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Anhedonia and Emotional Experience in Schizophrenia: Neural and Behavioral Indicators

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Abstract

Background—Emotional impairments such as anhedonia are often considered key features of schizophrenia. However, self-report research suggests that emotional experience in response to affect-eliciting stimuli is intact in schizophrenia. Investigation of neural activity during emotional experience may help clarify whether symptoms of anhedonia more likely reflect alterations of in-the-moment hedonic experience or impairments in other aspects of goal-directed behavior.

Methods—40 individuals with DSM-IV-TR schizophrenia or schizoaffective disorder and 32 healthy controls underwent fMRI while making valence and arousal ratings in response to emotional pictures, words, and faces. BOLD responses were compared between patients and controls, and were correlated with questionnaire measures of anhedonia.

Results—Patients showed some evidence of blunted valence, but not arousal, ratings in response to emotional stimuli as compared to controls. Higher anhedonia scores were associated with blunted valence ratings in both groups, and fully mediated the group differences in valence ratings. Functional activity was largely intact in patients, except for regions in right ventral striatum and left putamen, which showed reduced responses to positive stimuli. Higher anhedonia was associated with reduced activation to positive versus negative stimuli in bilateral amygdala and right ventral striatum in patients, and in bilateral caudate in controls.

Conclusions—Increased anhedonia is associated with a reduced experience of valence in both patients and controls, and group differences in experienced valence are likely driven by individual differences in anhedonia. Reduced activation of the striatum and amygdala may contribute to symptoms of anhedonia by failing to signal the salience of positive events.

Introduction

Anhedonia, or the inability to experience pleasure, is a long-established feature of schizophrenia (1, 2) that significantly impacts functional capacity and is resistant to treatment (3, 4). Surprisingly, however, a growing body of self-report (5, 6) and behavioral (7) data suggests that emotional experience in schizophrenia is intact. One possible

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explanation for this discrepancy is that in schizophrenia, clinical measures of anhedonia reflect not a deficit in consummatory pleasure, but a deficit in anticipatory pleasure or approach motivation (8–10). To investigate this possibility, we asked whether neural activity during emotional experience is also intact in schizophrenia.

In studying brain responses to affect-eliciting stimuli, several structures are of particular interest. First, the *striatum* has been associated with responses to “rewarding” or pleasurable stimuli (11–13). Further, reduced ventral striatal activity in response to positive stimuli has been associated with anhedonia in studies with both healthy (14) and depressed (15, 16) individuals. Most commonly, the mesolimbic dopamine system and its projections to the striatum are associated with reward prediction and incentive salience (11, 17, 18), suggesting that this region is instrumental to anticipatory pleasure and approach motivation. Second, *dorsomedial prefrontal cortex* (dmPFC) and *orbitofrontal cortex* (OFC) are active during emotional experience across a wide range of emotion elicitation studies (19). DmPFC may be involved in the introspective evaluation of one’s feelings, while OFC may be involved in establishing the threat or reward value of a stimulus (20). Third, the *amygdala* is implicated in processing survival-salient, arousing stimuli, both negative and positive (21). Finally, activity in the *rostral anterior cingulate cortex* (rACC) has been associated with subjective ratings of pleasantness (22, 23).

A number of studies have suggested that striatal activity during processing of positive stimuli may be altered in schizophrenia. For example, unmedicated patients have shown reduced ventral striatal activation during reward anticipation, which correlated with negative symptom severity (24). In emotion perception studies, patients failed to modulate nucleus accumbens activation when rating pleasant versus unpleasant odors (25), and demonstrated reduced phasic (but enhanced tonic) activity in the ventral striatum to both positive and aversive stimuli (26).

Studies of activity in dmPFC and OFC during emotional experience in schizophrenia have yielded mixed results. Given evidence of a dissociation between neural activity patterns during emotional experience versus emotion perception (27), we focus here on studies in which participants reported their own experienced emotion. Some of these studies found reduced activation of dmPFC and OFC during sadness in chronic and first-episode schizophrenia (28, 29). However, other studies failed to find group differences in these regions in patients (30, 31) and relatives (32). Functional neuroimaging studies of amygdala activation in schizophrenia have also given mixed results (33). Some emotional experience studies have shown reductions in amygdala activity in patients as compared to controls (28, 31), while others found no group differences (29, 30). Similarly, most emotion perception studies have found reduced amygdala activity in response to emotional stimuli in patients relative to controls (28, 34–38), and in paranoid vs. non-paranoid patients (39). However, some studies have shown increased (40, 41) or normal (42, 43) amygdala activation. The disparity in these results may reflect small sample sizes, differences in stimuli, clinical variation across samples, and the need for a low-level control condition given that neutral stimuli may elicit greater limbic activation in patients (44) and their relatives (45) than in controls. In the current study, we aimed to address these concerns by using a large sample and several types of stimuli, by conducting individual difference analyses, and by examining the pattern of responses across several emotional conditions rather than comparing emotional conditions to a neutral baseline.

This study aimed to address three questions. First, we asked whether self-reports of emotional experience are intact in patients. In keeping with previous data, we hypothesized that self-reports would be similar between patients and controls. Second, we asked whether neural activity during emotional experience was similar in patients and controls. If fMRI is

sensitive to differences in emotional experience not probed by self-report measures, we would expect to see differences in functional activity in regions associated with emotional experience. Third, we asked whether there was a relationship between questionnaire measures of anhedonia and individual differences in self-reported emotion or its associated neural activity.

Methods

Participants

Participants were 40 outpatients with DSM-IV-TR schizophrenia or schizoaffective disorder and 32 healthy community controls. All results reported below remained the same when schizoaffective patients were excluded (see Supplemental Materials). Controls were excluded if they had any history of, or first-order family member with, an Axis I psychotic disorder, or any current mood or anxiety disorder other than Specific Phobia. Other exclusions included: 1) DSM-IV substance abuse or dependence within six months; 2) any medical disorder that is unstable or severe, would confound the assessment of psychiatric diagnosis, or would make participation unsafe; 3) present or past head injury with neurological sequelae or causing loss of consciousness; and 4) DSM-IV mental retardation (mild or greater). The demographic and clinical characteristics of both participant groups are shown in Table 1. Groups were matched on age, parental education, gender, race, and handedness. All patients were taking antipsychotic medications, which were stable for least two weeks.

