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BRIEF REPORT

Social Anhedonia is Associated with Low Social Network Diversity in Trauma-Exposed Adults

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Social anhedonia has been proposed to contribute to social isolation in several psychiatric disorders, but it has not been examined in relation to deficits in social connection that also characterize posttraumatic stress disorder (PTSD). A growing body of evidence emphasizes the health importance of structural features of social networks, including their size and complexity. The current study examined the association between social anhedonia and social network features in a sample of trauma-exposed participants with and without PTSD as well as in non–trauma-exposed controls. Participants ($N = 101$; $n = 37$ healthy controls, $n = 23$ trauma-exposed without PTSD; $n = 41$ lifetime PTSD) completed self-report measures of social anhedonia (Revised Social Anhedonia Scale) and structural social network features, including social network size, diversity, and the number of embedded networks (Social Network Index). Relative to healthy controls, participants with PTSD reported significantly lower social network sizes and fewer embedded networks. In the combined trauma-exposed sample, higher ratings of social anhedonia were associated with lower social network diversity, $r(62) = -.43$, $p < .001$, an effect that remained statistically significant after controlling for PTSD and depression symptom severity. These results suggest that elevated social anhedonia in trauma-exposed individuals may contribute to disruptions in social network structure consistent with social isolation.

Social anhedonia refers to a reduced ability to experience pleasure and reward from social interactions (Barkus & Badcock, 2019). Although social anhedonia has been extensively studied in psychiatric disorders, including schizophrenia and major depression (Chapman et al., 1976; Kwapil et al., 2008), a growing literature acknowledges the importance of social anhedonia in posttraumatic stress disorder (PTSD) as a core component that may contribute to feelings of social detachment or estrangement (Nawijn et al., 2015; Olson et al., 2018); for example, a feeling of detachment from others was included as Criterion D6 of the PTSD diagnostic criteria in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association, 2013). However, social anhedonia has not been examined in relation to deficits in social connection that characterize PTSD. One approach to objectively measuring social connections is social network analysis (Bryant et al., 2017), which identifies the structural features of connections (i.e., networks) rather than focusing on the content or quality of social relationships (Hammer, 1981). These structural features assess social network size and complexity, including variables such as network density (i.e., the number of network members who know other members), diversity (i.e., the number of different social roles), and embeddedness (i.e., the number of different high-contact social roles; Cohen & Wills, 1985). In the general population, structural features of social networks are associated with poor mental health outcomes, such as higher suicide risk (Handley et al., 2012; Sripada et al., 2015). These structural features also predict increased physical morbidity, including reduced

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immune response and heightened cardiovascular risk (Ford et al., 2006; Molesworth et al., 2015). Despite evidence that social anhedonia contributes to PTSD symptoms, as well as broader research examining social network features as predictors of emotional well-being, no study has examined social anhedonia in relation to social network structure in PTSD.

An emerging body of literature points to associations between a PTSD diagnosis and alterations in structural features of social networks. Most prior studies of social connection in PTSD have focused on social support, which is an important function that social networks can provide. Kaniasty and Norris (2008) showed that higher levels of social support predict less symptom severity in the initial months after trauma exposure and that PTSD symptoms lead to a progressive erosion of social support at later stages of illness. In a study that examined structural features of supportive relationships in a recently traumatized sample, Lee and Youm (2011) found that Korean refugees with larger numbers of supportive connections had a lower risk of later developing PTSD. In a study that separately examined structural social network features versus social support, PTSD diagnosis was more strongly associated with low social network diversity than with low perceived availability of social support (Platt et al., 2014). Finally, Bryant et al. (2017) demonstrated that individuals with PTSD were less likely to be named, or "nominated," by other people as a member of their social network. Altogether, this literature suggests associations between an established PTSD diagnosis or longstanding symptoms and a loss of social connections and deterioration of social network structure. Thus, identifying cognitive–affective processes that might underlie the association between PTSD diagnosis and altered social network features is an important goal. In healthy populations, social reward valuation has been proposed as a core process that drives individual differences in social network size and complexity. Recent evidence suggests that affiliative processes influence individual differences in social network features (Bickart et al., 2012). Positive social stimuli, such as attractive faces, and positive outcomes of social interactions, such as approval and cooperation, are rewarding, and these social rewards may support social network size and complexity by increasing motivation for social interaction (Fareri & Delgado, 2014). A large recent online study of community adults demonstrated that social anhedonia was associated with social network features at the population level (Dodell-Feder et al., 2020). Together, these findings support an examination of the association between social anhedonia and social network features in trauma-exposed samples. Given the proposed role of social reward processing in influencing social network features via affiliative processes and the evidence that social anhedonia relates to social network structure in the general population, we hypothesized that social anhedonia would be associated with altered social network features in the context of trauma-related psychopathology.

