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Associations Between Childhood Area-Level Social Fragmentation, Maladaptation to School, and Social Functioning Among Healthy Youth and Those at Clinical High Risk for Psychosis

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***Background and Hypothesis*: Although studies have identified social fragmentation as an important risk factor for schizophrenia and other psychotic disorders, it is unknown whether it may impact social functioning. This study investigates whether social fragmentation during childhood predicts maladaptation to school as well as social functioning during childhood and adulthood. *Study Design*: Data were collected from the North American Prodrome Longitudinal Study. Participants included adults at clinical high risk for psychosis (CHR-P) and healthy comparisons (HC). Maladaptation to school and social functioning during childhood were assessed retrospectively and social functioning in adulthood was assessed at baseline. *Study Results*: Greater social fragmentation during childhood was associated with greater maladaptation to school (adjusted $\beta = 0.21$; 95% CI: 0.02 to 0.40). Social fragmentation was not associated with social functioning during childhood (unadjusted $\beta = -0.08$; 95% CI: −0.31 to 0.15). However, greater social fragmentation during childhood predicted poorer social functioning in adulthood (adjusted $\beta = -0.43$; 95% CI: −0.79 to −0.07). Maladaptation to school mediated 15.7% of the association between social fragmentation and social functioning. The association between social fragmentation and social functioning was stronger among adults at CHR-P compared to HC (adjusted $\beta = -0.42$; 95% CI: −0.82 to −0.02). *Conclusions*: This study finds that social fragmentation during childhood is associated with greater maladaptation to school during childhood, which in turn predicts poorer social functioning in adulthood. Further research is needed to disentangle aspects of social fragmentation that may contribute to social deficits, which would have implications for the development of effective interventions at the individual and community levels.**

Key words: clinical high risk for psychosis/maladaptation to school/social fragmentation/social functioning

Introduction

The impairments in social functioning associated with schizophrenia and other psychoses are strong predictors of the daily disability often observed in the disorder.[1] In addition, impairments in social functioning are long-lasting among many with schizophrenia.[2] Therefore, there has been a growing focus on improving social functioning and identifying its determinants. Deficits in social functioning have also been shown to predict the onset of psychosis.[3] Studies with youth at clinical high risk for psychosis (CHR-P) and those with first-episode psychosis have identified symptom severity, neurocognition, and

social cognition to be associated with social functioning.[4,5] However, little is known about the environmental factors that may contribute to impaired social functioning early in the pre-psychosis illness state.

Studies have suggested that social functioning does not depend exclusively on psychopathology, but may be influenced by a range of environmental characteristics including social and economic living conditions.[6,7] In nonhuman primates, early exposure to social adversity adversely impacts social functioning later in life.[8] For humans, exposure to adverse social environments during childhood and adolescence is a risk factor for schizophrenia,[9–11] and may also negatively impact social functioning.

One social environmental characteristic that has been shown to be associated with schizophrenia in a recent systematic review is area-level social fragmentation.[12] In this review, studies used various geographical area-level characteristics to construct social fragmentation indices to empirically measure the disruption of social ties in communities.[12] This construct is based on Social Disorganization Theory, which posits that social processes within communities, such as social cohesion, social norms, and collective efficacy, are likely structural factors that contribute to the well-being of children's development and health.[13,14] Studies that have used area-level residential instability (percentage of people who moved)[15,16] along with percentage of renter-occupied housing[16,17] and percentage of people divorced[18,19] to measure social fragmentation have demonstrated consistent associations with greater prevalence and incidence as well as earlier age at onset of non-affective psychotic disorders including schizophrenia. Another study showed that school-level social fragmentation (measured by percentage of children raised in single-parent households and percentage of children who moved) partially explained the association between urban upbringing and the onset of a non-affective psychotic disorder.[20]

