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Development of social functioning in preadolescent children at familial high-risk of schizophrenia or bipolar disorder – a 4-year follow-up study from age 7 to 11

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ABSTRACT

Social functioning is a major indicator of psychosis risk and evidence is lacking regarding social functioning development during preadolescence in children at familial high risk of schizophrenia (FHR-SZ) or bipolar disorder (FHR-BP). We aimed to investigate development of social functioning from age 7 to 11 in children at FHR-SZ or FHR-BP compared with population-based controls. At 4-year follow-up, 179 children at FHR-SZ (mean age 12.0 y, SD 0.3), 105 children at FHR-BP (mean age 11.9 y, SD 0.2), and 181 controls (mean age 11.9 y, SD 0.2) participated. We used the Vineland-II to measure social functioning. Development of social functioning was non-significantly different across groups on the Socialization Composite score as well as the subscales Interpersonal Relations, Play and Leisure, and Coping Skills. At 4-year follow-up, children at FHR-SZ demonstrated impaired social functioning, whereas children at FHR-BP displayed social functioning comparable to controls except from impaired coping skills. From age 7 to 11, the maturational pace of social functioning in children at FHR-SZ and FHR-BP is parallel to that of controls. Children at FHR-SZ show stable social functioning deficits, whereas children at FHR-BP show normal social functioning except from emergence of discretely impaired coping skills at age 11.

1. Introduction

Impaired social functioning, defined as adaptive social behavior and engagement in everyday social life (Sparrow et al., 2006), is a core feature of both schizophrenia (Harvey et al., 2012) and bipolar disorder (Sanchez-Moreno et al., 2009) and owing to their premorbid presence (Bearden et al., 2000; Cannon et al., 1997), social functioning deficits are considered endophenotypes of both disorders (Weiser et al., 2005). Poorer premorbid social functioning predicts later transitioning to schizophrenia in the general population (Tarbox and Pogue-Geile, 2008) and later transitioning to psychosis in individuals at clinical high-risk of psychosis (Cornblatt et al., 2012; Dragt et al., 2011). Similarly,

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premorbid impaired social functioning increases the risk of psychosis in individuals with bipolar disorder (Cannon et al., 1997). Moreover, social functioning deficits in individuals with bipolar disorder are present in the acute phase and in later illness stages as well as in euthymic phases and in fully remitted individuals (Sanchez-Moreno et al., 2009; Tatay--Manteiga et al., 2018). In a study of social functioning development, individuals at clinical high-risk for psychosis lagged behind controls from early adolescence (ages 12–15) and into young adulthood (ages 18–21) (Velthorst et al., 2018). The individuals at clinical high risk of psychosis, who later converted to psychosis, stagnated in their social functioning development in late adolescence (between the ages of 15–18) and then lagged further behind in adulthood (between the ages of 21–23) compared with the individuals at clinical high-risk, who did not convert to psychosis. Investigating heterogeneity in social functioning development from 6 months to 20 years of age, four cross-diagnostic trajectories were identified in first-admission individuals with affective and non-affective psychotic disorders when compared with a never-psychotic control group: preserved, moderately impaired, severely impaired, and profoundly impaired (Velthorst et al., 2017). Moreover, from early adolescence to first admission, a substantial decline in social functioning was followed by relatively stable social functioning after illness onset, i.e., with no further loss or regain of social functioning (Velthorst et al., 2017).

