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Social anhedonia is associated with neural abnormalities during face emotion processing

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ABSTRACT

Human beings are social organisms with an intrinsic desire to seek and participate in social interactions. Social anhedonia is a personality trait characterized by a reduced desire for social affiliation and reduced pleasure derived from interpersonal interactions. Abnormally high levels of social anhedonia prospectively predict the development of schizophrenia and contribute to poorer outcomes for schizophrenia patients. Despite the strong association between social anhedonia and schizophrenia, the neural mechanisms that underlie individual differences in social anhedonia have not been studied and are thus poorly understood. Deficits in face emotion recognition are related to poorer social outcomes in schizophrenia, and it has been suggested that face emotion recognition deficits may be a behavioral marker for schizophrenia liability. In the current study, we used functional magnetic resonance imaging (fMRI) to see whether there are differences in the brain networks underlying basic face emotion processing in a community sample of individuals low vs. high in social anhedonia. We isolated the neural mechanisms related to face emotion processing by comparing face emotion discrimination with four other baseline conditions (identity discrimination of emotional faces, identity discrimination of neutral faces, object discrimination, and pattern discrimination). Results showed a group (high/low social anhedonia) × condition (emotion discrimination/control condition) interaction in the anterior portion of the rostral medial prefrontal cortex, right superior temporal gyrus, and left somatosensory cortex. As predicted, high (relative to low) social anhedonia participants showed less neural activity in face emotion processing regions during emotion discrimination as compared to each control condition. The findings suggest that social anhedonia is associated with abnormalities in networks responsible for basic processes associated with social cognition, and provide a starting point for understanding the neural basis of social motivation and our drive to seek social affiliation.

Introduction

As fundamentally social creatures, humans are driven by the desire for meaningful and frequent social interaction (Baumeister and Leary, 1995). There are individual differences in the strength of this desire, however, and some individuals exhibit a significantly reduced drive for social affiliation known as social anhedonia (Brown et al., 2007; Kwapil, 1998; Kwapil et al., 2009). Social anhedonia (SA) has been characterized as a deficiency in the need to belong to a social group and is distinct from other constructs that might also predict abnormalities in social interaction such as social anxiety (Brown et al., 2007; Kwapil et al., 2009). Individuals high in SA exhibit a genuine preference for solitude, disengagement during social interactions (Brown et al., 2007), and reduced negative affect when alone (Kwapil et al., 2009). Higher levels of SA are related to lower levels of social support and social functioning (Blanchard et al., 2011). Reduced social support and smaller social networks are associated with differences in immune functioning and other clinically significant health outcomes (Miller et al., 2009). Furthermore, high SA has been identified as one of the single most predictive traits for future onset of schizophrenia spectrum disorders (Kwapil, 1998) and has long been recognized as a core attribute of psychosis vulnerability (Bleuler, 1950; Horan et al., 2007; Kraepelin and Gosline, 1919; Meehl, 1962; Rado, 1953; Stone et al., 2005). Altogether, existing evidence indicates that SA is a deviation in a psychologically and clinically important social and emotional process that has broad implications for our understanding of normal and abnormal functioning.

Despite evidence for serious physical and mental health difficulties associated with reduced desire for social affiliation, no research to our knowledge has been done exploring the neural basis of SA in nonclinical populations. In schizophrenia, SA is considered a negative symptom that is stable (Blanchard et al., 2001) and can be reliably assessed (Horan et al., 2006). Studies of SA in schizophrenia have indicated that a number of neural systems may be involved in reduced

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desire for social affiliation, including the amygdala (Becerril and Barch, 2010), caudate nucleus (Dowd and Barch, 2010), dorsolateral prefrontal cortex (Becerril and Barch, 2010), and somatosensory areas (Arnfred and Chen, 2004). However, disease-related confounds and secondary characteristics of schizophrenia illness, such as psychosocial stress and neurodegenerative processes, make it difficult to generalize these findings to SA among healthy individuals or to identify whether neural abnormalities associated with SA are associated with the psychosis vulnerability (Lenzenweger, 2006).

Differences in the neural processing of face emotion provide a potential starting point for identifying abnormalities associated with high SA. Accurate face emotion recognition is critical for recognizing and responding to other's mental states and is a building block to more complex social behaviors (Adolphs, 2002). Importantly, face emotion recognition ability (but not face identity processing ability) predicts social functioning in schizophrenia participants (Hooker and Park, 2002) and varies with psychometric psychosis-proneness in non-clinical populations (Germine and Hooker, 2011). Previous work has also shown that face emotion perception is abnormal in individuals high in social anhedonia (Luh and Gooding, 1999). Thus, individual differences in social anhedonia may be related to deficits in the neural mechanisms supporting face emotion recognition.

The neural substrates of face emotion recognition are well characterized in healthy and clinical populations. Previous work indicates that effective emotion recognition involves the recruitment of a network of regions, including the amygdala (Adolphs, 2002; Adolphs et al., 1994; Anderson and Phelps, 2001), superior temporal sulcus (Allison et al., 2000; Haxby et al., 2000), medial prefrontal cortex (Amodio and Frith, 2006; Blair et al., 1999; Dolan et al., 1996; Gorno-Tempini et al., 2001; Gur et al., 2002a; Phillips et al., 1998; Sprengelmeyer et al., 1998; Wright et al., 2002), and somatosensory-related cortices (including insula, S1, S2, and anterior supramarginal gyrus) (Adolphs, 2002; Adolphs et al., 2000). Using functional neuroimaging, researchers have consistently found abnormalities in these regions during emotion recognition in individuals with schizophrenia (Das et al., 2007; Farrer et al., 2004; Gur et al., 2002b, 2007; Hall et al., 2004; Hempel et al., 2003; Holt et al., 2006; Kosaka et al., 2002; Phillips et al., 1999; Pinkham et al., 2008; Schneider et al., 1998; Spence et al., 1997; Taylor et al., 2002; Waberski et al., 2004; Williams et al., 2004).

Deficits in emotion recognition have been associated with lesions to the amygdala (Adolphs et al., 1994), somatosensory and related cortices (Adolphs et al., 2000) and medial prefrontal cortex (Heberlein et al., 2008). The medial prefrontal cortex, in particular, likely plays a broad role in many social-cognitive processes and has been implicated in lower-level emotion perception as well as higher-level processes including theory of mind attributions (Gallagher et al., 2000), self-referential processing (Mitchell et al., 2005), and distinguishing between self and other (Heatherton et al., 2006; Ochsner et al., 2004). In terms of functional divisions, the anterior portion of rostral medial prefrontal cortex (arMFC) has been consistently identified in measures of social cognition and emotion processing (Amodio and Frith, 2006) and in social cognition in schizophrenia (Brunet-Gouet and Decety, 2006).

