al. (2012)	Social anxiety disorder	Emotion
Hamilton et al. (2015)	Social anxiety disorder	Emotion
Yin et al. (2017)	Generalized anxiety disorder	Emotion
Diwadkar et al. (2017)	Generalized anxiety disorder	Memory
Moon et al. (2016)	Generalized anxiety disorder	Emotion, Memory
Moon & Jeong (2017)	Generalized anxiety disorder	Emotion, Memory
Moon et al. (2017)	Generalized anxiety disorder	Emotion, Memory
<b>Eligible articles</b>	<b>Anxiety type</b>	<b>Task category</b>
Moon et al., (2015)	Generalized anxiety disorder	Emotion, Memory
Moon & Jeong (2015)	Generalized anxiety disorder	Emotion, Memory
Monk et al. (2008)	Generalized anxiety disorder	Emotion
Ottaviani et al. (2016)	Generalized anxiety disorder	Emotion, Attention
Fonzo et al. (2014)	Generalized anxiety disorder	Emotion
Price et al. (2011)	Generalized anxiety disorder	Emotion
Schlund et al. (2012)	Generalized anxiety disorder	Emotion
White et al. (2017)	Generalized anxiety disorder	Decision
Karim et al. (2016)	Generalized anxiety disorder	Emotion

Strawn et al. (2012)	Generalized anxiety disorder	Emotion
Paulesu et al. (2010)	Generalized anxiety disorder	Emotion
Palm et al. (2011)	Generalized anxiety disorder	Emotion
Robinson et al. (2013)	Induced – unpredictable shock	Emotion (due to threat)
Balderston et al. (2017a)	Induced – unpredictable shock	Emotion (due to threat)
Balderston et al. (2017b)	Induced – unpredictable shock	Emotion (due to threat)
Kirlic et al. (2017)	Induced – unpredictable shock	Emotion (due to threat)
Klumpers et al. (2017) – 2 studies	Induced – unpredictable shock	Emotion (due to threat)
Torrissi et al. (2016)	Induced – unpredictable shock	Emotion (due to threat)
Gold et al. (2015)	Induced – unpredictable shock	Emotion (due to threat)
Choi et al. (2012)	Induced – unpredictable shock	Emotion (due to threat)
Alvarez et al. (2015)	Induced – unpredictable shock	Emotion (due to threat)
Clarke & Johnstone (2013)	Induced – unpredictable shock	Emotion (due to threat)
Holtz et al. (2012)	Induced – unpredictable hyperventilation	Emotion (due to threat)
Drabant et al. (2011)	Induced – unpredictable shock	Emotion (due to threat)
Klumpers et al. (2010)	Induced – unpredictable shock	Emotion (due to threat)
Reicherts et al. (2017)	Induced – unpredictable thermal painful stimuli	Emotion (due to threat)
Yoshimura et al. (2014)	Induced – unpredictable shock	Emotion (due to threat)
Eser et al. (2009)	Induced – unpredictable CCK4-induced panic-like symptoms	Emotion (due to threat)
Yang et al. (2012)	Induced – unpredictable thermal painful stimuli	Emotion (due to threat)
Kalisch et al., 2006)	Induced – unpredictable shock	Emotion (due to threat)
Wittmann et al. (2018)	Panic disorder	Emotion
Feldker et al. (2018)	Panic disorder	Emotion
Held-Poschardt et al. (2018)	Panic disorder	Emotion, Decision
Reinecke et al. (2015)	Panic disorder	Emotion
Feldker et al. (2016)	Panic disorder	Emotion
Schwarzmeier et al. (2019)	Panic disorder	Emotion
Engel et al. (2016)	Panic disorder	Emotion
Petrowski et al. (2014)	Panic disorder	Emotion
Lueken et al. (2014)	Panic disorder	Emotion
Dresler et al. (2012)	Panic disorder	Emotion, Attention
van den Heuvel et al. (2011)	Panic disorder	Decision
Maddock et al. (2003)	Panic disorder	Emotion, Attention
Wintermann et al. (2013)	Panic disorder	Attention
Pfleiderer et al. (2010)	Panic disorder	Attention
Caseras et al. (2010a) – 2 studies	Specific phobia	Emotion
Caseras et al. (2010b) – 2 studies	Specific phobia	Emotion
Rivero et al. (2017)	Specific phobia	Emotion
Wiemer et al. (2015)	Specific phobia	Emotion
Scharmüller et al. (2014)	Specific phobia	Emotion
<b>Eligible articles</b>	<b>Anxiety type</b>	<b>Task category</b>
Lipka et al. (2014)	Specific phobia	Emotion
Schienle et al. (2013)	Specific phobia	Emotion
Schienle et al. (2007)	Specific phobia	Emotion
Straube et al. (2007)	Specific phobia	Emotion
Goossens et al. (2007)	Specific phobia	Emotion
Schienle et al. (2005)	Specific phobia	Emotion
Dilger et al. (2003)	Specific phobia	Emotion
Aue et al. (2019)	Specific phobia	Emotion
Hilbert et al. (2014)	Specific phobia	Emotion
Zilverstand et al. (2017)	Specific phobia	Emotion
Aue et al. (2015)	Specific phobia	Emotion
Barke et al. (2012)	Specific phobia	Emotion
Straube et al. (2011)	Specific phobia	Emotion, Attention