Participant diagnoses were based on a Structured Clinical Interview for DSM-IV-TR (46) and on information from medical records and corroborative sources. Clinical symptoms were rated using the Scales for the Assessment of Positive Symptoms (SAPS) (47) and Negative Symptoms (SANS) (48). We assessed anhedonia symptoms using the SANS global anhedonia score and the self-report Chapman physical and social anhedonia scales (49, 50), and handedness using the Edinburgh Index (51). See Supplemental Materials for details.

Materials and Tasks

All participants were scanned while making valence and arousal ratings of their own subjective responses to emotional pictures, words, and faces. Valence (pleasant-unpleasant) and arousal (activation-deactivation) are independent dimensions of affect (52) that are considered vital features of emotional experience (20). Participants rated their experience of each stimulus by button press as positive, negative, or neutral during valence runs, or as highly, slightly, or not aroused during arousal runs. Stimuli consisted of 50 each emotional words, pictures, and faces, 10 in each of the following categories: negative high arousal (NHA), negative low arousal (NLA), positive high arousal (PHA), positive low arousal (PLA), and neutral (NEU); see Supplemental Materials. Participants performed the task for 6 runs; one run each of arousal and valence judgments for each stimulus type (pictures, words, faces). Stimuli were presented for 2000 msec with a jittered inter-stimulus interval varying from 1000 to 10000 msec.

Behavioral data analysis

Valence and Arousal Ratings—We analyzed the valence and arousal ratings using repeated measures ANOVAs with group (schizophrenia, control) as a between-subjects factor and stimulus (picture, word, face) and condition (NHA, NLA, NEU, PLA, PHA) as within-subjects factors. To further characterize the pattern of responses as a function of condition, we created a priori contrasts that were sensitive to the valence and/or arousal characteristics of the stimuli. To examine the effect of valence irrespective of arousal, we

used a linear contrast with weights of $-1, -1, 0, 1$, and 1 for NHA, NLA, NEU, PLA, and PHA, respectively (valence contrast). To examine the effect of arousal irrespective of valence, we used a quadratic contrast with weights of $2, -1, -2, -1$, and 2 for NHA, NLA, NEU, PLA, and PHA, respectively (arousal contrast). To examine whether the valence ratings were influenced by both the valence and arousal characteristics of the stimuli, we also created a linear contrast in which valence was amplified by arousal, using weights of $-2, -1, 0, 1$, and 2 , for NHA, NLA, NEU, PLA, and PHA, respectively (valenceXarousal contrast). We tested the significance of these contrasts using univariate F-tests within each group.

Individual Difference Analyses—We conducted linear regression analyses to examine the extent to which anhedonia scores predicted valence ratings within each group. To determine whether the relationship between valence ratings in response to PHA/NHA stimuli and Chapman Physical/Social Anhedonia scores differed between groups, we conducted hierarchical regression analyses with anhedonia score (physical or social) and group entered in step 1, and groupXanhedonia interaction entered in step 2. We also examined whether anhedonia scores mediated the effect of group on valence ratings. To do this, we conducted two separate multiple mediation analyses using a Sobel procedure with bootstrapping (53), with PHA or NHA valence ratings as the dependent variable, group as the independent variable, and physical and social anhedonia scores as mediators. Within the patient group, we also conducted correlations between PHA/NHA valence ratings and SANS global anhedonia, and with SANS avolition, alogia, and affective flattening to examine the specificity to anhedonia.

fMRI analysis

For fMRI acquisition and image analysis, see Supplemental Materials. Functional activation was analyzed using the valence, arousal, and valenceXarousal contrasts in both region-of-interest (ROI) and whole-brain analyses. We examined voxelwise t-tests at the group level within predefined ROI masks including the amygdala, striatum, dmPFC, OFC, and rACC (see Supplemental Materials). Both the whole-brain and ROI analyses were corrected for multiple comparisons using combined p-value/cluster size thresholds, determined using Monte Carlo simulations to provide an overall false-positive rate of 0.05 (54, 55). These thresholds were $p < .01$ and 14 voxels for ROI analyses, and $p < .003$ and 30 voxels for whole-brain analyses. To identify regions whose activation patterns were consistent with the valence and arousal patterns of interest, we first conducted one-sample t-tests for each contrast on both groups combined. To identify regions showing group differences in activation, we also performed group t-tests on each contrast. In both analyses, significant regions were followed up with simple effects tests to determine the activation pattern within each group separately. To examine individual differences in functional activity, we also conducted voxelwise correlation analyses between contrast scores and anhedonia scores. Correlations were conducted within each group separately, and correlation coefficients were compared between groups using Fisher r-to-z transformations.

Results

Behavioral Results

Anhedonia Scores—Overall, individuals with schizophrenia had higher anhedonia scores than controls on both Chapman scales (Table 1).

Valence and Arousal Ratings—For valence (Figure 1a), there was a significant main effect of condition ($F(4, 280) = 659.85, p < .001$) and significant groupXcondition ($F(4, 280) = 13.49, p < .001$) and stimulusXcondition ($F(8, 560) = 12.70, p < .001$) interactions. Simple

effects tests revealed significant effects of condition for controls ($F(4, 280) = 386.85, p < .001$) and patients ($F(4, 280) = 273.95, p < .001$). However, comparisons within each condition revealed that patients' responses to negative stimuli were less negative ($F(1, 70) = 9.62, p < .004$ for NHA; $F(1, 70) = 7.87, p < .007$ for NLA), and to positive stimuli were less positive ($F(1, 70) = 15.77, p < .001$ for PLA, $F(1, 70) = 9.75, p < .004$ for PHA), than controls. Both the valence and valenceXarousal contrasts were significant for both groups, with similar effect sizes (valence contrast: $F(1, 70) = 521.50, p < .001, \eta_p^2 = 0.882$ for controls, $F(1, 70) = 368.23, p < .001, \eta_p^2 = 0.840$ for patients; valenceXarousal contrast: $F(1, 70) = 509.33, p < .001, \eta_p^2 = 0.879$ for controls; $F(1, 70) = 364.80, p < .001, \eta_p^2 = 0.839$ for patients). Because stimulus type (picture, word, face) did not interact with group in any of our analyses (behavioral or fMRI), stimulus effects are not discussed further here (but see Supplemental Materials).

