Impaired executive control of emotional information in social anhedonia

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A B S T R A C T

We examined the executive control of emotional information and its relationship to social functioning in individuals at risk for schizophrenia, defined by high social anhedonia (SA). Using the same structure as the Attentional Network Test (ANT), we developed a measure of executive control of emotional information (ANT-Emotion) in which subjects identify the direction of an arrow flanked by irrelevant angry or neutral faces. Subjects completed the ANT, ANT-Emotion, and the Social Adjustment Scale, Self-Report (SAS-SR), a measure of social functioning. While there were no group differences in the alerting, orienting, and executive control networks assessed by the ANT, high SA individuals exhibited a specific impairment in the executive control of emotional information. High SA individuals also reported poorer social functioning. However, executive control of emotional information did not mediate the relationship between SA and social functioning. These findings indicate that, in high-risk populations, the impaired ability to inhibit emotional information allows negative affective stimuli to exert inappropriate influence on cognitive processes. These results are consistent with studies indicating similar findings in schizophrenia patients, suggesting that impaired inhibition of negative emotion may be part of the liability for the disorder.

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1. Introduction

Social anhedonia (SA), a lack of pleasure from social interactions that is distinguishable from social anxiety (Brown et al., 2007), is thought to be a core feature of schizophrenia that contributes to the liability of developing the disorder, and a key factor underlying the social difficulties seen in the illness (Meehl, 1962). SA predicts social functioning in individuals with schizophrenia (Blanchard et al., 1998) and high-risk populations (Cohen et al., 2006), is associated with elevated symptoms (Horan et al., 2007), and predicts conversion to schizophrenia-spectrum disorders (Kwapil, 1998). Similar to schizophrenia patients, socially anhedonic individuals also exhibit deficits in attention (Gooding et al., 2006b) and emotion processing (Phillips and Seidman, 2008). In line with data demonstrating social cognitive processes act as mediators between cognitive impairments and functional outcome (Green et al., 2000) the present research examines attentional processes in relation to emotional information in individuals high in social anhedonia. We hypothesize that attentional deficits affect social functioning via a specific domain of social cognition, the executive control of socially relevant emotional information, and that this relationship is part of an underlying vulnerability observable in psychosis-prone populations.

1.1. Attention & the attention network test

Attentional deficits in schizophrenia and psychosis-prone populations are well documented (Heinrichs and Zakzanis, 1998; Nuechterlein et al., 1998) but the mechanisms by which attentional processes influence social functioning are unknown. To understand attentional mechanisms and how they contribute to social impairments in SA a measure that can dissociate between specific domains of attention is needed. A promising candidate is the Attention Network Test (ANT) (Fan et al., 2002), an experimental measure of three proposed attention networks: alerting, orienting and executive control (Posner and Petersen, 1990). The alerting network manages the ability to achieve and maintain an alert state, the orienting network manages the ability to select and focus on the to-be-attended stimulus, and the executive control network manages the ability to resolve conflict among responses and consequently the regulation of cognitions and emotions (Posner et al., 2002).

There is increasing evidence for a specific deficit in the executive control network, as measured by the ANT, in schizophrenia (Wang et al., 2005; Gooding et al., 2006a; Urbanek et al., 2009). However, it is unclear whether this deficit is a consequence of schizophrenia, or if it is part of the liability for the disorder. Although executive control has been examined using the ANT in positive schizotypy (Wan et al., 2006), and individuals high in physical anhedonia (Dubal et al., 2000), to our knowledge ANT performance has yet to be examined in SA. Moreover, little is known about the contribution of deficits in the executive control network to social impairments in SA as executive control mechanisms have not yet been directly tied to social cognition and functioning deficits. We sought to examine 1) whether

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executive control deficits are present in high SA individuals, and 2) the role of the executive control network in a specific social cognitive process, the executive control of emotional information.

1.2. Executive control of emotional information

Executive control of emotional information – operationalized here as the ability to control the extent that emotion influences cognition – is critical for successful decision making and social interactions; if an individual is unable to inhibit irrelevant emotional information in the social array this could adversely affect social interactions. In schizophrenia, neuroimaging studies show dysfunctional activity in the lateral prefrontal cortex (Barch, 2005), a region involved in the regulation of emotion on behavior (Ochsner and Gross, 2005). In behavioral studies, individuals with schizophrenia show increased latencies on the Stroop task for threat-related or paranoid words compared to depressed individuals and healthy controls (Bentall and Kaney, 1989), and are more influenced by negative affective primes when rating the trustworthiness of neutral faces (Hooker et al., 2011). Collectively, these findings are indicative of deficits in the executive control of emotional information in schizophrenia, and that these deficits directly affect social behavior, potentially contributing to social impairments. Thus, we were interested in investigating whether psychosis-prone individuals also exhibit impairments in the executive control of emotional information, and whether these impairments relate to social functioning.