Schweckendiek et al. (2011)	Specific phobia	Emotion
Hermann et al. (2007)	Specific phobia	Emotion
Lange et al. (2019)	Specific phobia	Emotion
Feldker et al. (2017)	Transdiagnostic anxiety	Emotion
Neumeister et al. (2018)	Transdiagnostic anxiety	Emotion
Williams et al. (2015)	Transdiagnostic anxiety	Emotion
Price et al. (2014)	Transdiagnostic anxiety	Emotion, Attention
Demencescu et al. (2011)	Transdiagnostic anxiety	Emotion
Carlisi et al. (2017)	Transdiagnostic anxiety	Emotion
Galván & Peris (2014)	Transdiagnostic anxiety	Decision
Smith et al. (2018)	Transdiagnostic anxiety	Emotion
Smith et al. (2019)	Transdiagnostic anxiety	Attention
Swartz et al. (2014a)	Transdiagnostic anxiety	Emotion
Swartz et al. (2014b)	Transdiagnostic anxiety	Emotion
Benson et al. (2015)	Transdiagnostic anxiety	Decision
Krain et al. (2008)	Transdiagnostic anxiety	Decision
Campbell-Sills et al. (2011)	Transdiagnostic anxiety	Emotion

**Table S3: Whole-brain meta-analysis of the anxiety disorders diagnostic groups in emotion tasks**

<b>MNI coordinates</b>	<b>Voxels</b>	<b>Z value</b>	<b>Description</b>	<b>Egger's bias</b>	<b>Egger's p value</b>
<b>Social anxiety disorder</b>					
-38,-80,26	1422	3.714	L. MOG, Angular g., MTG	0.01	0.996
42,20,-4	634	4.054	R. Insula, IFG all, STG pole	-0.04	0.971
4,-90,12	564	4.222	Bilateral Calcarine, cuneus	1.09	0.309
-20,0,-12	354	3.251	L. Amygdala, Hippocampus, Parahippocampal g., Olfactive b., Pallidum	0.60	0.614
58,-54,6	192	2.947	R. MTG	1.00	0.415
14,-60,-4	90	2.629	R. Lingual g.	-0.02	0.987
-40,26,-12	61	2.635	L. IFG orbital, triangular	-0.07	0.949
24,62,24	32	2.847	R. SFG, MFG	0.42	0.715
-8,-4,2	33	2.358	L. Thalamus	-0.07	0.956
14,-54,60	12	2.288	R. SPG, Precuneus	-0.32	0.784