For the arousal ratings (Figure 1b), there were significant main effects of stimulus ($F(2, 140) = 23.41, p < .001$) and condition ($F(4, 280) = 55.96, p < .001$), and significant stimulusXcondition ($F(8, 560) = 5.03, p < .001$) and groupXcondition ($F(4, 280) = 3.15, p < .009$) interactions. Simple effects tests revealed significant effects of condition within each group ($F(4, 280) = 39.35, p < .001$ for controls, $F(4, 280) = 17.72, p < .001$ for patients). Further, group comparisons within each condition revealed a significant group difference only for the NEU condition: compared to controls, patients showed higher arousal in response to neutral stimuli ($F(1, 70) = 6.87, p < .02$). The arousal contrast was significant for both groups ($F(1, 70) = 151.45, p < .001, \eta_p^2 = 0.654$ for controls; $F(1, 70) = 68.76, p < .001, \eta_p^2 = 0.496$ for patients). Taken together, these results indicate that patients showed blunted valence ratings in response to emotional stimuli. However, the patterns of both valence and arousal ratings as a function of emotional condition were similar between groups.

Individual Difference Analyses—We conducted hierarchical regression analyses using Chapman anhedonia scores to predict valence ratings to PHA and NHA stimuli in patients and controls (Table 2). In all of these analyses, anhedonia score and group accounted for a significant portion of the variance in the valence ratings, and adding a groupXanhedonia interaction term failed to account for significantly more variance. As expected, higher physical and social anhedonia scores were associated with less-positive responses to PHA stimuli and less-negative responses to NHA stimuli in both groups (Figure 2). Similarly, within the patient group, SANS anhedonia correlated negatively with PHA valence ratings ($r = -.37, p < .03$), though it failed to correlate with NHA valence ratings ($p > .16$). Together, these results suggest that within both patients and controls, higher levels of anhedonia are associated with less-valenced experiences of emotional stimuli.

Multiple mediation analyses revealed that for both PHA and NHA valence ratings, the effect of group was fully mediated by physical and social anhedonia scores (effect of group on valence rating, controlling for physical and social anhedonia: $t(69) = -1.2, p > .24$ for PHA, $t(69) = 1.0, p > .31$ for NHA). The total mediated effect was significant in both models (95% CI = $-0.27, -0.01$ for PHA; $0.02, 0.25$ for NHA). For PHA ratings, only physical anhedonia was significant as a specific mediator (95% CI = $-0.16, -0.003$), and for NHA ratings, neither specific mediator was significant alone.

To evaluate the specificity of these results to anhedonia, we correlated PHA and NHA valence ratings with SANS global avolition, alogia, and affective flattening in patients, and found that avolition also correlated negatively with PHA valence ratings ($r = -.34, p < .04$) and positively with NHA valence ratings ($r = .36, p < .03$). Aside from a trend-level correlation between alogia and NHA ratings ($r = .30, p < .07$), alogia and affective flattening failed to correlate with either measure ($p > .16$). Therefore, a reduced experience of positive

and negative emotion appears to be related to symptoms of anhedonia and amotivation, but not to other negative emotional symptoms.

fMRI Results

ROI Analyses: One-sample t-tests—As shown in Figure 3, one-sample t-tests identified several regions within the ROIs with activity patterns significant for the valence, arousal, and valenceXarousal contrasts. These regions and their activation patterns are detailed in Table 3.

Group t-tests—One region in right ventral striatum demonstrated a significant group difference in the valence contrast, and one in left putamen showed a group difference in the valenceXarousal contrast (Table 4 and Figure 4). As shown in Figure 4, in both of these regions, patients showed reduced activation as compared to controls for the positive conditions. Post-hoc tests revealed that in left putamen, PHA activation differed significantly between groups ($F(1,70)=5.05, p<.04$). In right ventral striatum, there were significant group differences in both PHA ($F(1,70)=5.58, p<.03$) and PLA ($F(1,70)=6.14, p<.03$).

Whole Brain Analyses—As shown in Table 5 and Figure 5, a number of regions were identified by the valence, arousal, and valenceXarousal contrasts in whole-brain one-sample t-tests. In the group t-tests, however, we did not find a single region that showed a significant group difference in any of the contrasts. To further examine whether the activity patterns were similar between groups, we conducted follow-up group analyses on each region identified in the one-sample t-tests. As shown in Table 5, overall activity differed between patients and controls in a number of these regions. However, in every region: (1) both patients and controls showed significant within-group effects of the relevant contrast, (2) there were no significant group differences in the magnitude of the contrast; and in all but two regions, (3) the pattern as a function of emotional condition was the same for both patients and controls. Thus, outside of the striatum, patients and controls demonstrated similar neural responses to both valence and arousal.

Individual difference analyses—We first conducted correlations between anhedonia scores and average activation contrast scores within the regions showing group differences in the contrasts. This analysis revealed a negative correlation between physical anhedonia and valenceXarousal contrast score in the right ventral striatum in patients ($r = -.36, p<.04$), indicating that patients with higher anhedonia scores showed less activation in this region in response to positive stimuli as compared to neutral and negative stimuli. This correlation was not significant in controls ($r = -.17, p>.34$), though the group difference in correlation coefficients was not significant ($p>.77$). We next conducted voxelwise ROI analyses (Table 6), in which physical anhedonia correlated negatively with the valence contrast in left amygdala, and with the valenceXarousal contrast in right amygdala, in patients. In controls, social anhedonia correlated negatively with the valence contrast in bilateral caudate. Comparison of correlation coefficients between groups revealed a significant difference in the right caudate ($p<.02$) and a trend level difference in left caudate ($p<.08$), but no difference in either amygdala region ($p>.70$).

Discussion

Behavioral Measures of Emotional Experience

In agreement with most clinical data, we found that individuals with schizophrenia self-reported more anhedonia than controls. Behaviorally, there was a groupXcondition interaction in the valence ratings, and post-hoc tests revealed that patients rated their

experience of the valenced stimuli as less valenced than controls. This finding is at odds with the majority of studies, which have shown intact responses to emotional stimuli. However, while the arousal ratings also showed a groupXcondition interaction, the only post-hoc group difference was heightened arousal ratings in response to neutral stimuli in patients. This finding suggests that patients' experience of arousal in response to emotional stimuli is intact, in agreement with previous literature. Furthermore, patients clearly showed modulation of both valence and arousal ratings as a function of the emotional content of the stimuli: when we conducted contrast analyses sensitive to valence, arousal, and valenceXarousal interaction, the relevant contrasts were significant within both groups, with similar effect sizes. Overall, while these findings suggest that the range of experienced emotion may be narrowed in patients, they also show that evoked arousal is relatively intact, and that affective stimuli modulate emotional experience in similar ways in patients and controls.