In this study, we defined childhood exposure to area-level social fragmentation as the disruption of social ties and relationships among residents and families in a community.[12] Social fragmentation, social disorganization,[21] and social isolation[22] share similar area-level variables, and are often used interchangeably. Although social capital is a related concept characterized by high levels of civic participation, social networks, and trust, it differs from social fragmentation— social capital is operationalized by voter turnout[23] and volunteering[24] while social fragmentation is operationalized by greater transiency and percent of the non-nuclear family.[12] Social disadvantage is another distinct term, measured using various individual-level characteristics such as living alone, single status, and unemployment.[25]

The area-level characteristics that have been used to measure social fragmentation may interact in complex processes, but prior studies have repeatedly found the social fragmentation index to be associated with psychotic disorders.[12] These studies postulated that growing up in socially fragmented communities may lead to a greater vulnerability toward social maladjustment.[12] Furthermore, chronic and repetitive adverse social stressors associated with poor peer relationships may increase the risk for psychosis among youth at CHR-P.[11] Similarly, these social stressors may also impact social functioning. For children to acquire social skills, they would need to make social decisions appropriate for the context they are in and behave in a manner that aligns with the social expectations of their peer group.[26,27] Social norms, which refer to the shared expectations regarding attitudes, beliefs, and behaviors in a given situation, can facilitate the process of social decision-making.[28,29] Children who struggle to comprehend and conform to the social norms of their group, especially in a normless social environment (ie, anomie),[30] may face adverse social outcomes like rejection from peers, negative emotions, and exclusion.[29,31,32] Children living in areas with greater anomie or social fragmentation may encounter more difficulties adapting to their social environment (eg, in schools),[33] and this in turn, may impact social functioning. However, the nature of the association between social fragmentation, maladaptation to school, and social functioning has not been examined in CHR-P individuals.

This study investigates whether exposure to area-level social fragmentation during childhood is associated with (1) maladaptation to school, (2) social functioning during childhood, and (3) social functioning later in young adulthood, which includes youth older than 19 years as defined by the World Health Organization.[34] Furthermore, we examine whether maladaptation to school is a mediator of the relationship between social fragmentation and social functioning. Because those at high-risk to develop schizophrenia and other psychotic disorders may have been more vulnerable to the impact of their social environment during childhood,[35] we also investigate CHR-P status as a potential moderator of the relationship between social fragmentation and social functioning. Whereas our prior analysis examined the association between residential instability and future risk for psychosis in a different subset of the sample (adolescents at CHR-P as opposed to adults at CHR-P and healthy comparisons (HC) in this current study),[11] in this current study, we focus on the social fragmentation construct in childhood as it predicts future social functioning among only adult participants at CHR-P and HC, which, to the best of our knowledge, has never been studied before.

In this study, we operationalized childhood exposure to area-level social fragmentation as communities with greater transiency (percentage of those who moved and those who are renting) and non-nuclear families (percentage of single-parent households and percentage of divorced) households and percent divorced.[12] We hypothesized that (1) greater area-level social fragmentation

during childhood would be associated with greater maladaptation to school, and impaired social functioning during childhood and young adulthood. In addition, the relationships between social fragmentation during childhood and social functioning during childhood and young adulthood would be, (2) mediated by poor adaptation to school, and (3) moderated by CHR-P status.

Methods

Participants

Participants were those with CHR-P status and healthy comparison (HC) individuals in the North American Prodrome Longitudinal Study Phase 2 (NAPLS2).[36] Both youth at CHR-P and HC were recruited from the same 8 sites and they were either referred by social service agencies, educators, or healthcare providers, or they self-referred after being informed through extensive community education initiatives.[36] Participants were young adults with ages greater than 19 and had available data on sociodemographic characteristics including childhood residence (cities/towns) suitable for geo-coding and clinical characteristics including premorbid adjustment during childhood and social functioning in young adulthood. We excluded participants who were not young adults (aged 19 and younger) because they did not have measures of social functioning in adulthood. Of the 413 adult participants in NAPLS2, only 233 were included in this study. 180 were excluded mainly due to missing data since majority of these participants were from the Canadian site and only those with available towns in the United States that could be geocoded to county-level characteristics were included. Baseline data were collected from June 2009 through April 2013.[36] CHR-P status was determined by the Criteria of Prodromal Syndromes, which is based on the Structured Interview for Psychosis Risk Syndromes (SIPS).[37,38] Exclusion criteria for youth CHR-P included IQ of 70 or less, lifetime psychotic disorder diagnosis, or significant central nervous system disorder. HCs were excluded if they were taking antipsychotics or have a first-degree relative with psychosis.[36] Institutional Review Board approval at each site was obtained.