High SA is associated with executive functioning impairments (Tallent and Gooding, 1999) and aberrant processing of affective information (Kerns and Berenbaum, 2000), indicating that dysregulation of inhibitory mechanisms, such as executive control, and emotion processing is present premorbidly. However, the interplay between affective information and executive control in SA has received limited attention in the literature. If high SA is related to difficulties in controlling the influence of emotional information on behavior, it may contribute to social deficits. Thus, we were interested in whether executive control of emotional information is impaired in socially anhedonic individuals, and whether this mediates the relationship between SA and social functioning.

1.3. The present research

The present research examined attentional processes, specifically the executive control of emotional information, and social functioning in high SA individuals and healthy controls. We developed a direct measure based on the ANT: the ANT-Emotion, a flanker task with orienting cues in which participants identify the direction of an arrow flanked by irrelevant neutral or angry faces. SA was assessed with the Revised Social Anhedonia Scale (Eckblad et al., 1982). We also administered the ANT as a measure of pure attentional processes, and the Social Adjustment Scale–Self Report (Weissman et al., 1978) as a measure of social functioning.

We directly tested the following predictions: 1) Compared to controls, high SA individuals will exhibit attentional deficits on both the ANT, specifically in executive control, and the ANT-Emotion, specifically in the executive control of emotional information. 2) High SA individuals will report poorer social functioning compared to controls. 3) Executive control of emotional information will mediate the relationship between SA and social functioning.

2. Methods

2.1. Participants

34 high SA and 29 control participants were recruited from the Greater Boston Area. Participants were recruited as part of a larger study ($N = 108$) investigating the relationship between psychosis-proneness, social cognition, and social functioning in which they were screened for SA with the Revised Social Anhedonia Scale (RSAS) (Eckblad et al., 1982), and also completed the Schizotypal Personality Questionnaire (SPQ) (Raine, 1991). Candidate high SA participants were selected from the larger study sample using the cut-off of 1.96 standard deviations above

the mean for their gender from normative data reported by Kwapil (1998): scores of 16 and above for females; 20 and above for males. In order to compare high SA individuals to individuals who are not at risk for schizophrenia, candidate control participants were individuals who scored below 1.96 standard deviations from the mean on the RSAS and within the 10% cutoffs of the SPQ from the normative data reported by Raine (1991) (SPQ scores less than 8 or greater than 42). Thus, control participants were individuals with no elevated risk for schizophrenia, scoring within the normal range of the SPQ and the RSAS. Exclusion criteria were: English as a second language, IQ below 70, history of head trauma, neurological illness, current or past axis I disorders, and active substance abuse within the past six months.

Demographic information and group differences are presented in Table 1. The study was approved by the ethical review board at Harvard University. After study procedures were explained, participants gave written informed consent.

2.2. Materials & assessments

2.2.1. Social anhedonia

The Revised Social Anhedonia Scale (RSAS) (Eckblad et al., 1982) is a 40 item true/false self-report scale comprising of questions measuring individuals' decreased pleasure from social interactions. Example items include: "I attach very little importance to having close friends" (keyed true) and "Just being with friends can make me feel really good" (keyed false).

2.2.2. Psychiatric illness

Axis I disorders were assessed using the Structured Clinical Interview for DSM-IV Axis I Disorders (First et al., 2002); clinical interviews were conducted by trained master's level doctoral students and supervised by a licensed clinical psychologist (CH). Reliability assessments conducted by an independent clinician on a random sample of ten clinical interviews revealed a kappa of 0.67, indicating substantial diagnostic agreement (Landis and Koch, 1977).

2.2.3. Intelligence

Full scale IQ scores were estimated using the vocabulary and matrix reasoning subtests of the Weschler Abbreviated Intelligence Scale (WASI) (Wechsler, 1999).

2.2.4. Social functioning

Social functioning was assessed with the Social Adjustment Scale – Self Report (SAS-SR) (Weissman et al., 1978). The SAS-SR consists of 54 questions assessing six areas of functioning: work, social and leisure activities, relationships with extended family, role as marital partner, parental role, and role within the family unit. Each area is rated across four categories of assessment: performance at expected tasks, level of conflict with people, interpersonal relations, and feelings and satisfactions. Area scores are averaged together to create one composite score of social functioning.