Method

Participants

Data from two studies of decision-making following trauma exposure were combined for the present analysis (total $N = 101$). The first study (i.e., Sample 1; $N = 56$) included healthy control (HC) participants ($n = 15$), trauma-exposed controls without PTSD (TENC; $n = 23$; i.e., no lifetime history of meeting the full criteria for PTSD), and participants with current (i.e., full criteria met for past-month symptoms; $n = 10$) or lifetime *DSM-5* PTSD (i.e., partially remitted or full criteria met for worst month but not past-month symptoms; $n = 8$). Inclusion criteria were (a) age of 20–50 years, (b) no history of lifetime Axis I diagnosis for HC group, and (c) trauma exposure consistent with group status. Exclusion criteria were (a) history of neurological disorder, (b) history of head trauma with loss of consciousness longer than 5 min, or (c) history of psychotic disorder, bipolar disorder, eating disorder, intellectual disability, pervasive developmental disorder, or obsessive–compulsive disorder. Psychotropic medications were exclusionary, aside from a stable (i.e., 6-week) dose of antidepressant medications in trauma-exposed groups.

The second study (i.e., Sample 2; $N = 45$) included an HC group ($n = 22$) and a group of symptomatic trauma-exposed (STE) participants ($n = 21$ who met the full criteria for current PTSD, $n = 2$ with current subthreshold symptoms). Inclusion criteria were (a) 18–45 years of age, (b) English as the individual's first language, and (c) trauma exposure consistent with group status. Exclusion criteria were (a) a history of neurological disorders, (b) a history of head trauma with loss of consciousness longer than 5 min, (c) estimated full-scale IQ less than 70, (d) a history of attention deficit hyperactivity disorder, (e) contraindications for magnetic resonance imaging, (f) left-handedness, (g) and alcohol and/or substance use disorder in the past year. For HC participants, a history of any *DSM-5* disorder was exclusionary, aside from alcohol or substance use disorder, before the past 12 months; for STE participants, a history of psychotic or bipolar disorder was exclusionary. For the STE group, a stable (i.e., 6-week) dose of antidepressant medication was permitted; other past-month psychotropic medication use was exclusionary.

The combined sample included 37 HC participants, 23 TENC participants, and 41 participants with a history of current or lifetime *DSM-5* PTSD. There were no group differences in gender distribution (HC: $n = 26$ out of 37 participants were women; TENC: $n = 14$ out of 23 participants were women; PTSD: $n = 35$ out of 41 participants were women), $\chi^2(2, N = 101) = 5.11$, $p = .078$, Cramer's $V = .225$. Demographic characteristics are summarized in Table 1. Participants reported race as follows: Asian ($n = 16$), Black/African American ($n = 10$), Native Hawaiian or Other Pacific Islander ($n = 2$), White ($n = 53$), Multiple races ($n = 11$), Other ($n = 2$), and not reported ($n = 7$). Regarding ethnicity, 13 participants reported Hispanic

Table 1
Demographic and Clinical Characteristics, by Group

	Total sample	HC		TENC[e]		PTSD[e]				
	(*n*)	(*N* = 37)		(*N* = 23)		(*N* = 41)		*F*(*df*, *df*)		
Variable		*M*	*SD*	*M*	*SD*	*M*[e]	*SD*		*p*	η_p^2
Age (years)		27.49	7.71	30.43	8.09	26.49	6.89	2.09 (2, 98)	.129	.041
WASI-II FSIQ[a]		113.14	15.15	106.57	16.16	111.75	15.59	1.33 (2, 97)	.270	.027
Social anhedonia (RSAS)[b]		0.74	0.26	0.90	0.24	1.11	0.31	17.61 (2, 98)	<.001	.264
Social network size (SNI)[b]		1.28	0.29	1.18	0.22	1.09	0.28	4.94 (2, 98)	.009[b]	.092
Number of embedded networks (SNI)[b]		0.45	0.20	0.38	0.22	0.30	0.20	5.18 (2, 98)	.007[d]	.096
Social network diversity (SNI)[b]		0.69	0.14	0.68	0.15	0.62	0.13	2.57 (2, 98)	.081	050
CAPS-5 total score				5.26	6.20	27.59	10.49	86.81 (1, 62)	<.001	.583

