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Original article

Social networks and symptomatology in recently hospitalized individuals with schizophrenia-spectrum disorders: A six-month longitudinal study

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ABSTRACT

Individuals with psychosis have reduced social networks; however, little is known about whether social networks change over time, particularly after an acute episode requiring hospitalization. Furthermore, longitudinal associations between social network engagement, symptom severity, and the impact of illness duration remains unclear. Using linear mixed models controlling for age and sex, we analyzed self-reported social network engagement, positive and negative symptom severity, and self-reported paranoia in individuals with a schizophrenia-spectrum disorder (SSD; $N = 68$) during six months following psychiatric hospitalization and compared them with non-clinical participants (NC; $N = 70$). SSD participants reported significantly lower social network scores than NC participants at every time point ($b = –5.77$), and significantly greater disruption in friendship than family ties ($b = – 2.00$). Social networks remained stable over time in both groups ($b = –0.01$), regardless of illness stage in the SSD group ($b = 8.08$). Notably, lower social engagement with family at baseline predicted greater increases in paranoia over time ($b = –1.01$). Conversely, more severe paranoia at baseline predicted greater reductions in friend networks over time ($b = –0.03$). Finally, social network related to negative symptom severity at baseline ($b = –0.42$) and longitudinally ($b = –0.18$). These findings suggest that clinical severity may influence the social network disruptions commonly found in psychotic disorders, especially during vulnerable periods of recovery following a psychotic exacerbation. Ultimately, this highlights the need for tailored post-discharge interventions that address social network deficits, particularly within families and in the context of severe paranoia.

1. Introduction

Social connection is a critical psychological need and is considered one of the most essential components to well-being and quality of life (Seppala et al., 2013). Individuals with psychosis experience disruptions in perception, cognition, and emotional processing which may lead to difficulty forming and maintaining social relationships. The literature suggests that the social networks of individuals with psychosis tend to be smaller, less dense, and composed of more family members than friends, when compared to the general population (Erickson et al., 1989; Macdonald et al., 2000; Okruszek et al., 2023; Palumbo et al., 2015; Reininghaus et al., 2008; Swinkels et al., 2023). Furthermore, the quality of social networks is often disrupted, with less frequent contact (Reininghaus et al., 2008), less emotional reliability (Macdonald et al., 2000), fewer reciprocal ties, and lower social support levels (Swinkels et al., 2023). In cross-sectional studies, these social network differences have been found to relate to positive symptomatology (Bell et al., 2023; Chau et al., 2019) and may contribute to the greater levels of loneliness in psychosis, which are predicted by social isolation (Shioda et al., 2016).

Despite the importance of social networks, little is understood about how they evolve over time in psychosis and what factors contribute to and result from such trajectories. A sample of studies examining people in their first episode of psychosis showed that social network size and quality began to deteriorate before the onset of symptoms and continued to decline during the early stages of the disorder (DeVylder and Gearing, 2013; Gayer-Anderson and Morgan, 2013). However, these changes were heterogeneous and are not observed across all cases. For instance, a

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study looking at the social networks of individuals with schizophrenia during the 12 months prior to and 15 months following their first hospitalization found that social network characteristics remained relatively stable over time (Horan et al., 2006). In addition, more recent work in non-clinical samples suggests that psychosis-spectrum symptoms (namely delusional ideation) predict worsening social networks over time (Marks et al., 2025). Therefore, in situations where clinical symptoms improve, it could be hypothesized that social networks would also improve. Furthermore, many studies have focused on the early stages of psychosis, but there is evidence to suggest that social network engagement may differ between early and chronic patients. Namely, early stages of psychosis may present a particularly malleable period, with those in the first five years of illness showing more pronounced improvements in clinical and functional parameters compared to those with chronic illness in response to intervention (Deste et al., 2019). This early period of resiliency may extend to social networks – with greater improvement in social networks following treatment. Overall, however, the lived experience of patients with psychosis is complex and mixed (Fusar-Poli et al., 2022). Additional research is needed to better understand the social networks of individuals with psychosis across the illness course.

One aspect of schizophrenia that may be important for understanding social networks is symptom severity. The period following hospitalization for an acute psychotic episode is a critical and unique window for examining how changes in symptom severity relate to social networks. Of the symptoms of psychosis, paranoia may be particularly important for understanding social networks. Paranoia can be understood as unfounded ideas that others intend a person harm (Freeman, 2016). Paranoia exists on a spectrum, with more severe manifestations taking the form of persecutory delusions. These delusions are present in over 70 % of individuals in their first episode of a psychotic disorder and can be detrimental to one's psychological well-being and quality of life (Freeman, 2016). Studies have shown that among individuals with psychosis, social isolation and loneliness predict increased paranoid thinking (Bell et al., 2023; Chau et al., 2019). It has been theorized that this relationship is bidirectional, in that people with paranoid thoughts may reduce social contact as a safety behavior to prevent the perceived threat. In turn, this withdrawal may decrease distress around the threat, thereby reinforcing the paranoid belief and further driving isolation (Fett et al., 2022). However, a recent study looking at social interactions found that individuals with a non-affective psychotic disorder experienced greater paranoia when alone compared to when they were with others, but that this did not predict future social isolation (Fett et al., 2022). Notably, the study sample consisted of stable outpatients with a psychotic disorder. Understanding changes in a more acute sample as their illness severity evolves may help elucidate the relationship between social isolation and paranoid symptomatology.