2.3. Tasks and stimuli

2.3.1. The ANT

The ANT (Fan et al., 2002) assesses the efficiency of the orienting, alerting, and executive control attentional networks by measuring differences in reaction times to indicate the direction of a central arrow across cue and flanker conditions. Participants identify the direction (left, right) of a target arrow that appears above or below a central fixation cross. The arrow is preceded by an asterisk cue that either alerts or orients participants to the upcoming target. There are four cue types: no cue (neither alerting nor orienting), central cue (on the fixation cross; alerting), double cue (two asterisks presented simultaneously above and below fixation; alerting), or spatial cue (a single asterisk above or below fixation in the location of the upcoming target; orienting). Immediately after the asterisk cue, the target arrow appears and is flanked by one of three flanker types: congruent (arrows in the same direction as the target arrow), incongruent (arrows in the opposite direction as the target arrow) and neutral (horizontal lines). The alerting and orienting networks are assessed via the different cue conditions, the executive control network is assessed via the different flanker conditions. Measures of the efficiency of the three attentional networks are obtained via simple subtractions of reaction times between conditions: alerting scores are calculated by subtracting reaction times in the double cue condition from reaction times in the no cue condition; orienting scores are calculated by subtracting reaction times in the spatial cue condition from reaction times in the center cue condition; executive control scores are calculated by subtracting reaction times in the congruent flanker condition from reaction times in the incongruent flanker condition.

The task consists of 288 trials; 72 in each of the four cue conditions. Each trial is 4000 ms beginning with a fixation cross, followed by one of the four cue types presented for 100 ms, followed by an average interval of 400 ms after which one of the three target stimuli is presented for 1700 ms or until the subject responds (congruent, incongruent, neutral). The task was presented on an IBM ThinkPad laptop using e-prime professional version 2.0.

2.3.2. The ANT-Emotion

Using the same structure as the ANT, the ANT-Emotion is designed to assess the orienting and executive control attentional networks in relation to emotional information. Orienting to emotional information is assessed by using faces (neutral, angry) as spatial cues for the upcoming target. Executive control of emotional information is assessed by using faces (neutral, angry) as flankers of the central target arrow. Participants

Table 1
Demographics and sample characteristics.

	Control group	High SA group	Differences between groups
N	29	34	
Gender (F/M)	20 F/9 M	19 F/15 M	χ^2 (1) = 1.136, P = 0.287
Age: mean (S.D.), [range]	28.76 (13.81),[18–65]	30.47 (14.04),[19–64]	t(61) = 0.486, P = 0.629
Education: mean (S.D.), [range]	14.86 (2.01), [12–20]	14.53 (2.16), [10–20]	t(60) = 0.612, P = 0.543
WASI IQ: mean (S.D.), [range]	119.52 (10.77), [92–137]	111.76 (11.89), [82–132]	t(61) = 2.692, P = 0.009, d = 0.69 [a]
Social anhedonia: mean (S.D.), [range]	6.21 (3.82), [0–14]	26.85 (6.41), [18–40]	t(61) = 15.184, P < 0.0001, d = 3.89
SPQ: mean (S.D.), [range]	19.97 (9.56), [9–40]	41.06 (15.47), [11 – 70]	t(61) = 6.372, P < 0.0001, d = 1.63
Social impairment: mean (S.D.), [range]	56.30 (11.67), [36–84]	70.68 (14.19), [39–109]	t(59) = 4.245, P < 0.0001, d = 1.11 [b]

[a] Cohen's d effect size.
[b] Two subjects did not complete the Social Adjustment Scale.

indicate the direction of a central arrow flanked by one of three flanker types: irrelevant angry faces, irrelevant neutral faces, or neutral lines. The flanker conditions are preceded by one of three cue types (asterisk, neutral face, angry face) presented in one of two cue positions (central, spatial – a single cue above or below fixation orienting participants to the location of the upcoming target). Face cues never precede face flankers: in face flanker conditions the cue presented is always an asterisk; in neutral line flanker conditions the cue presented is either an angry or neutral face (see Fig. 1a and b for task illustration).

Measures of the efficiency of each network are obtained through simple subtractions of reaction times between conditions. Executive control of emotional information scores are calculated by subtracting reaction times in the neutral face flanker condition from reaction times in the angry face flanker condition. Orienting to emotional information scores are calculated by subtracting reaction times in the neutral face spatial cue condition from the angry face spatial cue condition. Orienting to emotional information was included in the task in order to dissociate the attentional capture of emotional information from the executive control of emotional information. If negative emotional faces are simply more salient for high SA participants we would expect faster orienting to emotional information compared to controls; if, as predicted, high SA participants have difficulty inhibiting negative emotional faces we would expect impaired executive control of emotional information.