Instruments

Sociodemographic and clinical variables were obtained from self-report and interview-based measures at the time of baseline assessment and included age, sex, family history of mental illnesses, race/ethnicity, parental education, trauma, and city or town in which the participant lived the longest time during childhood.[36] The Family Interview for Genetic Studies was used to assess family history of mental illnesses, which included bipolar disorder, depression, or psychotic disorders among first- or second-degree family members.[39] High school parental education was computed as having completed high school or above as the highest education level attained by either the mother or father. Total traumatic events were assessed by using the Childhood Trauma and Abuse scale,[40] a semi-structured interview, which assesses trauma or abuse before the age of 16 years.[41] These events included bullying at school (psychologically), bullying at school (physically), emotional neglect from people at home, psychological abuse, physical abuse, and sexual abuse.

Area-Level Variables

Area-level characteristics, including social fragmentation, were derived from county-level data from the US Decennial Censuses.[42] Cities or towns where individuals spent the majority of their childhood were linked to the primary county 5-digit Federal Information Processing Standards codes.[43] Then, 1990 county-level characteristics were linked to those codes for participants born between 1985 and 1994, and 2000 county-level characteristics were linked to those born between 1995 and 2000. Censuses from these 2 time periods would capture the area (county) characteristics during the participants' childhood. Social fragmentation was measured as the average z-scores of the following area-level characteristics which have been previously shown to measure social fragmentation and to be associated with psychotic disorders: Residential instability (percent of residents who changed their address in the past 5 years),[15,16] percent of residents with renter-occupied housing,[16,17] percent of residents who are divorced,[18,19] and percent single-parent households.[20] Area-level socioeconomic (SES) deprivation was measured as the average z-scores of percent of residents with less than ninth-grade education, percent of residents living below the poverty line, and percent of residents who are unemployed.[44,45] A comparison of participants with available data and those with missing data are shown in supplementary table S1. And a comparison of demographic characteristics among adults at CHR-P and HC are shown in supplementary table S2.

Maladaptation to School and Social Functioning During Childhood

The Premorbid Adjustment Scale (PAS) is an interview-based rating scale designed to assess elementary school adjustment and social functioning retrospectively.[46,47] The PAS is rated on a 7-point scale based on an interview with the patient, with higher ratings representing greater maladjustment. The current study focused on the childhood (age 5–11) period of development to align with the childhood exposure to social fragmentation. The PAS assessed maladaptation to school during childhood with one item: Adaptation to school. The PAS assessed social functioning during childhood with the following 2 items: (1) sociability and withdrawal and (2) peer relationships.

We averaged these 2 items and as done in prior literature to measure overall social functioning during childhood.[48] This measure was also reversed such that higher ratings represented greater social functioning.

Social Functioning at Young Adulthood

Social functioning in young adulthood was assessed with the GF:Social scale, which ranges from 1 to 10, with higher ratings representing better social functioning.[49] The GF:Social scale assesses peer conflict, peer relationships, family involvement, and age-appropriate intimate relationships.

Statistical Analysis

Bivariate correlations of all variables assessed multicollinearity (supplementary table S3). Three generalized linear mixed regression models were fitted with social fragmentation during childhood as the independent variable and maladaptation to school, social functioning during childhood, and social functioning in young adulthood as 3 separate dependent variables. There were 88 unique counties, which were random intercepts in the models because individuals were clustered within counties. For models with significant associations, we then adjusted for 7 covariates including age, sex, family history of mental illnesses, White non-Hispanic race/ethnicity, high school parental education, total traumatic events, and area-level SES deprivation. For the associations that remained significant after adjusting for the 7 covariates, we then tested the associations between school maladaptation and social functioning during childhood and adulthood and tested maladaptation to school as a potential mediator of the association between social fragmentation during childhood and social functioning. Indirect effects were estimated using 5000 bootstrap replications, which produced 95% confidence intervals. To test the potential moderating role of CHR-P status on this association, the interaction term social fragmentation-by-CHR-P status was entered as a fixed factor. R statistical software package was used for all analyses.