-8,34,-8	12	2.170	L. MedFG orb., ACC	0.34	0.774
-12,-2,14	11	2.372	L. Caudate	0.09	0.936
<b>Panic disorder</b>					
-56,-6,-12	536	3.129	L. MTG, STG, ITG, STG pole, Insula	0.04	0.972
34,6,-6	204	3.089	R. Putamen, Insula	-0.10	0.937
-48,36,28	188	3.680	L. IFG triangular, MidFG	-0.45	0.709
26,-36,-16	82	2.245	R. Fusiform g.	0.22	0.863
-10,-32,-10	39	3.166	L. Cerebellum 4/5	1.05	0.413
-34,18,-14	20	2.373	L. Insula	0.60	0.684
44,-12,16	14	2.191	R. Rolandic operculum	1.46	0.253
34,14,26	12	2.325	R. IFG triangular, opercular	0.51	0.687
<b>Generalized anxiety disorder</b>					
58,-28,38	477	3.841	R. Supramarginal g., Postcentral g.	1.09	0.744
42,-28,-18	488	3.537	R. Parahippocampal, Angular g., Hippocampus	0.29	0.926
40,-68,14	241	3.416	R. MTG	0.71	0.837
48,-8,-12	209	3.095	R. MTG, STG, ITG	0.35	0.913
-26,-40,8	175	3.569	L. Hippocampus, Parahippocampal g., Lingual g.	0.25	0.941
44,-74,-2	180	3.107	R. MOG, IOG, ITG	0.49	0.880
-18,-76,-10	161	2.943	L. Lingual g.	1.21	0.688
-64,-14,-6	128	3.131	L. MTG, STG	0.36	0.912
-14,-26,16	101	2.740	L. Thalamus	0.61	0.846
54,10,40	90	2.712	R. Precentral g.	0.14	0.965
-26,-96,6	76	2.651	L. MOG	0.84	0.791
-2,-64,-28	74	2.575	Vermis 8	1.23	0.693
16,-92,-6	54	2.521	R. Calcarine, Lingual g.	0.27	0.936
-4,-50,-12	42	2.559	L. Cerebellum 4/5	0.18	0.957
30,-44,0	38	2.865	R. Hippocampus	0.18	0.957
6,-68,16	37	2.832	Calcarine	0.41	0.899
-60,-30,38	29	2.672	L. Supramarginal g.	0.58	0.856
2,-82,4	30	2.384	Calcarine	-0.31	0.921
-22,30,44	24	2.633	L. MidFG, SFG	-0.23	0.941
-18,-48,-14	15	2.252	L. Cerebellum 4/5, Fusiform g.	-0.32	0.925
<b>MNI coordinates</b>					
22,-78,-10	10	2.244	L. Lingual g., Fusiform g.	0.86	0.785
2,6,46	282	-2.843	MCC, SMA	-0.98	0.762
10,52,6	156	-3.273	R. MedFG orbital, ACC	1.07	0.750