The face stimuli are colored photographs of unfamiliar male and female faces taken from the NIMSTIM face set (Tottenham et al., 2009); 24 faces (12 female) were selected. In an effort to reflect the race and ethnicity demographics of the Greater Boston Area 50% are Caucasian, 25% are Asian/other, and 25% are African American (retrieved August 13, 2009 from http://www.helloboston.com/Census.Cfm). We chose to use open-mouth angry face stimuli as they are more reliably identified as angry than the closed-mouth angry stimuli (Tottenham et al., 2009). Similarly, we chose to use angry faces because they are more reliably identified correctly than fear faces (Tottenham et al., 2009). Each face is shown in both an angry expression and a neutral expression, totaling 48 face stimuli. Face photographs were made uniform by centering the face in a 1.68″ × 2.42″ box with a black background.

The task consists of 288 trials; 96 in each of the three cue types. Each trial begins with a fixation cross for 400 ms, followed by one of the four cue types presented for 100 ms, followed by an interval of 400 ms after which one of the three target stimuli (neutral line flanker, neutral face flanker, angry face flanker) is presented for 1700 ms or until the subject responds. The task was presented on an IBM ThinkPad laptop computer using e-prime professional version 2.0.

2.4. Data analysis

Data analysis was conducted with SPSS 18.0. All variables were screened for normalcy and outliers, defined as 2.5 or more standard deviations away from the mean of each group. Outliers were replaced with the group mean accordingly. Eight scores were replaced: five ANT scores (four high SA), and three ANT-Emotion scores (one high SA). Independent t-tests and χ^2 analyses were used to assess group differences on demographic variables. Univariate Analysis of Variance (ANOVAs) and Multivariate Analysis of Covariance (MANCOVAs) were used to assess group differences on the ANT and ANT-Emotion. Pearson correlations and linear regressions were calculated to assess the proposed mediation model between SA, executive control of emotional information, and social functioning.

3. Results

3.1. Hypothesis 1: compared to controls, high SA participants will exhibit attentional deficits on both the ANT, specifically in the executive control network, and the ANT-Emotion, specifically in the executive control of emotional information

Attention network scores and test statistics for group differences on the ANT are presented in Table 2. Due to group differences in IQ, IQ was entered as a covariate of no interest in all analyses. Results showed that there was no difference between high SA and control participants on any of the ANT scores. Specifically, ANOVAs revealed no group differences in overall reaction times [$F(1,54) = 0.397$, $P = 0.531$] or accuracy

[$F(1,54) = 0.036$, $P = 0.85$]. MANCOVA with attention network scores of the ANT (alerting, orienting, executive control) as dependent variables revealed no main effect of group [$F(3,52) = 0.767$, $P = 0.518$] or IQ [$F(3,52) = 0.563$, $P = 0.642$]. Similarly, follow-up ANOVAs revealed no group differences on alerting [$F(1,54) = 2.373$, $P = 0.129$], orienting [$F(1,54) = 0.42$, $P = 0.839$], or executive control network scores [$F(1,54) = 0.127$, $P = 0.723$]. Given that low IQ is a characteristic feature of schizophrenia liability (Woodberry et al., 2008), removing variance associated with IQ might be removing meaningful variance associated with the illness-related deficit under investigation (Miller and Chapman, 2009). Therefore, we also conducted these analyses without IQ as a covariate; results remained the same. Thus, our hypothesis that high SA individuals would exhibit deficits in the executive control network for non-emotional information was not supported (see Fig. 2).

Group means and test statistics for the ANT-Emotion are presented in Table 2. IQ was entered in all analyses as a covariate of no interest. Results showed a single group difference in executive control of emotional information, and no group differences on any other variables. Specifically, ANOVAs revealed no group differences in overall reaction times [$F(1,59) = 0.343$, $P = 0.56$], accuracy [$F(1,59) = 0.001$, $P = 0.976$], or orienting to emotional information [$F(1,59) = 0.139$, $P = 0.71$]. There were, however, group differences in executive control of emotional information: compared to controls, high SA participants were significantly slower to respond in the angry face flanker condition versus the neutral face flanker condition [$F(1,59) = 5.478$, $P = 0.023$] (see Fig. 3). To examine whether this effect was driven by differences in IQ we conducted these analyses without IQ as a covariate. Results remained the same: compared to controls, high SA participants showed impaired executive control of emotional information [$F(1,60) = 3.971$, $P = 0.05$].