In the present study, we used functional magnetic resonance imaging (fMRI) to examine differences in the neural circuitry underlying face emotion discrimination in otherwise normal individuals who were high versus low in SA. As our face emotion recognition task, we used the Queen Square Face Discrimination Test (QFDT; Garrido et al., 2009). Our primary hypothesis was that high SA would have specific deficits in face emotion processing even when controlling for broader, but equally complex aspects of face perception. The QFDT was chosen because it can dissociate face emotion processing and face identity processing (Banissy et al., 2011; Garrido et al., 2009; Germine and Hooker, 2011; Pitcher et al., 2008). In the QFDT, participants view sequentially presented emotional faces; in one condition they judge whether the two faces are expressing the same emotion, and in another condition they judge whether the two faces have the same identity. Importantly, the two conditions have identical stimuli and are equally difficult for healthy participants. As a result, any differences found between emotion discrimination and identity discrimination can be attributed to differences in specific cognitive processes related to emotion perception and cannot be attributed to differences in the stimuli, number of response options, or difficulty level of the two conditions. This feature of the task is an improvement over face processing studies where the experimental and control tasks differ along these dimensions (e.g. labeling emotional faces using four options vs. same/different identity of paired neutral faces). In the QFDT, the emotion recognition and identity recognition conditions use the same task structure (both are a forced choice same/different judgment) and the same stimuli. Therefore the comparison of emotion recognition and identity recognition of emotional faces isolates the specific cognitive processes for attending to, processing, and judging face emotions. Using a behavioral version of this task, we found that higher levels of psychosis risk (based on self-report of cognitive-perceptual, interpersonal, and disorganized psychosis-prone characteristics) are associated with reduced emotion discrimination performance, but normal identity discrimination performance (Germine and Hooker, 2011). The QFDT was also used by Pitcher et al. (2008), who found that applying transcranial magnetic stimulation (TMS) to the face area of somatosensory cortex impaired performance in the emotion discrimination condition, but not the identity discrimination condition. Thus, we have good reason to believe that the QFDT emotion discrimination condition depends on one or more processes specific to emotion processing that also vary with psychosis vulnerability. The current fMRI study included three additional control conditions designed to reveal potential group differences in the broader face emotion processing neural network. These conditions included identity discrimination of neutral faces, visual discrimination of objects, and visual discrimination of patterns. Given the putative relationship between SA and vulnerability for psychosis (Kwapil, 1998), we predicted that individuals high in SA would exhibit reduced recruitment of cortical regions involved in face emotion recognition, particularly superior temporal sulcus/gyrus, medial prefrontal cortex and somatosensory-related parietal regions, as well as reduced responses in the amygdala. Between group differences in one or more of these regions would indicate that higher levels of SA are associated with neural abnormalities during emotion perception, and help us better understand the neural basis of differences in the desire for social affiliation as well as psychosis vulnerability.

Material and methods

Participants

We recruited a community-based sample comprised of thirty participants who were high or low in social anhedonia based on their scores on the Revised Chapman Social Anhedonia Scale (RSAS; Chapman and Chapman, 1980). Fifteen high social anhedonia participants (high SA) were selected based on scoring in the top 10% on this measure (RSAS score>16 for females, >19 for males). Fifteen low social anhedonia participants (low SA) were selected based on having scores at or below the mean (RSAS score<7 for females and <9 for males). Participants were recruited from a combination of community advertisements and the community-wide university study pool. Community advertisements were posted on sites like Craigslist and targeted individuals with difficulties in interpersonal functioning associated with social anhedonia and psychosis risk (e.g. “People sometimes find me aloof and distant.”). In addition, items from the RSAS were used to pre-select individuals with high social anhedonia from the community-wide university study pool. Anyone who had a score of 16 or greater on the RSAS was invited to come into the lab for further screening. All participants took

the full RSAS after completing MR screening and demographic questionnaires. In total, 12/15 high SA individuals were recruited through community advertisements and 3/15 high SA participants came from prescreening of the community-wide university study pool using the RSAS. Participants in the low SA groups were recruited from prescreening of the community-wide university study pool for low-to-average levels of social anhedonia (based on the RSAS) and demographic characteristics similar to our high SA group. Altogether, 4/15 participants in our high SA group and 5/15 in our low SA group were university students. All participants were administered the Structured Clinical Interview for DSM-IV (SCID; First et al., 2002) and were excluded if they had any Axis I diagnosis, a history of alcohol or drug dependence, alcohol or drug abuse within the last six months, a past major head injury involving a loss of consciousness lasting more than 2–3 min, or did not speak English as a primary language. Socioeconomic status was assessed using the Hollingshead Index (Hollingshead, 1957). One participant in the high SA group and two participants in the low SA group were unable to give parental information, and so the average parental education/ socioeconomic status from that individual's SA group was used to replace the missing values (e.g. the mean parental socioeconomic status from the other 14 members of the high SA group was used in place of the final high SA participant's missing value). Groups did not differ significantly in terms of sex, age, education, parental education, socioeconomic status, or parental socioeconomic status (see Table 1).

Informed written consent was obtained from all participants after the nature of the study and procedures had been fully explained. The study was approved by the Institutional Review Boards at Harvard University and Massachusetts General Hospital (MGH) (Partners health care system).

Stimuli and experimental paradigm

In the scanner, participants were asked to judge whether two sequentially presented stimuli were the same or different on a specified characteristic. There were five different conditions presented in a block design. In the main condition of interest, participants were asked to discriminate the emotions of sequentially presented emotional faces (Emotional faces: Emotion discrimination — EE). In a comparison condition, participants were asked to discriminate the identities of the same set of sequentially presented emotional faces (Emotional faces: Identity discrimination — EI). These two conditions (EE and EI) were taken from the Queen Square Face Discrimination Task (QFDT; Garrido et al., 2009). Using this task, Pitcher et al. (2008) found specific deficits in face emotion discrimination but not face identity discrimination after transcranial magnetic stimulation (TMS) of the face area of somatosensory cortex, one of our regions of interest. Face stimuli were adapted from the images of six female models from Ekman and Friesen's(1976) facial affect series, expressing one of six emotions: happy, sad, surprise, fear, disgust, and anger. An example trial is shown in Fig. 1. The six facial expressions appeared an equal number of times in the EE task and the six models appeared an equal number of times in the EI task. The same images were used for both tasks, with identity varying between sample and target faces in all EE trials and expression varying between sample and target faces in all EI trials. The order of conditions was counterbalanced across runs. Out of four runs, two started with EE blocks whereas the other two started with EI blocks. We also included three additional baseline/control conditions: identity discrimination of sequentially presented neutral faces (Neutral faces: Identity discrimination — NI), visual discrimination of sequentially presented grayscale cars (Object discrimination — OD), and visual discrimination of sequentially presented patterns (Pattern discrimination — PD). The models used in the NI task were the same as those included in the EE and EI tasks. Objects used in the OD task were all side views of similar-looking sedans. Finally, the PD task used scrambled face images.

The structure of a single trial is shown in Fig. 1. Participants had to indicate whether the sample and target image depicted the same face emotion or different face emotion (EE), the same identity or a different identity (EI, NI tasks), or the same image or a different image (OD, PD

Table 1
Participant characteristics for low social anhedonia (low SA) and high social anhedonia (high SA) groups. Where applicable, values represent mean and standard deviation (in parentheses) for each group. All p values were based on two-tailed independent samples t-tests with df = 28. Socioeconomic status was based on the Hollingshead Index (Hollingshead, 1957), where numbers 1 to 7 were assigned to each occupational category with 1 for unskilled employees and 7 for higher executives, major professionals and proprietors. Levels of social anhedonia were determined by scores from the Revised Social Anhedonia Scale (RSAS; Chapman and Chapman, 1980).

