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Neural Activity to Positive Expressions Predicts Daily Experience of Schizophrenia-Spectrum Symptoms in Adults With High Social Anhedonia

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Social anhedonia (SA), the diminished pleasure from social relationships, is a prominent characteristic of the vulnerability and manifestation of schizophrenia disorder. However, SA can develop for multiple reasons and little is known about its neural basis; these 2 issues hinder the utility and sensitivity of SA as a marker of schizophrenia pathology. This study investigated whether lateral prefrontal cortex (LPFC) deficits in social reward processing are associated with both SA and other schizophrenia-spectrum symptoms. During functional MRI (fMRI), a community sample of healthy adults ($N = 30$) with high and low SA viewed positive, negative, and neutral facial expressions. Afterward, participants completed an online daily diary in which they rated schizophrenia-spectrum symptoms and occurrence of interpersonal conflict each day for 21 days. Compared with low SA, high SA participants had less ventral (V)LPFC activity to positive versus neutral expressions. In addition, participants with a combination of high SA and low VLPFC activity to positive versus neutral expressions had worse daily diary ratings of schizophrenia-spectrum symptoms, including worse cognition, paranoia, motivation/productivity, and vigor/positive affect (i.e., psychomotor activation). Finally, among high SA participants, VLPFC activity predicted the daily relationship between distress from interpersonal conflict and symptom-severity; specifically, high SA participants with low VLPFC activity had worse paranoia on days of high conflict distress. These findings indicate that VLPFC deficits in positive emotion are associated with both SA and other schizophrenia-spectrum symptoms and that understanding the interaction of SA, VLPFC function, and social stress could facilitate the use of SA in the prevention and treatment of schizophrenia.

Keywords: psychosis-proneness, schizotypy, fMRI, reward processing, social cognition

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Social anhedonia (SA), defined as diminished pleasure from social relationships, is a cardinal feature of schizophrenia-spectrum pathology. SA is a prominent, treatment-resistant characteristic of schizophrenia disorder that is evident prior to frank-psychosis and contributes to functional disability throughout illness (Blanchard, Mueser, & Bellack, 1998; Horan, Blanchard, Clark, & Green, 2008). Relatives of people with schizophrenia have abnormally high SA (Laurent et al., 2000). Otherwise healthy individuals with high SA exhibit schizophrenia-related problems, including cognitive deficits and psychotic-like experiences (Blanchard, Aghevli, Wilson, & Sargeant, 2010; Blanchard, Collins, Aghevli, Leung, & Cohen, 2011; Cohen, Leung, Saperstein, & Blanchard, 2006). High SA in college students prospectively predicts schizophrenia-spectrum disorders (Gooding, Tallent, & Matts, 2005, 2007; Kwapil, 1998), and high levels of asociality, which include social isolation and other SA-related characteristics, prospectively predict schizophrenia in both familial and clinical high-risk groups, as well as the general population (Tarbox & Pogue-Geile, 2008). Furthermore, although physical anhedonia is also associated with schizophrenia pathology, SA has greater influence on psychosis-risk and functional disability (Chapman, Chapman, Kwapil, Eckblad, & Zinser, 1994; Granholm, Ben-Zeev, & Link, 2009), suggesting that social consequences of SA impact disease expression (Blanchard et al., 2011; Horan, Brown, & Blanchard, 2007; Kwapil et al., 2009). Together these findings suggest that screening for SA could identify people at psychosis-risk who would benefit from preventive intervention, and that treating SA could yield functional benefits for people suffering from schizophrenia.

However, using SA as a marker of schizophrenia requires information about basic neural mechanisms of SA and how they relate to other schizophrenia-spectrum symptoms. SA can develop for reasons unrelated to schizophrenia pathology, including depressed mood (Blanchard, Horan, & Brown, 2001), social rejection

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(Baumeister & Leary, 1995), medication (Juckel et al., 2006), and internalized stigma (Yanos, Roe, Markus, & Lysaker, 2008). Thus, protocols that assess SA from behavioral reports, exclusively, will include people who developed SA for different reasons. As a consequence, prevention strategies that identify psychosis-risk from behavioral reports of SA could misclassify people, and longitudinal studies of psychosis-risk could underestimate the association between SA and schizophrenia. Moreover, neural treatment targets are difficult to identify because the multiple origins of SA obscure neural systems germane to schizophrenia, and benefits of neurally based treatments are hard to measure because behavioral assessments cannot determine whether SA improvements are due to enhanced neural function or other factors (Kirkpatrick, Fenton, Carpenter, & Marder, 2006). Therefore, neural deficits that are associated with both SA and other schizophrenia-spectrum symptoms would be a more specific marker to aid psychosis prevention and treatment.

The current study used functional MRI (fMRI) and experience sampling methods to test whether lateral prefrontal cortex (LPFC) deficits in social reward processing are associated with both SA and other schizophrenia-spectrum characteristics and whether the effect of LPFC deficits on schizophrenia-spectrum symptoms is influenced by social stress. We investigated these hypotheses in a community sample of adults that varied in SA—half with abnormally high SA and half with average or below average SA.

Psychometric High-Risk Approach

Investigating the neural basis of SA in a community sample that includes psychometrically defined high SA participants (i.e., scores >98% of the population) addresses several research barriers. First, SA is isolated by selecting participants who vary from normal to abnormal on this one characteristic of schizophrenia rather than selecting participants with a schizophrenia-spectrum disorder who, according to diagnostic criteria, have abnormal behavior in multiple domains. This construct-specific, dimensional approach enhances sensitivity of neural investigations because single behaviors, such as SA, map onto brain systems more accurately than diagnostic categories (Cuthbert & Insel, 2010; Insel et al., 2010).

Second, SA research with community samples is more generalizable than college student samples and more reliable than clinical samples. Most people with schizophrenia use antipsychotic medication and experience internalized stigma (Livingston & Boyd, 2010; Vauth, Kleim, Wirtz, & Corrigan, 2007)—both of which increase SA (Yanos et al., 2008). Moreover, antipsychotic medication reduces reward-related neural activity in the LPFC and other regions for both schizophrenia and healthy participants (Abler, Erk, & Walter, 2007; Juckel et al., 2006; Walter, Kammerer, Frasch, Spitzer, & Abler, 2009).

Finally, the psychometrically defined criterion ensures abnormally high SA scores in half the sample. Because SA has a taxonic structure in which only a small percentage of people have high SA, random-selection or a non-SA criterion, such as diagnosis, yields mostly low SA participants and requires a large sample to illustrate behavioral or neural effects (Blanchard, Gangestad, Brown, & Horan, 2000; Horan, Blanchard, Gangestad, & Kwapil, 2004). This is especially problematic for neuroimaging research because required resources limit sample size. Our approach oversampled high SA participants and included normal-to-low SA in the 'low' group to produce a wide range of scores. This strategy optimizes sensitivity to detect between-groups differences in neural function, as well as continuous relationships between SA, LPFC function, and schizophrenia-spectrum symptoms (DeCoster, Iselin, & Gallucci, 2009; Preacher, Rucker, MacCallum, & Nicewander, 2005).

LPFC Function in Reward Processing

Feeling pleasure from social interactions (and other events) arises from the interaction of neural structures that respond to reward, including ventromedial prefrontal cortex (VMPFC), and neural structures that control emotional experience from reward, including LPFC (Barrett, Mesquita, Ochsner, & Gross, 2007). LPFC, particularly ventral (V)LPFC, manages emotional experience by deploying cognitive skills, such as attentional control, to down-regulate negative emotion and up-regulate positive emotion (Kim & Hamann, 2007; Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008). LPFC regulatory functions also create, maintain, and retrieve representations of emotional experiences, such as pleasant memories, which motivate behavior (Miller & Cohen, 2001; Wallis & Miller, 2003).