Given that the two groups not only differed on SA, but also on SPQ scores (see Table 1), it is possible that other aspects of psychosis-proneness contribute to this group difference in executive control of emotional information. However, although SPQ scores did relate to SA ($r = 0.76$, $P < 0.0001$) and social impairments ($r = 0.52$, $P < 0.0001$), there was no significant relationship with executive control of emotional information ($r = -0.02$, $P = 0.89$). This indicates that the group difference in executive control of emotional information is not driven by other schizotypal traits that are measured in the SPQ.

Collectively these findings demonstrate that while high SA participants do not exhibit attentional deficits in relation to non-emotional information, they do exhibit a specific deficit in the executive control of emotional information. Moreover, this deficit appears to be driven by SA, rather than psychosis-proneness more generally.

3.2. Hypothesis 2: high SA individuals will report poorer social functioning compared to controls

Mean social functioning and group differences are displayed in Table 1. Consistent with our hypothesis, independent t-tests revealed high SA participants reported significantly poorer social functioning than controls [$t(59) = 4.245$, $P < 0.0001$]. This replicates previous findings that
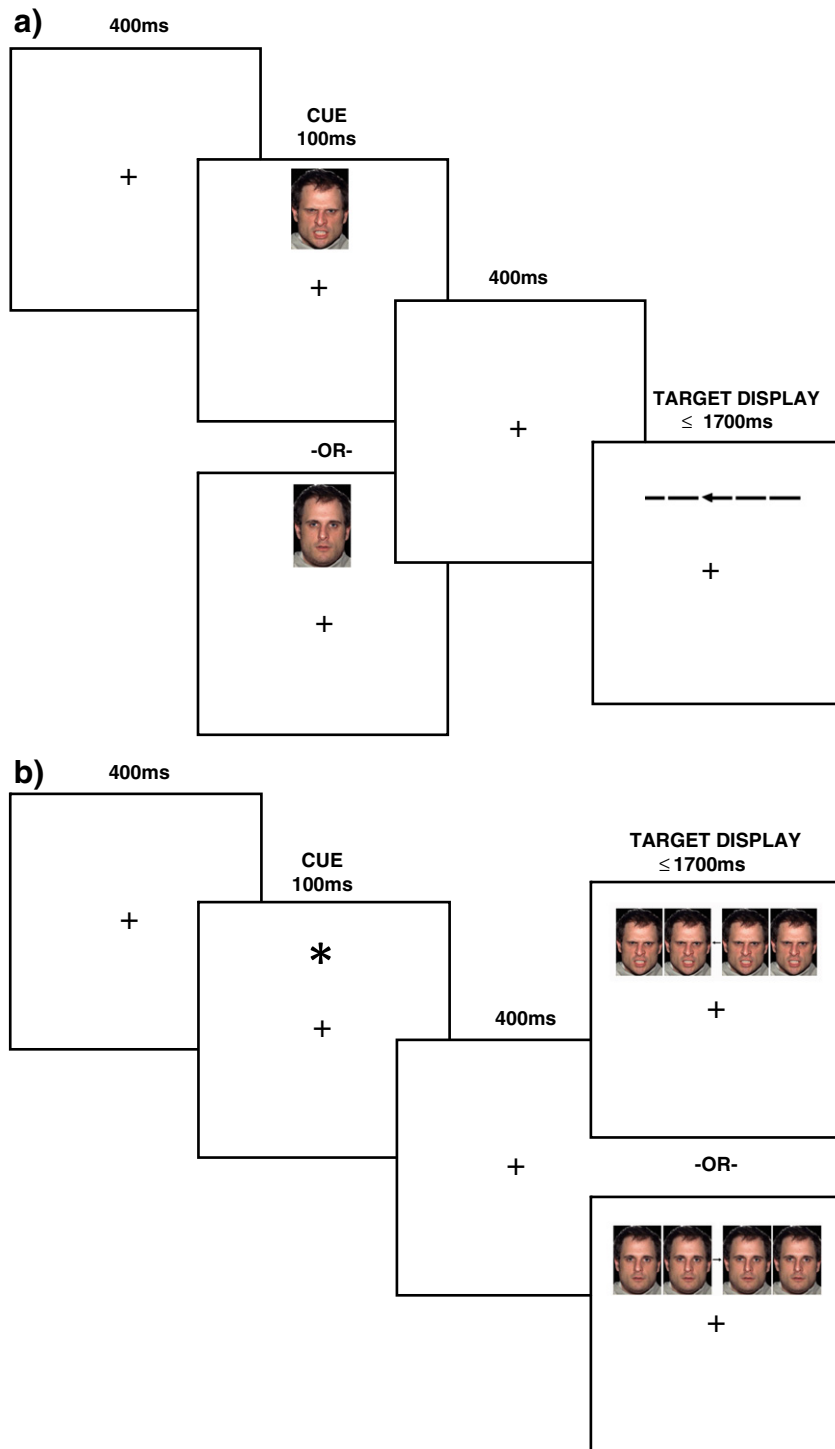
Fig. 1. a: Illustration of an ANT-Emotion trial assessing orienting to emotional information. Participants see a neutral or angry face cue followed by a target arrow flanked with neutral lines. Scores for orienting to emotional information are obtained through simple subtractions of RTs: RT angry face cue – RT neutral face cue. Lower scores indicate an orienting bias to negative emotional faces. b: Illustration of an ANT-Emotion trial assessing executive control of emotional information. Participants see an asterisk cue followed by a target arrow flanked by neutral or angry faces. Scores for executive control of emotional information are obtained through simple subtractions of RTs: RT angry face flankers – RT neutral face flankers. Higher scores indicate reduced ability to inhibit negative emotional faces.