	Low SA	High SA	p
Sex	7/15 male	7/15 male	–
Age	32.5 (12.5)	31.5 (10.7)	0.8
Education (participant; years)	14.7 (2.4)	15.5 (2.4)	0.4
Education (parental; years)	13.6 (2.1)	15.1 (1.9)	0.08
Socioeconomic status (participant)	4.3 (1.8)	4.7 (2.0)	0.8
Socioeconomic status (parental)	3.9(1.0)	4.4(1.3)	0.26
RSAS score	3.7 (2.9)	26.3 (6.6)	<0.001
Handedness	14/15 right-handed	14/15 right-handed	–

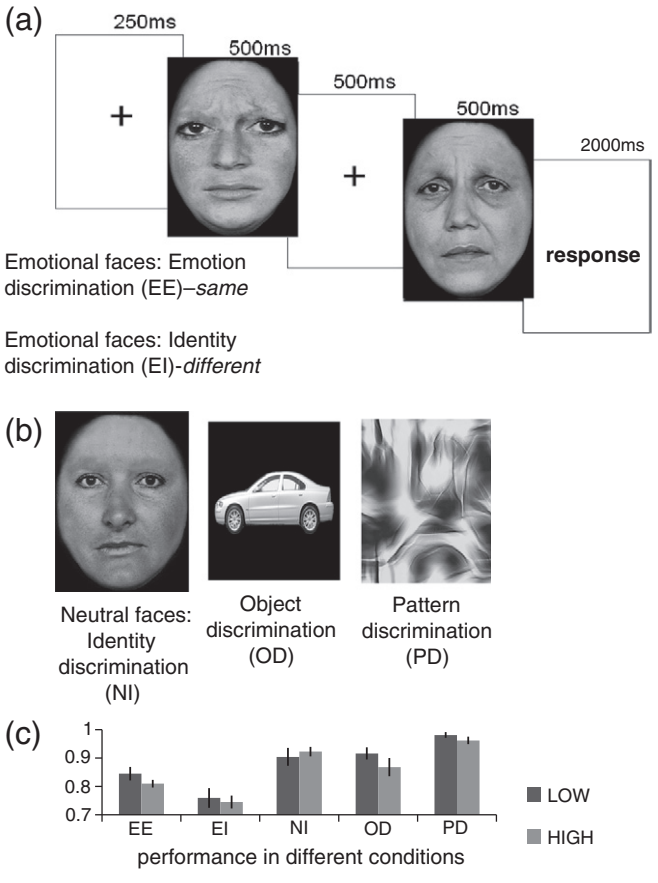

Fig. 1. Task stimuli and behavioral performance. (a) A single trial of the Emotional faces: Emotion discrimination (EE) condition. Stimuli were the same for EE and EI (Emotional faces: Identity discrimination) conditions. (b) Example stimuli from the three other comparison conditions. (c) Behavioral performance in terms of proportion correct for each condition. The darker bar represents performance for low social anhedonia participants, whereas the lighter bar represents performance for high social anhedonia participants. There were no between group differences in performance in any condition.

tasks). There were 72 trials of each task, with half requiring "same" judgments.

All participants were administered a brief practice including all conditions and correct/incorrect feedback before placement in the scanner. The practice and scanning experiments were administered using E-Prime software.

fMRI protocol

Scanning sessions lasted for 40 min, and consisted of 4 runs with 3 blocks of each task per run. Across the 4 runs, there were 72 trials of each task. Each block consisted of 6 trials of the same task and lasted 22.5 s, preceded by a task cue for 2.5 s and followed by a 12.5 second fixation period. While in the scanner, participants wore earplugs to muffle noise, and head fixation was ensured through foam padding in the head coil.

fMRI image acquisition

Brain imaging data were acquired using a 3.0 T Siemens Trio scanner employing a 12 channel whole-head coil. For functional scans, data were acquired in an oblique-axial plane using gradient echo planar imaging (EPI) with an echo time of 30 ms and repetition time of 2500 ms. Each volume comprised 41 slices with a 2.5 mm slice thickness and a gap of 0.8 mm giving coverage of the whole brain, except for the most superior portion of the posterior parietal lobe. Voxel size was $3.1 \times 3.1 \times 2.5$ mm and volumes were continuously acquired every 2.5 s in an interleaved fashion. Each run was preceded by 5 'dummy' scans to allow T1 equilibration. A structural scan sequence (MPRAGE) was conducted to obtain a T1-weighted anatomical image (128 saggital slices, voxel size $1.3 \times 1.0 \times 1.3$ mm, flip angle = 7 degrees, TR = 2530 ms, TE = 3.39 ms).

fMRI: functional activation analyses

We analyzed the data using SPM8 (Wellcome Department of Cognitive Neurology, London, United Kingdom; http://www.fil.ion.ucl.ac.uk/spm/software/spm8). Preprocessing included realignment to the first volume acquired, coregistration of the structural to functional scans, normalization to a structural template (re-sampled voxel size after normalization was $2 \times 2 \times 2$ mm), and smoothing with a 6 mm Gaussian kernel. Analyses were conducted with a general linear model framework. Vectors of onset times with durations of 22.5 s were defined for all five tasks: EE, EI, NI, OD, and PD. These onset vectors were convolved with the SPM8 canonical hemodynamic response function (HRF) using a box-car function. Additionally, regressors were created using an artifact detection tool (ART; Whitfield-Gabrieli, 2009) to exclude scans with gross motion (>0.6 mm relative to previous time frame) or spiking artifacts (global mean image intensity greater than 2.5 SD from mean of the entire time series within a scan) from analysis. Where this procedure resulted in omission of more than 10% of time frames, filters were adjusted to bring the number of excluded scans to approximately 10%. There were no between group differences in number of outliers identified (high SA max = 83; low SA max = 92) or filter parameters. A high-pass frequency filter (200 s) was also applied to the time series. For each subject, contrast images were calculated for each of the five tasks (EE, EI, NI, OD, and PD) relative to baseline (blocks of fixation).

Second-level analysis

To verify that our task was activating our regions of interest, we conducted a one sample t-test of EE vs. baseline (fixation) across all participants. To assess whether our EE task was isolating regions for face emotion processing relative to face identity processing, we also conducted a one sample t-test of the contrast of EE versus EI across participants. To look at between group differences, we implemented a flexible factorial design in SPM8. Our hypothesis was a group (high/low SA) × condition (EE/control) interaction, such that high SA would show less activity for EE as compared to each control condition. Four group × condition analyses were conducted: EE vs. EI, EE vs. NI, EE vs. OD, and EE vs. PD. All group maps were thresholded at $p<0.001$ uncorrected with an extent threshold of 5 voxels. For regions of interest where suprathreshold clusters were identified in our flexible factorial analysis, small volume corrections were performed using Family Wise Error correction (FWE, $p<0.05$). The WFU Pickatlas (Maldjian et al., 2003, 2004) was used to create anatomically defined masks of the right superior temporal gyrus, bilateral postcentral gyri (somatosensory cortices), and bilateral supramarginal gyri (somatosensory-related or somatosensory association cortices). A mask of the anterior portion of rostral medial prefrontal cortex (arMFC) was defined based on Amodio and Frith (2006). This mask was drawn to include all voxels in prefrontal cortex with MNI coordinates of $x<20$ and $x>-20$, $y>20$, and $z>0$ (4013 voxels total). Any voxel in the left or right hemisphere that fell within these coordinate boundaries was included in a single, bilateral arMFC mask. For regions of interest showing a significant interaction, contrast estimates were extracted from the peak of primary clusters in order to conduct post hoc comparisons.