Results

This study included 223 young adults with 122 males (54.7%), 113 (50.7%) white non-Hispanics, 138 (61.9%) individuals with a family history (first- and second-degree family members) of mental illnesses, 67 (30%) with a family history of psychosis, and 32 (14.3%) with a parental history of psychosis. The average age was 21.8 and there were 133 youth at CHR-P and 90 HC. A summary of the rest of the demographic characteristics is detailed in table 1. For area-level characteristics, social fragmentation was positively and significantly correlated with SES deprivation (Pearson correlation coefficient = 0.491).

Main Effects

Greater social fragmentation was associated with greater maladaptation to school in childhood (unadjusted β = 0.25; 95% CI: 0.08 to 0.42) even after adjusting for 7 covariates (adjusted β = 0.21; 95% CI: 0.02 to 0.40) (table 2). However, social fragmentation was not associated with social functioning in childhood (unadjusted β = −0.08; 95% CI: −0.31 to 0.15) (table 3). In contrast, greater social fragmentation during childhood predicted poorer social functioning in young adulthood (unadjusted β = −0.56; 95% CI: −0.86 to −0.26) even after adjusting for 7 covariates (adjusted β = −0.43; 95% CI: −0.79 to −0.07) (table 3).

Mediator Analysis

Greater maladaptation to school during childhood predicted poorer social functioning in adulthood (adjusted β = −0.38; 95% CI: −0.61 to −0.15). Maladaptation to school had a significant indirect effect on the association between social fragmentation during childhood and social functioning in adulthood (adjusted β = −0.07; 95% CI: −0.17 to <−0.01) and explained 15.7% of this association (figure 1).

Moderator Analysis

CHR-P status significantly interacted with social fragmentation during childhood in predicting poorer social functioning in adulthood (adjusted β = −0.42; 95% CI: −0.82 to −0.02) such that the slope between social fragmentation and social functioning was more negative among youth at CHR-P compared with HC (figure 2). For secondary analysis, we conducted bivariate correlations of sociodemographic and clinical characteristics among CHR-P and HC separately (supplementary tables S4 and S5).

Discussion

Area-level social fragmentation has long been linked with schizophrenia and other psychotic disorders, but whether it may impact social functioning has not been known. This is the first study to show that greater area-level social fragmentation predicts social functioning impairment in young adulthood even after adjusting for covariates including area-level SES deprivation. This finding builds upon previous literature demonstrating the associations between measures of social fragmentation and non-affective psychosis incidence, prevalence, and onset of disorder.[12] Measures of social fragmentation and urban upbringing have also been shown to be associated with reductions of gray matter volume in similar brain areas that are involved with exposure to social stress.[50,51,52] It has been postulated from these studies that it may be particularly difficult for youth to fit in or

Table 1. Sociodemographic Characteristics (n = 223)