Here, we longitudinally examined the social networks of individuals with an SSD during the first six months following an inpatient psychiatric hospitalization. Furthermore, we were interested in whether social networks vary between individuals in their first episode of psychosis versus those in a chronic stage, to possibly explain the heterogeneity observed in previous work. Finally, we aimed to understand whether severity of paranoia can successfully predict social network engagement. We hypothesized that: 1) the level of reported social network disruption in individuals with an SSD will decrease in the six months following inpatient psychiatric hospitalization, 2) this trend will be stronger in individuals earlier on in their illness course, and 3) variability in social network disruption will relate to severity of paranoia, in that lower social engagement will correspond with greater paranoia at baseline and decreasing social network disruption will be associated with improving paranoia over time.

2. Methods

2.1. Participants and protocol

At baseline, 78 individuals with a schizophrenia spectrum disorder (SSD) and 73 non-clinical (NC) comparison participants were enrolled. Within the SSD group, 27 were classified as experiencing early psychosis (EP; less than two years since first psychotic episode), and 51 were classified as experiencing chronic psychosis (CP; more than two years since first psychotic episode). SSD participants were recruited from Vanderbilt University Medical Center during an inpatient hospitalization for an acute psychotic exacerbation and were enrolled within approximately 8 weeks of discharge (average = 4.43 weeks, range = 0 – 14.9 weeks). Eight participants were enrolled prior to discharge. NC participants were recruited from existing study registries.

All participants were between the ages 18-55, had no past or present neurological illness or significant traumatic head injury, and had an estimated premorbid IQ >70 as determined by the Wechsler Test of Adult Reading (Ginsberg et al., 2003). SSD diagnoses were determined by the discharge diagnosis given by the treating inpatient provider during the individual's hospitalization. In some cases, participants had previously taken part in other studies, and if a Structured Clinical Interview of the DSM-IV-TR or DSM-5 (SCID) completed by a trained rater was available, then the SCID diagnosis was used (N = 2). The clinical sample at baseline consisted of 38 individuals with schizophrenia, 13 with schizophreniform disorder, 20 with schizoaffective disorder, and 1 with delusional disorder. In addition, three with a discharge diagnosis of brief psychotic disorder and three with a psychotic disorder not otherwise specified were included. NC participants were determined via the SCID to be free of a lifetime mental health diagnosis, a first degree-relative with a psychotic disorder, or any current psychotropic medication use. Groups did not differ on age, sex, race, or parental education; full demographic comparison can be found in Table 1.

Study protocols were approved by the Vanderbilt Institutional Review Board (IRB #202462, 3/3/2021), and all participants provided written informed consent prior to completing study activities. All participants were invited to complete six visits: a baseline visit and subsequent visits one, two, three, eight, and 24 weeks later. More frequent visits at the start of the study were designed to capture the more rapid changes expected after discharge. Within the final SSD sample, 98.5 % completed a baseline visit, 77.9 % completed a week one visit, 69.1 % completed a week two visit, 67.7 % completed a week three visit, 67.7 % completed a week eight visit, and 50 % completed a week 24 visit. Within the final NC sample, 100 % completed a baseline visit, 97.1 % completed a week one visit, 95.7 % completed a week two visit, 95.7 % completed a week three visit, 95.7 % completed a week eight visit, and 80 % completed a week 24 visit. Clinical assessments and self-report questionnaires, described below, were completed at every visit. Participants had the option to complete visits virtually if they were unable to attend in person (i.e., due to distance), resulting in 28 % of visits completed virtually across groups. Demographics and social network scores between those who completed their baseline visit virtually or in-person are included in the Supplement (Table S2), as are differences between those who completed all visits versus those who were lost to follow-up (Table S1).

2.2. Study measures

2.2.1. Social networks

Social networks were measured using the Lubben Social Network Scale – 6 (LSNS-6), a six-item self-report measure that assesses the size and level of social engagement with friend and family ties (Lubben et al., 2006). Family and Friend relationships are separately assessed via the following three questions: 1) How many [relatives/friends] do you see or hear from at least once a month?, 2) How many [relatives/friends] do

Table 1
Participant demographics.

	NC, $n = 70$	SSD, $n = 68$	Statistic (t)	p-value
Age, y				
Mean (SD)	29.3(7.6)	28.6 (8.3)	0.50	.615
Parental Education, y				
Mean (SD)	15.4 (2.8)	15.0 (2.5)	0.91	.366
Premorbid IQ				
Mean (SD)	115 (9.9)	104 (12.7)	5.4	**<.001**
	N (%)	N (%)	Test	p-value
Sex			Fisher's exact	.82
Male	49 (70)	45 (66.18)		
Female	21 (30)	23 (33.82)		
Race			Fisher's exact	.46
Asian	4 (5.71)	2 (2.94)		
Black	19 (27.14)	26 (38.24)		
Middle Eastern	1 (1.43)	1 (1.47)		
Multiracial	0 (0)	2 (2.94)		
Other	3 (4.29)	3 (4.41)		
White	43 (61.43)	34 (50)		
Visits completed in-person/virtual			Fisher's exact	.35
Baseline	51 (72.86) / 19 (21.14)	53 (79.1) / 14 (20.9)		
Week 1	50 (73.53)/ 18 (26.47)	39 (73.58) / 14 (26.42)		
Week 2	48 (71.64)/ 19 (28.36)	34 (72.34)/ 13 (27.66)		
Week 3	50 (74.63)/ 17 (25.37)	35 (76.09)/ 11 (23.91)		
Week 8	49 (73.13)/ 18 (26.87)	32 (69.57)/ 14 (30.43)		
Week 24	40 (71.43)/ 16 (28.57)	11 (32.35)/ 23 (67.65)		
Illness chronicity				
Early psychosis	N/A	27 (39.71)		
Chronic psychosis	N/A	41 (60.29)		
PANSS Positive				
Mean (SD)	N/A	20.4 (5.9)		
PANSS Negative				
Mean (SD)	N/A	14.4 (5.0)		
PANSS General				
Mean (SD)	N/A	35.1 (7.6)		
BL Isolation (LSNS-6 Total <12)	8 (11.43)	30 (44.12)	Fisher's exact	**<.001**
BL Clinically Severe Paranoia (R-GPTS-b >18)	0 (0)	22 (32.35)	Fisher's exact	**<.001**