Individual difference analyses revealed that higher anhedonia was associated with blunted responses to emotional stimuli within both patients and controls. Furthermore, the group differences in valence ratings were fully mediated by anhedonia scores. Together, these results indicate that the level of anhedonia, rather than simply the diagnosis of schizophrenia, may underlie the blunted responses to emotional stimuli seen in patients. This finding highlights the importance of including sufficiently powered individual difference analyses in future work.

fMRI Measures of Emotion Processing

fMRI analysis revealed that brain activity is largely intact during emotional experience in schizophrenia. On whole-brain analysis, we did not find any regions that showed group differences in any contrast, suggesting similar patterns of neural activity in patients and controls. On ROI analysis, however, right ventral striatum and left putamen showed reduced activation to positive stimuli in patients as compared to controls. Given past research showing that striatal activation is associated with the anticipation (56) and receipt (23) of pleasurable stimuli, this finding may represent a failure to respond to positive experiences that contributes to an inability to anticipate or want such experiences in the future (57).

In support of this interpretation, reduced activation to positive versus negative stimuli in the same ventral striatal region was also associated with higher physical anhedonia in patients. This finding suggests that the group differences in activation seen in this region may be driven by individual differences in anhedonia. Similarly, bilateral amygdala activation to positive versus negative stimuli was reduced in patients who were higher in physical anhedonia. Within controls, greater social anhedonia was associated with decreased bilateral caudate activation in response to positive relative to negative stimuli. Because the amygdala (21) and striatum (58) are thought to be involved in salience attribution, these results may indicate that these regions fail to mark positive events as salient in anhedonic individuals, leading to a blunted experience of emotion and a reduced ability to seek out similar events in the future.

Given that the ventral striatum is typically associated with reward processing, it is interesting to speculate on how these results relate to findings of reduced ventral striatal activation during reward anticipation in schizophrenia (24). Notably, the reduced ventral striatal activation to positive stimuli seen here in patients may represent a deficit in motivational or reward-prediction processes, rather than in hedonic processes per se. During learning, dopaminergic neurons initially fire to unexpected positive stimuli, shifting over time to fire to cues that predict these rewards (18). Thus, the deficient right ventral striatal activation reported here could reflect a failure of this initial dopaminergic firing to unpredicted positive stimuli, potentially impairing reward prediction/incentive salience and

leading to reduced anticipatory activation. Importantly, this impairment in predictive or motivational processes may be independent of the hedonic response to the reward, allowing a normal experience of “liking” combined with reduced “wanting”. This is consistent with the view that consummatory pleasure is intact in schizophrenia while anticipatory pleasure is impaired (10).

Given the finding of group differences in striatal activity, a major limitation of this study is that all patients were taking medications that block dopamine receptors, potentially altering striatal function. However, the majority of patients were taking atypical antipsychotics, which have a lesser effect on striatal activity during reward processing than typical antipsychotics (59, 60). Further, when we removed from analysis all patients taking typical antipsychotics or risperidone (which are pharmacologically similar), the group differences and correlations remained significant. In addition, neural activity did not correlate with antipsychotic dose within the regions showing group differences (see Supplemental Materials). While the possibility of medication effects cannot be ruled out without examination of unmedicated patients, we feel that these results provide reasonable evidence that the findings reported here were not driven by medications.

In summary, this study makes several important contributions to the literature on emotional experience and its related brain activity in schizophrenia. First, while patients showed blunted responses to emotional stimuli as compared to controls, these group differences in ratings were clearly mediated by the level of anhedonia displayed by the participants. Second, the pattern of brain activity in response to emotional stimuli was largely intact, with the exception of two striatal regions that showed reduced responses to positive stimuli. Third, blunted activation to positive vs. negative stimuli correlated with anhedonia in the amygdala and right ventral striatum in patients, and in the caudate in controls, suggesting that failure to mark stimuli as salient or rewarding may contribute to symptoms of anhedonia. Clinically, these results highlight the importance of individual differences, suggesting that optimal treatment strategies are best tailored to the individual symptomatology of the patient. Future work examining the relationship between reduced neural responses to positive stimuli and deficits in motivated behavior, using paradigms that probe for reward anticipation and reinforcement learning in anhedonic individuals, may shed additional light on the questions raised here.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. Bleuler, E. *Dementia Praecox, or the Group of Schizophrenias*. New York: International Universities Press; 1911.
2. Kraepelin, E. *Dementia Praecox and Paraphrenia*. Edinburgh: E. & S. Livingstone; 1971.
3. Herbener ES, Harrow M, Hill SK. Change in the relationship between anhedonia and functional deficits over a 20-year period in individuals with schizophrenia. *Schizophr Res*. 2005; 75:97–105. [PubMed: 15820328]
4. Shoichet RP, Oakley A. Notes on the treatment of anhedonia. *Can Psychiatr Assoc J*. 1978; 23:487–492. [PubMed: 709497]