Studies on social functioning in children of parents with schizophrenia and bipolar disorder are few. Cross-sectional evidence indicates that from early childhood through adolescence, children at familial high-risk of schizophrenia demonstrate social functioning deficits (Cohen's *d* ranges 0.25–0.85) that are predictive of later transition to psychosis (Hameed and Lewis, 2016; Horton et al., 2014; Niemi et al., 2005; Niemi et al., 2003). Cross-sectional evidence of impaired social functioning (Cohen's $d = 0.75$) has also been reported in unaffected adult first-degree relatives (age range 20–46 years) of individuals with bipolar disorder (Gkintoni et al., 2017), whereas unaffected offspring (age range 11–26 years) of mothers with bipolar disorder did not demonstrate social functioning deficits (Reichart et al., 2007). These cross-sectional findings may indicate a normal development of social functioning during childhood or a very slowly lag in development, where deficits do not emerge until adulthood in first-degree relatives of individuals with bipolar disorder. In our baseline study, we also identified social functioning deficits in 7-year-old children of parents with schizophrenia (Cohen's *d* range 0.43–0.50) but not in 7-year-old children of parents with bipolar disorder (Cohen's *d* range 0.15–0.17) (Christiani et al., 2019). However, development of social functioning in preadolescence remains to be investigated in prospective follow-up studies of children at familial high-risk of schizophrenia or bipolar disorder, which may help elucidate the distinct developmental pathways of these FHR populations. Due to the high degree of shared genetic risk factors between the two disorders, we included both children at familial high risk of schizophrenia and children at familial high risk of bipolar disorder. Evidence from a population-based study suggests a genetic correlation of 0.60 (r_g) with the shared genetic effects explaining 52% of the genetic variance in schizophrenia and 69% of the genetic variance in bipolar disorder (Lichtenstein et al., 2009). Thus, the existence of both shared and non-shared genetic underpinnings may affect the development of social functioning in offspring at FHR-SZ and FHR-BP in both comparable and distinct ways.

We aimed to investigate development of social functioning during preadolescence from age 7 to 11 in children at familial high-risk of schizophrenia (FHR-SZ) or bipolar disorder (FHR-BP) compared with population-based controls (hereafter controls). We hypothesized that children at FHR-SZ would display a developmental lag (i.e., slower maturational growth) in their social functioning compared with controls and that children at FHR-BP would display intermediate social functioning maturation.

2. Methods

2.1. Participants

In this population-based, prospective cohort study, baseline data were collected from January 2013 to January 2016 as part of the Danish High Risk and Resilience Study – VIA 7 (hereafter the VIA 7 study) (Thorup et al., 2015). Of 11,959 eligible children, 944 were approached. 249 refused and 173 were non-respondents. Participants included 522 children aged 7, of whom 202 children had at least one parent with schizophrenia spectrum psychosis (ICD-10 codes F20, F22, F25 or ICD-8 codes 295, 297, 298.29, 298.39, 298.89, 298.99), 120 children had at least one parent with bipolar disorder (ICD-10 codes F30, F31 or ICD-8 codes 296.19, 296.39), and 200 children had parents without any of these two disorders. There were nine children (eight children at FHR-SZ and one child at FHR-BP), where both parents were diagnosed with either schizophrenia or bipolar disorder. If one parent had schizophrenia and the other bipolar disorder, the child was assigned to the schizophrenia high risk group as per the ICD-10 hierarchy. Parental psychiatric diagnoses were re-assessed using the diagnostic interview Schedule for Clinical Assessment in Neuropsychiatry, version 2.1 (Wing, 1998) conducted by blinded and trained assessors who were either psychologists, doctors, or nurses supervised at clinical conferences by experienced users (specialist in child and adolescent psychiatry, AAET, and specialist in psychiatry OM). In the majority of cases, the register diagnosis was confirmed but there were also parents who no longer fulfilled the diagnostic criteria for the diagnosis in the register and cases with a diagnostic change. However, we kept the register-based diagnosis as an indication of the familial risk of the offspring because the index parent previously fulfilled the diagnostic criteria for either schizophrenia or bipolar disorder. Moreover, previous evidence has shown that the validity of the schizophrenia and bipolar diagnoses in the Danish Psychiatric Registers is high (Kessing, 1998; Uggerby et al., 2013). Of the entire sample, we obtained social functioning data on 192 children at familial high risk of schizophrenia (FHR-SZ), 107 children at familial high risk of bipolar disorder (FHR-BP), and 188 controls (Christiani et al., 2019). The first follow-up took place from March 2017 to June 2020 as part of the Danish High Risk and Resilience Study – VIA 11 (hereafter the VIA 11 study) (Thorup et al., 2018). The total sample at follow-up comprised 465 children aged 11, of whom 179 children at FHR-SZ, 105 children at FHR-BP, and 181 controls participated (89,1% over all retention rate). We obtained permission from the Danish Ministry of Health to retrieve the cohort from the Danish registers, i.e., the Danish Civil Registration System (Pedersen et al., 2006) and the Danish Psychiatric Central Research Register (Mors et al., 2011). The Danish Data Protection Agency approved the study, and the guidelines of the National Committee for Health Research Ethics were followed. Due to the observational nature of the study, formal approval was not deemed necessary by his authority. Prior to enrolment, the participants received written and oral information about the study. The legal guardians of the children gave written consent, and the children gave assent to participate. The controls were matched to children at FHR-SZ on age, sex, and municipality. The FHR-BP group was unmatched, but comparable to the other two groups concerning age and sex. All children had Danish as their first language.