Note: F, female; M, male; IQ, intelligence quotient; y, years; PANSS, Positive and Negative Syndrome Scale; LSNS, Lubben Social Network Scale; R-GPTS-b, Revised Green et al., Paranoid Thoughts Scale-Part b (persecutory ideation); BL, baseline.

you feel close to such that you could call on them for help?, and 3) How many [relatives/friends] do you feel at ease with that you can talk to about private matters? Every item has the same five answer options: 0 = none, 1 = one, 2 = two, 3 = three or four, 4 = five through eight, and 5 = nine or more. Family and Friend subscale scores are calculated by summing scores from the three questions designated for each respective category. Total LSNS-6 scores are derived from the sum of each subscale and range from 0 – 30, with higher scores indicating greater social integration and scores below 12 indicating a high risk for social isolation (Lubben et al., 2006). The LSNS-6 and its two subscales have demonstrated strong reliability and validity across a variety of populations (Lubben et al., 2006), including individuals with psychotic disorders (Świtaj et al., 2021). Our sample replicated strong internal consistency at baseline (SSD: $\alpha = 0.83$, NC: $\alpha = 0.81$).

2.2.2. Psychotic symptoms

SSD participants were interviewed by a trained rater using the Positive and Negative Syndrome Scale (PANSS) (Kay et al., 1987) to measure psychotic symptom severity across several domains, including positive, negative, and general symptoms. The PANSS includes 30 items scored on a 7-point Likert scale from Absent to Extreme to measure the severity of positive (7 items), negative (7 items), and general (16 items) symptoms. Higher scores reflect greater symptom severity, with total positive and total negative symptom scores ranging from 1 to 49 and total general symptom scores ranging from 1 to 112.

2.2.3. Paranoia

Self-reported paranoia was measured across all participants using the revised Green et al. Paranoid Thoughts Scale (R-GPTS) (Freeman et al., 2021), which contains 18 items measuring ideas of reference and ideas of persecution. The survey instructs participants to "Think about the last month and indicate the extent of these feelings from "0 (Not at all)" to "4 (Totally)". Participants are presented with eight items assessing ideas of reference (R-GPTS-a) and 10 items assessing ideas of persecution (R-GPTS-b). All items are measured on a 5-point Likert scale with total scores ranging from 0 to 40. The R-GPTS has demonstrated high reliability across the spectrum of paranoia severity in both clinical and non-clinical settings (Freeman et al., 2021). Our sample replicated strong internal consistency at baseline (SSD: $\alpha = 0.96$, NC: $\alpha = 0.80$). An R-GPTS-b score of 18+ indicates clinically severe paranoid ideation (Freeman et al., 2021).

Interviewer-rated paranoia was also assessed in the SSD group using the Suspiciousness/Persecution Item (P6) of the PANSS (Kay et al., 1987). Higher P6 scores reflect more severe persecutory delusions. Examination of P6 scores is included in Supplementary File 1.

2.3. Statistical analyses

Data analysis was conducted in RStudio (R Core Team [v.4.4.2], 2024) using a final sample of 68 SSD participants (EP, N = 27; CP, N = 51) and 70 NC participants, after removing 10 SSD and three NC participants for missing LSNS-6 data.

In baseline analyses, we constructed linear models controlling for age and sex to evaluate group differences (NC vs. SSD and EP vs. CP) in social network scores, and then to examine relationships between SSD participants' social network scores and clinical characteristics. The following clinical symptoms at baseline were modeled as independent predictors of baseline social network scores: R-GPTS scores, PANSS P6 scores, PANSS positive symptom subtotal scores, and PANSS negative symptom subtotal scores.

In longitudinal analyses, time was modeled continuously for each visit as weeks since baseline, with variable time intervals amongst participants determined by the date of each visit. To investigate how social network scores changed over time in our sample, we constructed linear mixed effect models that account for within-subject correlation and handle missing visits under the missing-at-random assumption. Each model included a random intercept for participant and fixed effects for time, age, and sex. This was first done across the whole sample, with group included as a fixed effect, and then within each group separately. Finally, within the SSD-only model, illness chronicity (EP and CP) was examined as a fixed effect. Post-hoc pairwise comparisons were performed in each subgroup separately.