5. Kring AM, Moran EK. Emotional response deficits in schizophrenia: Insights from affective science. *Schizophr Bull.* 2008; 34:819. [PubMed: 18579556]
6. Cohen AS, Minor KS. Emotional experience in patients with schizophrenia revisited: Meta-analysis of laboratory studies. *Schizophr Bull.* 2008
7. Heerey EA, Gold JM. Patients with schizophrenia demonstrate dissociation between affective experience and motivated behavior. *J Abnorm Psychol.* 2007; 116:268–278. [PubMed: 17516760]
8. Burbridge JA, Barch DM. Anhedonia and the experience of emotion in individuals with schizophrenia. *J Abnorm Psychol.* 2007; 116:30–42. [PubMed: 17324014]
9. Horan WP, Green MF, Kring AM, Nuechterlein KH. Does anhedonia in schizophrenia reflect faulty memory for subjectively experienced emotions? *J Abnorm Psychol.* 2006; 115:496–508. [PubMed: 16866590]
10. Gard DE, Kring AM, Gard MG, Horan WP, Green MF. Anhedonia in schizophrenia: distinctions between anticipatory and consummatory pleasure. *Schizophr Res.* 2007; 93:253–260. [PubMed: 17490858]
11. Schultz W. Multiple dopamine functions at different time courses. *Annu Rev Neurosci.* 2007; 30:259–288. [PubMed: 17600522]
12. O'Doherty JP. Reward representations and reward-related learning in the human brain: insights from neuroimaging. *Curr Opin Neurobiol.* 2004; 14:769–776. [PubMed: 15582382]
13. Smith KS, Berridge KC. Opioid limbic circuit for reward: interaction between hedonic hotspots of nucleus accumbens and ventral pallidum. *J Neurosci.* 2007; 27:1594–1605. [PubMed: 17301168]
14. Wacker J, Dillon DG, Pizzagalli DA. The role of the nucleus accumbens and rostral anterior cingulate cortex in anhedonia: integration of resting EEG, fMRI, and volumetric techniques. *Neuroimage.* 2009; 46:327–337. [PubMed: 19457367]
15. Keedwell P, Andrew C, Williams S, Brammer M, Phillips M. The neural correlates of anhedonia in Major Depressive Disorder. *Biol Psychiatry.* 2005; 58:843–853. [PubMed: 16043128]
16. Epstein J, Pan H, Kocsis JH, Yang Y, Butler T, Chusid J, et al. Lack of ventral striatal response to positive stimuli in depressed versus normal subjects. *Am J Psychiatry.* 2006; 163:1784–1790. [PubMed: 17012690]
17. Berridge KC. Motivation concepts in behavioral neuroscience. *Physiol Behav.* 2004; 81:179–209. [PubMed: 15159167]
18. Schultz W, Dayan P, Montague PR. A neural substrate of prediction and reward. *Science.* 1997; 275:1593–1599. [PubMed: 9054347]
19. Kober H, Barrett LF, Joseph J, Bliss-Moreau E, Lindquist K, Wager TD. Functional grouping and cortical-subcortical interactions in emotion: a meta-analysis of neuroimaging studies. *Neuroimage.* 2008; 42:998–1031. [PubMed: 18579414]
20. Barrett LF, Mesquita B, Ochsner KN, Gross JJ. The experience of emotion. *Annu Rev Psychol.* 2007; 58:373–403. [PubMed: 17002554]
21. Zald DH. The human amygdala and the emotional evaluation of sensory stimuli. *Brain Research Reviews.* 2003; 41:88–123. [PubMed: 12505650]
22. Rolls ET, Kringelbach ML, de Araujo IE. Different representations of pleasant and unpleasant odours in the human brain. *Eur J Neurosci.* 2003; 18:695–703. [PubMed: 12911766]
23. Rolls ET, Grabenhorst F, Parris BA. Warm pleasant feelings in the brain. *Neuroimage.* 2008; 41:1504–1513. [PubMed: 18468458]
24. Juckel G, Schlagenhauf F, Koslowski M, Wüstenberg T, Villringer A, Knutson B, et al. Dysfunction of ventral striatal reward prediction in schizophrenia. *Neuroimage.* 2006; 29:409–416. [PubMed: 16139525]
25. Crespo-Facorro B, Paradiso S, Andreasen NC, O'Leary DS, Watkins GL, Ponto LL, et al. Neural mechanisms of anhedonia in schizophrenia: a PET study of response to unpleasant and pleasant odors. *JAMA.* 2001; 286:427–435. [PubMed: 11466121]
26. Taylor SF, Phan KL, Britton JC, Liberzon I. Neural response to emotional salience in schizophrenia. *Neuropsychopharmacology.* 2005; 30:984–995. [PubMed: 15689961]

27. Wager, TD.; Barrett, LF.; Bliss-Moreau, E.; Lindquist, K.; Duncan, S.; Kober, H.; Joseph, J.; Davidson, M.; Mize, J. The Neuroimaging of Emotion. In: Lewis, M.; Haviland-Jones, JM.; Barrett, LF., editors. *Handbook of Emotion*. 3rd Edition. New York: Guilford; 2008. p. 249-271.
28. Takahashi H, Koeda M, Oda K, Matsuda T, Matsushima E, Matsuura M, et al. An fMRI study of differential neural response to affective pictures in schizophrenia. *Neuroimage*. 2004; 22:1247–1254. [PubMed: 15219596]
29. Reske M, Kellermann T, Habel U, Jon Shah N, Backes V, von Wilmsdorff M, et al. Stability of emotional dysfunctions? A long-term fMRI study in first-episode schizophrenia. *J Psychiatr Res*. 2007; 41:918–927. [PubMed: 17467008]
30. Schneider F, Habel U, Reske M, Toni I, Falkai P, Shah NJ. Neural substrates of olfactory processing in schizophrenia patients and their healthy relatives. *Psychiatry Res*. 2007; 155:103–112. [PubMed: 17532193]
31. Schneider F, Weiss U, Kessler C, Salloum JB, Posse S, Grodd W, et al. Differential amygdala activation in schizophrenia during sadness. *Schizophr Res*. 1998; 34:133–142. [PubMed: 9850979]
32. Habel U, Klein M, Shah NJ, Toni I, Zilles K, Falkai P, et al. Genetic load on amygdala hypofunction during sadness in nonaffected brothers of schizophrenia patients. *Am J Psychiatry*. 2004; 161:1806–1813. [PubMed: 15465977]
33. Aleman A, Kahn RS. Strange feelings: do amygdala abnormalities dysregulate the emotional brain in schizophrenia? *Prog Neurobiol*. 2005; 77:283–298. [PubMed: 16352388]
34. Gur RE, McGrath C, Chan RM, Schroeder L, Turner T, Turetsky BI, et al. An fMRI study of facial emotion processing in patients with schizophrenia. *Am J Psychiatry*. 2002; 159:1992–1999. [PubMed: 12450947]
35. Hempel A, Hempel E, Schonknecht P, Stippich C, Schroder J. Impairment in basal limbic function in schizophrenia during affect recognition. *Psychiatry Res*. 2003; 122:115–124. [PubMed: 12714175]
36. Johnston PJ, Stojanov W, Devir H, Schall U. Functional MRI of facial emotion recognition deficits in schizophrenia and their electrophysiological correlates. *Eur J Neurosci*. 2005; 22:1221–1232. [PubMed: 16176365]
37. Taylor SF, Liberzon I, Decker LR, Koeppe RA. A functional anatomic study of emotion in schizophrenia. *Schizophr Res*. 2002; 58:159–172. [PubMed: 12409155]
38. Paradiso S, Andreasen NC, Crespo-Facorro B, O'Leary DS, Watkins GL, Boles Ponto LL, et al. Emotions in unmedicated patients with schizophrenia during evaluation with positron emission tomography. *Am J Psychiatry*. 2003; 160:1775–1783. [PubMed: 14514490]
39. Williams LM, Das P, Harris AW, Liddell BB, Brammer MJ, Olivieri G, et al. Dysregulation of arousal and amygdala-prefrontal systems in paranoid schizophrenia. *Am J Psychiatry*. 2004; 161:480–489. [PubMed: 14992974]
40. Kosaka H, Omori M, Murata T, Iidaka T, Yamada H, Okada T, et al. Differential amygdala response during facial recognition in patients with schizophrenia: an fMRI study. *Schizophr Res*. 2002; 57:87–95. [PubMed: 12165379]
41. Holt DJ, Kunkel L, Weiss AP, Goff DC, Wright CI, Shin LM, et al. Increased medial temporal lobe activation during the passive viewing of emotional and neutral facial expressions in schizophrenia. *Schizophr Res*. 2006; 82:153–162. [PubMed: 16377154]
42. Plailly J, d'Amato T, Saoud M, Royet JP. Left temporo-limbic and orbital dysfunction in schizophrenia during odor familiarity and hedonicity judgments. *Neuroimage*. 2006; 29:302–313. [PubMed: 16099179]
43. Surguladze S, Russell T, Kucharska-Pietura K, Travis MJ, Giampietro VP, David AS, et al. A reversal of the normal pattern of parahippocampal response to neutral and fearful faces is associated with reality distortion in schizophrenia. *Biol Psychiatry*. 2006; 60:423–431. [PubMed: 16487943]
44. Hall J, Whalley HC, McKirdy JW, Romaniuk L, McGonigle D, McIntosh AM, et al. Overactivation of fear systems to neutral faces in schizophrenia. *Biol Psychiatry*. 2008; 64:70–73. [PubMed: 18295746]