2.2. Procedures

The assessments were carried out at two research sites in Copenhagen and Aarhus, Denmark or, in some cases, in suitable surroundings in the homes of the families. The assessors were trained psychologists, physicians, or nurses that were instructed and supervised by a specialist in child neuropsychology (J.R.M.J.), a clinical psychologist (C.J.C.), and a specialist in clinical child psychology (N.H.). Inter-rater reliability was calculated using Krippendorff's Alpha on 10 interviews (on the Socialization subdomain from the Vineland Adaptive Behavior Scales, Second

Edition (Vineland-II) (Sparrow et al., 2006)) rated by all assessors and showed an acceptable agreement with $\alpha > 0.80$ across the three subscales (Subscale I $\alpha = 0.98$; Subscale II $\alpha = 0.88$; Subscale III $\alpha = 0.96$).

2.3. Measures

2.3.1. Assessment of global functioning, dimensional psychopathology, and general intelligence

We used the Children's Global Assessment Scale (CGAS) (Shaffer et al., 1983) to assess current level of global functioning (higher scores indicate better functioning), the Child Behavior Checklist School-Age Version (CBCL) (Achenbach and Rescorla, 2001) to assess the level of problem behavior (higher scores indicate more problem behavior) rated by the primary caregiver (defined as the parent/legal guardian spending the most time with the child), and the Reynold's Intelligence Screening Test (RIST) (Reynolds and Kamphaus, 2003) to asses an IQ estimate.

2.3.2. Assessment of social functioning

Social functioning was measured using the subdomain Socialization from the Vineland-II (Sparrow et al., 2006). The subdomain Socialization includes the three subscales Interpersonal Relationships, i.e., how the child interacts with others (39 items), Play and Leisure, i.e., how the child plays and uses leisure time (31 items), and Coping Skills, i.e., how the child regulates behavior and demonstrates responsibility to others (29 items). Response options to all items are 2 (usually), 1 (sometimes or partially), 0 (never), or "don't know". A higher score indicates a higher level of social functioning. The test-retest reliability is > 0.85 on all subscales (Sparrow et al., 2006). At follow-up (age 11), the Vineland-II domain Socialization was ascertained through a semi-structured interview with the primary caregiver to establish the level of the child's adaptive behavior within the domain of social functioning. At baseline (age 7), social functioning was assessed with the Vineland-II rating form for parents/caregivers, which covers the same content as the interview format. Upon oral instructions by the assessor, the questionnaire was filled in by the primary caregiver (Christiani et al., 2019). The test-retest reliability coefficients between the parent-rated questionnaire and interview-administered version of the Vineland-II subscales Interpersonal Relations, Play and Leisure, and Coping Skills are 0.76, 0.75, and 0.81, respectively, as reported in the Vineland-II manual (pp 93–96) (Sparrow et al., 2006).