Results

Behavioral

No significant between group differences were found in performance on any condition (EE, EI, NI, OD, and PD) (all $p>0.2$). Details of performance in each condition for each group are shown in Fig. 1.

Effects of task across all participants

We conducted one sample t-tests on EE (vs. baseline) contrasts across all participants to verify that the EE task was inducing BOLD signal changes in the expected face emotion and face processing regions. This analysis verified task-related activity in a network of regions that included the right superior temporal sulcus, left postcentral gyrus (left primary somatosensory cortex), bilateral supramarginal gyri (somatosensory-related or somatosensory association cortices), bilateral fusiform gyri, and bilateral medial prefrontal cortex, including the anterior rostral medial prefrontal cortex (arMFC) (see Table 2). No suprathreshold voxels were detected in the amygdala.

To verify that the EE task was uniquely associated with activity in our emotion perception regions of interest relative to other tasks, we also conducted a one sample t-test on the EE vs. EI contrast across all participants. This analysis revealed a network of activation for the EE task (relative to EI) that included the right superior temporal sulcus, bilateral postcentral gyri (primary somatosensory cortices), left supramarginal gyrus (somatosensory-related cortex), and bilateral medial prefrontal cortices, including anterior rostral medial prefrontal cortex (arMFC) (see Table 2). As in our EE vs. baseline contrast, no suprathreshold voxels were detected in the amygdala. With the exception of the amygdala, these results demonstrate that the EE task was associated with activation across all of our regions of interest in the distributed emotion perception network, above and beyond the most closely matched control condition (EI). Given that this is the first time this task has been used in the scanner, our EE vs. EI contrast serves as validation that the EE task is suited to tapping into neural networks associated with emotion perception.

Group comparisons

The main hypothesis of the study was that participants with high social anhedonia would show reduced activity in emotion processing regions during emotion discrimination. Regions showing a significant

Table 2
Emotional faces: Emotion discrimination (EE) related fMRI BOLD responses across all participants. Neural activity clusters are based on one sample t-tests across all participants with a significance threshold of $p<0.001$ uncorrected and an extent threshold of 5 voxels. Neuroanatomical labels, MNI coordinates, and t-values are provided for the peak voxel of each cluster. Clusters that include *a priori* regions of interest are italicized. Superscripts denote clusters containing areas of activation that survive small volume correction based on regions of interest: (1) Includes voxels in anterior rostral medial frontal cortex (arMFC) that survive small volume correction over a bilateral region defined by voxels with MNI coordinates of $|x|<20$, $y>20$, and $z>0$ (Amodio and Frith, 2006); (2) Includes voxels in postcentral gyrus/somatosensory cortex that survive small volume correction over anatomically defined bilateral postcentral gyri; (3) Includes voxels in right superior temporal gyrus that survive small volume correction over anatomically defined right superior temporal gyrus; (4) Includes voxels in supramarginal gyrus/somatosensory-related/somatosensory-association cortex that survive small volume correction over anatomically defined bilateral supramarginal gyri.

Brain region	Brodmann areas	MNI coordinates			t value	Cluster size in voxels
		x	y	z		
Emotional faces: Emotion discrimination (EE) vs. baseline–all participants						
L middle occipital gyrus	19	−22	−98	0	17.21	13,498
L medial frontal gyrus[1]	8	−6	14	48	12.33	2042
L superior parietal lobule[2]	7	−28	−58	48	11.36	10,028
R inferior frontal gyrus	9	42	10	26	10.58	6471
R inferior parietal lobule[4]	40	36	−52	48	10.17	1306
R superior temporal gyrus[3]	42	50	−38	10	5.25	128
L cuneus	18	−20	−70	8	4.16	43
pons	NA	0	−30	−34	3.98	54
Emotional faces: Emotion discrimination (EE) vs. Emotional faces: Identity discrimination (EI)—all participants						
L inferior frontal gyrus	45	−54	30	4	8.37	2877
L superior frontal gyrus/arMFC[1]	6	−10	8	60	7.06	1877
L superior temporal sulcus[4]	22	−42	−40	2	6.35	1538
R cerebellum	NA	30	−70	−36	5.84	402
L caudate	NA	−8	14	8	5.18	150
R cerebellum	NA	12	−38	−48	4.67	57
R middle frontal gyrus	47	52	38	−2	4.6	189
R superior temporal sulcus/gyrus[3]	22	50	−34	0	4.5	283
R superior temporal gyrus[3]	38	40	6	−22	4.39	22
R postcentral gyrus/somatosensory cortex[2]	3	20	−40	54	4.37	147
L cingulate gyrus	24	−4	−12	36	4.24	54
L postcentral gyrus/somatosensory cortex[2]	4	−18	−28	70	4.24	52
L cingulate gyrus	31	−16	−22	44	4.13	11
L cerebellum	NA	−26	−76	−38	3.84	14
L superior temporal gyrus	38	−48	8	−24	3.64	5
L medial frontal gyrus	6	−8	−30	62	3.63	10
L middle frontal gyrus	10	−38	60	−2	3.6	8
L superior frontal gyrus/arMFC[1]	9	−12	54	28	3.54	6

interaction in the expected direction, i.e. where low SA had greater activity than high SA participants for emotion discrimination (Emotional faces: Emotion discrimination condition − EE) versus the control condition, are listed in Table 3. Regions where there was a significant group × condition interaction but with the opposite pattern (i.e. where high SA had more activity than low SA for EE>control) are shown in Table 4. None of these regions (where high SA>low SA during EE) occurred in our regions of interest. Clusters within a priori regions of interest were investigated by extracting contrast estimates from the peak of the cluster. Primary clusters showing significant group x condition interactions are shown together with contrast estimates in Fig. 2, organized by region of interest. Finally, Table 5 shows results of post hoc comparisons applied to the contrast estimates in primary clusters located in our regions of interest.

Emotional faces: Emotion discrimination (EE) vs. Emotional faces: Identity discrimination (EI)

For the comparison of EE and EI, there was a significant group x condition interaction (where low SA>high SA) in arMFC (see Fig. 2), but not other regions of interest. Post hoc comparisons were conducted to investigate the interaction. Statistics for each comparison are in Table 5. As predicted, the results showed that high SA participants had reduced arMFC activity during emotion discrimination. In addition, low SA participants had greater arMFC activity for emotion discrimination as compared to identity discrimination of emotional faces (i.e. among low SA: EE>EI). However, high SA participants deactivated the arMFC during EE, such that high SA had significantly less activity for emotion discrimination as compared to identity discrimination of emotional faces (i.e. among high SA: EE<EI). Moreover, the direct comparison of emotion discrimination between the groups showed that low SA participants had significantly greater arMFC activity than high SA participants (i.e. low SA EE>high SA EE). There was no significant difference between low SA and high SA participants for arMFC activity during identity discrimination of emotional faces (EI condition) (see Table 5).