Using fMRI and daily diary methods, Hooker, Gyurak, Verosky, Miyakawa, and Ayduk (2010) demonstrated that greater VLPFC activity to positive and negative (vs. neutral) expressions from a romantic partner predicted better emotion regulation after a conflict with that person in daily life. Detailed analyses revealed that VLPFC activity to positive expressions predicted up-regulation of positive mood but not down-regulation of negative mood. There was also no correlation between VLPFC activity to positive expressions and VLPFC activity to negative expressions. These findings suggest that engaging LPFC function to control positive emotion is a valence-specific trait that can be quantified by imaging.

LPFC Function and Reward Processing in Schizophrenia-Spectrum Populations

Deficits in LPFC-dependent cognitive skills, such as attention and working memory, are well documented in schizophrenia-spectrum populations (Barch, 2005; Giuliano et al., 2012; Keshavan et al., 2010), including people with high SA (Cohen, Couture, & Blanchard, 2012; Gooding, Matts, & Rollmann, 2006; Gooding & Tallent, 2003). Although LPFC function in reward processing is understudied in psychosis-risk, research in schizophrenia suggests that SA is associated with problems using LPFC functions to control positive emotion. Schizophrenia participants with higher SA have worse working memory and other LPFC-mediated cognitive skills (Strauss & Gold, 2012). While schizophrenia participants, as a group, may not differ from controls in their immediate affective response to emotion probes (Cohen & Minor, 2010), higher SA, across both schizophrenia and healthy participants, is related to lower positive affect in response to positive stimuli (Cohen, Callaway, Najolia, Larsen, & Strauss, 2012; Dowd & Barch, 2010; Strauss & Herbener, 2011). Even when positive affect is experienced normally at first, schizophrenia participants with higher SA are less able to amplify their positive emotion (Henry et al., 2007), remember positive experiences (Herbener, 2009; Herbener, Rosen, Khine, & Sweeney, 2007), anticipate future pleasure (Gard, Kring, Gard, Horan, & Green, 2007), and use positive experiences to motivate behavior (Gold, Waltz, Prentice, Morris, & Heerey, 2008; Strauss & Gold, 2012). FMRI

research indicates that schizophrenia participants have reduced LPFC activity during a delay between viewing positive pictures and reporting emotional response, which suggests difficulty maintaining representations of positive emotion, and lower LPFC activity is related to worse anhedonia/asociality (Ursu et al., 2011). In monetary incentive delay tasks, higher SA is related to abnormal LPFC activity for expected rewards (i.e., when actual earnings match the initial cue), indicating problems creating and/or maintaining representations of reward value (Walter et al., 2010; Walter et al., 2009). These data support the proposal that LPFC deficits in schizophrenia contribute to negative symptoms, such as anhedonia, asociality, and amotivation, which are associated with problems processing reward-related information and using it to motivate goal-directed behavior (Barch & Dowd, 2010; Strauss & Gold, 2012).

Psychometrically defined high SA participants have similar problems with positive emotion. They report less positive affect to positive pictures, films, and words (Kerns, Docherty, & Martin, 2008; Leung, Couture, Blanchard, Lin, & Llerena, 2010; Mathews & Barch, 2006), less attention to their positive emotions, and less consummatory and anticipatory pleasure (Kerns, 2006; Martin, Becker, Cicero, Docherty, & Kerns, 2011). Diary studies confirm less positive affect in daily life and less pleasure from daily events (Brown, Silvia, Myin-Germeys, & Kwapil, 2007; Kwapil, Brown, Silvia, Myin-Germeys, & Barrantes-Vidal, 2012). High SA participants also have difficulty controlling the influence of emotional information on behavior (Martin, Cicero, & Kerns, 2012; Tully, Lincoln, & Hooker, 2012).

Together these data indicate that SA may result from deficits engaging LPFC functions to manage positive affect during social encounters. Because LPFC dysfunction is central to schizophrenia pathology, people who experience SA because of LPFC deficits should have the highest degree of schizophrenia-spectrum symptoms. That is, although SA can develop from other sources, the combination of high SA and low LPFC function may be specific to schizophrenia pathology, and, therefore, predict the day-to-day expression of schizophrenia-spectrum characteristics, especially negative symptoms.

Influence of Social Stress

Distressing social interactions, such as interpersonal conflict, can precipitate or exacerbate psychotic symptoms in schizophrenia-spectrum populations (Hooley, 2007). High SA is not only related to fewer social interactions but also worse quality interactions when they occur (Brown et al., 2007; Kwapil et al., 2012), resulting in more interpersonal conflict and less social support (Blanchard et al., 2011). LPFC functions control the impact of social stress (Hooker et al., 2010), suggesting that high SA individuals with low LPFC function might be especially vulnerable to stress-induced exacerbation of psychotic-like symptoms.

Current Study

During fMRI, high and low SA participants viewed videos (from a standard stimulus set) of interpersonally relevant positive, negative, and neutral facial expressions and rated how accepted or rejected they felt. Afterward, in an online daily diary, they reported severity of schizophrenia-spectrum symptoms, including cognition, paranoia, odd perceptual experiences, negative affect, vigor/positive affect, and motivation/productivity every evening for 21 days. Hypotheses focused on LPFC activity to positive expressions. Although deficits in other reward processing regions were expected, LPFC deficits are most directly associated with schizophrenia-spectrum pathology, and, therefore, should best predict schizophrenia-spectrum symptoms. Similarly, although high SA may have LPFC deficits to negative expressions, reduced response to positive cues is closer to the phenomenology of SA and should best reflect the core problem.

Specific hypotheses were as follows: (1) In a between-groups analysis, high (vs. low) SA participants will have less LPFC activity to positive (vs. neutral) expressions; (2) Using SA as a continuous variable, participants with higher SA and lower LPFC activity to positive (vs. neutral) expressions will have more severe schizophrenia-spectrum symptoms; and (3) the interaction of SA, LPFC activity, and conflict distress will predict more severe schizophrenia-spectrum symptoms, such that participants with higher SA and lower LPFC activity will have worse schizophrenia-spectrum symptoms on days of high conflict distress.

Method

Participants

Thirty healthy adults from Greater Boston participated ($N = 15$ high SA; $N = 15$ low SA). High SA participants were recruited with targeted advertisements (e.g., "Do you prefer to be alone?"). High SA was defined as >1.96 *SD*s above the population mean on the Revised Social Anhedonia Scale (RSAS; females:16+, males: 20+); low SA was defined as equal to or less than 1 *SD* above the population mean (females:12 or less, males:14 or less; Eckblad, Chapman, Chapman, & Mishlove, 1982). Low SA criteria was intended to yield participants in the normal range while excluding individuals (>1 *SD*) who might be categorized as "high SA" by other criteria in the literature. Inclusion criteria: 18–60 years old, primary English speaker. Exclusion criteria: IQ <70, head trauma, neurological illness, substance abuse within 6 months, or current/past Axis I or II disorder.[1] Participants gave written informed consent.

Assessments included: Structured Clinical Interview for *DSM–IV* (SCID) (First, Spitzer, Gibbon, & Williams, 2002) and Schedule for Nonadaptive and Adaptive Personality (SNAP-2; Clark, 2006) for psychopathology; RSAS for participant selection and analyses.[2] Standard measures of schizoptypy and trait affect were used to assess construct validity of the daily diary constructs, including Perceptual Aberration Scale (Chapman, Chapman, & Raulin, 1978), Magical Ideation Scale (Eckblad & Chapman, 1983), Schizotypal Personality Questionnaire (Raine, 1991), Referential Thinking Scale (Lenzenweger, Bennett, & Lilenfeld, 1997), and the Big Five Inventory (John & Srivastava, 1999); Global Functioning Social and Role scales (Cornblatt et al., 2007) were used to

[1] Cluster A disorders were allowed in the high SA group, but no participants had these disorders.