-8,-34,54	165	-2.984	L. Precuneus, MCC	-0.59	0.861
26,54,0	145	-3.397	R. SFG orb., MidFG orb., SFG, MidFG	0.26	0.938
40,16,-14	105	-2.910	R. Insula, STG pole	-0.47	0.885
44,-60,-20	81	-3.526	R. Fusiform g., Cerebellum 1	0.55	0.875
16,-24,-22	66	-3.100	R. Cerebellum 3, possible Parahippocampal g.	-1.40	0.675
12,12,-10	63	-2.509	R. Caudate	-0.29	0.932
12,16,2	61	-2.896	R. Caudate	0.24	0.944
-10,-70,54	59	-2.991	L. Precuneus	-0.54	0.862
-32,-38,66	40	-2.395	L. Postcentral g.	0.19	0.955
-28,-44,52	38	-2.394	L. IPG, Postcentral g.	-0.20	0.952
2,-12,2	21	-2.438	Bilateral thalamus	-0.49	0.889
36,-16,6	18	-2.540	L. Insula	-3.62	0.185
34,46,30	15	-2.400	R. MidFG	-1.37	0.686
<b>PTSD</b>					
50,4,-6	1671	5.105	R. STG, STG pole, Insula, Heschl's g., Rolandic operculum, IFG opercular, MTG, MTG pole	1.35	0.319
-42,-8,6	283	3.059	L. Insula, STG pole, STG, Heschl's g., Rolandic operculum,	-0.39	0.805
-52,4,34	39	2.567	L. Precentral g.	-0.41	0.805
-44,-60,36	18	-2.285	L. Angular g.	-0.24	0.905
-62,-54,32	11	-2.269	L. Supramarginal g.	-0.15	0.937
<b>Specific phobia</b>					
-4,28,36	4369	4.906	MCC, ACC, MedFG sup., SMA	0.62	0.590
-6,-70,-12	2357	3.651	Cerebellum 6, 3-5, 1, Vermis 6, Lingual g.	0.79	0.523
-44,16,-2	2073	3.825	L. Insula, IFG all, STG, STG pole, Heschl's g., Rolandic operculum, Amygdala, Hippocampus, Putamen	1.09	0.382
14,8,14	1177	3.475	Bilateral Caudate	0.80	0.492
38,20,-8	658	3.237	R. Insula, IFG all	1.32	0.329
36,-76,-14	628	2.902	R. Cerebellum 6, 1, Fusiform g., IOG	0.17	0.883
36,-54,54	174	2.832	R. IPG, Angular g.	-0.41	0.760
12,-14,-24	172	2.714	R. Parahippocampal g., Hippocampus	0.61	0.675
-48,-46,46	138	2.735	L. IPG	0.01	0.993
-18,-90,-8	115	2.723	L. Lingual g., IOG	0.90	0.461
24,-36,-34	86	2.641	R. Cerebellum 4/5	0.91	0.437
56,-50,6	80	2.684	R. MTG	-0.10	0.931
-60,-34,8	50	2.535	L. MTG	0.24	0.841
-44,-64,-16	39	2.358	L. Fusiform g.	-0.31	0.806
-20,30,42	30	2.673	L. MidFG/SFG	-0.31	0.797
-36,-64,42	31	2.275	L. Angular g., IPG	0.02	0.988
44,10,-22	30	2.412	R. STG, MTG pole	0.64	0.633
42,38,20	26	2.361	R. MidFG	0.76	0.514
-32,2,48	24	2.287	L. Precentral g./MidFG	0.33	0.770
-6,48,38	18	2.271	L. MedFG	0.25	0.834
<b>MNI coordinates</b>	<b>Voxels</b>	<b>Z value</b>	<b>Description</b>	<b>Egger's bias</b>	<b>Egger's p value</b>
48,-12,50	15	2.231	R. Precentral g.	0.15	0.899
-6,-32,-30	13	2.264	Undefined – L. Pons suggested	0.68	0.583
40,-38,48	10	2.090	R. IPG	-0.60	0.612
<b>Transdiagnostic anxiety</b>					
-34,6,-10	224	2.837	L. Insula, Putamen	0.31	0.838

10,-66,18	184	2.996	R. Calcarine, Cuneus, Lingual g., Precuneus	0.69	0.681
2,20,40	132	2.651	MCC, MedFG	1.03	0.504
12,-36,-24	52	2.409	R. Cerebellum 3, 4/5	1.44	0.325
-4,22,54	42	2.389	R. SMA, MedFG	0.57	0.711
-16,-28,-28	21	2.350	L. Cerebellum 4/5	0.24	0.885
-30,24,38	18	2.355	L. MidFG	0.84	0.608

$p \leq 0.005$ ,  $k \geq 10$ . Exploratory Egger's tests are reported for meta-analytic clusters.

ACC: anterior cingulate cortex; MCC: midcingulate cortex; MidFG: middle frontal gyrus; MedFG: Medial frontal gyrus; SMA: Supplementary motor area; STG: superior temporal gyrus; MTG: Middle temporal gyrus; ITG: inferior temporal gyrus; SPG: superior parietal gyrus; IPG: inferior parietal gyrus; SOG: superior occipital gyrus; MOG: middle occipital gyrus; IOG: inferior occipital gyrus