Activation in arMFC did not survive small volume correction (over bilateral arMFC; see Group comparisons section) in this contrast ($p=0.15$).

Emotional faces: Emotion discrimination (EE) vs. Neutral faces: Identity discrimination (NI)

When comparing EE with NI, there was a significant group × condition interaction (where low SA>high SA) in arMFC and right superior temporal gyrus (see Fig. 2). Removing the extent threshold ($p<0.001$ uncorrected, $k=0$) revealed an additional suprathreshold cluster in left somatosensory cortex (left postcentral gyrus). Post hoc comparisons (see Table 5) revealed the same overall pattern in arMFC as was observed in the EE vs. EI analysis (see Emotional faces: Emotion discrimination (EE) vs. Emotional faces: Identity discrimination (EI) section). In right superior temporal gyrus and left somatosensory cortex, high SA participants deactivated regions of both right superior temporal gyrus and left somatosensory cortex during EE relative to NI (i.e. among high SA: EE<NI) whereas low SA participants did not show differences between these two conditions (i.e. among low SA: EE = NI). As predicted, low SA was associated with greater activity in right superior temporal gyrus and left somatosensory cortex during EE (i.e. low SA EE>high SA EE), but not differences during NI (i.e. low SA NI = high SA NI).

Both arMFC and right superior temporal gyrus clusters survived small volume correction in this comparison ($p<0.05$).

Table 3
Group × condition interactions: Regions where low social anhedonia > high social anhedonia during Emotional faces: Emotion discrimination (EE). Neural activity clusters are areas where significant group (low social anhedonia vs. high social anhedonia) × condition (emotion discrimination vs. control) interactions were detected at $p<0.001$ (uncorrected) with an extent threshold of 5 voxels. Neuroanatomical labels, MNI coordinates, and t-values are provided for the peak voxel of each cluster. Regions indicated with a ^ only showed significant interactions when the extent threshold was removed. Small volume correction was applied to the regions corresponding to (1) the anterior portion of rostral medial prefrontal cortex (arMFC) bilaterally as defined by Amodio and Frith (2006) (region defined as all voxels with MNI coordinates: $|x|<20$, $y>20$, $z>0$), (2) right superior temporal gyrus and (3) bilateral postcentral gyri (somatosensory cortices). Regions indicated with an asterisk * survived small volume corrections (family-wise error corrected, $p<0.05$). Clusters that occur in *a priori* regions of interest are italicized.

Brain region	Brodmann areas	MNI coordinates			t value	Cluster size in voxels
		x	y	z		
Emotional faces: Emotion discrimination (EE) vs. Emotional faces: Identity discrimination (EI)						
R medial frontal gyrus	10	16	56	12	3.75	6
Emotional faces: Emotion discrimination (EE) vs. Neutral faces: Identity discrimination (NI)						
*R superior frontal gyrus(arMFC)**	10	16	62	14	4.99	112
R caudate	8	24	18	18	4.24	52
*R superior temporal gyrus**	42	66	−24	10	3.98	50
R fusiform gyrus	18	38	−72	−12	3.85	22
L medial superior frontal (arMFC)	10	−14	56	2	3.77	9
L superior frontal gyrus (arMFC)	10	−16	50	26	3.75	23
R superior temporal gyrus	41	42	−30	8	3.69	5
R supenor temporal gyrus	22	60	−8	0	3.67	5
L precentral gyrus	6	−40	−10	38	3.65	7
*L postcentrol gyrus (somatosensory Cortex)^*	3	−54	−14	50	3.64	4
Emotional faces: Emotion discrimination (EE) vs. Object discrimination (OD)						
R anterior cingulate/subgenual cortex	25	2	16	−12	4.58	85
*L superior frontal gyrus (arMFC)**	10	−16	52	26	4.11	32
R superior temporal gyrus	22	66	−20	6	3.84	16
L middle frontal gyrus	8	−28	30	48	3.74	8
*L. postcentral qyrus (somatosensory cortex)^*	3	−44	−22	62	3.5	1
Emotional faces: Emotion discrimination (EE) vs. Pattern discrimination (PD)						
*R superior temporal gyrus**	22	66	−18	4	4.93	115
L superior frontal gyrus (arMFC)	10	−16	48	24	4.16	52
L posterior cingulate	30	−10	−54	16	4.1	45
R anterior cingulate/subgenual cortex	25	2	10	−8	4.02	36
R superior temporal gyrus	22	60	−8	0	3.98	29
R precentral gyrus	6	62	0	36	3.67	5
L anterior cingulate	32	−10	42	−4	3.61	6
R medial frontol gyrus (arMFC)	10	18	54	16	3.52	5

Emotional faces: Emotion discrimination (EE) vs. Object discrimination (OD)

When comparing EE with OD, there was again a significant group x condition interaction (where low SA>high SA) in arMFC and right superior temporal gyrus (see Fig. 2). As in the comparison of EE and NI, removing the extent threshold ($p<0.001$ uncorrected, $k=0$) also revealed an additional suprathreshold cluster in left somatosensory cortex (left postcentral gyrus). Post hoc comparisons (see Table 5) revealed the same overall pattern in arMFC and right superior temporal gyrus as was observed in the comparison of EE and NI (Emotional faces: Emotion discrimination (EE) vs. Neutral faces: Identity discrimination (NI) section). In left somatosensory cortex, significant differences were observed in all post hoc comparisons. High SA participants deactivated this region during EE relative to OD

Table 4
Group × condition interactions: Regions where high social anhedonia > low social anhedonia during Emotional faces: Emotion discrimination (EE). Neural activity clusters are areas where significant group (high social anhedonia>low social anhedonia) x condition (emotion discrimination vs. control) were detected at $p<0.001$ (uncorrected) with an extent threshold of 5 voxels. Neuroanatomical labels, MNI coordinates, and t-values are provided for the peak voxel of each cluster. No clusters showing high social anhedonia>low social anhedonia occurred in any of our regions of interest.

