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Association of enhanced limbic response to threat with decreased cortical facial recognition memory response in schizophrenia

Theodore D. Satterthwaite, M.D., M.A.^{a,c}, Daniel H. Wolf, M.D., Ph.D.^a, James Loughhead, Ph.D.^a, Kosha Ruparel, M.S.^a, Jeffrey N. Valdez, M.S.^a, Steven J. Siegel, M.D., Ph.D.^a, Christian G. Kohler, M.D.^a, Raquel E. Gur, M.D., Ph.D.^{a,b,c}, and Ruben C. Gur, Ph.D.^{a,b,c}

^aDepartment of Psychiatry, University of Pennsylvania, Philadelphia PA 19104, USA

^bDepartment of Radiology, University of Pennsylvania, Philadelphia PA 19104, USA

^cPhiladelphia Veterans Administration Medical Center, Philadelphia PA 19104, USA

Abstract

Objective—Recognition memory of faces is impaired in patients with schizophrenia, as is the neural processing of threat-related signals, but how these deficits interact to produce symptoms is unclear. Here we used an affective face recognition paradigm to examine possible interactions between cognitive and affective neural systems in schizophrenia.

Methods—fMRI (3T) BOLD response was examined in 21 controls and 16 patients during a two-choice recognition task using images of human faces. Each target face had previously been displayed with a threatening or non-threatening affect, but here were displayed with neutral affect. Responses to successful recognition and for the effect of previously threatening vs. non-threatening affect were evaluated, and correlations with total BPRS examined. Functional connectivity analyses examined the relationship between activation in the amygdala and cortical regions involved in recognition memory.

Results—Patients performed the task more slowly than controls. Controls recruited the expected cortical regions to a greater degree than patients, and patients with more severe symptoms demonstrated proportionally less recruitment. Increased symptoms were also correlated with augmented amygdala and orbitofrontal cortex response to threatening faces. Controls exhibited a negative correlation between activity in the amygdala and cortical regions involved in cognition, while patients showed a weakening of that relationship.

Conclusions—Increased symptoms were related to an enhanced threat response in limbic regions and a diminished recognition memory response in cortical regions, supporting a link between two brain systems often examined in isolation. This finding suggests that abnormal

Please address correspondence to: Theodore D. Satterthwaite, University of Pennsylvania School of Medicine, Department of Psychiatry, 3535 Market Street, 2nd Floor, Philadelphia, PA 19104, Ted.Satterthwaite@uphs.upenn.edu.

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processing of threat-related signals in the environment may exacerbate cognitive impairment in schizophrenia.

INTRODUCTION

Patients with schizophrenia show impairments in cognition (1, 2) and emotion processing (3, 4) that affect functional outcomes. Despite some discussion of relationships between these deficits (5), few studies have investigated the interaction between cognitive and emotional processing demands in schizophrenia. Studies in healthy people suggest that cognitive functioning and emotional processing are intimately related. For example, responses to threatening stimuli impedes performance on cognitive tasks (6). fMRI studies have identified a potential neural basis for this interaction, demonstrating that increased responses to threat in ventral limbic regions inhibit activation in cortical regions involved in cognition (7, 8).

Recognition memory is prominently affected in schizophrenia (1, 9), and is related to dysregulation of multiple brain regions (10–13). In a series of experiments in healthy individuals, Buckner and colleagues demonstrated that medial and lateral parietal regions are the most reliably recruited brain regions by recognition memory tasks (14, 15). These regions are functionally connected with the hippocampus (16) and abnormalities within this network correlate with symptoms of dementia (17). Notwithstanding the traditional focus on fronto-temporal abnormalities in schizophrenia, parietal regions merit increasing scrutiny following findings of parietal abnormalities in the 'default network' in schizophrenia (18). In contrast, emotional paradigms in schizophrenia consistently demonstrate altered recruitment of limbic regions including the amygdala and orbitofrontal cortex (19–21).

The present study examined the interaction between emotion processing and recognition memory systems in schizophrenia using an affective face recognition memory paradigm. Subjects made a simple binary 'old' vs. 'new' choice regarding previously viewed (target) or novel faces, all shown with a neutral expression. However, the target faces were initially displayed with intense threatening (angry or fearful) or non-threatening (happy or sad) affect. Thus, the task has the advantage of integrating both cognitive (old vs. new) and affective (threat vs. non-threat) components. In healthy subjects, this task recruits frontoparietal regions involved in recognition memory, and also produces an amygdala and orbitofrontal cortex response to faces initially seen with a threatening expression (22).