**Table S4: Whole-brain meta-analysis of anxiety induced by threat-of-shock only**

MNI coordinates	Voxels	Z value	Description	Egger's bias	Egger's p value
42,16,8	8111	6.145	Bilateral Insula, IFG all, STG pole, Putamen, Rolandic operculum, Caudate, Pallidum, Thalamus, Vermis 3, R Lingual g., L.STG	0.84	0.466
2,-4,48	7499	6.011	MCC, SMA, MedFG, ACC, R. SFG	0.06	0.960
58,-44,36	2081	6.505	R. Supramarginal g., Angular g., IPG, STG, MTG, Rolandic operculum	0.75	0.500
32,42,18	888	3.967	R. MidFG, SFG	0.48	0.738
-60,-38,26	743	4.986	L. Supramarginal g., STG	0.69	0.516
18,-62,34	378	4.276	R. Precuneus, Cuneus, SOG	-0.07	0.956
-38,44,26	265	3.598	L. MidFG	1.08	0.333
-42,-52,-30	161	2.758	L. Cerebellum 6, Cerebellum crus 1	0.61	0.624
-34,-54,-48	76	2.794	Cerebellum 8	0.59	0.605
0,-50,-26	65	2.749	Vermis 9, Vermis 4-5	1.08	0.391
54,-30,-10	30	2.643	R. MTG	0.13	0.918
-10,-34,-48	24	2.749	Undefined – Cerebellum 9 suggested	0.58	0.630
28,-48,70	22	2.443	R. SPG	-0.23	0.855
-10,-70,46	17	2.465	L. Precuneus	0.83	0.493
54,-60,-8	2227	-4.151	R. ITG, MTG, MOG, SOG, IOG	-0.60	0.616
-48,-70,0	2130	-4.123	L. IOG, ITG, MTG, MOG, SOG, IPG, SPG, Angular g.	-0.76	0.496
-56,-22,48	1101	-4.630	L. Postcentral g., Precentral g., IPG	-0.76	0.506
-8,50,-22	1074	-4.518	Rectus g., Bilateral SFG orb	-0.15	0.906
-22,-16,-22	899	-4.237	L. Hippocampus, Parahippocampal g., Fusiform g, Lingual g, Calcarine	-0.61	0.605
-42,-10,16	295	-3.506	L. Insula, Heschl's g., Rolandic operculum	-0.37	0.747
52,-2,24	285	-3.829	R. Postcentral, Precentral	-0.67	0.549
26,-6,-28	276	-3.600	R. Parahippocampal g. Hippocampus	-0.40	0.731
-12,-54,18	195	-3.150	L. Calcarine, Cuneus, Precuneus	0.02	0.987
12,-54,12	179	-3.127	R. Calcarine, Cuneus, Precuneus	0.01	0.994
-4,-32,66	109	-2.287	Paracentral lobule	-0.23	0.907
26,-62,-6	101	-2.735	R. Fusiform g., Lingual g.	-0.58	0.620
-22,32,40	33	-2.336	L. MidFG	0.24	0.889
28,-56,10	15	-2.311	R. Calcarine	-0.41	0.747
30,-76,14	13	-2.160	R. MOG	-0.45	0.697

$p \leq 0.005$ ,  $k \geq 10$ . Exploratory Egger's tests are reported for meta-analytic clusters.

ACC: anterior cingulate cortex; MCC: midcingulate cortex; MidFG: middle frontal gyrus; MedFG: Medial frontal gyrus; SMA: Supplementary motor area; STG: superior temporal gyrus; MTG: Middle temporal gyrus; ITG: inferior temporal gyrus; SPG: superior parietal gyrus; IPG: inferior parietal gyrus; SOG: superior occipital gyrus; MOG: middle occipital gyrus; IOG: inferior occipital gyrus



**Table S5: Whole-brain pairwise analysis of convergence between induced anxiety and pathological anxiety with exclusions**