Finally, we conducted an analysis of longitudinal relationships between social network and paranoia and negative symptom subtotal scores in SSD participants using linear mixed effect models including participant as a random effect and time, age, and sex as fixed effects. To identify more nuanced, individual-level patterns in social network and symptom change that may have been masked by conventional grouped mixed effects modeling, change scores were constructed by subtracting baseline scores from week 24 scores for social networks and self-reported paranoia (NC, $N = 56$; SSD, $N = 34$). Therefore, higher change scores indicate greater improvement in social networks or paranoia by the end of the study. In the SSD group, linear models

controlling for age and sex were used to assess the relationships between 1) baseline social networks and changes in paranoia, and 2) baseline paranoia and changes in social network. R-GPTS total scores were examined, combining both subscales: A) ideas of reference and B) ideas of persecution. Examination of relationships with each subscale separately are included in Supplementary Files 1, 2, 3 and Supplementary Figs. 2, 3, 4.

2.3.1. Model diagnostics

Post-hoc Shapiro-Wilk tests indicated that all model residuals were normally distributed (all p >.05), and visual inspection of residual plots supported the assumption of linearity across all modeled variables, thus justifying the use of linear and mixed effects modeling.

All statistically significant results examining nested relationships within diagnostic groups (NC and SSD) and illness stage groups (EP and CP) were Bonferroni-corrected to account for multiple comparisons.

3. Results

3.1. Baseline group differences in social networks

At baseline, average LSNS-6 total scores in the SSD group were 12.2 ($SD = 5.89$; min: 0, $n = 2$; max: 27, $n = 1$) and in the NC group 18.3 (SD: 5.16; min: 4, $n = 1$; max: 30, $n = 1$). Compared to NC comparisons, participants with SSDs had significantly lower baseline LSNS-6 total ($b =$ -6.03, $SE = 0.96$, $t(132) = -6.28$, $p < .001$), Friend ($b = -4.07$, $SE = 0.60$, $t(132) = -6.76$, $p < .001$), and Family ($b = -1.97$, $SE = 0.52$, $t(132) = -3.76$, $p < .001$) scores. More SSD participants were at high risk for isolation (LSNS-6 < 12) than NC participants (χ^2 (2) = 19.46, $p < .001$). In addition, there was a significant interaction between group and score type ($b = -2.00$, $SE = 0.79$, $t(266) = -2.52$, $p = .012$), driven by a greater deficit in Friend-networks compared to Family-networks in SSD, which was not observed in the NC group (see Fig. 1).

3.2. Longitudinal changes in social networks

Participants with SSD had significantly lower LSNS-6 total ($b =$ –5.77, $SE = 0.93$, $t(132) = -6.22$, $p < .001$), Friends ($b = -3.98$, $SE =$ 0.58, $t(132) = -6.92$, $p < .001$), and Family ($b = -1.78$, $SE = 0.45$, $t(132) = -3.95$, $p < .001$) scores over time, compared with NC (main effect of group). However, social networks were stable over time across the whole sample (main effect of time for LSNS-6 total: $b = -0.01$, $SE = 0.01$, $t(552) = -1.07$, $p = .284$; Friends: $b = -0.004$, $SE = 0.01$, $t(551) = -0.58$, $p = .562$; Family: $b = -0.01$, $SE = 0.01$, $t(555) = -1.02$, $p = .310$) (see Fig. 2). We also did not find evidence for a significant difference in how social networks changed over time between groups (group by time interaction: $b = 0.02$, $SE = 0.02$, $t(553) = 1.00$, $p = .320$), suggesting that LSNS-6 total scores were stable over time for both NC and SSD participants.

3.3. Social networks and illness stage

When comparing the illness chronicity of our SSD participants at baseline, we found no significant difference in the proportion of isolated individuals (χ^2 (1) = 1.36, $p = .244$), and in LSNS-6 total ($b =$ -0.76, $SE =$ 1.74, $t(59) = -0.44$, $p = .662$), Friend ($b = -0.76$, $SE = 1.06$, $t(59) =$ -0.72, $p = .473$), and Family ($b = -0.001$, $SE = 0.94$, $t(59) = -0.001$, $p =$ .999) scores between EP and CP subgroups, indicating illness duration did not impact SSD participants' social networks.

Furthermore, illness chronicity did not have a significant impact on how SSD participants' social networks changed over time, as the interaction between illness stage (EP and CP) and time was not significant ($b = 8.08$, $SE =$4.67, $t(2.18) = 0.17$, $p = .863$), and both EP and CP participants' LSNS-6 total, Friends, and Family scores remained stable longitudinally ($ps > .05$).