Brain region	Brodmann areas	MNI coordinates			t value	Cluster size in voxels
		x	y	z		
Emotional faces: Emotion discrimination (EE) vs. Emotional faces: Identity discrimination (EI)						
L precuneus	7	−18	−56	50	4.12	16
L fusiform gyrus	37	−38	−50	−8	3.93	11
R cingulate gyrus	31	22	−34	32	3.81	9
L cerebellum	NA	−12	−66	−26	3.79	11
L cerebellum	NA	−22	−64	−28	3.65	5
Emotional faces: Emotion discrimination (EE) vs. Neutral faces: Identity discrimination (NI)						
None						
Emotional faces: Emotion discrimination (EE) vs. Object discrimination (OD)						
R precentral gyrus	44	56	10	10	3.5	5
Emotional faces: Emotion discrimination (EE) vs. Pattern discrimination (PD)						
R middle frontal gyrus	9	38	10	34	4.4	49
L middle frontal gyrus	10	−28	52	−8	4.3	22
R inferior frontal gyrus	44	50	6	18	4.18	61
R precentral gyrus	44	46	18	10	3.77	12
R middle frontal gyrus	10	44	48	24	3.75	10
L fusiform gyrus	37	−38	−60	−10	3.66	5

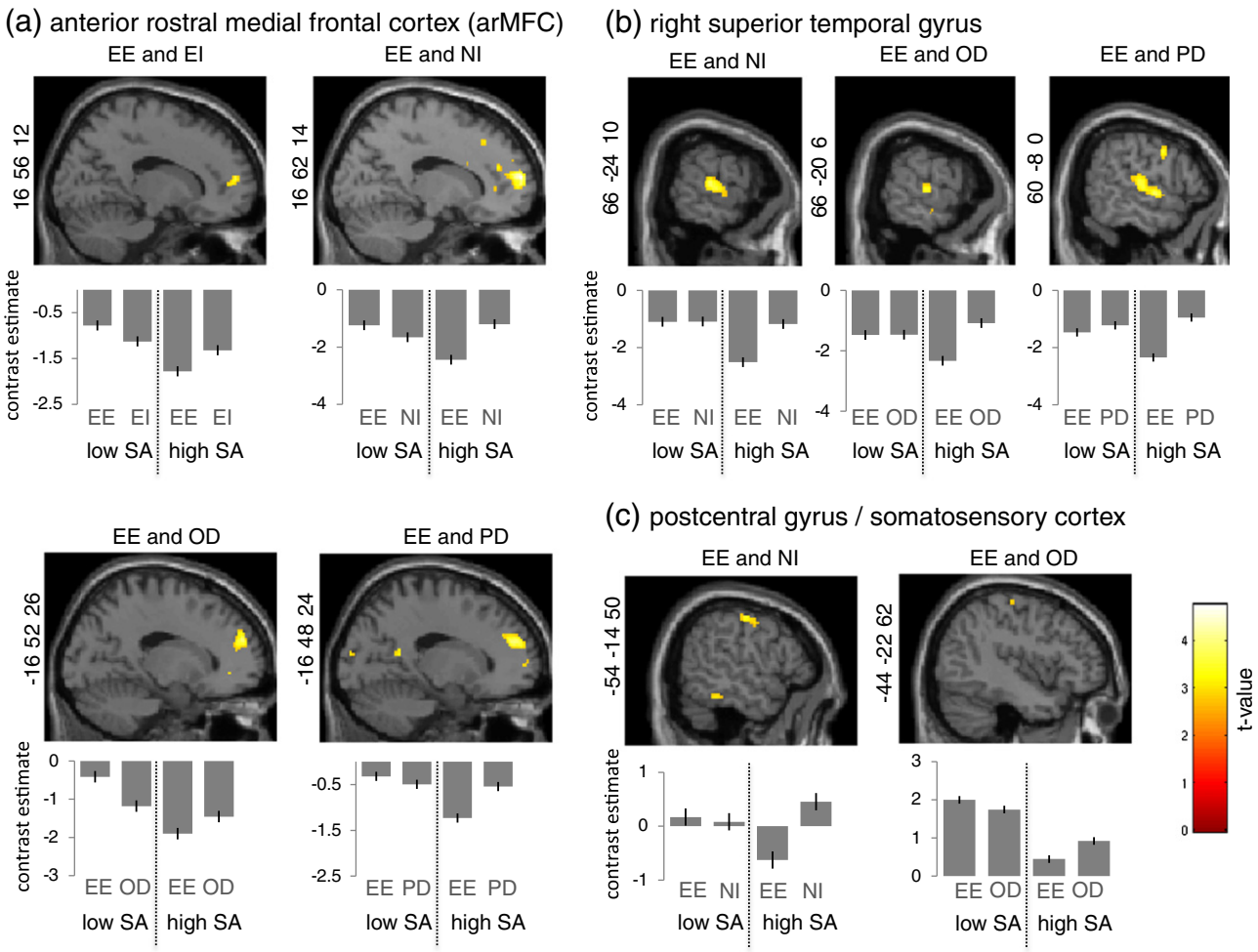

Fig. 2. fMRI BOLD responses associated with Emotional faces: Emotion discrimination (EE) in low vs. high social anhedonia (SA) groups. Activation patterns and contrast estimates associated with group × condition interaction effects in our regions of interest are shown. The Emotional faces: Emotion discrimination (EE) condition was compared with Emotional faces: Identity discrimination (EI), Neutral faces: Identity discrimination (NI), Object discrimination (OD), and Pattern discrimination (PD), for low social anhedonia (low SA) and high social anhedonia (high SA) participants. Significant interactions were observed in: (a) anterior rostral medial frontal cortex (arMFC) when comparing Emotion discrimination (EE) with EI, NI, OD, and PD; (b) right superior temporal gyrus when comparing EE with NI, OD, and PD; and (c) left postcentral gyrus/somatosensory cortex when comparing EE with NI and OD. MNI coordinates (x y z) of peak voxels for each cluster are shown to the left of each image. Contrast estimates were extracted from the peak voxel of the cluster and plotted for each group and condition. All results shown above are based on a full flexible factorial model implemented in SPM 8, with a significance threshold of $p<0.001$ uncorrected. Clusters are displayed at $p<0.005$ to show activation extent.

(i.e. among high SA: EE<OD) whereas low SA participants showed greater activation of this region during EE relative to OD (i.e. among low SA: EE>OD). As predicted, low SA showed greater activity in somatosensory cortex during EE than high SA (i.e. low SA EE>high SA EE), but also during OD (i.e. low SA OD>high SA OD).

Differences in arMFaC in the comparison of EE and OD survived small volume correction at the trend level ($p=0.06$), whereas differences in right superior temporal gyrus did not ($p=0.25$).

Emotional faces: Emotion discrimination (EE) vs. Pattern discrimination (PD)

When comparing EE with PD, there was again a significant group x condition interaction (where low SA>high SA) in arMFC and right superior temporal gyrus (see Fig. 2). Removing the extent threshold did not reveal any additional suprathreshold clusters in other regions of interest. Post hoc comparisons (see Table 5) revealed that, as predicted, low SA had greater activation during EE than high SA in arMFC and right superior temporal gyrus (i.e. low SA EE>high SA EE). High SA also showed deactivation during EE as compared with PD (i.e. among high SA: EE<PD). Among low SA, there was no difference between EE and PD in these regions (i.e. among low SA: EE = PD) and no differences in PD between low and high SA (i.e. low SA PD = high SA PD).

Differences in right superior temporal gyrus survived small volume correction ($p<0.05$) in this comparison. Differences in arMFC survived small volume correction at the trend level ($p=0.06$).

Further analyses

Amygdala

Although the amygdala was one of our a priori regions of interest, we found no within-subjects differences in this region when comparing EE with any of our control conditions (EI, NI, OD and PD) across all participants. We also failed to detect any significant group × condition interactions in the amygdala.