MNI coordinates	Voxels	Description
<b>Induced vs. Pathological anxiety (youth patient sample excluded)</b>		
-10,-4,68	2442	SMA, MCC, MedFG sup., ACC
46,14,-14	2150	R. Insula, IFG all, Rolandic operculum, STG pole, STG
-40,20,-10	1009	L. Insula, IFG all, Putamen, Rolandic operculum, STG pole
-4,-22,-10	615	R. Thalamus, Lingual g., Vermis 3
50,4,46	183	R. Precentral g., MidFG
12,-26,40	49	R. MCC
<b>Induced vs. Pathological anxiety (medicated patient sample excluded)</b>		
-2,0,40	2778	MCC, MedFG sup., ACC, SMA
44,20,-2	1720	R. Insula, IFG all, Rolandic operculum, STG pole, STG, Putamen
-48,16,2	1362	L. Insula, IFG all, Rolandic operculum, STG pole, Putamen, Pallidum
-2,-22,-10	517	R. Thalamus, Lingual g., Vermis 3
-6,6,2	192	L. Caudate, Pallidum
-10,-4,68	172	L. SMA

$p \leq 0.005$ ,  $k \geq 10$ .

ACC: anterior cingulate cortex; MCC: midcingulate cortex; MidFG: middle frontal gyrus; MedFG: Medial frontal gyrus; SMA: Supplementary motor area; STG: superior temporal gyrus; ITG: inferior temporal gyrus

**Table S6: Whole-brain pairwise analyses of convergence between induced anxiety and anxiety disorders diagnostic groups in emotion tasks**

MNI coordinates	Voxels	Description
<b>Induced vs. Specific phobia</b>		
2,28,16	5043	MCC, MedFG sup., ACC, SMA
38,20,-8	1274	R. Insula, IFG all, STG pole, STG, Putamen
2,-20,-8	997	Bilateral Thalamus, Caudate, L. Pallidum
-42,18,-4	846	L. Insula, IFG all, Putamen, Rolandic operculum, STG pole
-38,4,-14	51	L. Putamen, Insula
<b>Induced vs. Panic disorder</b>		
34,6,-6	490	R. Insula, Putamen, STG pole
-34,18,-14	131	L. Insula, IFG orbital, STG pole
<b>Induced vs Generalized anxiety disorder</b>		
58,-30,34	205	R. Supramarginal g.
<b>Induced vs. PTSD</b>		
52,8,-6	689	R. Rolandic operculum, Insula, IFG opercular, STG pole, STG
-50,8,2	36	L. Rolandic operculum, Insula, IFG opercular, STG pole
<b>Induced vs. Social anxiety disorder</b>		
48,18,-12	954	R. IFG all, Insula, STG pole
-56,-46,32	225	L. Supramarginal g., STG, MTG
-42,26,-10	82	L. IFG orbital
-8,-2,64	34	L. SMA
<b>Induced vs. Transdiagnostic anxiety</b>		
2,20,40	621	SMA, MCC, MedFG
-26,10,-2	193	L. Insula, putamen, STG pole

$p \leq 0.005$ ,  $k \geq 10$ .

ACC: anterior cingulate cortex; MCC: midcingulate cortex; MidFG: middle frontal gyrus; MedFG: Medial frontal gyrus; SMA: Supplementary motor area; STG: superior temporal gyrus; MTG: Middle temporal gyrus; ITG: inferior temporal gyrus; SPG: superior parietal gyrus; IPG: inferior parietal gyrus; SOG: superior occipital gyrus; MOG: middle occipital gyrus; IOG: inferior occipital gyrus

**Table S7: Whole-brain meta-analysis of PTSD contrasting patients vs. traumatized controls in emotion tasks**

<b>MNI coordinates</b>	<b>Voxels</b>	<b>Z value</b>	<b>Description</b>	<b>Egger's bias</b>	<b>Egger's p value</b>
-2,-30,48	193	2.745	MCC, Precuneus, PCC	0.57	0.663
-48,-68,14	42	2.519	L. MTG, MOG	1.19	0.389
-16,-64,54	38	2.654	L. SPG, Precuneus	1.12	0.398
-26,-70,50	32	2.429	L. SPG	1.08	0.420
-14,46,4	28	2.684	L. ACC, MedFG orb.	0.64	0.622
10,-24,44	28	2.447	R. MCC	0.68	0.597
-14,-74,14	21	2.460	L. Calcarine	1.16	0.355
6,-80,20	21	2.396	Cuneus, Calcarine	1.91	0.113
48,26,28	16	2.535	R. IFG triangular	0.36	0.770
44,-18,50	16	2.281	R. Postcentral g., Precentral g.	-0.73	0.586
32,42,14	14	2.596	R. MidFG	1.27	0.288
0,-68,12	11	2.191	Calcarine	1.59	0.203
4,34,0	197	-3.948	ACC (pregenual), MedFG orb.	-1.13	0.371
56,-30,16	11	-2.529	R. STG	-0.90	0.462

$p \leq 0.005$ ,  $k \geq 10$ . Exploratory Egger's tests are reported for meta-analytic clusters.