[2] Clinical interviews were conducted by trained clinical psychology doctoral students and supervised by a licensed clinical psychologist (CIH).

validate expected poor social functioning in high SA. IQ was assessed with the Wechsler Abbreviated Scale of Intelligence (WASI). Standard measures of cognitive control skills, including Color Stroop, Wechsler Adult Intelligence Scale Fourth Edition (WAIS IV) letter-number sequencing, and digit span, as well as a trait measure of anticipatory and consummatory pleasure, the Temporal Experiences of Pleasure Scale (TEPS; Gard et al., 2007), were used to characterize participants and to facilitate interpretation of observed LPFC activity. Participant characteristics are shown in Table 1.

fMRI Task

The fMRI task was designed to measure LPFC response to social reward. During the scan, participants viewed short videos of interpersonally relevant positive (e.g., caring, encouraging), negative (e.g., disapproving, contemptuous), and neutral facial expressions. Participants were told to imagine they were interacting with the person and then rate how accepted or rejected they felt. Emotional expressions simulated interpersonal praise and acceptance or criticism and rejection. Behavioral ratings of acceptance/rejection were meant to focus participants' attention on their emotional response. Videos were 3 s (+1-s ISI); presentation was blocked by condition. After viewing 6 videos within a condition-type (e.g., 6 positive expressions), a 5-point rating scale appeared (1 = *very rejected*; 3 = *neutral*; 5 = *very accepted*; 3 s), followed by 12 s of "rest" (white fixation-cross/black background). Twelve blocks of each condition were presented across three fMRI runs.

Facial expression videos were selected from the Mind Reading Library (Baron-Cohen, Hill, & Wheelwright, 2003). Because "happy" is the only positive facial expression included in most commonly used stimulus sets of static basic facial expressions, we tried to maximize social reward processing by using dynamic stimuli in which the individual looks directly at the viewer, as if communicating directly, with a range of positive expressions. An independent sample (N = 29) verified that the videos elicited target feelings of acceptance, rejection, and neutrality (data in Supplemental Materials). The final task included five male and five female actors who appeared in each condition.

fMRI Data Acquisition and Analysis

Participants were scanned on a 3Tesla Siemens TimTrio at Harvard University. Echoplanar image (EPI) acquisition parameters: 40 oblique-axial slices with 3 × 3 × 3 mm isotropic voxels; time-repetition (TR) = 2,560 ms; time-echo (TE) = 30 ms, flip angle = 85°, field-of-view (FOV) = 216 × 216 mm. Anatomical T1-weighted high resolution scan (MEMPRAGE) acquisition parameters: 176 axial slices; 1 × 1 × 1 mm voxels; TE (multiecho): 7.22 ms; TR: 2,530 ms; flip angle = 7°; FOV = 256 mm × 256 mm.

MR data was processed and analyzed with SPM8. EPI volumes for each subject were corrected for slice timing, realigned, coregistered to the structural scan, normalized to Montreal Neurological Institute (MNI) template space, and smoothed (8-mm full-width-half-maximum kernel). Hemodynamic response was modeled at the onset of each condition-block for 24 s, which was the period in which participants viewed the facial expressions. Data was high-pass filtered at 128 s. There were three conditions: (1) positive, (2) negative, and (3) neutral expressions. Movement and other arti-

Table 1
Participant Characteristics and Behavioral Data From Questionnaires, Cognitive Tests, and Acceptance/Rejection Ratings

Behavioral assessment	Low social anhedonia (N = 15) High social anhedonia (N = 15) Between group test[a]
Gender (F/M)	10/5 8/7 $\chi^2 = 0.56, p = .46$
Age	30.27 (10.47), [19–51] 32.00 (12.75), [20–52] $t(28) = 0.41, p = .69$
Education	15.60 (2.67), [12–20] 14.67 (2.23), [10–20] $t(28) = 1.04, p = .31$
Parental socioeconomic status	9.33 (9.91) 8.10 (1.95) $t(28) = 0.47, p = .64$
IQ[b]	114.13 (11.35), [89–133] 116.00 (12.67), [82–132] $t(28) = 0.43, p = .67$
Revised Social Anhedonia Scale	2.67 (2.53), [0–10] 24.60 (5.63), [18–38] $t(28) = 13.77, p < .0001^*$
Perceptual Aberration Scale[c]	0.60 (1.12), [0–4] 1.79 (2.01), [0–6] $t(27) = 1.98, p = .06$
Magical Ideation Scale[c]	1.80 (1.61), [0–5] 4.21 (3.45), [1–12] $t(27) = 2.44, p = .02^*$
Referential Thinking Scale[c]	0.40 (.91), [0–3] 4.21 (5.49), [0–17] $t(27) = 2.65, p = .01^*$
Color Stroop (incongruent-congruent reaction time)	84 (22.7) [59–134] 81.9 (21.4) [37–104] $t(28) = .26, p = .78$
Letter-number sequencing	10.73 (2.5) [5–14] 11 (2.7) [6–17] $t(28) = .22, p = .72$
Digit span	12.06 (2.8) [8–18] 12.0 (2.3) [9–16] $t(28) = .06, p = .95$
GF: Role functioning[c]	8.93 (.96) [6–10] 7.93 (1.21) [6–10] $t(27) = 2.49, p = .02$
GF: Social functioning[c]	9.40 (.74) [8–10] 6.79 (1.63) [3–9] $t(27) = 5.64, p < .0001^*$
TEPS anticipatory	46.00 (4.90), [37–54] 36.86 (5.68), [29–44] $t(28) = 4.65, p < .001^*$
TEPS consummatory	36.27 (5.47), [20–41] 32.86 (7.76), [15–42] $t(28) = 1.38, p = .18$
fMRI task ratings (scale: 1 = *very rejected*; 5 = *very accepted*)	
Positive expressions	4.71 (.42) [3.42–5] 4.46 (.44) [3.17–5] $t(29) = 1.5, p = .14$
Negative expressions	1.36 (.35) [1–2.11] 1.56 (.49) [1–2.83] $t(29) = 1.26, p = .22$
Neutral expressions	3.05 (.39) [2.25–3.56]

(*table continues*)

Table 1 (*continued*)

Behavioral assessment	Low social anhedonia (N = 15) / High social anhedonia (N = 15) / Between group test[a]
	2.89 (.28) [2.08–3.17] $t(29) = 1.3, p = .21$

Note. TEPS = Temporal Experiences of Pleasure Scale. Data is used for descriptive purposes and/or supporting analyses. Data shown is mean (*SD*) [Range].
[a] Results are not corrected for multiple comparisons. [b] Full-scale IQ calculated from Wechsler Abbreviated Scale of Intelligence (WASI) Matrix Reasoning and Vocabulary subscales. [c] One high SA participant did not complete these measures.
* $p < .05$ (two-tailed test).

facts were controlled for by regressing out movement over time (SPM movement parameters) and single volumes that differed ±4 *SD* in signal intensity from the global mean or differed from the previous volume by >3 mm movement (x, y, z planes) or 0.02 degrees (pitch, roll, yaw; identified with Artifact Detection Tool [ART], Gabrieli-Whitfield; http://www.nitrc.org/projects/artifact_detect/). Movement and other artifacts were minimal; ART removed <5% of epi volumes per participant (<1% for most participants). There were no group differences in movement (in any direction) or the number of volumes removed by ART.

Between-groups differences were investigated with flexible factorial analysis of variance (ANOVA) models in SPM8. Each model had three factors: subject, group (low SA/high SA), and condition (e.g., positive/neutral). Group × Condition interaction effects were investigated with three models: (1) positive versus neutral; (2) negative versus neutral; and (3) positive versus negative. In each model, contrast files representing neural activity for each condition relative to the 'rest-period' baseline was entered for each subject. Expected task-related activity was verified in each group using one sample T tests of the main contrasts (results in Supplemental Tables 1–2).