		N (%)
Age		
	19.0 to 21.0	100 (44.8)
	21.1 to 23.0	76 (34.1)
	23.1 to 35.0	47 (21.1)
Sex		
	Male	122 (54.7)
	Female	101 (45.3)
Race		
	East Asian (eg, Chinese, Japanese, Korean)	10 (4.5)
	Southeast Asian (eg, Cambodian, Indonesian, Vietnamese)	5 (2.2)
	South Asian (eg, East Indian, Pakistani, Sri Lankan)	9 (4)
	Black (eg, African, African Caribbean)	39 (17.5)
	Central/ South American	9 (4)
	West/Central Asia and Middle East (eg, Egyptian, Lebanese, United Arab Emirates, Afghanistan, and Iranian)	1 (0.4)
	White (European)	122 (54.7)
	Native Hawaiian or Pacific Islander	1 (0.4)
	Interracial	27 (12.1)
Hispanic/Latino ethnicity		
	Yes	40 (17.9)
	No	183 (82.1)
Family history of mental illnesses		
	Yes	138 (61.9)
	No	85 (38.1)
Highest parental education		
	Completed high school or above	211 (94.6)
	Did not complete high school	12 (5.4)
Trauma		
	None known	101 (45.3)
	Experienced one or more traumatic events	122 (54.7)
		Mean (SD)
Area-level characteristics in childhood		
	Percentage of residents who lived in a different address in the past 5 years	50.1 (8.7)
	Percentage of residents who lived in renter-occupied housing	59.2 (11.3)
	Percentage of single-parent household	7.8 (2.0)
	Percentage of residents who are divorced	8.4 (1.6)
	Percentage of residents who are unemployment	5.8 (1.6)
	Percentage of residents who lived under the 185% poverty line	23.6 (7.8)
	Percentage of residents who did not complete ninth grade	9.2 (3.9)
Clinical characteristics in childhood		
	Maladaptation to school	0.8 (1.0)
	Social functioning	5.8 (1.2)
Clinical characteristics in adulthood		
	Social functioning	7.4 (1.8)
		N (%)
	Clinical high risk for psychosis	133 (59.6)
	Healthy comparisons	90 (40.4)

Table 2. Generalized Linear Mixed Models Predicting Maladaptation to School in Childhood

	Univariate Model Predicting Maladaptation to School in Childhood				Multivariable Model Predicting Maladaptation to School in Childhood			
Parameters	Estimate	SE	95% CI	*P*	Estimate	SE	95% CI	*p*
Age	<0.01	0.04	−0.08 to 0.07	.957	0.02	0.04	−0.06 to 0.09	.648
Female sex	**−0.34**	**0.13**	**−0.58 to −0.09**	**.009**	**−0.30**	**0.13**	**−0.54 to −0.05**	**.021**
White non-Hispanic	0.03	0.13	−0.22 to 0.28	.800	0.07	0.13	−0.18 to 0.32	.599
Family history of mental illnesses	**0.04**	**0.13**	**−0.22 to 0.30**	**.006**	**0.28**	**0.13**	**0.02 to 0.54**	**.035**
High school parental education	0.26	0.28	−0.29 to 0.82	.355	0.40	0.28	−0.16 to 0.95	.161
Total traumatic events	**0.13**	**0.04**	**0.05 to 0.22**	**.002**	**0.10**	**0.04**	**0.02 to 0.18**	**.017**
Area-level SES Deprivation	0.12	0.09	−0.05 to 0.29	.168	0.03	0.09	−0.15 to 0.20	.776
Area-level Social Fragmentation	**0.25**	**0.09**	**0.08 to 0.42**	**.009**	**0.21**	**0.10**	**0.02 to 0.40**	**.040**

All models used generalized linear mixed models with counties as the random intercept.
All significant associations ($P < .05$) are shown in bold.

integrate into communities with greater social fragmentation. Furthermore, chronic and repetitive exposure to adverse social environments over time would in turn lead to impaired social functioning, which along with genetic vulnerability may play a role in the development of psychosis.[35,53]

Moreover, we show that the relationship between greater social fragmentation during childhood and impaired social functioning in young adulthood is mediated by greater maladjustment to school during childhood. These findings build upon a study demonstrating that social fragmentation measured at the school level partially explained the association between urban upbringing and non-affective psychotic disorder.[20] Since our measure of social fragmentation during childhood was measured at the county level, perhaps, it may be a proxy for social fragmentation at the school level, which may be reflected by high turnover of students and teachers, leading to the disruption of the social connections and friendships in schools, and making it more difficult for children to socially adjust.