We hypothesized that the symptoms of schizophrenia might reflect an imbalance between emotional and cognitive responses, such that as symptoms become more severe, limbic responses predominate at the expense of cortical recruitment by cognitive demands. Specifically, we examined brain responses using fMRI to test three predictions. First, we expected that greater symptom severity would be correlated with diminished recruitment of parietal regions known to be involved in recognition memory (14, 23, 24). Second, as suggested by previous work (21, 25), we expected that response to threat in limbic regions would be correlated with symptom severity. Third, we used a functional connectivity analysis to test for disruption of the relationship between parietal regions involved in recognition memory and limbic regions involved in threat response.

METHODS

Subjects

The sample included 16 patients (60% male) with schizophrenia (n=12) or schizoaffective disorder depressed type (n=4) and 21 healthy comparison subjects (48% male). Groups were demographically balanced (see Supplementary Methods). After a complete description of the study, subjects provided written informed consent. Standardized assessment followed previously reported procedures (see Supplementary Methods). No subjects met criteria for a depressive episode. All except two unmedicated patients were receiving treatment with antipsychotics (1 first-generation, 12 second-generation, and 1 combined). The average daily dose in chlorpromazine equivalents was 290 (SD=254) mg/day.

Task

The face recognition experiment was preceded by an emotion identification task (26–28), where subjects viewed 30 faces displaying happy, sad, angry, fearful, or neutral affect, and were asked to label the emotion displayed (Figure 1A). Stimuli validation are described in detail elsewhere (29). The face recognition experiment examined here (Figure 1B) presented 30 faces from the emotion identification phase (targets) along with 30 novel faces (foils); subjects made a simple ‘old’ vs. ‘new’ judgment using a two-button response pad. In the current face recognition experiment, all faces were displayed with neutral expressions; target faces had previously been shown in the identification experiment with an emotional expression. However, neutral target faces were displayed with the same expression in both experiments, making neutral trials difficult to compare to other targets; these were therefore excluded from all analyses. Previous studies in our laboratory (26, 27) indicated the utility of dividing emotions by threat-relatedness, as suggested by Gray (30) and others (31, 32). Target faces originally displayed with an angry or fearful affect were modeled together as THREAT; faces originally displayed with a happy or sad affect were modeled as NON-THREAT. There were 6 trials per emotion in the emotion identification experiment, yielding 12 THREAT and 12 NON-THREAT trials.

Performance analysis

Percent correct and median response time were calculated by group (patients vs. controls), prior facial affect (THREAT vs. NON-THREAT), and face recognition response (correct vs. incorrect). Differences in accuracy and response time among the conditions were evaluated with a 2×2×2 (group × affect × response) repeated measures ANOVA, implemented in STATA (College Station, Texas). Response bias was evaluated by calculating *Br* (33), which provides an independent measure of the overall tendency of subjects to make ‘old’ or ‘new’ responses regardless of accuracy.

$$Br = \text{Incorrect foils} / [1 - (\text{Correct targets} - \text{False alarms})] - 0.5$$

Positive values correspond to a liberal familiarity bias (i.e., more likely to say ‘old’ to a new item) whereas negative values correspond to a conservative novelty bias (i.e., more likely to say ‘new’ to an old item). Association with symptoms was evaluated by correlating each patient’s total BPRS score with accuracy and response time for all trials as well as targets and foils separately. Exploratory analyses of the influence of paranoia (as measured by the

“suspiciousness” item on the BPRS) and medication dosage (chlorpromazine equivalents) was also conducted in similar fashion (34). All performance analyses were evaluated at significance level of $p=0.05$ (uncorrected).

Image analysis

fMRI data were preprocessed and analyzed using FMRI Expert Analysis Tool Version 5.9, part of FMRIB's Software Library (www.fmrib.ox.ac.uk/fsl) with standard fMRI settings (see Supplementary Methods) (22). To study both recognition memory and prior facial affect, single subject analyses were carried out separately using two general linear models. The first model examined recognition memory, considering four trial types as regressors of interest: correct to target (HIT), incorrect to target (MISS), correct to foil (CORRECT REJECTION), and incorrect to foil (FALSE ALARM). Analysis of this model focused on the standard recognition memory contrast of HIT>CORRECT REJECTION. The second model examined prior facial affect, and included each original target emotion (happy, sad, anger, fearful) separately as regressors of interest. The main contrast of interest in this model, THREAT>NON-THREAT, was composed of (anger and fear)>(happy and sad). In this analysis correct and incorrect trials were modeled together. Additionally, both models included neutral trials, non-responses, temporal derivatives of each variable, and six rigid-body movement parameters as covariates of no interest.