high SA is associated with poorer social functioning (Blanchard et al., 1998; Cohen et al., 2006).

3.3. Hypotheses 3: executive control of emotional information will mediate the relationship between social anhedonia and social functioning

Following the steps required for determining mediation (see Baron and Kenny, 1986), we conducted a series of correlation and regression analyses across all 63 participants to determine if there were significant associations between 1) SA and social functioning, 2) SA and executive control of emotional information, 3) executive control of emotional information and social functioning, and 4) SA and social functioning whilst controlling for executive control of emotional information. All four steps must be met for a mediation model to be supported. A variable is considered a statistical mediator if the relationship between the independent (i.e. SA) and the dependent

Table 2

Attention network RT scores and accuracy of high SA and control participants.

	Control group	High SA group	Differences between groups
ANT: mean (S.D.)			
Alerting	33.98 (19.25)	42.88 (23.49)	$F(1,54) = 2.373, P = 0.129$[a]
Orienting	46.30 (25.88)	49.55 (26.84)	$F(1,54) = 0.42, P = 0.839$
Executive control	114.18 (40.31)	112.75 (41.76)	$F(1,54) = 0.127, P = 0.723$
Overall RT	549.42 (70.23)	566.72 (72.12)	$F(1,54) = 0.397, P = 0.531$
Accuracy (%)	97.53 (2.24)	97.53 (4.49)	$F(1,54) = 0.036, P = 0.850$
ANT-Emotion: mean (S.D.)			
Executive control of emotional information	−0.76 (16.98)	7.85 (16.86)	$F(1,59) = 5.478, P = 0.023, \eta_p^2 = 0.085$[b,c,d]
Orienting to emotional information	0.03 (15.07)	−0.04 (17.40)	$F(1,59) = 0.139, P = 0.71$
Overall RT	520.87 (73.1)	541.59 (88.14)	$F(1,59) = 0.343, P = 0.56$
Accuracy (%)	95.92 (2.86)	95.99 (2.53)	$F(1,59) = 0.001, P = 0.976$

[a] Six control participants did not complete the ANT.
[b] η_p^2 = partial eta squared effect size.
[c] IQ was entered as a covariate of no interest. There was no main effect of IQ. Results remained the same in all analyses when IQ was not entered as a covariate.
[d] One subject did not complete the ANT-Emotion.

(i.e. social functioning) variable is significantly reduced when the mediator (i.e. executive control of emotional information) is controlled for (Baron and Kenny, 1986). Pearson correlations revealed social anhedonia correlated significantly with social impairment ($r = 0.59$; $P < 0.0001$), but executive control of emotional information did not correlate significantly with either social anhedonia ($r = 0.21, P = 0.1$) or social impairment ($r = -0.17, P = 0.19$). Linear regression revealed social anhedonia significantly predicted social functioning [$F(1,59) = 31.215$, $P < 0.0001$; $R^2 = 0.35$] such that higher social anhedonia predicted greater social impairment, satisfying step 1 of the mediation model. However, social anhedonia did not significantly predict executive control of emotional information [$F(1,60) = 2.741$, $P = 0.103$; $R^2 = 0.04$]. Similarly, executive control of emotional information did not significantly predict social functioning [$F(1,58) = 1.803$, $P = 0.185$; $R^2 = 0.03$]. Thus, the mediation model was not supported. These non-significant findings could be due to a small sample size; power analysis indicates that our sample size has sufficient power to detect large and medium effects (0.99 and 0.70 respectively), but low power to detect small effects (0.12).