2.4. Statistical analysis

Demographic characteristics, global functioning, dimensional psychopathology, and general intelligence were compared using univariate analysis of variance and X^2 test as appropriate. Log transformation was applied to approximate a normal distribution (CBCL Total Score). Dropout analyses were performed with the X^2 test and independent samples *t*-test as appropriate. At both baseline (age 7) and follow-up (age 11), the three subscales from the Vineland-II Socialization subdomain were standardized into *z* scores using the control group means and standard deviations at baseline as reference groups. Finally, a Socialization composite score was generated by summing the three subscale *z* scores (in line with the Vineland-II manual (Sparrow et al., 2006)) followed by re-standardization. Multiple imputation followed by multi-level mixed effects linear regression models with maximum likelihood estimation were performed using familial high-risk group and time x group interaction as fixed factors to examine the development of social functioning (the Socialization composite score and the three subscale scores) from age 7 and 11 and the between-group differences at age 11. Owing to the inclusion of 16 sibling pairs, the multilevel mixed models were applied with each child nested in their family. Alpha was set to < 0.05. Effect size estimates were calculated using Cohen's *d*. In exploratory analyses, we examined the prevalence rate of children with abnormal social functioning development from age 7 to 11. Hence, we used regression equations to examine individual change in social functioning from age 7 to 11 by calculating the difference between the predicted score (based on the baseline score at age 7) and the observed score of each case at follow-up (age 11) ad modum Crawford and Garthwaite (Crawford and Garthwaite, 2007) (Supplementary Text). We used SPSS Statistics, version 28 (IBMCorp., 2021) and Stata/SE 15.1 (StataCorp, 2017) to conduct all statistical analyses.

3. Results

3.1. Sample characteristics at 4-year follow-up (age 11)

We found no significant between-group differences regarding sex, age, or general intelligence (Table 1). Both FHR groups demonstrated significantly more problem behavior and poorer global functioning compared with controls (all *P*-values $< .001$) but did not differ significantly from each other. We retrieved social functioning data on 169 children at FHR-SZ, 103 children at FHR-BP, and 171 controls. The largest fraction on missing information (FMI) ranged between 0.1900 and 0.2424 on the three Vineland subscales. There were no significant differences between children whose social functioning was assessed at age 11 and dropouts regarding sex (X^2 (1) = 0.126, $P = .72$) or FHR group (X^2 (2) = 0.228, $P = .89$). Those who were not assessed with the three Vineland-II subscales at age 11 did not significantly differ on global functioning at age 7 from those who participated, whereas they demonstrated significantly more problem behavior and significantly lower social functioning at age 7 compared with those who participated at follow-up (Supplementary Text).

3.2. Social functioning development from baseline to follow-up (age 7 to 11)

There were no significant time x group interactions in the Socialization Composite score (F(2, 13,061.7) = 1.46, $P = .23$), the Interpersonal Relations subscale (F(2,11,920.6) = 1.13, $P = .32$), the Play and Leisure subscale (F(2,15,335.0) = 2.32, $P = .10$), or the Coping Skills subscale (F(2,10,160.2) = 0.91, $P = .40$).

3.3. Social functioning at 4-year follow-up (age 11)

At 4-year follow-up, children at FHR-SZ demonstrated significant impairments in overall Socialization skills (Cohen's $d = 0.40$; $P = <.001$), Interpersonal Relations (Cohen's $d = 0.33$; $P = .001$), Play and Leisure (Cohen's $d = 0.30$ $P = .001$), and Coping Skills (Cohen's $d = 0.39$ $P < .001$) compared with controls. Children at FHR-BP did not perform significantly different from controls except in Coping Skills where they showed significant impairments (Cohen's $d = 0.24$; $P = .04$) (Table 2). Children at FHR-SZ displayed significantly poorer functioning than children at FHR-BP in Play and Leisure (Cohen's $d = 0.22$; $P < .05$) but did not perform significantly different in overall Socialization skills and the other two subdomains.