Interestingly, area-level social fragmentation during childhood was not associated with social functioning during childhood. This lack of association may indicate that certain social deficits (eg, in peer relationships) may not be observed immediately after exposure to adverse social environments. These findings parallel research on the impact of social isolation on social deficits in mice.[54] In this mouse model, sociability deficits caused by social isolation during the juvenile period were not observed immediately after a period of social isolation, but only later in adulthood. The study authors suggested that the observed social deficits may be caused not by a lack of social experiences per se, but by the need to adapt to the environment.[54] Furthermore, they propose that mice have a juvenile window that serves as a sensitive period for behavioral plasticity. Once this window closes, the mice may not possess the ability to modify their social strategy.[54] Similarly in humans, it could be that social functioning deficits from repetitive exposure to social fragmentation during a sensitive period (eg, childhood) would not be observed immediately after childhood, but later in adulthood. Recent studies demonstrate that greater moving distances as well as greater number of moves during childhood and adolescence predicted psychosis onset even after adjusting for socioeconomic status and parental history of severe mental illnesses including psychotic disorders.[10,55] However, greater cumulative distances moved during adulthood was not associated with increased psychosis risk.[10] Perhaps, the stress from disruption of social networks during specific developmental periods (eg, childhood), which has been measured at the individual level (eg, number of moves) or at the community level (eg, social fragmentation), may be critical in the development of psychosis later in life.

In this study, we also found that CHR-P status during young adulthood moderated the association between social fragmentation during childhood and social functioning in young adulthood such that this association is more negative among youth at CHR-P compared with HC. These results suggest that youth at high risk for psychosis may be more vulnerable to the adverse effects of social fragmentation during childhood. It is not clear whether social fragmentation may have impacted children to develop prodromal symptoms along with having poorer social functioning or if there may be an interactive effect of environment and genetic liability in impacting social functioning. Also, it is possible that families with greater genetic liability may have moved to more socially fragmented areas. Future studies should investigate the longitudinal trajectories of children who live in socially fragmented areas and track their prodromal symptoms and social functioning to better understand this.

There are several limitations to this study. First, the data were collected at baseline and reports of premorbid adjustment during childhood could have been influenced by recall error. However, childhood exposure to social fragmentation was linked to an external data source that

Table 3. Generalized Linear Mixed Models Predicting Social Functioning in Childhood and Adulthood

	Univariate Model Predicting Social Functioning in Childhood				Multivariable Model Predicting Social Functioning in Childhood				Univariate Model Predicting Social Functioning in Adulthood				Multivariable Model Predicting Social Functioning in Adulthood			
Parameters	Estimate	SE	95% CI	*P*	Estimate	SE	95% CI	*P*	Estimate	SE	95% CI	*P*	Estimate	SE	95% CI	*P*
Age	−0.01	0.05	−0.11 to 0.09	.874	0.00	0.05	−0.09 to 0.10	.926	−0.13	0.07	−0.27 to 0.02	.090	−0.10	0.07	−0.24 to 0.03	.131
Female sex	0.09	0.17	−0.23 to 0.41	.589	0.02	0.17	−0.31 to 0.34	.916	**0.63**	**0.24**	**0.16 to 1.10**	**.009**	**0.57**	**0.22**	**0.13 to 1.00**	**.011**
White non-Hispanic	0.28	0.16	−0.04 to 0.60	.084	0.24	0.17	−0.09 to 0.57	.158	0.21	0.24	−0.27 to 0.69	.387	0.00	0.23	−0.44 to 0.44	.992
Family history of mental illnesses	0.23	0.17	−0.10 to 0.57	.172	−0.14	0.17	−0.48 to 0.20	.420	**−0.94**	**0.25**	**−1.43 to −0.46**	**<.001**	**−0.63**	**0.24**	**−1.10 to −0.17**	**.008**
High school parental education	0.10	0.37	−0.62 to 0.82	.784	−0.07	0.37	−0.79 to 0.66	.859	**1.35**	**0.53**	**0.30 to 2.39**	**.012**	0.90	0.49	−0.07 to 1.87	.070
Total traumatic events	**−0.22**	**0.05**	**−0.32 to −0.12**	**<.001**	**−0.20**	**0.06**	**−0.31 to −0.10**	**<.001**	**−0.48**	**0.08**	**−0.62 to −0.33**	**<.001**	**−0.38**	**0.07**	**−0.53 to −0.24**	**<.001**
Area-level SES Deprivation	−0.07	0.11	−0.28 to 0.14	.522	−0.07	0.11	−0.30 to 0.15	.520	−0.07	0.17	−0.41 to 0.27	.678	0.16	0.16	−0.16 to 0.48	.336
Area-level Social Fragmentation	−0.08	0.12	−0.31 to 0.15	.501	0.03	0.13	−0.21 to 0.28	.796	**−0.56**	**0.15**	**−0.86 to −0.26**	**<.001**	**−0.43**	**0.18**	**−0.79 to −0.07**	**.022**