Note. HC = healthy control; TENC = trauma-exposed non-PTSD; PTSD = posttraumatic stress disorder; CAPS-5 = Clinician-Administered PTSD Scale for *DSM-5*; WASI-II = Wechsler Abbreviated Scale of Intelligence (second ed.); FSIQ = Full-Scale IQ; RSAS = Revised Social Anhedonia Scale; SNI = Social Network Index.
[a]WASI-II FSIQ is a two-subtest estimate (Vocabulary and Matrix Reasoning). Data from one participant in the PTSD group participant are missing. [b]These are transformed scores: log(raw value + 1). The constant was added to all scores to avoid log0. [c]HC < TENC < PTSD. [d]HC > PTSD. [e]12 participants in the PTSD group and 0 participants in the TENC group reported antidepressant use.

ethnicity, 78 reported non-Hispanic ethnicity, and 10 participants did not respond. Comorbid diagnoses and index traumatic events are presented in the Supplementary Materials.

Procedure

Participants were recruited from advertisements in the Boston metropolitan area and provided written informed consent to protocols approved by the institutional review board of Partners Healthcare. Doctoral-level psychologists conducted interviews, including the Structured Clinical Interview for *DSM-5* (SCID-5; First et al., 2015) and, for trauma-exposed participants, the Clinician-Administered PTSD Scale for *DSM-5* (CAPS-5; Weathers et al., 2018).

Measures

Social Anhedonia

The Revised Social Anhedonia Scale (RSAS; Eckblad et al., 1982) is a self-report questionnaire consisting of 40 items that are answered as "true" or "false." Items are scored as 0 or 1, with 18 reverse-keyed items, and higher scores reflect a higher level of social anhedonia (i.e., social withdrawal and/or apathy). In the current data set, internal reliability was good, Cronbach's $\alpha = .92$.

Social Networks

The Social Network Index (SNI; Cohen et al., 1997) is a self-report questionnaire used to assess the extent of individuals' social contact. Participants were asked to respond to 12 questions regarding social roles (e.g., family relationships, friendships, group or class membership, employment, neighbors). Derived scales were (a) social network size, as the number of people in the social network (i.e., the total number of people who participants indicated having regular contact with, defined as at least once every 2 weeks), (b) the number of embedded networks (range: 0–8; i.e., the number of different high-contact network domains), and (c) the diversity of the social network (range: 0–12; i.e., the number of social roles in which participants reported contact at least every 2 weeks with at least one person). Given the structure of the questionnaire, the Cronbach's alpha values were low (i.e., below .50); see the Supplementary Material for a discussion.

Depression

The Beck Depression Inventory (BDI-II; Beck et al., 1996) is a widely used self-report measure of depression. Total scores were computed by summing responses, rated on a scale of 0–3, on 21 items measuring depression symptoms. Higher scores reflect a higher level of depression severity. In the current data set, the internal reliability was good, Cronbach's $\alpha = .92$.

Data Analysis

For *t* tests, when Levene's test for equality of variances was violated, adjusted degrees of freedom (equal variances not assumed) are reported. Regarding missing data, SNI data were missing for three of 101 cases: Two participants skipped an item pertaining to contact with in-laws, and a third participant indicated that they worked but did not provide the number of supervisees or work contacts. Because missing data were rare and there is no clear method for prorating, those missing data were scored "0" (i.e., no contacts in that domain). For two TENC participants, a single CAPS-5 item was omitted; these items were scored 0. For the RSAS, six participants were missing one item out of 40. Total scores were prorated per participant as follows: prorated score = 40 × (raw total)/39. Social network features and RSAS scores were not normally distributed and were therefore log-transformed. Because the raw values included 0, a constant (1) was added to every value prior to transformation to avoid log(0). The resulting transformed variables were normally distributed.

For between-group comparisons, analyses of variance (ANOVAs) were followed by least-significant difference post hoc tests. We examined Pearson correlations between social network index measures, social anhedonia, and, for trauma-exposed participants, overall PTSD symptom severity. Significant associations between social network parameters and social anhedonia were then entered into a regression that included PTSD symptom severity as an additional predictor in order to assess whether those associations remained statistically significant after accounting for overall symptom severity. The regression was repeated after controlling for gender, age, and severity of depression symptoms.

Results

Group Differences

There was a significant group difference in CAPS-5 total scores, which were higher in the PTSD group than in the TENC group (Table 1). There also was a significant group difference in RSAS scores, $F(2, 98) = 17.61$, $p < .001$, $\eta_p^2 = .264$ (large effect size); post hoc tests showed that participants in the PTSD group reported significantly more social anhedonia than those in the TENC group, $p = .004$, and that individuals in the TENC group had significantly more social anhedonia than those in the HC group, $p = .031$.