3.4. Individual change in social functioning from baseline to follow-up

Observed scores were available both at baseline (age 7) and follow-up (age 11) for a total of 419 children on the Interpersonal Relations subscale (FHR-SZ: $N = 162$; FHR-BP: $N = 95$; controls: $N = 162$), 391 children on the Play and Leisure subscale (FHR-SZ: $N = 151$; FHR-BP: $N = 87$; controls: $N = 153$), and 409 children on the Coping Skills subscale (FHR-SZ: $N = 158$; FHR-BP: $N = 93$; controls: $N = 158$). All except from three children had no significant difference between their predicted and obtained score at age 11 (data not shown). On the Interpersonal Relations subscale, one child had a significant difference between its predicted and obtained score (FHR-SZ: $N = 0$; FHR-BP: $N = 0$; controls: $N = 1$; [0,6%]) and on the Play and Leisure subscale, the same child and two other children had a significant difference between their predicted and obtained score (FHR-SZ: $N = 2$ [1,3%]; FHR-BP: $N = 0$; controls: $N = 1$;

Table 1
Demographic and Clinical Characteristics of Study Population at Follow-up (Age 11).

Variable	Controls	FHR-SZ	FHR-BP	P-value	Controls vs FHR-SZ	Controls vs FHR-BP	FHR-SZ vs FHR-BP
Children, No.	181	179	105	NA	NA	NA	NA
Female, No. (%)	83 (46)	85 (48)	46 (44)	.83	NA	NA	NA
Age, mean (SD)	11.93 (0.23)	11.96 (0.27)	11.93 (0.23)	.57	NA	NA	NA
CGAS[a], mean (SD) (Controls: $N = 175$; FHR-SZ: $N = 173$; FHR-BP: $N = 104$)	75.17 (13.97)	64.63 (15.61)	68.12 (14.94)	<.001	<.001	<.001	.06
CBCL[b] Total Score, mean (SD) (Controls: $N = 173$; FHR-SZ: $N = 165$; FHR-BP: $N = 102$)	12.75 (12.66)	23.70 (20.60)	21.61 (21.24)	<.001	<.001	<.001	.36
IQ estimate age 11 (RIST Index), mean (SD) (Controls: $N = 178$; FHR-SZ: $N = 170$; FHR-BP: $N = 102$)	97.54 (11.16)	95.35 (10.00)	97.17 (9.83)	.13	NA	NA	NA

Abbreviations: FHR-SZ, familial high risk of schizophrenia; FHR-BP, familial high risk of bipolar disorder; NA = Not applicable; CGAS, Children's Global Assessment Scale; CBCL, Child Behavior Checklist School-Age Version.
[a] The scale ranges from 1 to 100 with higher scores indicating a higher level of functioning.
[b] The scale ranges from 0 to 226 with higher scores indicating a higher level of problem behavior.

Table 2
Social Functioning in Children at FHR-SZ. FHR-BP, and Controls at Baseline (age 7) and Follow-Up (age 11).