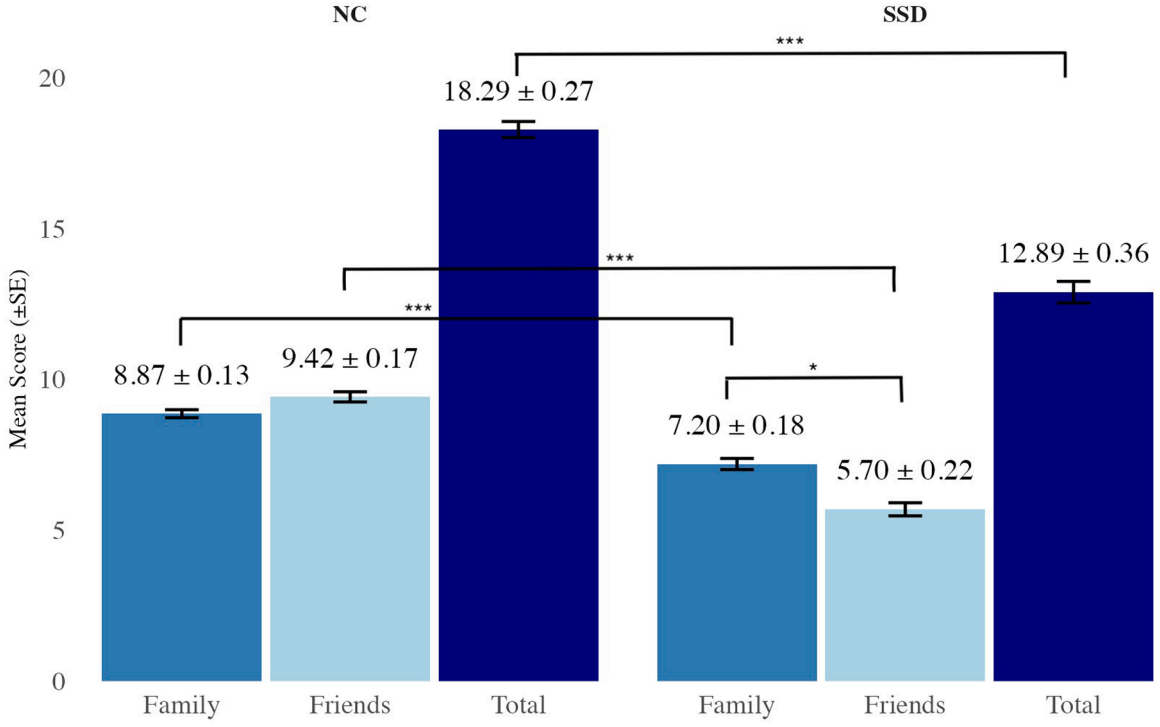

Fig. 1. *Group Differences in Social Network. Note:* Diagnostic group differences in mean social network scores (±SEM) across three domains: Family, Friends, and Total. Social network scores were derived from the LSNS-6 and are displayed separately for individuals with schizophrenia-spectrum disorders (SSD, right) and non-clinical (NC, left) comparisons. Higher scores reflect greater social connectedness. Error bars denote standard errors of the mean. Horizontal bars represent significant group differences at $p < .05$.

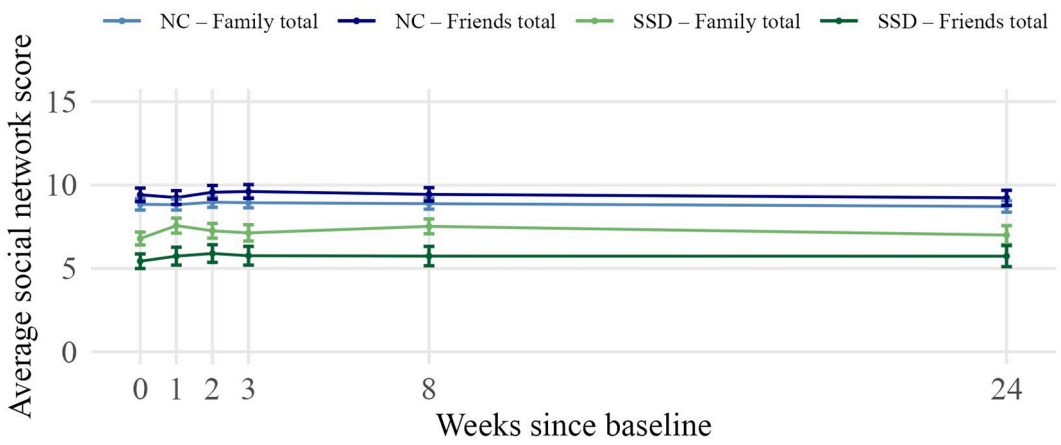

Fig. 2. *Average Social Network Scores Over Time by Diagnostic Group. Note*: Diagnostic group-level trajectories of friend and family scores over 24 weeks for participants with schizophrenia-spectrum disorders (SSD) and non-clinical (NC) comparisons. Each line demonstrates no significant change in LSNS-6 friend and family subscores in either diagnostic group, $p > .05$. Error bars represent standard errors of the mean.

3.4. Baseline associations between social networks and paranoia

At baseline, average R-GPTS Total scores in the SSD group were 25.2 (*SD*: 21.3; min: 0, $n = 6$ participants; max: 68, $n = 1$) and in the NC group 4.01 (*SD*: 5.24; min: 0, $n = 24$; max: 27, $n = 1$).

Social network engagement at baseline was not significantly associated with paranoia in the SSD group (R-GPTS total: $b = –0.01$, $SE = 0.04$, $t(61) = –0.29$, $p = .774$). Similarly, isolated SSD participants did not differ from non-isolated SSD participants in paranoia severity (R-GPTS total: $b = 1.37$, $SE = 5.39$, $t(61) = 0.25$, $p = .801$).