Statistical threshold was set at $p < .001$ (uncorrected for multiple comparisons) with cluster extent of 5 voxels/135 mm. Regions showing a significant Group × Condition interaction are listed in Table 2. Neural activity (i.e., percent signal change) from the peak voxel of the significant cluster is plotted in Figures 1 and 2; these barplots show average neural activity for each group and each condition. Each participant's level of neural activity in the peak voxel was extracted and the difference between conditions calculated (e.g., Positive – Neutral); this relative activity for positive-neutral was used as a predictor in the mixed model analyses with the daily diary ratings.

Daily-Diary Questionnaire

Following the scan, participants completed an online daily diary questionnaire each evening (between 5 p.m. and 3 a.m.) for 21 days. Questions (Table 3) assessed characteristics relevant to schizophrenia-spectrum disorders, including cognition, paranoia, odd perceptual experiences, negative affect, vigor/positive affect, and motivation/productivity. (Disorganized symptoms are difficult to assess with self-report measures so were not included). Vigor/positive affect included the energy associated with positive affect and served as an assessment of the negative symptom psychomotor retardation. Motivation/productivity assessed daily productivity (the behavioral output of motivation) and served as an assessment of the negative symptom amotivation. Participants rated their experience/symptom-level on a 1–5 scale (1 = *not at all*; 5 = *extremely*), reported occurrence of interpersonal conflict (yes/no), and degree of distress (1–5) the conflict caused. Like previous research (Myin-Germeys, Birchwood, & Kwapil, 2011; Myin-Germeys & van Os, 2007), ratings of conflict distress were used as

Table 2
Brain Regions Showing a Significant Group × Condition Interaction Effect

Anatomical region	R/L	BA	Volume in voxels/mm^3	Coordinates x, y, z	t[a]
Group × Condition interaction in expected direction: low SA > high SA					
Positive > Neutral					
Insula–posterior	R	48	79/2133	45, −4, 10	4.28
Transverse temporal/Superior temporal gyrus	L	48	10/270	−45, −10, 1	4.03
Superior frontal gyrus/Middle frontal sulcus	L	47/11	10/270	−21, 50, −2	3.92
Inferior frontal gyrus–triangularis[β]	L	45	8/216	−33, 41, 13	3.86
→ventral lateral prefrontal cortex (VLPFC)					
Insula–anterior	R	48	5/135	39, 14, −8	3.72
Insula–posterior	L	48	12/324	−48, −1, −2	3.64
Negative > Neutral					
Middle frontal gyrus[c]	R	46	1/27	27, 47, 22	3.56
Positive > Negative					
Insula/Superior temporal gyrus	R	48	24/648	48, −7, 4	3.78
Middle cingulate cortex	L	23	8/216	−3, −7, 37	3.72
Anterior cingulate cortex	L	32	5/135	−6, 38, 7	3.58
→ ventromedial prefrontal cortex (VMPFC)					
Group × Condition interaction in unexpected direction: high SA > low SA					
No significant findings					

[a] Statistical threshold is, $t(28) = 3.41$, $p < .001$ (uncorrected for multiple comparisons, Cluster extent 5 voxels/135 mm). [b] Ventral lateral prefrontal cortex (VLPFC) activity to positive > neutral expressions was used in main analyses with daily-diary. [c] Does not meet cluster threshold.

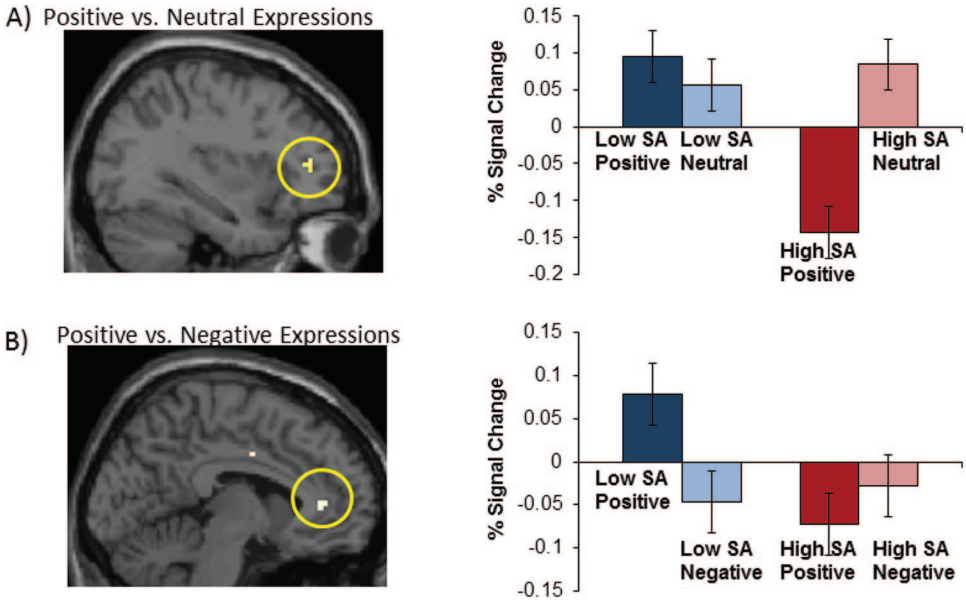

Figure 1. (A) The Group × Condition analysis of variance (ANOVA) for positive and neutral expressions shows a significant interaction in the expected direction (Low SA [Positive – Neutral] − High SA [Positive – Neutral]) in the ventral lateral prefrontal cortex (VLPFC), specifically the left inferior frontal gyrus triangularis (BA 45). Neural activity from this region is plotted for each group and condition and shows that people with high SA deactivate the inferior frontal gyrus in response to positive social cues. (B) The Group × Condition ANOVA for positive and negative expressions shows a significant interaction in the expected direction (Low SA [Positive – Negative] − High SA [Positive – Negative]) in the medial prefrontal cortex, specifically the rostral portion of the anterior cingulate cortex. Neural activity from this region is plotted for each group and condition and indicates that people with low social anhedonia have greater activity in this region for positive versus negative social signals, whereas people with high social anhedonia show a trend in the opposite direction.

the dependent measure (range in mixed models: 0–5; 0 = *no conflict*). Daily social contact (*yes*/*no*) was reported and used to validate expected SA-related behavior in the sample.

To ensure data quality and participant compliance, research staff monitored daily diary progress each morning, and sent a reminder e-mail if an entry was missed. Participants were excluded if they missed more than 6 days (i.e., 15 dairy-days was the minimum).

Analysis of fMRI and Daily-Diary

Because the data are hierarchically organized (i.e., 21 days are nested within-participant) and include relationships between within-person and between-person variables, we used the mixed procedure in SAS, which is based on a hierarchical linear modeling (HLM) approach and permits simultaneous analysis of within- and between-person variation (Kenny, Kashy, & Cook, 2006).

Lower-level (*within-person*) analyses modeled as random effects generated independent estimates of each participant's average level of a diary variable (e.g., average paranoia across 21 days) and the relationship among diary variables (e.g., relationship between conflict distress and paranoia for that person). Then higher-level (*between-person*) analyses examined whether these within-person processes were a function of between-subjects variables, such as SA and/or LPFC activation (e.g., whether the relationship between conflict distress and paranoia differed as a function of SA and LPFC activity). All variables are continuous and grand-mean centered. Simple slopes analyses for high and low groups for each variable (e.g., high/low SA; high/low LPFC) were tested at 1 *SD* above and below each centered mean (Aiken & West, 1991).

Test–retest reliability (i.e., stability of diary ratings) was examined by correlating average daily diary ratings for the first and second halves of the daily diary period. Construct validity was examined by correlating average daily diary ratings of schizophrenia spectrum symptoms with standard measures of trait schizotypy and trait affect. Standardized alpha coefficients are provided for internal consistency.