Additionally, the groups differed significantly with regard to some of the social network features assessed by the SNI (Table 1). First, there were group differences in social network size, $F(2, 98) = 4.94$, $p = .009$, $\eta_p^2 = .092$ (medium effect size), with participants in the PTSD group reporting significantly fewer people in their network compared to those in the HC group, $p = .002$. There were no significant differences in social network size between the HC and TENC or between the TENC and PTSD groups. Second, there were group differences in the number of embedded networks, $F(2, 98) = 5.18$, $p = .007$, $\eta_p^2 = .096$ (medium effect size), with a significantly lower number of embedded networks among participants in the PTSD group compared to those in the HC group, $p = .002$, but

Table 2
Pearson Correlations Between Social Anhedonia and Social Network Features

Measure	1	2	3	4	CAPS-5
1. Social anhedonia (RSAS)	–	−.25*	−.24	−.43***	.44***
2. Social network size (SNI)	*−.32**	–	.77***	.61***	−.23
3. Number of embedded networks (SNI)	*−.35****	*.77****	–	.40*	−.30*
4. Social network diversity (SNI)	*−.40****	*.61****	*.52****	–	−.20

Note. The combined three-group sample ($N = 101$) is presented below the diagonal in italics. The trauma-exposed sample (i.e., combined TENC and PTSD groups; $n = 64$) is presented above the diagonal. HC = healthy controls; TENC = trauma-exposed non-PTSD group; PTSD = posttraumatic stress disorder; CAPS-5 = Clinician-Administered PTSD Scale for *DSM-5*; RSAS = Revised Social Anhedonia Scale; SNI = Social Network Index.
*$p < .05$. **$p < .001$.

no significant differences in the number of embedded networks between HC and TENC participants or between TENC and PTSD participants. Finally, there was no significant difference in social network diversity between any of the groups.

Correlations Between Social Anhedonia and Social Network Features

As hypothesized, across the entire sample, RSAS social anhedonia scores were associated with structural network features, including smaller social network size, lower number of embedded networks, and lower social network diversity (all medium effect sizes; Table 2). When considering only the trauma-exposed participants (i.e., TENC and PTSD), the association between RSAS score and lower social network size also was statistically significant, $r(62) = -.25$, $p = .043$, as was the association between RSAS score and lower social network diversity, $r(62) = -.43$, $p < .001$. There also was a significant positive correlation between RSAS and CAPS-5 scores, $r(62) = .44$, $p < .001$.

Regression Analysis

Because social anhedonia was related to social network features (i.e., size and diversity) as well as overall PTSD symptom severity in the combined trauma-exposed sample, regressions were performed within this combined sample ($n = 62$) to identify separate effects of PTSD symptom severity (i.e., CAPS-5 total score) and social anhedonia (i.e., RSAS score) on social network features (i.e., SNI score). The model that included both RSAS score and CAPS-5 total score as predictors of social network size was not significant, $F(2, 61) = 2.684$, $p = .076$, $R^2 = .081$, $f^2 = 0.09$. However, the model that included both RSAS score and CAPS-5 total score as predictors of social network diversity was significant, $F(2, 61) = 6.891$, $p = .002$, $R^2 = .184$, $f^2 = 0.23$. Social anhedonia predicted social network diversity while accounting for PTSD symptoms; that is, the association between RSAS score and social network diversity score was statistically significant after controlling for total CAPS-5 score (Table 3). The model also was significant after additionally controlling for gender, age, and overall depression symptom severity (i.e., BDI-II total score), $F(5, 58) = 3.443$, $p = .009$, $R^2 = .229$, $f^2 = 0.30$; and, again, RSAS score was the only variable that contributed significantly to the prediction of social network diversity. These results were unchanged after omitting the 12 participants in the PTSD group who were taking antidepressants (see Supplementary Materials).

To examine whether the association between social anhedonia and social network diversity varied between groups, we used a general linear model with social network diversity as the dependent variable and group (i.e., three categories), RSAS score, and Group × RSAS interaction as predictors. There was a significant effect of RSAS score, $F(1, 95) = 14.006$, $p < .001$, $\eta_p^2 = .128$. The effect of group was not significant, $F(1, 95) = 1.041$, $p = .357$, $\eta_p^2 = .021$, nor was the Group × RSAS interaction, $F(1, 95) = 0.875$, $p = .420$, $\eta_p^2 = .018$. Thus, the strength of the association between social anhedonia and social network diversity did not differ between groups.