3.5. Overall longitudinal changes in associations between social networks and paranoia

In the SSD group, we found that R-GPTS total scores decreased over time ($b = -0.35$, $SE = 0.09$, $t(230) = -3.98$, $p_{corrected} < .001$), but there was not a significant relationship between LSNS-6 and R-GPTS total scores over time (LSNS-6 total/R-GPTS total: $b = -0.06$, $SE = 0.21$, $t(273) = -0.27$, $p = .778$; LSNS-6 friends/R-GPTS total: $b = -0.30$, $SE = 0.37$, $t(265) = -0.82$, $p = .412$; LSNS-6 family/R-GPTS total: $b = 0.13$, $SE = 0.35$, $t(282) = 0.35$, $p = .724$).

3.6. Predictive longitudinal relationships between social networks and paranoia

Analyses of change scores revealed that lower LSNS-6 family scores at baseline, but not total or friend scores (*ps* >.05), predicted greater increases in paranoia over time ($b = -1.01$, $SE = 0.45$, $t(191) = –2.27$, $p_{corrected} = .048$) (see Fig. 3). This suggests that individuals with SSD starting the study with less family social engagement experienced subsequent worsening of paranoia throughout the six months following hospitalization.

The reverse relationship was then examined. This revealed a significant negative association between R-GPTS total scores at baseline and subsequent change in LSNS-6 total scores in SSD participants ($b = -0.04$, $SE = 0.01$, $t(191) = –2.68$, $p_{corrected} = .016$). (see Fig. 4a). This relationship was primarily driven by SSD participants' relationships with friends ($b = -0.03$, $SE = 0.01$, $t(191) = –4.02$, $p_{corrected} < .001$), not family ($p = 0.65$) (see Fig. 4b-c), at enrollment. This indicates that in highly paranoid SSD participants, relationships with friends were more vulnerable to deterioration in the six months following hospitalization, whereas familial relationships tended to be more stable.

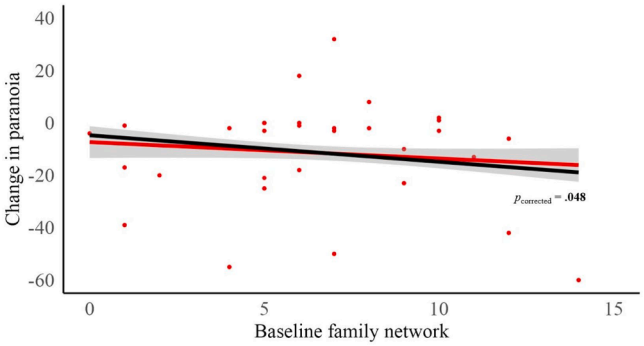

Fig. 3. *Model-Adjusted Relationship Between Baseline Family Networks and Change in Paranoia in SSD. Note:* Model-adjusted relationship between baseline family network and change in paranoia among participants with schizophrenia-spectrum disorders (SSD). Change in paranoia is calculated as the difference in R-GPTS total scores from week 0 to week 24. Predicted R-GPTS total values derived from a linear model adjusting for age and sex are reflected in black. Raw data and trend reflected in red. Higher baseline LSNS-6 family scores were associated with greater decreases in paranoia over time, $p_{corrected} < .05$.

3.7. Exploratory associations between social networks and other symptomatology

Social network engagement at baseline was not significantly associated with overall positive symptoms (PANSS Positive Total: $b = –0.07$, $SE = 0.13$). Similarly, isolated SSD participants did not differ from non-isolated SSD participants in positive symptom severity (PANSS positive total: $b = –2.25$, $SE = 1.41$, $t(62) = –1.59$, $p = .116$).

In contrast, PANSS negative symptoms were significantly negatively associated with LSNS-6 total scores ($b = –0.42$, $SE = 0.14$, $t(62) = –2.95$, $p_{corrected} = .01$), indicating that greater negative symptom severity related to less overall social network engagement in our SSD group at baseline (see Supplementary Figure 1). Isolated SSD participants scored higher on PANSS negative symptoms compared to non-isolated SSD participants, although this difference did not survive correction for multiple comparisons ($b = -2.67$, $SE = 1.20$, $t(62) = -2.22$, $p_{corrected} = .06$).

Longitudinally, PANSS negative symptoms significantly decreased over the course of the study ($b = -0.06$, $SE = 0.02$, $t(230) = -2.57$, $p_{corrected} = .022$). In line with baseline findings, the significant relationship between social LSNS-6 scores and PANSS negative symptoms extended longitudinally (LSNS-6 total: $b = -0.18$, $SE = 0.05$, $t(259) = -3.40$, $p_{corrected} = .002$; LSNS-6 friends: $b = -0.23$, $SE = 0.09$, $t(246) =$