45. Seifert NY, Pauly K, Habel U, Kellermann T, Jon Shah N, Ruhrmann S, et al. Increased neural response related to neutral faces in individuals at risk for psychosis. *Neuroimage*. 2008; 40:289–297. [PubMed: 18187342]
46. First, M.; Spitzer, R.; Gibbon, M.; Williams, J. Structured clinical interview for DSM-IV-TR axis I disorders-patient edition. Biometrics Research Department, New York State Psychiatric Institute; 2001.
47. Andreasen, NC. The scale for the assessment of positive symptoms (SAPS). Iowa City: University of Iowa; 1983.
48. Andreasen, NC. The scale for the assessment of negative symptoms (SANS). Iowa City: University of Iowa; 1983.
49. Chapman LJ, Chapman JP, Raulin ML. Scales for physical and social anhedonia. *J Abnorm Psychol*. 1976; 85:374–382. [PubMed: 956504]
50. Eckblad M, Chapman LJ, Chapman JP, Mishlove M. The revised social anhedonia scale. 1982
51. Oldfield RC. The assessment and analysis of handedness: the Edinburgh inventory. *Neuropsychologia*. 1971; 9:97–113. [PubMed: 5146491]
52. Russell JA. A circumplex model of affect. *J Pers Soc Psychol*. 1980; 39:1161–1178.
53. Preacher KJ, Hayes AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behav Res Methods*. 2008; 40:879–891. [PubMed: 18697684]
54. McAvoy MP, Ollinger JM, Buckner RL. Cluster size thresholds for assessment of significant activation in fMRI. *Neuroimage*. 2001; 13:S198.
55. Forman SD, Cohen JD, Fitzgerald M, Eddy WF, Mintun MA, Noll DC. Improved assessment of significant activation in functional magnetic resonance imaging (fMRI): Use of a cluster-size threshold. *Magn Reson Med*. 1995; 33:636–647. [PubMed: 7596267]
56. O'Doherty JP, Deichmann R, Critchley HD, Dolan RJ. Neural responses during anticipation of a primary taste reward. *Neuron*. 2002; 33:815–826. [PubMed: 11879657]
57. Berridge KC, Kringelbach ML. Affective neuroscience of pleasure: reward in humans and animals. *Psychopharmacology (Berl)*. 2008; 199:457–480. [PubMed: 18311558]
58. Zink CF, Pagnoni G, Martin-Skurski ME, Chappelow JC, Berns GS. Human striatal responses to monetary reward depend on saliency. *Neuron*. 2004; 42:509–517. [PubMed: 15134646]
59. Juckel G, Schlagenhauf F, Koslowski M, Filonov D, Wüstenberg T, Villringer A, et al. Dysfunction of ventral striatal reward prediction in schizophrenic patients treated with typical, not atypical, neuroleptics. *Psychopharmacology (Berl)*. 2006; 187:222–228. [PubMed: 16721614]
60. Kirsch P, Ronshausen S, Mier D, Gallhofer B. The influence of antipsychotic treatment on brain reward system reactivity in schizophrenia patients. *Pharmacopsychiatry*. 2007; 40:196–198. [PubMed: 17874350]

Figure 1.

Valence and arousal ratings as a function of condition in individuals with schizophrenia (SCZ) and controls (CON).

(a) Average valence ratings (1=negative, 2=neutral, 3=positive) collapsed across stimulus type (pictures, words, faces) for each emotional condition: negative high arousal (NHA), negative low arousal (NLA), neutral (NEU), positive low arousal (PLA), and positive high arousal (PHA).

(b) Average arousal ratings (1=highly aroused, 2=slightly aroused, 3=not aroused) collapsed across stimulus type (pictures, words, faces) for each emotional condition.

* $p < .05$. Error bars represent standard error.

Figure 2.

Scatterplots of (a) average valence ratings to positive high arousal (PHA) stimuli as a function of Chapman physical anhedonia, (b) average valence ratings to positive high arousal (PHA) stimuli as a function of Chapman social anhedonia, (c) average valence ratings to negative high arousal (NHA) stimuli as a function of Chapman physical anhedonia, and (d) average valence ratings to negative high arousal (NHA) stimuli as a function of Chapman social anhedonia, in individuals with schizophrenia (SCZ; circles) and controls (CON; triangles).