Multiple Test Correction

To reduce the possibility of false positive findings (i.e., Type I error), the adaptive False Discovery Rate (FDR) procedure (Benjamini & Hochberg, 2000) was implemented in SAS to correct for the number of tests conducted on each predictor of daily diary schizophrenia-spectrum symptoms. Four predictors were examined. Specifically, analyses tested whether the six daily diary symptoms were predicted by: (1) SA, (2) LPFC, and (3) the interaction of SA × LPFC. Raw (unadjusted) p values are reported for each predictor (Tables 3 and 4). These six p values (corresponding to the six daily diary symptoms for each predictor) were entered in the adaptive FDR procedure to verify that results remained significant ($p < .05$, two-tailed) after correcting for six tests. The influence of predictor (4) the interaction of SA × LPFC × Conflict distress was only examined on symptoms that were significantly predicted by the interaction of SA × LPFC.

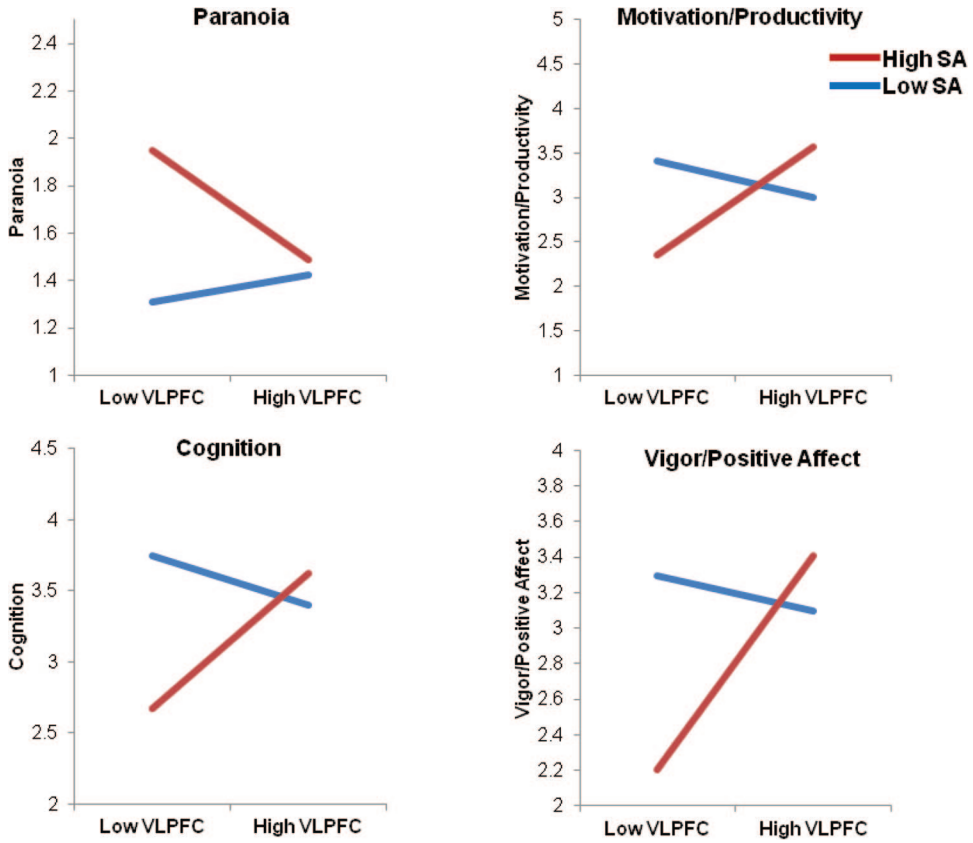

Figure 2. Daily diary ratings of schizophrenia-spectrum symptoms are predicted by the interaction of social anhedonia (SA) × ventral lateral prefrontal cortex (VLPFC) activity to positive > neutral expressions. The average daily rating for each symptom is plotted on the *y*-axis. VLPFC activity is on the *x*-axis. High SA participants are shown with a red line and low SA participants are shown with a blue line. In each case, participants with higher SA and lower VLPFC have worse schizophrenia-spectrum symptoms.

Four symptoms were significant, so results of the 3-way interaction (SA × LPFC × Conflict distress) corrected for four tests. Multiple test correction was not conducted on follow-up simple slopes analyses, which were used to better understand the significant interactions.

Analyses that did not include daily diary ratings are reported as significant at $p < .05$ (two-tailed), and results were not corrected for multiple comparisons. These analyses do not test the main hypotheses, but instead provide supporting information, such as characteristics of the sample (e.g., trait schizotypy questionnaires) and reliability/validity analyses to examine data quality.

Results

Behavior

Table 1 shows participant characteristics and behavioral results. There were no differences between high SA and low SA groups on demographic characteristics or quantitative measures of cognitive-control. There were also no group differences in accept/reject ratings in the fMRI task. High SA participants reported less anticipatory pleasure than low SA but similar consummatory pleasure. As expected, high SA also had more schizotypal traits and worse social and role functioning.

fMRI Between-Group Differences: Low SA Versus High SA

Positive versus neutral. fMRI analyses investigated the hypothesis that high SA participants have deficient neural response to positive expressions. Specifically, we expected that low SA (vs. high SA) would have greater LPFC activity for positive versus neutral expressions.

Group × Condition ANOVA results show the predicted interaction in left ventral LPFC (VLPFC), specifically inferior frontal gyrus-triangularis (BA 45). As Figure 2 illustrates, the interaction is characterized by high SA deactivating to positive expressions. Each participant's VLPFC activity for positive minus neutral expressions (i.e., positive > neutral) was used in analyses with behavioral variables and daily diary ratings.

Group × Condition interactions were also observed in posterior insula, superior temporal gyrus, and superior frontal gyrus. No regions showed an interaction in the opposite direction (i.e., high > low SA; positive > neutral).

Table 3
Descriptive Information Regarding Daily-Diary Questions and Average Response Across the 21 Diary-Days

	Diary questions (rating scale: 1 = *not at all*; 5 = *extremely*)	All participants	High social anhedonia (SA)	Low SA
Schizophrenia-spectrum symptom				
Cognition	My memory was good today; My concentration was good today; I was able to stay focused when I wanted to (α .78)	3.20 (0.66) [1.73–4.27]	2.87 (0.64) [1.73–3.95]	3.52 (0.53) [2.83–4.27]
Paranoia	I had a sense that people were looking at me oddly because of my appearance or something I did; I felt that others dislike me; I felt trusting (reverse coded); I felt that I had to be "on guard" even with my friends (α .65)	1.62 (0.32) [1.01–2.66]	1.84 (0.27) [1.55–2.67]	1.39 (0.16) [1.01–1.57]
Odd perceptual experiences	I heard voices or whispers that didn't seem to be coming from anywhere identifiable; I had a strange or otherworldly feeling in my body (e.g., feelings of déjà vu); I saw a "vision" or hallucination even though I was not taking drugs (α .35)	1.04 (0.09) [1.0–1.38]	1.07 (0.12) [1.0–1.38]	1.002 (0.01) [1.0–1.03]
Motivation/productivity	I was productive today	2.89 (0.67) [1.67–4.07]	2.61 (0.64) [1.67–3.91]	3.16 (0.59) [2.24–4.07]
Vigor/positive affect	I felt invigorated; I felt cheerful; I felt lively; I felt happy (α .86)	2.82 (0.98) [1.12–4.38]	2.46 (0.73) [1.12–3.75]	3.19 (0.57) [2.25–4.38]
Negative affect	I felt anxious; I felt sad; I felt discouraged; I felt angry (α .71)	1.31 (0.24) [1–1.87]	1.40 (0.26) [1.05–1.87]	1.23 (0.20) [1–1.63]
Daily events				
Conflict occurrence[a,b] (# of days with conflict)	Did you have a disagreement, irritation, annoyance or other negative encounter with another person today?	4.33 (4.21) [0–16]	4.93 (3.94) [1–16]	3.73 (4.53) [0–15]
Conflict distress (No conflict coded as 0)	If yes, how distressing was this encounter? (rated: 1–5) [Range: lowest - highest rated conflict out of all conflicts]	2.22 (1.08) [1–5]	2.09 (1.03) [1–5]	2.38 (1.13) [1–5]
Social activity[c,d] (% of days socialized)	Did you socialize with another person or group of people today?	.88 (.15) [.38–1.0]	0.81 (0.19) [.38–1.0]	0.96 (.06) [.83–1.0]