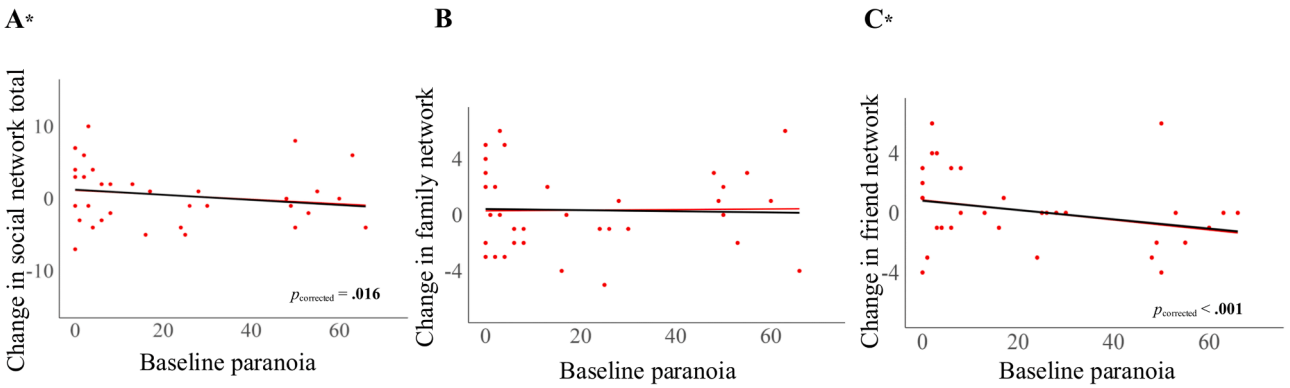

Fig. 4. *Model-Adjusted Relationships Between Baseline Paranoia and Change in Social Networks in SSD. Note*: Model-adjusted relationship between baseline paranoia (R-GPTS total scores) and change in social network (LSNS-6 total, family, friends scores) among participants with schizophrenia-spectrum disorders (SSD). Change in social network is calculated as the difference in LSNS-6 scores from week 0 to week 24. Predicted LSNS-6 scores derived from a linear model adjusting for age and sex are indicated in black. Raw data and trend are reflected in red. Fig. 4a, c: greater baseline R-GPTS total scores were associated with greater decreases in LSNS-6 total and friend scores over time, $p_{corrected} < .05$. Fig. 4b: no significant relationship between baseline R-GPTS total scores and change in LSNS-6 family scores.

-2.62, $p_{corrected} = .019$; LSNS-6 family: $b = -0.27$, $SE = 0.09$, $t(285) = -3.06$, $p_{corrected} = .005$), suggesting that decline in negative symptoms was related to increase in social network.

Finally, due to attrition in the SSD sample, we compared demographic characteristics, LSNS-6 total scores, R-GPTS total scores, PANSS negative total scores, and PANSS positive total scores between those that completed a Week 24 visit and those that did not in Supplementary Table 1. No significant differences in these variables were observed.

4. Discussion

Using data from a sample of recently hospitalized individuals with SSDs, we characterized social networks during the six months following an acute psychotic episode. Compared to NC counterparts, individuals with SSDs reported reduced social networks that remained stable over time, regardless of illness stage. Symptom analyses revealed that greater paranoia predicted declining friendships, while lower baseline family engagement predicted stable or worsening of paranoia. Reduced social networks were also significantly related to greater negative symptom severity. These findings suggest that family support may buffer against worsening paranoia, whereas social networks are especially vulnerable to disruption with greater symptom burden.

In line with extensive literature on social functioning in psychosis (e.g., Erickson et al., 1989; Macdonald et al., 2000; Okruszek et al., 2023; Palumbo et al., 2015; Reininghaus et al., 2008), we found individuals with SSDs experienced significantly greater disruption in their relationships with friends and family compared to their NC counterparts at enrollment, and significantly more SSD participants met LSNS-6 criteria for being at high risk for social isolation. These network alterations were driven largely by less social engagement with friends than family, replicating previous findings (e.g., Horan et al., 2006; Okruszek et al., 2023).

Contrary to our hypotheses, social networks remained stable over the six months following hospitalization, suggesting that network reductions are entrenched prior to illness exacerbation. This finding corroborates a previous cohort study showing social network size stability across the year following individual's first hospitalization for psychosis (Renwick et al., 2017). Additionally, we did not find group differences in social networks between those in the early stages of illness and those experiencing chronic psychosis, suggesting that the processes important in fostering and maintaining social connection begin to deteriorate prior to illness onset. Indeed, prior literature has shown social difficulties that manifest early and persist throughout the prodromal, early, and chronic phases of psychotic illness (e.g., Gayer-Anderson and Morgan, 2013; Horan et al., 2012, 2006). This finding contrasts with historical theories, including the "social network crisis model", which proposes that the progressive impact of illness duration breaks down social connections over time (Beels, 1979). In support of this theoretical framework, Lipton et al.'s (1981) early study found greater social network reductions in individuals with schizophrenia with multiple admissions compared those at first admission (Lipton et al., 1981). However, these findings were limited to a small sample size ($N = 30$) and did not account for variability in clinical characteristics. Our results suggest that a complex interplay of factors may influence individual social network variability, including fluctuations in symptom severity.

Including a sample of recently hospitalized individuals with SSDs presented a unique opportunity to explore the relationship between psychopathology and social networks in a particularly acute sample. During this period of recovery, we hypothesized that recent disruptions to daily routines, occupational roles, and social engagement from hospitalization may render social networks most vulnerable to changes in clinical status, particularly paranoia. However, our initial findings did not support a baseline relationship between severity of self-reported paranoia and social network quality in SSD participants. Rather, we found that those with an SSD who began the study with more severe paranoia reported greater decline in relationships with friends, but not in relationships with family. This finding supports the idea that social withdrawal, particularly from friends, may partially reflect a safety behavior in which individuals avoid social engagement in periods of heightened paranoia (Berkhof et al., 2024; Fett et al., 2022; Freeman et al., 2007). Highly paranoid individuals may view others as a threat and assume that social withdrawal will alleviate their discomfort (Fett et al., 2022). However, as Fett et al. (2022) demonstrated, social interactions can reduce paranoia, possibly by providing opportunities for corrective feedback that disconfirms paranoid beliefs or by reducing the salience of threatening interpretations.