Within-group and between-group mixed-effects analyses were performed to conduct one-sample t-tests on subject-level whole-brain contrasts. As described below, for each contrast a hypothesis-driven analysis within *a priori* regions of interest (ROIs) was followed by a whole-brain exploratory analysis. To examine individual differences in clinical severity, BOLD response in HIT>CORRECT REJECTION and THREAT>NON-THREAT contrasts were correlated in separate voxel-wise analyses with total BPRS score as an additional covariate of interest. Peak r values are reported for each significant correlation with total BPRS (but see Vul et al. (35)). In an exploratory analysis, we examined the influence of paranoia by adding the suspiciousness item (34) as an additional covariate. In order to contain type I error, correlations were evaluated only within *a priori* ROI; individual items beyond paranoia were not evaluated. Finally, we investigated the effect of medication dosage by adding each patient's antipsychotic dose (chlorpromazine equivalents) as a covariate to each correlation analysis.

We corrected for multiple comparisons using Monte Carlo simulations (AFNI AlphaSim, R. W. Cox; National Institutes of Health) at a Z threshold of 2.33 and a probability of spatial extent $p<0.05$. For display purposes, all figures were smoothed using a $6\times6\times6$ mm kernel and thresholded at $p<0.05$ (uncorrected) using MANGO (J. L. Lancaster and J. Martinez; University of Texas, San Antonio). Identified clusters were anatomically labeled using the Talairach Daemon database (36). Coordinates are reported in Montreal Neurological Institute coordinate space. Peak voxel % signal change was plotted against total BPRS score for each subject for correlation scatter plots.

ROI definition

We identified parietal regions previously implicated in other studies of recognition memory as our *a priori* ROI for the HIT>CORRECT REJECTION analysis (14, 15, 23, 24). This ROI was constructed using a contiguous mask of regions from the Talairach Daemon atlas (36). The large cortical mask (25,098 2×2×2 mm voxels) included the lateral parietal cortex (inferior parietal lobule, angular gyrus, supramarginal gyrus) and medial parietal cortex (precuneus and posterior cingulate). For the THREAT>NONTREAT contrast, we identified bilateral amygdala and orbitofrontal cortex as *a priori* regions of interest (22), anatomically defined using the Harvard-Oxford atlas, thresholded at 0. For both contrasts of interest, we followed the *a priori* analysis with an exploratory whole-brain analysis to identify significant effects outside the *a priori* ROIs. As noted above correlation analyses were conducted for *a priori* ROIs only.

Functional connectivity analysis

In order to examine interactions between limbic regions involved in emotional processing and cortical regions involved in recognition memory, we conducted a functional connectivity analysis using the methods described by Fox et al. (16, 17, 37). To remove confounding sources of correlation, we included three regressors in addition to motion parameters in the model: mean whole brain signal, mean signal within the lateral ventricles, and mean signal within a white matter ROI. We examined correlations with a left amygdala ‘seed’ region, defined anatomically using the Harvard-Oxford Subcortical Atlas at a threshold of 0.75. Finally, in patients, we examined the relationship of connectivity to symptom severity (total BPRS), paranoia, and medication dosage within the *a priori* parietal ROI using the methods described above.

RESULTS

Performance

Behavioral results are displayed in Table 1. Target accuracy was limited by a conservative response bias, indicating that subjects were more likely to judge old target faces as ‘new.’ Patients displayed a somewhat less conservative response bias, although there was not a significant group difference ($p=0.14$). As expected, both patients and controls recognized previously non-threatening faces more accurately ($f[1,35]=7.95$, $p<0.01$). While there was no accuracy difference between groups, patients performed the task more slowly than controls ($f[1,35]=9.04$, $p=0.005$). Furthermore, symptom burden correlated with overall response time ($r=0.56$, $p=0.01$); this BPRS~response time correlation was present for both targets ($r=0.46$; $p=0.03$) and foils ($r=0.45$, $p=0.04$). No other group differences, main effects, interactions, or significant correlations were present.