All models used generalized linear mixed models with counties as the random intercept.
All significant associations ($P < .05$) are shown in bold.

provided an objective measure. Second, the sample size in this study was modest. For secondary analysis among the CHR-P status subgroup, we were not able to observe significant correlations between maladaptation to school in childhood and social functioning in adulthood likely due to the sample size being underpowered. However, the negative directionality of this association was consistent with the main group. Third, the area-level social fragmentation construct may be a proxy for an objective environmental exposure to social deprivation and isolation, but we did not collect information about perceived social deprivation and isolation at the neighborhood and school levels during childhood and therefore, it is unknown at which of these levels social fragmentation may be more relevant as it relates to social functioning. Future studies should collect social fragmentation scores at the census tract and school levels and at various time periods longitudinally to assess its impact on social functioning and psychosis risk. Fourth, social functioning is a broad concept with several different components, but our assessment may be limited by using a brief rating scale. We assessed social functioning in childhood by averaging the scores on 2 items (sociability and withdrawal, and peer relationships) while maladaptation to school was measured with one item: Adaptation to school. Further studies should use more detailed assessments to examine various domains of social functioning.

This study is situated within a growing body of literature on the social determinants of psychosis and delves deeper into the longstanding known relationship between social fragmentation and psychosis.[12,56] There are several potential implications from these findings. First is that by further disentangling the different social determinants of psychosis, we are able to better understand the mechanisms and psychopathology of schizophrenia and the social deficits of this disease. Understanding mechanisms would then have implications for developing effective interventions at the individual and community levels. For example, if those with chronic exposure to social fragmentation during childhood have greater negative core schemas or poorer social cognition, targeted interventions at specific developmental time periods may help prevent impairment of social functioning and help youth live more meaningful lives. In addition, allocation of mental health resources that improve social connections may be guided by measures of social fragmentation in our communities.

Conclusion

This is the first study to find associations between area-level social fragmentation during childhood and social functioning in early adulthood. This association is mediated by greater maladaptation to school during childhood. Moreover, this association is moderated by CHR-P status such that childhood social fragmentation has an even stronger negative association with adult social

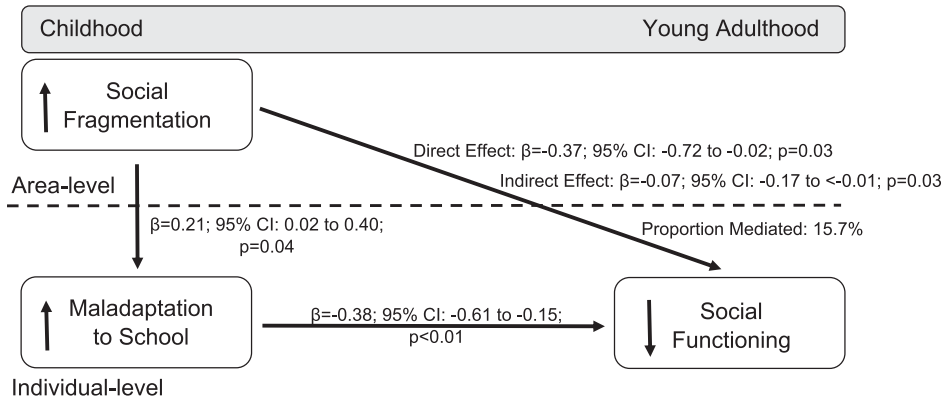

Fig.1. Greater maladaptation to school in childhood mediates the association between higher levels of social fragmentation and poorer social functioning.