Furthermore, we found that SSD participants who began the study with less family social engagement experienced greater increases in paranoia by the end of the study, while this relationship was not found with friends. These results complement Fett et al.'s (2022) findings by suggesting the inverse: during the vulnerable period following hospitalization, the absence of social opportunities with family could potentially exacerbate one's paranoid symptomatology. Although the directionality of this relationship cannot be fully determined, it may suggest that lower family engagement and heightened paranoia reinforce one another, particularly during early recovery. Because social networks remained stable over time in the clinical group, the absence of an association between changes in paranoia and changes in social networks was unsurprising. However, the change score analyses revealed

important nuances by further characterizing the post-discharge social environments in which paranoia was most vulnerable to change, and the clinical profiles for which social networks were susceptible to change, despite overall group stability.

Additionally, our study highlights the specificity of social connection type (friends, family) on the association between social networks and paranoia, in that familial relationships tend to be particularly resilient. Strong familial involvement in the care of individuals with psychosis has been extensively studied as an influential psychosocial factor relating to one's prognosis. While there is robust evidence that interpersonal processes within the family environment can pose as a risk factor that exacerbates symptom severity (e.g., high levels of expressed emotion and perceived criticism) (Bebbington and Kuipers, 1994), there is also evidence to suggest that positive family characteristics can aid in recovery (González-Pinto et al., 2011; López et al., 2004; Soundy et al., 2015). Active family participation in care can lead to better outcomes from and adherence to treatments (Alston et al., 2019; Glick et al., 2011; Leclerc et al., 2015), and better quality of life (Butler et al., 2019; Fleury et al., 2008), all of which are key factors in promoting sustained clinical stability.

Our findings comparing friend and family relationships recapitulate some of what is understood about social outcomes in individuals with psychosis, particularly as it relates to negative symptoms of schizophrenia. Negative symptoms in psychosis – including apathy and reductions in emotional engagement – are significant predictors of poor outcomes and psychosocial functioning (Hunter and Barry, 2012). Our findings extend this literature by demonstrating a robust baseline and longitudinal relationship between greater negative symptom severity and lower social network engagement during the period following an acute psychotic exacerbation. As a whole, this work emphasizes the importance of considering the impact of both positive and negative symptomatology in social outcomes for those with an SSD.

4.1. Clinical implications

Among the variety of interventions employed to treat those with psychosis, a subset is focused on improving socialization, including social recovery therapy and social skills training (Vinu and Georgiades, 2025), both of which are a part of the greater landscape of cognitive behavioral therapy (CBT). Our results support the need for more personalized post-discharge care plans following psychiatric hospitalization that target deficits in one's social network, especially with family. Notably, family interventions are often poorly implemented in clinical practice, in part because they require organization-wide training to effectively facilitate a therapeutic alliance between the clinical team, patients, and their families (Eassom et al., 2014). Our findings contribute to the growing body of evidence advocating for more effective implementation of family-focused care, especially in psychiatric discharge planning.

4.2. Limitations and future directions

Of note, several limitations should be considered. First, the LSNS-6 is limited to self-perceived social network characteristics and therefore does not capture reciprocity between participants and social ties, nor a collateral assessment of social network from family and/or friends. There may be utility in including these perspectives to capture the full picture of one's social network, as discrepancies between patient and family perspectives of social functioning and relationships can be common (Cowan et al., 2023; EnglandKennedy and Horton, 2011). Further, the LSNS-6 scale limits our findings to quantitative interpretations of social engagement, and even items capturing subjective measures such as "closeness" and "ease" in relationships are reported quantitatively. This makes it difficult to decipher whether the observed relationships between paranoia and social network are driven by the size of one's social network or by the perceived quality. Lastly, statistical power was constrained by sample size – particularly for subgroup analyses of EP and CP groups – and by incomplete longitudinal data, with Week 24 data available for only 50 % of the clinical sample (SSD; $N = 34$).

In sum, social networks are severely disrupted in those with SSDs, and these disruptions remain stable during recovery from a psychotic exacerbation, regardless of illness duration. Individuals who discharge with less engagement with family may be at a higher risk for worsening paranoia, while those with higher levels of paranoia after discharge may experience difficulties maintaining relationships with friends. Reduced social networks were also significantly related to greater negative symptom severity. Future research is needed to incorporate more robust qualitative and functional assessments of social networks. Better understanding the differential impact of qualitative versus quantitative social network characteristics on symptom severity may be important when developing effective, targeted interventions. Finally, based on our assertion that social networks breakdown early, potentially preceding illness onset in SSDs, future research should aim to tailor social engagement interventions for individuals at clinical high risk.

CRediT authorship contribution statement

Gabriella V. Schock: Writing – original draft, Visualization, Methodology, Formal analysis, Conceptualization. **Andrew R. Kittleson:** Writing – review & editing, Methodology, Formal analysis, Conceptualization. **Annalise S. Halverson:** Writing – review & editing, Methodology, Formal analysis, Conceptualization. **Jinyuan Liu:** Writing – review & editing, Methodology, Formal analysis, Conceptualization. **Julia M. Sheffield:** Writing – review & editing, Supervision, Resources, Project administration, Methodology, Funding acquisition, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.psychres.2026.116960.

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