ACC: anterior cingulate cortex; MCC: midcingulate cortex; PCC: posterior cingulate cortex; MidFG: middle frontal gyrus; MedFG: Medial frontal gyrus; STG: superior temporal gyrus; MTG: Middle temporal gyrus; SPG: superior parietal gyrus; MOG: middle occipital gyrus

**Table S8: Whole-brain meta-analysis of pathological anxiety across disorders for non-emotion tasks**

MNI coordinates	Voxels	Z value	Description	Egger's bias	Egger's p value
<b>Attention</b>					
<i>No peaks</i>					
<b>Decision</b>					
20,10,-10	69	-2.636	R. Olfactory c., Putamen	0.98	0.743
26,48,20	45	-2.594	R. MidFG	-1.01	0.730
-24,46,30	13	-2.327	L. MidFG, SFG	-0.31	0.891
<b>Memory</b>					
-28,-40,-2	98	3.298	L. Hippocampus, Parahippocampal g.	0.15	0.962
58,12,30	94	2.984	R. IFG opercular, Precentral g.	0.68	0.840
2,4,40	647	-4.344	Bilateral MCC, SMA, ACC	-0.61	0.846
-20,-90,22	459	-4.712	L. SOG, MOG, Cuneus	-1.61	0.584
12,-44,40	363	-3.048	R. Precuneus, MCC, PCC	-1.90	0.526
14,18,4	287	-4.135	R. Caudate	-2.50	0.373
48,42,4	250	-3.690	R. MidFG, IFG orbital, triangular	0.23	0.945
26,-86,32	233	-3.169	R. SOG, Cuneus, MOG	-2.19	0.506
-32,46,32	197	-3.032	L. MidFG, IFG triangular	0.05	0.988
34,-4,16	167	-3.066	L. Insula, Rolandic operculum, Putamen, Heschl's g.	-1.72	0.576
-44,-56,20	153	-3.357	L. MTG, Angular g.	-0.79	0.773
6,36,32	124	-2.890	L. ACC, MCC, MedFG sup.	2.08	0.578
14,10,-10	106	-2.986	R. Caudate, Putamen, Olfactory c., Rectus	-0.46	0.888
-40,44,8	104	-3.165	L. MidFG, IFG triangular, MidFG orbital	-0.42	0.882
16,-78,48	91	-2.854	R. Cuneus, Precuneus, SPG	-0.01	0.996
-28,-14,-20	63	-2.778	L. Hippocampus	-0.47	0.888
-10,-74,56	55	-3.151	L. Precuneus	-0.81	0.791
40,34,30	54	-2.727	R. MidFG, IFG triangular	0.11	0.973
46,30,10	37	-2.659	R. IFG triangular	1.35	0.720
52,-2,8	19	-2.470	R. Rolandic operculum	-1.17	0.699
10,-14,70	13	-2.372	R. SMA	-0.65	0.842

$p \leq 0.005$ ,  $k \geq 10$ . Exploratory Egger's tests are reported for meta-analytic clusters.

ACC: anterior cingulate cortex; MCC: midcingulate cortex; MidFG: middle frontal gyrus; MedFG: Medial frontal gyrus; SMA: Supplementary motor area; STG: superior temporal gyrus; MTG: Middle temporal gyrus; ITG: inferior temporal gyrus; SPG: superior parietal gyrus; IPG: inferior parietal gyrus; SOG: superior occipital gyrus; MOG: middle occipital gyrus; IOG: inferior occipital gyrus

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