Table 3
Linear Regression Model of the Effects of Social Anhedonia and CAPS-5 Scores on Social Network Diversity in the Trauma-Exposed Sample

Variable	β	*B*	95% CI *B*	*t*(61)	*p*
Constant		0.849	[0.733, 0.966]	14.54	<.001
Social Anhedonia	−.421	−0.197	[−0.317, −0.076]	−3.26	.002
CAPS-5 Total	−.018	0.000	[−0.003, 0.002]	−0.14	.892

Note. $n = 64$, representing the posttraumatic stress disorder (PTSD) and trauma-exposed, non-PTSD (TENC) groups combined.
CAPS-5 = Clinician-Administered PTSD Scale for *DSM-5*.

Because there is no existing literature on the association between social anhedonia and social network features, we could not estimate the expected effect size or perform an a priori power analysis. Observed post hoc power for the correlation between RSAS score and social network diversity, $r = -.43$, $n = 64$, was 0.95 (two-tailed; calculated in G*Power 3.1).

Discussion

In the present study, individuals with PTSD endorsed significantly more social anhedonia than trauma-exposed participants without PTSD, who endorsed significantly more social anhedonia than healthy controls. Compared to healthy controls, individuals with PTSD had altered structural features of their social networks, including smaller social network size and lower numbers of embedded networks. Across trauma-exposed participants, social anhedonia was associated with less social network diversity, an effect that was statistically significant even after accounting for overall PTSD and depression symptom severity. In the context of longitudinal studies demonstrating that PTSD symptoms predict deterioration of social relationships over time (Kaniasty & Norris, 2008; King et al., 2006), our results suggest that social anhedonia may explain certain aspects of social isolation in traumatic stress samples. Specifically, social anhedonia that occurs after trauma exposure may contribute to individual differences in the diversity of one's social roles. This finding may be particularly important given evidence that similar social network metrics are associated with adverse health outcomes, including suicidality (Handley et al., 2012).

Social network diversity may be more closely related to social anhedonia as a dimensional process that is independent of psychiatric diagnosis than to the clinical diagnosis of PTSD. Indeed, we did not find a significant group difference in social network diversity between participants in the PTSD, TENC, and HC groups. However, lower social network diversity was significantly correlated with higher ratings of social anhedonia across the entire sample and within trauma-exposed participants. Our results are consistent with findings that suggest affiliative processes contribute to individual differences in social networks among healthy adults (Bickart et al., 2012; Fareri & Delgado, 2014); our results extend these findings to a trauma-exposed sample. To our knowledge, there are no prior reports of an association between social anhedonia and structural features of social networks in PTSD. This motivates further inquiry into whether aberrant social reward processing leads to social isolation across a range of trauma exposure and psychopathology.

Trauma-exposed participants without PTSD were more socially anhedonic than those in the HC group, a finding that may be related to multiple factors. Unlike individuals in the HC group, some members of the TENC group had other psychiatric disorders that can involve anhedonia, such as major depressive disorder and alcohol and/or substance use disorders. Moreover, trauma exposure itself has been associated with alterations in mood and social factors (Barzilay et al., 2019). Although these questions were beyond the scope and statistical power of the present study, they motivate further inquiry into these possibilities.

The present study had several limitations. First, the assessment of social anhedonia was limited to self-report. It will be important to extend the present findings to behavioral measures of social anhedonia. Second, we did not collect information on income or socioeconomic status, and the possible effects of these additional demographic and socioeconomic variables should be examined in future studies. Finally, most participants (i.e., 52 out of 62 trauma-exposed participants, with available data on time since trauma exposure) were assessed 2 or more years posttrauma, so we were not able to examine how associations between social anhedonia and social network structure may evolve over the early posttraumatic period. Future longitudinal studies that examine how social anhedonia, social network disruption, and PTSD symptomatology evolve are warranted.

Despite these limitations, the finding that social anhedonia was associated with lower social network diversity in the present sample has important theoretical and possible clinical implications. It may be important to consider the contribution of social anhedonia to individual differences in social network features. Social anhedonia and low social network diversity may reciprocally drive each other over time, progressively exacerbating social isolation. From a clinical perspective, our results identify social anhedonia as a potential target in addressing social dysfunction in individuals who have been exposed to trauma. Forms of therapy designed to increase social enjoyment (e.g., behavioral activation) may help prevent the deterioration of social networks after trauma exposure.

Open Practices Statement

The study reported in this article was not formally preregistered. Neither the data nor the materials have been made available on a permanent third-party archive. Requests for the data or materials can be sent via email to the first author at eaolson@mclean.harvard.edu.

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