Note. Data shown is mean (*SD*) [range].
[a] This question was the section heading and was followed by examples of specific types of conflicts; e.g., I felt someone was hostile toward me (yes/no). [b] There was no difference between groups in number of days with conflict, $t(28) = .78$, $p = .45$, or percentage of days with conflict, $t(28) = .74$, $p = .46$. [c] High SA participants socialized significantly less than low SA participants, $t(28) = 3.15$, $p = .004$. [d] "Socialize" was defined as interacting with others for purely social reasons. The main question (Did you socialize ..?) was followed by examples of social interactions (e.g. "I went to a party and socialized"). The construct was coded 1 if they had any social interaction that day and 0 if they had no social interaction.

Negative versus neutral. Group × Condition ANOVA results (low > high SA for negative > neutral) revealed one voxel in right LPFC. This does not meet cluster threshold; thus it is not significant. However, it suggests that LPFC deficits are not specific for positive expressions. No regions showed the opposite interaction.

Positive versus negative. Group × Condition ANOVA results for positive versus negative expressions (i.e., low > high SA for positive > negative) revealed significant interactions in rostral anterior cingulate cortex (i.e., VMPFC), middle cingulate cortex, and posterior insula. No regions showed the opposite interaction.

Correlation Between VLPFC Activity and Behavioral Variables

To better understand the function of observed VLPFC activity to positive > neutral expressions, we examined the correlations between VLPFC activity and relevant behavioral variables. There were no significant correlations between VLPFC activity to positive > neutral expressions and standard cognitive-control tests, including Stroop, $r(28) = -0.06$, digit span, $r(28) = -0.10$, and letter-number sequencing, $r(28) = 0.13$, and no relationship with IQ, $r(28) = 0.03$. VLPFC activity to positive > neutral expres-

Table 4
Results From Preliminary Analyses on Expected Relationships Between Higher Social Anhedonia (SA) and Worse Daily Experience of Schizophrenia-Spectrum Symptoms, as Well as Lower Ventral Lateral Prefrontal Cortex (VLPFC) Activity and Worse Daily Experience of Schizophrenia-Spectrum Symptoms

	SA (df = 28)			VLPFC activity to positive-neutral expressions (df = 28)		
Daily-diary variable	*b (SE)*	*F*	*p*	*b (SE)*	*F*	*p*
Cognition	−0.02 (0.01)	6.43	**.02**	1.17 (0.55)	4.61	**.04**
Paranoia	0.02 (0.004)	24.47	**<.0001**	−0.81 (0.24)	11.05	**.002**
Odd experiences	0.003 (0.001)	4.29	**.05**	−0.12 (0.08)	2.34	.14
Motivation/productivity	−0.02 (0.01)	3.45	.07	1.18 (0.54)	4.71	**.04**
Vigor/positive affect	−0.03 (0.01)	6.14	**.02**	1.61 (0.60)	7.31	**.01**
Negative affect	0.008 (0.004)	4.54	**.04**	−0.28 (0.22)	1.64	.21

Note. Significant results are shown with p values in bold type. All results remain significant after correcting for the six tests conducted.

sions was significantly related to TEPS anticipatory pleasure, $r(28) = 0.59$, $p < .001$, such that higher VLPFC activity was related to more anticipatory pleasure; there was no relationship between VLPFC activity and consummatory pleasure, $r(28) = 0.24$.

Daily-Diary Ratings of Schizophrenia-Spectrum Symptoms

Data quality and preliminary analyses. Daily diary data was inspected for data quality, including reliability and validity (Supplemental Tables 3 and 4) and expected daily diary characteristics of the sample were verified (Tables 3 and 4). In summary, diary compliance was high; most participants (N = 27/30) completed 20–21 diary-days. Number of diary-days did not differ between high and low SA groups, High SA: X = 20.5 (1.6); Low SA: X = 20.1 (1.6); $t(28) = 0.57$, $p = .57$. High SA reported less social contact than low SA, $t(28) = 3.15$, $p = .004$, confirming expected differences in SA-related behavior. A total of 87% (26 participants) reported at least one conflict, and 50% had 4 or more conflicts, providing adequate data to examine conflict distress. There were no group differences in number of days with conflict, $t(28) = .78$, $p = .45$, or percentage of days with conflict, $t(28) = .74$, $p = .46$. Internal consistency was acceptable for all constructs except odd perceptual experiences ($\alpha = .35$); odd experiences tended to occur within a single sensory domain, so items across domains were not correlated. Test–retest reliability was high (e.g., rs > 0.70), indicating stable estimates for daily diary variables (Supplemental Table 3). Diary ratings correlated with corresponding schizotypal and affective traits (for example, paranoia correlated with SPQ Suspiciousness), indicating high construct validity (Supplemental Table 4). Preliminary HLM analyses confirmed expected behavioral associations between SA and daily experience. Higher SA was related to worse symptom-severity for every schizophrenia-spectrum symptom. VLPFC activity was related to all schizophrenia-spectrum symptoms except negative affect and odd perceptual experiences (Table 4).

Hypothesis testing. To test Hypothesis 2, we used mixed-model analyses to examine whether schizophrenia-spectrum symptoms were predicted by the interaction of SA and VLPFC activity to positive > neutral expressions. Results showed that the interaction of SA × VLPFC activity significantly predicted cognition, paranoia, motivation/productivity, and vigor/positive affect. These results remained significant after multiple test correction. As Figure 2 illustrates, in all cases, higher SA and lower VLPFC was related to worse symptoms. The interaction of SA × VLPFC activity did not predict odd perceptual experiences or negative affect (Table 5; Figure 2).

To better understand this finding, follow-up analyses examined the simple slopes of each SA × VLPFC interaction (Aiken & West, 1991). Statistics are reported in Table 6. VLPFC activity was expected to predict schizophrenia-spectrum symptoms for high SA but not low SA. Thus, we tested the effect of VLPFC activity on schizophrenia-spectrum symptoms for high and low SA participants separately. VLPFC activity did not predict schizophrenia-spectrum symptom in low SA participants. However, VLPFC activity was a significant predictor of each symptom in high SA participants. Specifically, among high SA participants, lower VLPFC activity was related to worse cognition, paranoia, motivation/productivity, and vigor/positive affect. Next, we tested the effect of SA on people with high and low VLPFC activity. Among individuals with low VLPFC activity, people with high SA had significantly worse schizophrenia-spectrum symptoms than people with low SA. However, among individuals with high VLPFC activity, people with high and low SA did not differ in their level of schizophrenia-spectrum symptoms. These findings confirm that individuals with higher SA and lower VLPFC activity to positive > neutral expressions experience a greater degree of schizophrenia-spectrum symptoms in their daily lives.

To test Hypothesis 3, we examined whether the four schizophrenia-spectrum symptoms identified in Hypothesis 2 (i.e., cognition, paranoia, motivation/productivity, and vigor/positive affect) were predicted by the interaction of SA, VLPFC activity to positive > neutral expressions, and conflict distress. We expected that among high SA participants, lower VLPFC activity would be related to more severe symptoms on days of highly distressing interpersonal conflict. Results showed that the interaction of SA, VLPFC activity, and conflict distress significantly predicted paranoia (Table 5; Figure 3). This finding remained significant after multiple test correction.