Variable	Study Group, Estimated Means (95% CI), *z* Scores[a]				Pairwise Comparisons Between Groups					
	Controls $N = 200$	FHR-SZ $N = 202$	FHR-BP $N = 120$	*P*-value	Controls vs FHR-SZ		Controls vs FHR-BP		FHR-SZ vs FHR-BP	
					P-value	Cohen's *d*[b]	*P*-value	Cohen's *d*[b]	*P*-value	Cohen's *d*[b]
Baseline (age 7)										
Socialization Composite	0.02 (−0.18–0.21)	−0.69 (−0.89- −0.49)	−0.20 (−0.46- 0.07)	<.001[c]	<.001[c]	0.50	.21	0.16	.003[c]	0.34
Socialization Interpersonal Relations	0.01 (−0.19- 0.21)	−0.63 (−0.83–0.43)	−0.20 (−0.46- 0.07)	<.001[c]	<.001[c]	0.45	.21	0.14	.01[c]	0.29
Socialization Play and Leisure	0.02 (−0.16- 0.20)	−0.56 (−0.74- −0.38)	−0.17 (−0.41- 0.07)	<.001[c]	<.001[c]	0.46	.22	0.15	.01[c]	0.30
Socialization Coping Skills	0.01 (−0.18- 0.19)	−0.68 (−0.87- −0.50)	−0.18 (−0.43- 0.06)	<.001[c]	<.001[c]	0.51	.23	0.14	.001[c]	0.35
Follow-up (age 11)										
Socialization Composite	4.27 (4.10- 4.43)	3.76 (3.59- 3.93)	4.03 (3.81- 4.24)	<.001[c]	<.001[c]	0.40	.08	0.19	.06	0.22
Socialization Interpersonal Relations	5.73 (5.55- 5.92)	5.29 (5.09- 5.48)	5.55 (5.31- 5.79)	<.001[c]	.001[c]	0.33	.23	0.14	.10	0.19
Socialization Play and Leisure	3.71 (3.58- 3.84)	3.41 (3.27- 3.55)	3.63 (3.46- 3.80)	<.001[c]	.001[c]	0.30	.46	0.08	.05[c]	0.22
Socialization Coping Skills	2.36 (2.13- 2.58)	1.72 (1.50- 1.95)	1.97 (1.67- 2.27)	<.001[c]	<.001[c]	0.39	.04[c]	0.24	.21	0.15

Abbreviations: FHR-SZ, familial high risk of schizophrenia; FHR-BP, familial high risk of bipolar disorder.
[a] The three subscales from the Vineland-II Socialization domain were standardized into *z* scores using the control group mean at baseline (age 7) as reference group. Finally, a Socialization composite score was generated by summing the three subscale *z* scores followed by re-standardization.
[b] Due to the use of multiple imputations the baseline Cohen's *d* levels reported in the current study vary slightly from those reported in a previous publication.[18].
[c] *P*-value significant at the 0.05 level.

[0,7%]). All three children improved their performance from 1 to 3 SDs below their respective group mean at baseline to 0,5–1 SD above their respective group mean at follow-up.

4. Discussion

This prospective, population-based cohort study is the first to conjointly examine social functioning development in preadolescent, same-aged children of parents with schizophrenia or bipolar disorder using a well-validated and detailed assessment method. Development of social functioning did not differ across children at FHR-SZ or FHR-BP and controls from age 7 to 11. Importantly, this finding indicates that the maturational gain or speed is normal in both FHR groups with no signs of developmental lags during middle childhood. The low prevalence of children (< 1% across groups) that differed significantly from their predicted scores was in accordance with our group mean level findings of non-differential development across groups. Thus, heterogeneity in the development of social functioning seems very low during this developmental phase.

Our finding of stable deficits that neither progressed nor decreased in children at FHR-SZ, is comparable to cross-sectional evidence from familial high-risk studies of schizophrenia (Hameed and Lewis, 2016; Niemi et al., 2003). Other than discretely impaired coping skills at age 11, we did not detect any social impairments in children at FHR-BP. The latter part is in keeping with the limited cross-sectional evidence of normal social functioning in young offspring (ages 11–26) of mothers with bipolar disorder, whereas the finding of impaired coping skills is not (Reichart et al., 2007). This difference may be due to the wider age range in the study by Reichart et al. (Reichart et al., 2007), which may obscure a putative developmental lag in social functioning that is potentially present during adolescence and young adulthood. Differences in the assessment methods applied to measure social functioning may also explain the differing outcomes. The considerable number of items of the Vineland-II subscales (ranging between 29 and 39 items) may be more sensitive towards more subtle differences. The onset of discretely impaired coping skills (of small effect size) by age 11 in