HIT > CORRECT REJECTION

Controls activated the expected parietal regions (Figure 2A), including the posterior cingulate ($Z_{\max}=3.80$, 1220 voxels, coordinates: -4 , -36 , 24) and the left inferior parietal lobule ($Z_{\max} 4.21$, 1093 voxels, -54 , -44 , 42). The exploratory whole-brain analysis (Table S1) implicated other regions beyond the parietal ROIs, including bilateral middle frontal

gyrus and the anterior cingulate. As in prior experiments (14, 38), the left inferior parietal lobule demonstrated the greatest response to HIT, an intermediate response to MISS or FALSE ALARM, and the least response to CORRECT REJECTION (Figure 2B). Patients activated the same network to a lesser extent, including the bilateral inferior parietal lobule (left: $Z_{\max}$ 3.49, 162 voxels, coordinates: $-36, -54, 46$; right: $Z_{\max}$ 3.71, 91 voxels, $54, -36, 22$) and left angular gyrus ($Z_{\max}$ 2.96, 73 voxels, $54, -36, 22$). Group comparisons within the *a priori* parietal ROI demonstrated that controls activated the posterior cingulate more than patients ($Z_{\max}$ 3.23, 184 voxels, $-6, -40, 26$; Figure 2C). Whole-brain analysis revealed a significant group difference in the right middle frontal gyrus ($Z_{\max}$ 3.52, 147 voxels, $46, 24, 32$).

Patients with more severe symptoms demonstrated diminished HIT>CORRECT REJECTION response: there were significant total BPRS correlations (Figure 2D) in the lateral parietal cortex ($Z_{\max}$ -3.8 , 305 voxels, $52, -66, 34$; peak $r = -0.84$) and the posterior cingulate ($Z_{\max}$ -3.03 , 74 voxels, $2, -22, 40$; peak $r = -0.69$). No correlations were found with paranoia. Patients with a higher dosage of medication demonstrated a diminished HIT>CORRECT REJECTION response in the right precuneus ($Z_{\max}$ -3.8 , 77 voxels, $22, -78, 44$). Total BPRS ~ HIT>CORRECT REJECTION correlations remained significant when medication dosage was considered in the same model.

THREAT>NON-THREAT

As we reported previously (22), controls showed significant THREAT>NON-THREAT responses in the left amygdala ($Z_{\max}$ 3.45, 54 voxels, $-30, -6, -28$; Figure 3A), right orbitofrontal cortex ($Z_{\max}$ 3.22, 137 voxels, $44, 56, -12$) as well as a left lateral temporal region (Table S2). In contrast, patients did not show significant THREAT>NON-THREAT differences in either the *a priori* ROIs or in the exploratory whole-brain analysis; there were no group differences between patients and controls. A follow-up analysis examining THREAT and NON-THREAT separately also did not find group differences. Furthermore, when the four schizoaffective (depressed type) patients were excluded, between-groups results remained unchanged. However, there was a significant correlation between THREAT>NON-THREAT response and total BPRS in the right amygdala ($Z_{\max}$ 3.57, 125 voxels, $30, 4, -14$; peak $r = 0.87$; Figure 3B), the right orbitofrontal cortex ($Z_{\max}$ 3.75, 242 voxels, $32, 22, 2$; peak $r = 0.79$), as well as a sub-threshold cluster present in the left amygdala ($Z_{\max}$ 3.33, 46 voxels, $-18, -12, -16$). Furthermore, paranoia was correlated with THREAT>NON-THREAT response in the right amygdala ($Z_{\max}$ 3.68; 113 voxels; $28, 12, -24$). Medication dosage attenuated the THREAT>NON-THREAT response in the left amygdala ($Z_{\max}$ 3.35; 115 voxels; $-28, -8, -14$) and in the left orbitofrontal cortex ($Z_{\max}$ 3.24; 62 voxels; $-44, 24, 2$). Total BPRS correlations with THREAT>NON-THREAT remained significant when paranoia and medication dose were each included in the same model.

Functional connectivity analysis

In controls, amygdala activity was positively correlated with other limbic regions and negatively correlated with cortical regions, including parietal areas activated in the HIT>CORRECT REJECTION analysis (Figure 4A). Patients showed a similar pattern, but

with stronger connectivity between the left amygdala and other ventral limbic regions, as well as a diminished negative correlation with cortical regions (Figure 4B). A direct between-group comparison (Figure 4C) in the *a priori* parietal ROI demonstrated that patients had increased connectivity (in the form of diminished negative correlation) between the amygdala and the right inferior parietal lobule ($Z_{\max}$ 3.22, 97 voxels, 50, -58, 40). In the whole brain analysis, patients demonstrated increased connectivity in the right middle frontal gyrus as well as limbic regions, including the right orbitofrontal cortex, right insula, and midbrain (Table S3). No symptom or medication-dose correlations were present.