Table 5
Main Results Testing the Hypotheses That Schizophrenia-Spectrum Symptoms Are Predicted by the Interaction of Social Anhedonia (SA) × Ventral Lateral Prefrontal Cortex (VLPFC) Activity (Hypothesis 2) and the Interaction of SA × VLPFC Activity × Conflict Distress (Hypothesis 3)

	SA*VLPFC activity (df = 27)			SA × VLPFC activity × Conflict distress (df = 27)		
Daily-diary variable	*b (SE)*	*F*	*p*	*b (SE)*	*F*	*p*
Cognition	0.13 (0.06)	5.71	**.02**	0.03 (0.02)	1.96	.17
Paranoia	−0.06 (0.02)	8.48	**.007**	−0.02 (0.008)	5.24	**.03**
Odd experiences	−0.01 (0.01)	1.29	.27	—	—	—
Motivation/productivity	0.17 (0.05)	9.80	**.004**	0.05 (0.04)	1.92	.12
Vigor/positive affect	0.14 (0.06)	5.38	**.03**	0.04 (0.03)	1.38	.25
Negative affect	0.01 (0.04)	0.18	.68	—	—	—

Note. Only variables showing a significant relationship with SA × VLPFC activity were examined in the 3-way interaction. Significant results are shown with *p* values in bold type. All results remain significant after correcting for number of tests.

Follow-up tests used simple slopes analyses to examine this 3-way interaction. Statistics for all effects are reported in Supplemental Table 5. Given results (above) that high SA have worse symptoms than low SA, analyses reported here focus on high SA participants. We first examined the effect of VLPFC activity. On days of high conflict distress, VLPFC activity significantly predicted paranoia for high SA participants, such that lower VLPFC activity was related to worse paranoia, $b = -1.62$ (*SE* 0.41), $t(27) = 3.99$, $p = .0004$. On days of low conflict distress, VLPFC activity in high SA participants was only weakly related to paranoia, $b = -0.62$ (*SE* 0.31), $t(27) = 2.00$, $p = .06$. Examination of the effect of conflict distress showed that conflict distress significantly predicted paranoia for participants with high SA and low VLPFC activity, $b = 0.12$ (*SE* 0.02), $t(27) = 5.44$, $p = .0001$; these participants experienced worse paranoia on days of high-conflict distress relative to days of low-conflict distress. However, conflict distress was not significantly related to paranoia for participants with high SA and high VLPFC activity, $b = -0.08$ (*SE* 0.05), $t(27) = 1.74$, $p = .09$. As expected, paranoia in low SA participants was not influenced by VLPFC activity or conflict distress (all *p* values >.15).

Discussion

Using a multimethod approach in a community sample of healthy adults, this study investigated the relationship between neural deficits associated with SA and the daily experience of schizophrenia-spectrum symptoms. Three main findings emerged. First, compared with low SA, high SA participants had less activity to positive (vs. neutral) expressions in the ventral lateral prefrontal cortex (VLPFC; i.e., inferior frontal gyrus-triangularis, BA45). Second, the interaction of SA and this VLPFC activity to positive expressions predicted daily diary ratings of schizophrenia-spectrum symptoms; participants with high SA and low VLPFC activity had worse cognition, paranoia, vigor/positive affect, and motivation/productivity. Third, among high SA participants, VLPFC activity predicted the daily relationship between conflict distress and paranoia. Specifically, high SA participants with low VLPFC activity had worse paranoia on days of high-conflict distress compared with days of low-conflict distress.

These findings reveal a connection between LPFC deficits and SA—two characteristics of schizophrenia that, historically, were thought to arise from different behavioral and neural pathways. The data here indicate that reduced VLPFC engagement when processing positive emotion could be a component of schizophrenia liability that contributes to both SA and other schizophrenia-spectrum symptoms. Furthermore, the observed interaction between SA, VLPFC activity, and conflict distress suggests that high SA individuals with VLPFC deficits in emotion processing are especially susceptible to the negative impact of interpersonal conflict.

Table 6
Results From Follow-Up, Simple Slopes Analyses Examining Schizophrenia–Spectrum Symptoms Predicted by the Interaction of Social Anhedonia (SA) and Ventral Lateral Prefrontal Cortex (VLPFC) Activity to Positive Expressions

	Effect of VLPFC activity for people with high SA			Effect of VLPFC activity for people with low SA			Effect of SA for people with low VLPFC activity			Effect of SA for people with high VLPFC activity		
	b (SE)	*t*	*p*	*b (SE)*	*t*	*p*	*b (SE)*	*t*	*p*	*b (SE)*	*t*	*p*
Cognition	2.26 (0.87)	2.59	**.02**	−0.83 (0.84)	−0.99	0.33	−0.05 (0.02)	−2.99	**.006**	0.009 (0.02)	0.62	0.54
Paranoia	−1.10 (0.32)	3.46	**.002**	0.27 (0.31)	0.89	0.38	0.027 (0.006)	4.90	**<.001**	0.003 (0.006)	0.50	0.62
Motivation/Productivity	2.88 (0.84)	3.45	**.002**	−0.99 (0.81)	1.23	0.23	−0.05 (0.02)	3.07	**.005**	0.02 (0.01)	1.65	0.10
Vigor/Positive Affect	2.87 (0.97)	2.96	**.01**	−0.46 (0.93)	0.49	0.63	−0.05 (0.02)	2.73	**.01**	0.01 (0.02)	0.78	0.45

Note. Significant findings are shown with *p* values in bold type. Degrees of freedom (df) = 27 for all analyses. Multiple comparison correction was not conducted. Simple slope analyses for the interaction of SA, VLPFC activity, and conflict distress is reported in the main text and Supplemental Table 5.

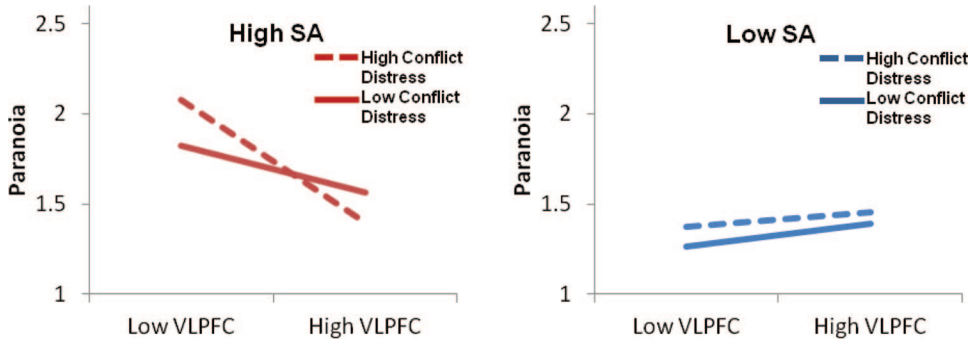

Figure 3. Daily diary ratings of paranoia are plotted as a function of social anhedonia (SA), ventral lateral prefrontal cortex (VLPFC) activity to positive > neutral expressions, and conflict distress. Symptom severity is plotted on the *y*-axis. VLPFC activity is on the *x*-axis. High SA participants are shown in red and low SA in blue. Days of high conflict distress are shown with a dashed line and low conflict distress with a solid line. As the graph shows, paranoia severity in high SA participants was influenced by both VLPFC activity and conflict distress, such that high SA participants with low VLPFC activity have the worst paranoia on days of high conflict distress. Paranoia severity in low SA participants was not influenced by VLPFC activity or conflict distress.