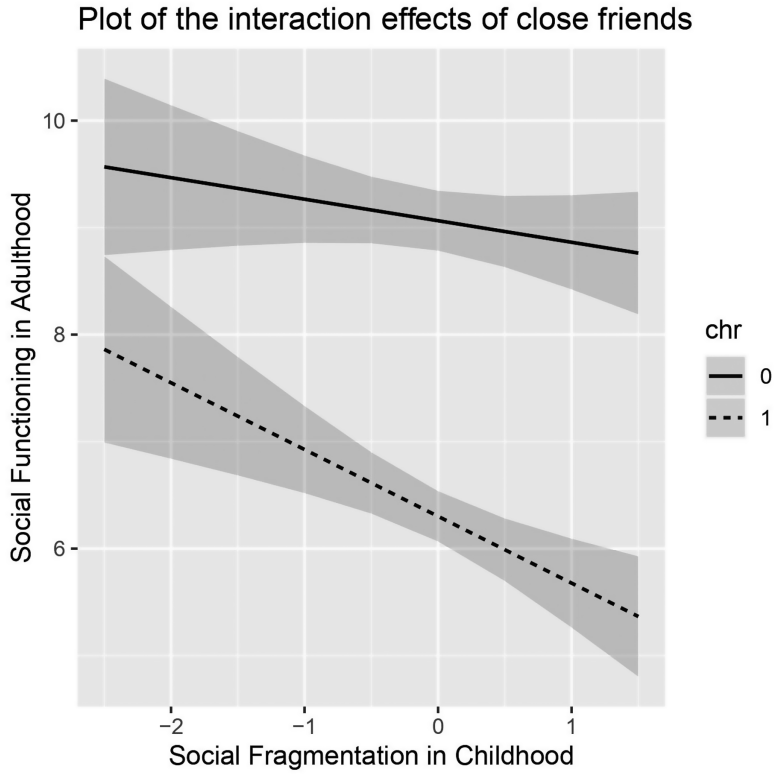

Fig. 2. Interaction between social fragmentation in childhood and clinical high risk for psychosis status predicts social functioning in adulthood. The solid line represents the best-fit line for healthy comparisons. The dotted line represents the best-fit line for adults at clinical high risk for psychosis. Clinical high risk for psychosis status was determined at baseline (adulthood).

functioning among youth at CHR-P compared to HC. Perhaps, exposure to cumulative social stressors from area-level social fragmentation during childhood might impact social functioning through repeated and chronic adverse social experiences (eg, lack of feeling of belongingness or not fitting in) at school. Further research will be needed to explore the mechanisms of these associations and better understand the perceived experiences of area-level social fragmentation, the specificity of the developmental time period of exposure in relation to its impact on social functioning, and how social fragmentation and genetic vulnerability might interact in contributing to poorer social functioning.

Supplementary Material

Supplementary material is available at https://academic.oup.com/schizophreniabulletin/.

Conflict of Interests Statement

Disclosures statement: Dr. Cannon has served as a consultant for Boehringer-Ingelheim Pharmaceuticals and Lundbeck A/S. Dr. Mathalon has served as a consultant for Aptinyx, Boehringer-Ingelheim Pharmaceuticals, Cadent Therapeutics, and Greenwich Biosciences. Dr. Perkins has served as a consultant for Sunovion and Alkermes, has received research support from Boehringer-Ingelheim, and has received royalties from American Psychiatric Association Publishing. Dr. Woods has received investigator-initiated research support from Pfizer and sponsor-initiated research support from Auspex and Teva; he has served as a consultant for Biomedisyn (unpaid), Boehringer-Ingelheim, and Merck and as an unpaid consultant to DSM-5; he has been granted a patent for a method of treating prodromal schizophrenia with glycine; and he has received royalties from Oxford University Press. The other authors report no financial relationships with commercial interests.

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