As the amygdala is considered a central part of the extended face emotion perception network, we conducted further analyses to explore whether the combination of all conditions using faces (EE, EI, and NI) would show suprathreshold amygdala activation when compared with baseline. Using a within-subjects one sample t-test across all participants, we again found no significant differences in the amygdala ($p<0.001$). An analysis of signal-to-noise in the right amygdala as compared with right superior temporal gyrus in our sample suggested

Table 5
Post hoc comparisons for group x condition interactions. Four post hoc comparisons were performed for each cluster showing a group x condition interaction effect shown in Fig. 2. The five conditions examined were Emotional faces: Emotion discrimination (EE), Emotional faces: Identity discrimination (EI), Neutral faces: Identity discrimination (NI), Object discrimination (OD), and Pattern discrimination (PD). Low SA signifies the low social anhedonia group, whereas high SA signifies the high social anhedonia group. Where the comparison was within the same group (e.g. low SA: EE vs. low SA: EI), paired t-tests were used (two-tailed; df = 14). Where the comparison was between groups (e.g. low SA: EE vs. high SA: EE), independent sample t-tests (two-tailed; df = 28) were used.

Brain region	MNI coordinates			Contrast	t value	p value
	x	y	z			
(1) anterior rostral medial frontal cortex (|x|<20,y>20, z>0)	16	56	12	Low SA: EE–low SA: EI	3.23	0.006
				High SA: EE–high SA: EI	−4.27	0.0008
				Low SA: EE–high SA: EE	6.56	<0.0001
				Low SA: EI–high SA: EI	0.26	0.23
	16	62	14	Low SA EE–low SA NI	2.46	0.03
				High SA EE–high SA: NI	−7.3	<0.0001
				Low SA: EE–high SA: EE	5	0.0002
				Low SA: NI–high SA: NI	−1.9	0.08
	−16	52	26	Low SA: EE–low SA: OD	5.12	0.0002
				High SA EE–high SA OD	−3	0.01
				Low SA: EE–high SA:EE	7.02	<0.0001
				Low SA: OD–high SA: OD	1.29	0.22
	−16	48	24	Low SA: EE–low SA:PD	1.7	0.11
				High SA: EE–high SA: PD	−6.7	<0.0001
				Low SA: EE–high SA: EE	6.2	<0.0001
				Low SA: PD–high SA: PD	0.34	0.74
(2) right superior temporal gyrus	66	−24	10	Low SA: EE–low SA: NI	−0.05	0.96
				High SA: EE–high SA: NI	−7.8	<0.0001
				Low SA: EE–high SA EE	5.86	<0.0001
				Low SA: NI–high SA: NI	0.36	0.72
	66	−20	6	Low SA: EE–low SA: OD	−0.06	0.95
				High SA: EE–high SA: OD	−7.8	<0.0001
				Low SA: EE–high SA:EE	4.76	0.002
				Low SA: OD–high SA: OD	−1.7	0.11
	60	−8	0	Low SA: EE–low SA:PD	−1.7	0.11
				High SA: EE–high SA: PD	−9.7	<0.0001
				Low SA: CE–high SA: EE	4.3	<0.0001
				Low SA: PD–high SA: PD	−1.3	0.2
(3) postcentral gyrus/somatosensory cortex	−54	−14	50	Low SA: EE–low SA: NI	0.55	0.59
				High SA: EE–high SA: NI	−6.76	<0.0001
				Low SA EE–high SA: EE	3.53	0.003
				Low SA: NI–high SA: NI	−1.65	0.12
	−44	−22	62	Low SA: EE–low SA: OD	2.43	0.03
				high SA EE–high SA: OD	−4.56	0.0004
				Low SA: EE–high SA: EE	10.54	<0.0001
				Low SA: OD–high SA: OD	5.6	<0.0001

significantly lower signal-to-noise in the amygdala (paired samples t-test: $t(29) = 11.8$; $p < 0.0001$). Previous research has indicated that the amygdala habituates rapidly to emotional information (Breiter et al., 1996) and may show decreased activity during emotion labeling as compared with other forms of encoding (Lieberman et al., 2007). Low signal-to-noise combined with our use of a block design, continuous presentation of faces, and possible emotion labeling demands may have interfered with our ability to detect amygdala differences.

Fusiform gyrus

Given its role in face processing more generally, we also looked at differences in the degree to which emotion modulated BOLD responses in the fusiform gyrus (Vuilleumier et al., 2001) in high vs. low SA participants. We observed a group x condition interaction in both left and right fusiform gyri. In the right fusiform gyrus, the low SA group showed greater BOLD response for EE>NI as compared to the high SA group. These results are consistent with our hypothesis that high SA (vs. low SA) would be associated with reduced neural responses during emotion discrimination. We also found, though, that high SA was associated with greater BOLD response in the left fusiform gyrus for EE>NI and EE>PD as compared to the low SA group. Since face processing is associated more strongly with right fusiform responses in most individuals (Kanwisher et al., 1997; McCarthy et al., 1997), our observation of fusiform gyrus response differences suggests variations in lateralization that may relate to level of SA. These results are difficult to interpret, however, and warrant further investigation.

Correlations with behavioral performance during emotion discrimination (EE)

To explore whether any of our task-related regions showed significant correlations with performance, we extracted contrast estimates from clusters in our regions of interest that were significantly associated with EE vs. EI across all participants (right superior temporal sulcus, somatosensory cortices, and arMFC). None of these regions showed a significant or trend relationship with EE performance across participants.

Discussion

Although social anhedonia has long been recognized as a key feature of schizophrenia illness and liability, there is surprisingly little known about its underlying neural substrates. In this study we investigated whether otherwise healthy individuals with high social anhedonia (SA) had deficient neural activity during face emotion discrimination — a social cognitive process associated with robust behavioral and neural deficits in schizophrenia. The results show that people with high SA have reduced neural response in emotion perception regions during discrimination of emotional faces. Compared to low SA, high SA was associated with reduced neural activity in the anterior portion of the rostral medial prefrontal cortex (arMFC), right superior temporal gyrus, and left somatosensory cortex during emotion discrimination relative to control conditions. Deficient activity for emotion discrimination in the high SA group was most

consistent in arMFC. High SA participants showed reduced recruitment of this region when emotion discrimination was compared to each control condition, including identity discrimination of emotional faces — a condition that is comparable in difficulty and uses the exact same emotional stimuli as the emotion discrimination condition.

Detailed examination of neural activity in the regions that showed a group × condition interaction (arMFC, right superior temporal gyrus, and somatosensory cortex) revealed a few consistent patterns. First, as predicted, there were significant between group differences during emotion discrimination, such that low SA participants had more neural activity than high SA in these regions. Second, the high SA group showed significantly *less* neural activity of these same regions during emotion discrimination compared to the control conditions. In the arMFC, low SA group showed greater neural activity for the emotion discrimination as compared to the control conditions. There were no consistent differences between low SA and high SA in the control conditions. Interestingly, these findings suggest that high SA participants were *deactivating* these regions during emotion perception. It is unclear based on the current work whether these differences were related to differences in strategy during emotion discrimination (e.g. attending to low-level features to perform the task and thus down-regulating activity in emotion processing regions) or other differences in emotional information processing. For example, previous studies have shown that patients with schizophrenia exhibit abnormal visual scanpaths of emotional faces (Loughland et al., 2002) and show greater interference from face identity information during emotion matching compared to healthy control participants (Baudouin et al., 2002).