DISCUSSION

This study explored the sparsely investigated intersection between emotion and cognition in schizophrenia. To probe this connection, we investigated the impact of a prior exposure to an intense facial affect on neural activation during a subsequent face recognition task. There were three main findings. First, patients with schizophrenia showed impaired recruitment of regions involved in recognition memory, and the degree to which activation was reduced correlated with overall symptom severity. Second, symptom severity correlated with heightened amygdala and orbitofrontal cortex responses to previously threatening faces. Third, patients showed a weakening of the normally negatively correlated relationship between the amygdala and frontoparietal regions involved in cognition, and also displayed increased amygdala connectivity with other limbic regions. The results have several implications and also certain limitations that are discussed below.

Patients with schizophrenia show impaired brain responses to face recognition

Behavioral deficits in facial recognition memory in schizophrenia have been demonstrated in the past (2). Here, we report on potential neural substrates of this performance deficit, as patients showed reduced recruitment of parietal and frontal regions involved in recognition memory. Further, the degree to which recruitment was impaired correlated with symptom severity. Previous literature has emphasized frontotemporal abnormalities during recognition memory in schizophrenia (10–12); this study provides evidence that parietal regions may also contribute to these deficits. We also observed similar effects in the middle frontal gyrus, a region implicated in the cognitive control of recognition memory (24). Notably, hippocampus is *not* reliably activated by the standard HIT>CORRECT REJECTION contrast in recognition memory studies, which might reflect robust task-induced hippocampal activation regardless of recognition performance (14). Previous studies in schizophrenia have shown impaired recruitment of the hippocampus across the task (12), which was also seen in the present study (data not shown).

Threat response in schizophrenia is associated with severity of symptoms

Previous studies have shown abnormalities in affective threat processing in schizophrenia (19–21), and suggested that abnormalities are symptom-related (25). However, all prior studies gathered data *during* the display of threatening affect; here we instead examined patient's response to neutral faces that were *previously* displayed with a threatening expression. This difference is important, as facial affective memory is likely to play a role in the development and maintenance of psychotic symptoms (39). We found that greater

response in the amygdala to prior threat is associated with both greater symptom burden and higher levels of paranoia, suggesting an emergence of symptoms with increased sensitivity to social signals of threat. Unlike studies examining direct displays of threat, we did not find group differences in amygdala responses, although this may be due to the lack of explicit attention to affect or to the heterogeneity of symptom-mediated threat response in patients.

The finding that increased symptoms both reduces activation during recognition memory in cortical regions and augments response to threat in limbic regions suggests a link between these two systems. However, group-level correlations cannot illuminate potential within-subject interactions between the amygdala and cortical regions involved in recognition memory. We investigated this relationship directly using a functional connectivity analysis.

Patients with schizophrenia demonstrate abnormal amygdalo-cortical connectivity

Substantial behavioral evidence indicates that threat-sensitivity may impair recognition memory (6). Furthermore, in healthy subjects, Dolcos et al. (8) found that dorsal cortical regions were activated by a working memory task while ventral limbic regions responded to threatening emotional distracters; greater limbic responses to threat were correlated with impaired memory performance. Here we found that activity in emotion-responsive limbic regions is negatively correlated with activity in dorsal cortical regions implicated in cognition. Patients showed a markedly different pattern, with increased amygdalo-limbic *and* increased amygdalo-cortical functional connectivity, indicating a disruption of the normally anti-correlated amygdalo-cortical relationship observed in controls. Notably, group differences in amygdala functional connectivity occur within lateral parietal cortex and middle prefrontal gyrus; these same regions demonstrated group differences and symptom correlations in the HIT>CORRECT REJECTION contrast. Although firm mechanistic interpretations are premature, these results suggest that increased sensitivity of the amygdala to potentially threatening stimuli may negatively impact the function of cortical regions involved in cognition.