Deficits in LPFC-dependent cognitive skills are a central feature of schizophrenia-spectrum populations. However, most evidence of LPFC deficits in high SA is limited to behavioral studies. Daily diary ratings not only confirm the subjective experience of worse cognition in high SA but also demonstrate that individuals with higher SA and lower VLPFC activity experience the worst day-to-day cognitive function. The findings suggest that reduced VLPFC activity to positive expressions reflects a deficit engaging VLPFC-dependent cognitive functions to process positive social signals.

Measuring VLPFC response in a relatively unstructured task probably enhanced ability to detect the relationship between VLPFC activity and daily cognition. Participants were not instructed to regulate emotional response; instead, we measured spontaneous neural activity when viewing positive, negative, and neutral expressions with the idea that participants' natural tendency to engage VLPFC-dependent cognitive functions during the task would best predict the application of those cognitive functions in daily life. The current findings are consistent with our previous study which showed a correlation between spontaneous VLPFC activity to a partner's positive expressions and self-reported attentional control (Hooker et al., 2010). Thus, while the lack of instruction to regulate leaves ambiguity about the psychological process engaged in the task, it may have allowed relevant individual differences to emerge. Surprisingly, high SA participants did not have worse performance on standard cognitive-control tests and there was no correlation between VLPFC activity and cognitive-control performance. This suggests that VLPFC deficits associated with high SA might be most apparent in unstructured environments when control-related mechanisms have to be both initiated and applied.

High SA and low VLPFC activity to positive expressions was related to less vigor/positive affect—a construct which includes the psychomotor activation and arousal associated with positive affect. These results are consistent with evidence that VLPFC regulates positive emotion. Among couples, VLPFC activity to a partner's positive expression predicted up-regulation of positive mood after conflict (Hooker et al., 2010). In people with depression, increased VLPFC activity to positive stimuli after SSRI treatment and after neurofeedback predicted increased positive affect (Johnston et al., 2011; Light et al., 2011; Linden et al., 2012). It is interesting that neurofeedback participants reported using memories of positive experiences to improve their mood (Johnston et al., 2011).

Internal representations of positive emotion, such as memories of rewarding experiences, motivate goal-directed behavior (Barch & Dowd, 2010; Miller & Cohen, 2001; Wallis & Miller, 2003). Our findings regarding daily productivity suggest that reduced VLPFC activity associated with SA creates weak reward-representations, which then compromises goal-directed behavior. High SA individuals reported less daily productivity—a finding similar to another experience sampling study demonstrating that healthy adults with higher schizotypy were more likely to be "doing nothing" at various times throughout the day (Husky, Grondin, & Swendsen, 2004). This lack of goal-directed behavior may be best explained by the interaction of schizotypal traits and LPFC function. We found that participants with high SA and low VLPFC activity to positive expressions reported the worst daily productivity. This suggests that VLPFC deficits when creating reward-representations may, ultimately, lead to lower motivation to accomplish daily tasks. Behavioral data from the fMRI task and the Temporal Experience of Pleasure Scale, a trait measure of anticipatory and consummatory pleasure, support this interpretation. High SA participants reported low anticipatory pleasure, and across both groups, individuals with lower VLPFC activity when viewing positive expressions reported less anticipatory pleasure; that is, they were less likely to get excited about future events. At the same time, there were no group differences in consummatory pleasure or acceptance ratings during the fMRI task, and VLPFC activity was not correlated with either variable. This suggests that VLPFC activity during positive events helps create a neural representation of the experience that is later retrieved to motivate behavior. Together these findings illustrate a relationship between anhedonia, LPFC function in reward-processing, and goal-directed behavior. Despite theoretical proposals linking these behavioral and neural processes (Barch & Dowd, 2010; Gold et al., 2008),

concrete data are minimal. The results here provide evidence for a possible mechanism underlying the negative symptoms (including anhedonia, amotivation, and asociality) that contribute to functional disability in schizophrenia-spectrum populations.

As expected, higher SA was related to higher levels of paranoia and odd perceptual experiences. These data confirm prior findings that, even though SA is considered a negative symptom of schizophrenia, healthy high SA individuals—identified by abnormal scores on this single dimension—have higher than expected levels of positive symptoms (Blanchard et al., 2011). This co-occurrence of symptoms provides evidence that high SA is associated with schizophrenia liability (Schürhoff et al., 2003).

We provide additional evidence of schizophrenia liability by showing that among high SA individuals those with a second psychosis-risk factor—low VLPFC function—have the most severe paranoia. Mechanisms by which VLPFC function contribute to paranoia are not well understood. However, VLPFC controls the influence of emotion on social judgment, including evaluations of trustworthiness (Beer, Knight, & D'Esposito, 2006; Hooker & Knight, 2006). VLPFC deficits could contribute to paranoia through the exaggerated influence of negative affect or negative social environments on interpersonal judgment (Hooker et al., 2011).

Negative social environments, especially interpersonal conflict, are associated with more severe paranoia in community-based high SA individuals (Blanchard et al., 2011) and the exacerbation of paranoia in schizophrenia and other high-risk populations (Hooley, 2007). We found that, among high SA participants, lower VLPFC activity was related to worse paranoia on days of high conflict distress. This provides initial evidence that psychosis-risk populations with LPFC deficits are susceptible to an exacerbation of paranoia after interpersonal conflict (Hooley, 2007). However, because participants completed the daily diary each evening about events that day, causal direction cannot be determined. One interpretation, consistent with prior research (Hooker et al., 2010), is that participants with high SA and low VLPFC activity experienced an increase in paranoia *after* distressing conflicts. Alternatively, on days when paranoia is high, participants with low VLPFC activity may have more severe conflicts and/or experience conflicts as more distressing.

Collecting daily diary reports multiple times a day might help identify causal influences. Studies of schizophrenia-spectrum populations that collect diary-data 6–10 times/day demonstrate that paranoia increases after social and nonsocial stressors (Myin-Germeys et al., 2011; Myin-Germeys & van Os, 2007) and increases more after social interactions with unfamiliar people than familiar people (Collip et al., 2011; Verdoux, Husky, Tournier, Sorbara, & Swendsen, 2003).

Limitations of the psychometric high-risk approach may have also suppressed the influence of conflict distress and VLPFC activity on other symptoms. Participants were healthy, free of psychological disturbance, and included ages beyond the risk-period for psychosis. Consequently, their schizophrenia-spectrum symptoms were relatively mild and stable, making it difficult to detect symptom increases associated with conflict. Ratings of odd perceptual experiences were especially low which could explain the nonsignificant relationship with VLPFC activity. In addition, while the psychometric high-risk approach minimizes confounding factors associated with illness, the findings are not immediately applicable to clinical populations. Characteristics of schizophrenia disorder may cause different dynamics between SA, VLPFC, and conflict. Moreover, the results here may not be specific to schizophrenia. SA and the schizophrenia-spectrum symptoms we measured are associated with several psychological disorders. A similar issue is that high SA participants were elevated on several other trait measures, so even though SA was the independent variable that differentiated the two groups, the results here do not demonstrate the absence of a relationship between VLPFC and other traits. Finally, while our multimethod approach provides the benefit of a detailed picture of brain-behavior relationships, the number of analyses conducted on a relatively small sample is a limitation.

Nonetheless, results here provide an initial model for understanding the relationship between SA, LPFC, and schizophrenia-spectrum symptoms. The findings fit with current neurodevelopmental theories that the biologically based vulnerability of schizophrenia manifests as relatively stable behavioral deficits in cognition, hedonic capacity, and social functioning (Cornblatt et al., 2003; Stone, Faraone, Seidman, Olson, & Tsuang, 2005; Stone et al., 2012). Our results suggest that VLPFC dysfunction could be a core vulnerability that contributes to all three of these deficits and that understanding the interaction of VLPFC function, hedonic capacity and social interactions might facilitate early identification of psychosis-risk and treatment development in schizophrenia-spectrum populations.

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