4. Discussion

This study examined social functioning and executive control of emotional information in individuals at risk for developing schizophrenia. Attentional processes were assessed using the ANT and our new measure of executive control of emotional information, the ANT-Emotion. Two main findings emerged: first, while there were no group differences in alerting, orienting, or executive control networks assessed by the ANT, high SA individuals did exhibit a specific impairment in the executive control of emotional information relative to controls. Second, high SA individuals reported significantly greater social functioning impairments relative to controls.

These findings suggest that there are specific deficits in the executive control of emotional information observable in individuals with vulnerability factors for schizophrenia. This deficit was only apparent in the presence of irrelevant angry faces, and not in the executive control network assessed by the ANT. This suggests that high SA individuals' executive control may be intact but vulnerable, such that deficits are not detected by pure cognitive tasks, rather they only emerge when affective and cognitive information interacts. Moreover, high SA participants did not exhibit faster orienting to emotional information indicating that their slower reaction times in the presence of angry face flankers is not simply due to an orienting bias towards angry faces. We interpret these findings as indicating that the impaired ability to inhibit emotional information allows negative affective stimuli to exert inappropriate influence on cognitive processes. This is consistent with prior studies indicating that schizophrenia patients have an impaired ability to regulate the influence of irrelevant negative affective information on cognitive
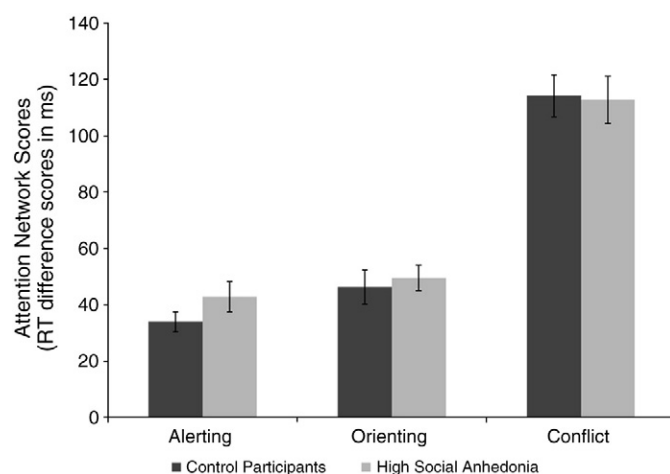

Fig. 2. There were no differences between control and high SA participants on any of the three attentional networks of the ANT, indicating that high SA is not characterized by attentional deficits in relation to nonemotional stimuli.
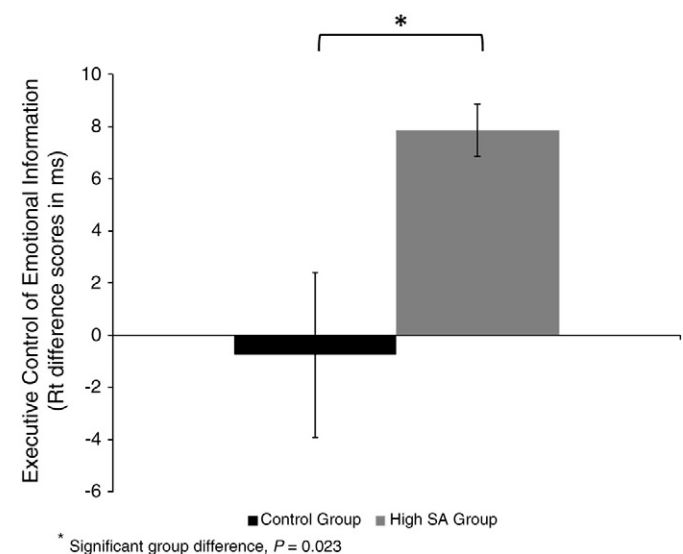

Fig. 3. High SA participants demonstrated impaired executive control of emotional information: compared to control participants, high SA participants were significantly slower to respond to the target in the angry face flanker condition versus the neutral face flanker condition.

processes: patients show increased latencies on the emotional Stroop (Bentall and Kaney, 1989), and rate faces as less trustworthy following negative affective primes (Hooker et al., 2011). Moreover, this impairment may be specifically associated with SA: Suslow et al. (2003) examined the influence of subliminal affective face primes on valence judgments of neutral Chinese characters in schizophrenia patients with and without SA. Compared to patients without SA, high SA patients showed persistent negative evaluative shifts, indicating that impaired regulation of irrelevant negative affect may be characteristic of high SA. Our results are consistent with this, and indicate that it may also be characteristic of individuals at risk for developing schizophrenia.