These findings have important implications for our understanding of the mechanisms underlying individual differences in SA, as well as the clinical and functional consequences of these differences. The medial prefrontal cortex, superior temporal gyrus, and somatosensory cortices are part of a network of brain regions that process face emotions. The medial prefrontal cortex plays a role in emotion recognition (Heberlein et al., 2008), emotion experience (Heberlein et al., 2008), mentalizing (Gallagher et al., 2000), and self-other processing (Heatherton et al., 2006; Ochsner et al., 2004). The arMFC region, in particular, has been found in previous studies to be consistently associated with mentalizing and other aspects of social cognition (Amodio and Frith, 2006). This region has also been associated with abnormalities during social-cognitive processing in schizophrenia samples (Brunet-Gouet and Decety, 2006). Lesions to ventral (but not lateral or dorsal) regions of the medial prefrontal cortex are also associated with impairments in emotion recognition (Heberlein et al., 2008). The superior temporal gyrus and sulcus are involved in perceptual processing of dynamic social stimuli including facial expressions of emotion and eye gaze (Haxby et al., 2000; Hooker et al., 2003, 2008, 2010). Finally, somatosensory cortex and related areas are thought to contribute to emotion processing by allowing facial expressions to be understood using an internal representation of a facial expression maintained in one's own somatosensory cortex (Adolphs et al., 2000; Heberlein et al., 2008; Hooker et al., 2008). Disruption of activity in somatosensory cortex leads to impairments in emotion discrimination of the same emotional face stimuli used in the present study (the Queen Square Face Discrimination Task, QFDT; Pitcher et al., 2008) and lesions to somatosensory and somatosensory-related areas are likewise associated with emotion recognition deficits (Adolphs et al., 2000). As somatosensory cortex and medial prefrontal cortex are involved in both emotion experience and emotion recognition, researchers have suggested that these regions are involved in understanding other's mental states through simulation mechanisms (Adolphs, 2002; Adolphs et al., 2000; Heberlein et al., 2008; Hooker et al., 2008). Given these previous findings, our results suggest that social anhedonia is related to differences in the neural substrates responsible for self/other representation and social perception, perhaps through their common relationship with simulation mechanisms.

As a personality trait, SA is specifically related with schizophrenia and not to other disorders with anhedonic symptoms (Blanchard et al., 2001). Given this relationship, our findings can also be interpreted in the context of schizophrenia vulnerability. Individuals with schizophrenia have abnormalities in medial prefrontal cortex responses during emotion perception (Hempel et al., 2003) and intention attribution (Brunet et al., 2003). Structural abnormalities have also been consistently identified in superior temporal regions in individuals with schizophrenia and schizophrenia spectrum disorders (Davidson and Heinrichs, 2003; Dickey et al., 2002a, 2002b, 2003; Downhill et al., 2001; Siever and Davis, 2004; Wright et al., 2000). Superior temporal gyrus abnormalities may be related to deficits in both emotion perception (Edwards et al., 2002; Hooker and Park, 2002; Mandal et al., 1998; Mueser et al., 1996) and gaze perception (Hooker et al., 2003; Hooker and Park, 2005) observed in individuals with schizophrenia. In addition, deficits in somatosensory processing (e.g. differences in two point discrimination) are often associated with schizophrenia and schizophrenia vulnerability (Chang and Lenzenweger, 2001, 2004, 2005; Hooley and Delgado, 2001; Lenzenweger et al., 2003).

Our neural findings from individuals with high levels of SA are consistent with a relationship between SA and schizophrenia vulnerability (Kwapil, 1998; Stone et al., 2005). High SA in young adults prospectively predicts schizophrenia diagnosis ten years later (Kwapil, 1998). In addition, first-degree relatives of schizophrenia patients have abnormally high anhedonia levels (Stone et al., 2005). Understanding the neural basis of individual differences in SA may thus contribute to our understanding of schizophrenia liability and development.

Although our results indicate a relationship between SA and differences in neural networks related to basic emotion recognition, it is unclear whether these neural response differences are a cause or a consequence of varying levels of SA. High SA is identified by self-report of reduced approach motivation in social situations. If emotion recognition and social approach motivation rely on shared neural substrates, lack of approach motivation may be intrinsically related to reduced recruitment of social cognitive networks. For example, a reduced tendency or ability to simulate the mental states of others might result in both reduced social approach motivation as well as reduced emotion processing/recognition through the same basic mechanisms. Alternatively, over the course of development, social isolation associated with high SA may contribute to reduced engagement of social cognition systems during social interaction. The result of this could then be a reduced tendency of these systems to respond to even straightforward emotion recognition demands. Finally, it is also possible that abnormalities in the neural networks responsible for processing social and emotional stimuli lead to high-level trait differences in SA. That is, reduced responses in social perception networks may create a predisposition to experience lower levels of pleasure from social interaction and thus reduced drive for social affiliation. It is not possible to distinguish between these possibilities based on the current study. Future work might address these questions by looking at how differences in brain function predict differences in social pleasure over hours, days, or years.

The present study has several limitations. First, our use of a block design did not permit us to look at the relationship between brain activation and accuracy on individual trials. Using a block design also meant that we were unable to investigate emotion-specific effects (e.g. positive vs. negative valence) from emotion processing more generally. As social anhedonia is defined by lack of pleasure from social interactions rather than increased negative affect during social interactions, it is possible that our results were driven by abnormalities in neural responses to positive emotional faces rather than emotional faces more generally. Due to the limited number of trials per emotion and the use of same/different responses, we were not able to look at emotion-specific brain responses and between-group differences in these responses. It is thus unclear whether our results were driven by differences associated with a specific emotion.

Another limitation was that our task failed to produce suprathreshold activity in the amygdala in either low or high SA groups. In

addition to possible habituation effects from our use of a block design and continuous face presentation, possible emotion labeling demands (Lieberman et al., 2007) and reduced signal-to-noise in this region may have compromised our ability to detect amygdala differences. Thus it is difficult to interpret our lack of between group differences in medial temporal lobe areas.

Finally, although we assessed Axis I disorders in all participants, we did not conduct a comprehensive assessment of Axis II personality disorders. One of the symptoms of Schizoid Personality Disorder, in particular, is high levels of social anhedonia. It is possible that some of our participants met criteria for this disorder. Although not a form of psychosis, Schizoid Personality Disorder is considered a schizophrenia spectrum disorder and this diagnostic information would have been useful for exploring differences between disordered and nondisordered forms of SA in our sample.

Conclusion

The wide range of physical and mental health outcomes arising from differences in social affiliation and social support argues that the experience of pleasure that accompanies social interaction is a vital component of a functioning social cognitive system (Brown et al., 2007; Kwapil et al., 2009) with broad and meaningful health consequences. Social impairments and low levels of social affiliation are related to increased risk of mental illness (Hooley, 2010), as well as differences in immune functioning and mortality (Miller et al., 2009). Understanding the neural basis of differences in SA is thus both psychologically and clinically important. Our results indicate that individual differences in SA are related to observable differences in neural responses to social-emotional stimuli, especially in systems responsible for emotion perception and higher-level social cognitive functions. Future work elucidating the neural mechanisms underlying SA will have critical implications for our understanding of normal and abnormal social functioning, and the basic processes that fuel our fundamental drive to be social beings.

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