Figure 3.

Results of ROI analyses of valence, arousal, and valenceXarousal contrasts in the total sample (both patients and controls). Regions are described in Table 3. Images are shown in neurological orientation.

(a) Regions showing significant activation in the valence contrast. Positive z-scores (red) indicate greater activation to positively valenced stimuli than to negatively valenced stimuli; negative z-scores (blue) indicate greater activation to negatively valenced stimuli than to positively valenced stimuli.

(b) Regions showing significant activation in the arousal contrast. Positive z-scores (red) indicate greater activation to high arousal stimuli relative to neutral and low-arousal stimuli.

(c) Regions showing significant activation in the valence-by-arousal contrast. Negative z-scores indicate greater activation to negative high arousal stimuli than to positive and/or low-arousal stimuli.

Figure 4.

Results of ROI analyses of group t-tests between patients and controls for the valence and valenceXarousal contrasts. Regions are described in Table 4. Images are shown in neurological orientation.

(a) Right ventral striatal region demonstrating a group difference in the valence contrast. Activation did not differ significantly in the negative high arousal (NHA), negative low arousal (NLA), or neutral (NEU) conditions, but was significantly lower in patients than in controls for the positive low arousal (PLA) and positive high arousal (PHA) conditions.

(b) Left putamen region demonstrating a group difference in the valenceXarousal contrast. Activation did not differ significantly in the negative, neutral, or PLA conditions, but was significantly lower in patients than in controls in the PHA condition.

* $p < .05$. Error bars represent standard error.

Figure 5.

Results of whole-brain analyses of valence, arousal, and valenceXarousal contrasts in the total sample (both patients and controls). Regions are described in Table 5. Images are shown in neurological orientation.

(a) Regions showing significant activation in the valence contrast. Positive z-scores (red) indicate greater activation to positively valenced stimuli than to negatively valenced stimuli; negative z-scores (blue) indicate greater activation to negatively valenced stimuli than to positively valenced stimuli.

(b) Regions showing significant activation in the arousal contrast. Positive z-scores (red) indicate greater activation to high arousal stimuli relative to neutral and low-arousal stimuli; negative z-scores (blue) indicate greater activation to neutral and low-arousal stimuli than to high-arousal stimuli.

(c) Regions showing significant activation in the valenceXarousal contrast. Negative z-scores indicate greater activation to negative high arousal stimuli than to positive and/or low-arousal stimuli.

Table 1

Clinical and demographic characteristics

	Mean (SD)		t or X ²	df	p
	CON	SCZ			
Age	36.25 (10.85)	36.8 (8.99)	0.24	70	0.82
Education	15.53 (4.29)	13.05 (2.27)	3.15	70	0.002*
Highest Parental Education	12.76 (2.76)	13.35 (3.83)	-1.65	65	0.1
Gender (% Male)	65.6	65	0.003	1	0.95
Race (% Caucasian)	53.1	47.5	0.225	1	0.64
Chapman Social Anhedonia	2.35 (2.06)	5.28 (2.17)	-5.748	69	.001*
Chapman Physical Anhedonia	3.45 (2.99)	7.23 (4.18)	-4.253	69	.001*
SANS Global Anhedonia	-	2.68 (1.21)	-	-	-
Positive symptoms	-	1.83 (0.93)	-	-	-
Negative symptoms	-	1.81 (1.37)	-	-	-
Disorganization symptoms	-	1.17 (0.80)	-	-	-
Duration of Illness (years)	-	17.73 (11.25)	-	-	-
Diagnosis subtype (%)	-	-	-	-	-
Schizoaffective - bipolar	-	7.5	-	-	-
Schizoaffective - depressive	-	17.5	-	-	-
Schizophrenia - undifferentiated	-	42.5	-	-	-
Schizophrenia - residual	-	15	-	-	-
Schizophrenia - paranoid	-	15	-	-	-
Schizophrenia - disorganized	-	2.5	-	-	-
Medication (% Taking)	-	-	-	-	-
Aripiprazole	-	22.5	-	-	-
Clozapine	-	5.0	-	-	-
Ziprasidone	-	2.5	-	-	-
Haloperidol	-	5.0	-	-	-
Iloperidone	-	2.5	-	-	-
Risperidone	-	30.0	-	-	-
Quetiapine	-	17.5	-	-	-

	Mean (SD)		t or χ^2	df	p
	CON	SCZ			
Trifluoperazine	-	2.5	-	-	-
Olanzapine	-	12.5	-	-	-
Average Dose (CPZ equivalents)	-	452.20 (369.60)	-	-	-

CON = control, SCZ = schizophrenia, SD = standard deviation

SANS = Scale for the Assessment of Negative Symptoms

SAPS = Scale for the Assessment of Positive Symptoms

CPZ = chlorpromazine; df = degrees of freedom

*
p < .05

Positive symptoms were the sum of global scores for hallucinations and delusions; negative symptoms were the sum of global scores for alogia, anhedonia, avolition, affective flattening and attentional impairment; and disorganization symptoms were the sum of global scores for bizarre behavior, positive thought disorder, and inappropriate affect.

Table 2

Hierarchical regression analyses using group and anhedonia scores to predict valence

Dependent Variable	Predictors	R ²	R ² Change	β
PHA Valence Rating	Step 1	.229****		
	Group			-.191
	Physical Anhedonia			-.360***
	Step 2	.231***	.002	
	Group × Physical Anhedonia			-.127
PHA Valence Rating	Step 1	.192***		
	Group			-.177
	Social Anhedonia			-.313*
	Step 2	.206***	.014	
	Group × Social Anhedonia			-.357
NHA Valence Rating	Step 1	.199***		
	Group			.191
	Physical Anhedonia			.326**
	Step 2	.205***	.006	
	Group × Physical Anhedonia			-.219
NHA Valence Rating	Step 1	.190***		
	Group			.151
	Social Anhedonia			.332**
	Step 2	.197**	.007	
	Group × Social Anhedonia			-.246

*
p < .05,**
p < .01,***
p < .005,****
p < .001; PHA = positive high arousal; NHA = negative high arousal.