children at FHR-BP may point towards a much earlier onset than previous evidence of social functioning deficits (of medium to large effect size) has shown in unaffected adult first-degree relatives (age 20–45) of individuals with bipolar disorder (Gkintoni et al., 2017) and the children at FHR-BP may be on a course of an increasing lag in this subdomain. Moreover, children at FHR-BP demonstrated significantly better social functioning in all domains at baseline than children at FHR-SZ, whereas at follow-up this was only the case for the subdomain Play and Leisure. Altogether, the latter findings may suggest that the FHR-BP group is starting to approximate the FHR-SZ group.

An increasing lag in social functioning from 12 years of age and into adulthood is present in youth at clinical high-risk of non-affective and affective psychosis (Velthorst et al., 2018). Ongoing and planned follow-up studies in the current cohort (at ages 15 (Thorup et al., 2022) and 19 respectively) will examine whether this may also be the case for children at FHR-SZ and/or FHR-BP and whether the established early impairments in social functioning (or later emerging impairments in the development of social functioning) are indicative of a higher risk for later transition to psychosis as evidenced in clinical and familial high-risk populations (Cornblatt et al., 2012; Dragt et al., 2011; Niemi et al., 2005; Niemi et al., 2003). Finally, heterogeneity in social functioning development is well documented in individuals with non-affective and affective psychosis (Velthorst et al., 2017) and may also become evident at a later developmental period in children at FHR-SZ or FHR-BP. The potential identification of distinct trajectories of social functioning and thereby, potentially, a subgroup with developmental lag among these FHR groups will be of clinical value and allow for more differentiated intervention studies targeting the developmentally most severely affected subgroups.

4.1. Strengths and limitations

Among the strengths of this study are the large, same-aged sample, the high retention rate, and the inclusion of two FHR groups and a control group being investigated prospectively at two time points in preadolescence. Limitations include the smaller sample size of the FHR-BP group and a relatively long follow-up period, which may prohibit the detection of more heterogeneous pathways. Attrition bias was indicated with more problem behavior and poorer social functioning at baseline in those children who dropped out than those who participated, although effect sizes were small. Despite the adequate to good test-retest reliability coefficients between the parent-rated questionnaire and interview-administered version of the Vineland-II, this difference in administration may have caused less accurate ratings at baseline, since the primary caregivers do not have the clinical experience and training of the assessors. Further, the use of a measurement method based on parent-report and a semi-structured interview with the parent may potentially introduce a risk of bias. However, a performance-based measure may be less ecologically valid and therefore less generalizable than a behavior-rated assessment method. Finally, the data extract from the Danish registers did not include information about psychotic features being present or not in the parents with bipolar disorder and thus additional analyses examining trajectories of social functioning among children with a parent with any psychotic disorder (BP or SZ) compared to those without psychosis were not conducted.

4.2. Conclusions

In this prospective, population-based cohort study, we identified non-differential development of social functioning during preadolescence across children at FHR-SZ, FHR-BP, and controls. Children at FHR-SZ displayed stable deficits compared with controls, whereas children at FHR-BP demonstrated stable social functioning comparable to controls except from the onset of impaired coping skills at age 11. Whether the latter finding reflects early evidence of a recent and discrete onset of a developmental lag and whether this finding and the identified stable deficits in social functioning throughout preadolescence in children at FHR-SZ are predictive of later transition to psychosis remains to be investigated in ongoing and planned follow-up studies at age 15 (Thorup et al., 2022) and 19. Finally, irrespective of whether the identified social functioning deficits are risk markers or antecedents of later transition to psychosis, our findings may inform intervention studies aiming at improving social functioning in the impaired FHR children.

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CRediT authorship contribution statement

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Declaration of Competing Interest

All contributing authors declare no conflicts of interest.

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Supplementary materials

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