Limitations and summary

Several limitations of this study should be acknowledged. First, our grouping of stimuli into THREAT and NON-THREAT, while suggested by earlier work (31), may obscure relevant differences between emotions. For example, while an angry face represents a direct threat indicated by gaze, a fearful face indicates a more ambiguous environmental threat (40). Second, the low accuracy in target recognition led our study to be underpowered for certain performance-based comparisons. Performance was limited by the general difficulty of face recognition tasks (2) and by the fact that subjects were not notified that they would be asked to later recognize the faces from the emotion identification task. We observed a conservative response bias, suggesting low target accuracy was not due to guessing. While poor performance likely prevented discernment of group differences in accuracy, group differences in response time were observed. In the future it will be important to further assess the effects of correct recognition on threat response with a design that produces more correct trials and incorporates behavioral metrics of recognition confidence. Third, in order to constrain multiple comparisons, we did not examine individual components of symptomatology beyond overall severity and paranoia. Future studies should investigate the

effects of paranoia on threat-responsiveness with more detailed assessments in a larger sample, as well as relationships with other specific symptoms. Finally, although our findings remained significant even when medication dose was added as a covariate to the models, we cannot exclude the influence of medication in this sample of patients. Interestingly, we found that increased doses of medication was correlated with diminished threat response in the amygdala and OFC, but also diminished recognition response in the parietal cortex. Although preliminary, this finding corroborates evidence that antipsychotics may reduce psychotic symptoms but also impair cognition at high doses.

Notwithstanding these limitations, this study demonstrates that patients with schizophrenia have symptom-related abnormalities in both cognitive and affective components of face recognition memory. The functional connectivity analysis indicates these deficits may be related: an abnormal response to the emotional elements of the task may impair performance of the task's cognitive demands. These results link patient's deficits in recognition memory to parietal abnormalities, extending previous results relating cognitive deficits to fronto-temporal abnormalities. Furthermore, the data also suggests that some degree of cognitive impairment may be related to abnormalities in threat processing. Thus, development of new treatments for cognitive impairment in schizophrenia should consider affective dysregulation as well.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1. Experimental paradigm

A. Encoding task. Subjects initially performed an emotion identification task in which they identified the facial affect displayed. Four emotional labels were available, including two non-threatening affects (happy and sad), two threatening affects (angry and fearful), and neutral. Subjects were not instructed to remember the faces displayed. Emotion identification and face recognition experiments were separated by a 10-minute diffusion tensor imaging acquisition. **B.** Face recognition task. Following the affect identification task, subjects were asked to make a forced-choice facial recognition judgment. Thirty faces

(targets) from the affect identification task and thirty novel faces (foils) were displayed for 2 seconds each. Subjects made a simple 'old' vs. 'new' judgment as to whether the face had been previously displayed in the affect identification task. Faces were separated by 60 variable (0–12s) intervals where a crosshair fixation point was displayed on a complex background (degraded face). Task duration was 4 minutes, 16 seconds.

Figure 2. HIT > CORRECT REJECTION contrast

A. Controls activate expected frontoparietal memory network, including lateral and medial parietal regions. B. Response of left inferior parietal lobule. As in other studies, the inferior parietal lobule responded robustly to HIT but not CORRECT REJECTION; MISS and FALSE ALARMS demonstrated an intermediate response. Error bars represent standard deviation. C. Controls demonstrate a greater response than patients in the posterior cingulate and the right middle frontal gyrus. D. Patients with more severe symptoms demonstrated a reduced HIT > CORRECT REJECTION response in both lateral and medial parietal regions.

Figure 3. THREAT > NON-THREAT contrast

A. Controls demonstrate a THREAT > NON-THREAT response in the left amygdala and right orbitofrontal cortex. B. In patients, severity of symptoms correlates with increased response to THREAT in right amygdala and right orbitofrontal cortex.

Figure 4. Functional connectivity analysis

A. Controls demonstrate a positive correlation with activity in a left amygdala seed in ventral limbic regions, and a negative correlation in cortical regions involved in cognition. B. The negative correlation between amygdala and left parietal clusters is related to performance in controls: subjects who performed less well demonstrated diminished anti-correlation. C. Patients show increased functional connectivity between the amygdala and other limbic regions as well as increased connectivity (reduced negative correlation) with cortical regions. D. Between-groups contrast. Compared to controls, patients show significantly

reduced anti-correlation between the left amygdala and the right middle frontal gyrus and right inferior parietal lobule, as well as increased connectivity between the amygdala and the right orbitofrontal cortex, right insula, and midbrain.

Table 1

Performance measures during face recognition task

Performance Measure	Patients (n=16)	Controls (n=21)
<i>Median % Correct (S.D.)</i>		
Non-threat	29 (17)	33 (19)
Threat	18 (17)	25 (14)
Foils	65 (15)	77 (17)
<i>Mean Response Time, ms (S.D.)</i>		
Non-threat	1128 (169)	1020 (211)
Threat	1169 (180)	1069 (149)
Foils	1095 (151)	1032 (133)