Table 3
Results of ROI analyses of valence, arousal, and valenceXarousal contrasts in the total sample

Brain Region	Talairach Coordinates	BA	# Voxels	Z	Activation Pattern		Regional Analyses				
					Valence	Arousal	Main Effect of Group	Group X Condition Interaction	Group Difference in Contrast	Within CON Contrast	Within SCZ Contrast
Valence Contrast											
R OFC	41, 24, -4	47	27	-3.98	neu > neg > pos	-	***	NS	NS	***	***
rACC	1, 35, 3	32	17	4.61	deactivation: neu = neg > pos	-	NS	NS	NS	*****	**
Arousal Contrast											
R Amygdala	26, -14, -8	-	24	3.39	-	ha > la = neu	NS	NS	NS	****	*
dmPFC	-1, 51, 23	9	79	3.68	-	ha > la = neu	NS	NS	NS	NS	*****
ValenceXArousal Contrast											
R amygdala	28, -10, -15	-	21	-3.41	neg > neu = pos	nha > nla = neu	NS	NS	NS	*	**

* p<.05,

** p<.01,

*** p<.005,

p<.001; NS = not significant; BA= Brodmann Area, CON = control, SCZ= schizophrenia, R = Right, L = Left, rACC = rostral anterior cingulate cortex, OFC = orbitofrontal cortex, dmPFC = dorsomedial prefrontal cortex

Table 4

Results of ROI analyses of group t-tests between patients and controls for the valence and valenceXarousal contrasts

Brain Region	Talairach Coordinates	# Voxels	Z
Valence Contrast			
R Ventral Striatum	7, 3, -4	14	2.80
Valence X Arousal Contrast			
L Putamen	-28, -14, -1	17	3.44

R = Right, L = Left

Table 5

Results of whole-brain analyses of valence, arousal, and valenceXarousal contrasts in the total sample

Brain Region	BA	Talairach Coordinates	# Voxels	Z	Activation Pattern		Regional Analyses				
					Valence	Arousal	Main Effect of Group	Group X Condition Interaction	Group Difference in Contrast	Within CON Contrast	Within SCZ Contrast
Valence Contrast:											
Midbrain	-	1, -27, -3	45	-4.50	neg > neu=pos	-	***	*	NS	****	*
R Fusiform Gyrus	37	39, -50, -13	144	-3.66	neg=neu > pos	-	NS	NS	NS	****	****
R Orbital Frontal Cortex	47	45, 28, -4	82	-3.25	neg=neu > pos	-	***	*	NS	***	****
R Inferior Frontal Gyrus	9	45, 15, 29	242	-3.98	neg=neu > pos	-	***	NS	NS	****	****
L Inferior Frontal Gyrus	9	-47, 13, 26	163	-2.96	neg=neu > pos	-	***	NS	NS	***	****
L Postcentral Gyrus	40	-54, -24, 23	516	4.02	pos > neu=neg	-	NS	NS	NS	****	****
R Central Operculum	43	51, -5, 11	130	3.56	pos=neu > neg	-	**	NS	NS	****	****
R Supramarginal Gyrus	40	59, -29, 30	167	3.61	pos=neu > neg	-	*	NS	NS	****	****
R Supramarginal Gyrus	40	45, -43, 49	69	2.69	pos=neu > neg	-	NS	NS	NS	**	****
R Superior Parietal Lobule	7	16, -60, 63	88	2.46	pos=neu > neg	-	**	NS	NS	*	****
L Superior Parietal Lobule	7	-18, -55, 64	70	2.89	pos=neu > neg	-	*	NS	NS	***	***
Rostral Anterior Cingulate	24	-1, 31, 6	112	3.98	deactivation: neg > pos	-	NS	NS	NS	****	**
Arousal Contrast:											
R Middle Occipital Gyrus	37	53, -64, 7	210	5.26	-	ha > la > neu	NS	NS	NS	****	****
R Cerebellar Declive	-	3, -77, -14	191	4.65	-	ha > neu=la	NS	NS	NS	*	****
R Cuneus	18	5, -90, 10	205	4.33	-	ha > neu=la	NS	NS	NS	***	***
L Posterior Cingulate	29	-2, -43, 2	60	3.87	-	ha > neu=la	*	NS	NS	***	*
L Middle Occipital Gyrus	37	-54, -66, 6	114	4.45	-	ha > neu=la	NS	NS	NS	****	*
R Middle Frontal Gyrus	9	46, 31, 27	49	-4.21	-	neu > la > ha	**	NS	NS	***	***
Dorsomedial Prefrontal Cortex	9	-1, 54, 21	140	4.20	-	deactivation: neu > la; activation: ha	NS	NS	NS	*	****
L Paracentral Lobule	31	-2, -28, 45	133	4.86	-	deactivation: neu=la; activation: ha	NS	NS	NS	****	***

Brain Region	BA	Talairach Coordinates	# Voxels	Z	Activation Pattern		Regional Analyses				
					Valence	Arousal	Main Effect of Group	Group X Condition Interaction	Group Difference in Contrast	Within CON Contrast	Within SCZ Contrast
L Precuneus	7	-1, -55, 49	30	4.25	-	deactivation: neu=la > ha	NS	NS	NS	****	****
ValenceX Arousal Contrast:											
L Fusiform Gyrus	37	-39, -62, -15	123	-4.78	neg=neu > pos	nha > nla=neu	NS	NS	NS	*	****

* p<.05,

** p<.01,

*** p<.005,

**** p<.001; CON = control; SCZ = schizophrenia; R = Right; L = Left; pos = positive; neg = negative; neu = neutral; ha = high arousal; la = low arousal; NS = not significant; BA = Brodmann Area

Table 6
ROI results of correlation analyses between anhedonia scores and fMRI valence and valenceXarousal contrast scores

Contrast	Anhedonia Measure	Talairach Coordinates	Region name	# Voxels	r	Z
Control						
Valence	Chapman social anhedonia	8, -1, 14	R Caudate	14	-0.550	-3.26
		-13, -2, 18	L Caudate	28	-0.539	-3.18
Schizophrenia						
Valence	Chapman physical anhedonia	-14, -9, -15	L Amygdala	17	-0.446	-2.89
ValenceXArousal	Chapman physical anhedonia	17, -14, -11	R Amygdala	15	-0.413	-2.58

R = right, L = left