Our findings also suggest that the deficit in executive control may be specific to the inhibition of socially relevant emotional information. Using the emotion Stroop with non-socially specific negative words, Mohanty et al. (2008) did not find inhibitory deficits in SA. However, our findings, and those of Suslow et al. (2003), suggest that there is a deficit in the presence of socially relevant stimuli (i.e. negative emotional faces). This could contribute to the social withdrawal and dysfunction characteristic of high SA: if individuals struggle to inhibit socially relevant negative affective information, it follows that they would withdraw from unnecessarily unpleasant experiences (Suslow et al., 2003). Future studies could examine this hypothesis further by adapting the ANT-Emotion to contain both social and non-social flankers, or by using an emotional Stroop with categories of social and non-social negative words.

Consistent with our second hypothesis, high SA related to poorer social functioning. This replicates previous findings that high SA is associated with social impairments (Blanchard et al., 1998; Cohen et al., 2006), providing further evidence for the presence of social deficits in people at increased risk for schizophrenia. However, executive control of emotional information did not mediate the relationship between SA and social functioning. There are two possible explanations for this. First, it could be that there is a mismatch between the specificity of the domain measured by the ANT-Emotion and the broad assessment of social functioning obtained by the SAS-SR. We chose the SAS-SR because it is a standard measure used in the field. However, like many social functioning assessments, it is broad in scope. Our finding suggests that developing measures of social functioning that more accurately reflect the underlying social cognitive processes necessary for successful social interactions may result in more success in revealing any underlying relationship between SA, social cognitive deficits, and social impairments. Second, although our sample size is appropriate to detect mediation of large and medium effects, it is not big enough to detect small effects. Future research should examine the relationship between executive control of emotional information and social functioning using alternate measures of social impairments in sample sizes large enough to detect small effects.

Several limitations of the current study should be acknowledged. First, the ANT-Emotion is a newly created task; replications in independent samples are needed and improvements may be warranted. Of particular interest is the effect of stimulus onset asynchrony (SOA). Unlike the ANT, the ANT-Emotion uses a constant SOA (400 ms). This might allow participants to predict the onset of target stimuli, possibly masking any orienting biases that might exist in the sample, or even reducing group differences on executive control of emotional information. However, despite these potential improvements, the task provides valid and compelling data that high SA individuals have impaired executive control of emotional information. The ANT-Emotion is based on the ANT – a well-validated and reliable measure of attention networks, of which the executive control piece is based on another well-validated task – the Eriksen flanker (Eriksen and Eriksen, 1974). Furthermore, the interference effect of irrelevant affective information has been established in other tasks such as the emotional Stroop, therefore it is unlikely that our findings are a consequence of an invalid task. A second limitation is that the ANT-Emotion currently only uses angry faces, hence it is unclear if

our findings would generalize to other facial expressions, both negative and positive. In light of the "anhedonia paradox" (Horan et al., 2008), which suggests that the anticipation of reward, not consummation, is the primary deficit in schizophrenia, the relationship between SA and the regulation of positive emotional information is of particular interest. Our findings indicate that high SA is associated with difficulties regulating negative affective information and could suggest a complementary deficit in the facilitation of positive affective information, thought to underlie the observed deficit in anticipatory pleasure in anhedonia (Pizzagalli, 2010). Future studies could examine both inhibitory and facilitatory mechanisms in the context of emotional information and SA to delineate this further. Finally, there were group differences on the SPQ. Although these differences are expected given the close relationship between positive and negative aspects of schizotypy (Wuthrich and Bates, 2006), and they were shown to have no impact on the group differences in executive control of emotional information, future studies could investigate the specific influence of different aspects of psychosis-proneness on executive control of emotional information.

In summary, the current study demonstrates a deficit in the executive control of socially relevant emotional information in socially anhedonic individuals. Additionally, SA predicted poorer social functioning, consistent with the idea that social impairments are a core characteristic of psychosis-prone populations (Meehl, 1962). These findings have implications for how high-risk populations process negative affective information in social contexts. Further research is needed to identify the precise nature of these deficits, and how they might contribute to the social dysfunction characteristic of schizophrenia.

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