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School mobility during childhood predicts psychotic symptoms in late adolescence

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Background: Recently, school mobility was identified as a risk factor for psychotic symptoms in early adolescence. The extent to which this risk continues into late adolescence and the trajectories via which this risk manifests remain unexplored. **Methods:** Psychotic symptoms in 4,720 adolescents aged 18 were ascertained by trained psychologists using the Psychosis-Like Symptoms Interview. Mothers reported on sociodemographic factors (i.e., family adversity, ethnicity and urbanicity) from pregnancy to 4 years; child's involvement in bullying at age 6–7 years; residential mobility at 11 years and school mobility at 11–12 years. Young people reported on their friendships at 8 years, and antisocial behaviour and cannabis use at 15 years. **Results:** School mobility across childhood significantly predicted psychotic symptoms at 18 years (adjusted odds ratio = 2.15; 95% confidence intervals = 1.06, 4.40). Within path analysis, school mobility (β = .183, p = .035), involvement in bullying (β = .133, p = .013), antisocial behaviour (β = .052, p = .004), cannabis use (β = .254, p = .020) and female sex (β = .420, p < .001) significantly predicted psychotic symptoms. Residential mobility (β = .375, p < .001), involvement in bullying (β = .120, p = .022) and poor friendships (β = .038, p = .014) significantly predicted school mobility. Residential mobility indirectly increased the risk of psychotic symptoms via school mobility (β = .069, p = .041). **Conclusions:** Children who move schools often are more likely to have experienced peer problems. School mobility, in turn, appears to be a robust marker for psychotic symptoms in late adolescence. Clinicians and teachers should consider school mobility as an important risk indicator for both peer problems and psychopathology. **Keywords:** ALSPAC; school mobility; adolescence; psychotic symptoms; bullying.

Introduction

Psychotic symptoms exist on a continuum within the general population (Zammit et al., 2013). Evidence indicates that psychotic symptoms increase the risk of psychotic disorder (Poulton et al., 2000; Rössler et al., 2007) and that subclinical and clinical psychosis share similar risk factors (Van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009). Consequently, researchers have examined psychotic symptoms in community populations to further understand the aetiology of psychosis (Zammit et al., 2013). By capturing individuals earlier in the developmental trajectory, community studies facilitate the examination of prospective pathways to psychosis while incorporating a range of psychosocial risk factors (Boyd et al., 2013).

Several environmental risk factors have been associated with psychosis, including residential mobility (Pedersen & Mortensen, 2001), urban upbringing (Pedersen & Mortensen, 2001), bullying involvement (Schreier et al., 2009), socioeconomic disadvantage (Wicks, Hjern, Gunnell, Lewis, & Dalman, 2005) and family breakdown (Van Os et al., 2009). A common theme underpinning these risk factors is that they appear to elicit feelings of 'social defeat', or of being an outsider (Selten & Cantor-Graae, 2007). Within this context, nonpromotional school mobility (i.e., school moves not related to being promoted to the next school level) was recently identified as another risk factor for psychotic symptoms in early adolescence. Singh, Winsper, Wolke, and Bryson (2014) found that school mobility at age 9 was significantly associated with psychotic symptoms at age 12 following control for a wide range of confounders, including family adversity items (e.g., single status and financial adversity), urbanicity, bullying, poor friendships, ethnicity, sex and residential mobility. DeVylder, Oh, Pitts, and Schiffman (2015), in contrast, did not find a significant association between school mobility and psychotic symptoms in adulthood in unadjusted or adjusted analyses. Assessment of school mobility in this study, however, relied on retrospective reports from adults up to 25 years later. Therefore, the extent to which school mobility prospectively predicts psychotic symptoms later in development and the trajectories via which this risk may manifest remain unknown. This question warrants further investigation, given the potential negative effects of school mobility on mental health (Herbers, Reynolds, & Chen, 2013) and the relatively high prevalence of school mobility observed across populations (DeVylder et al., 2015; Rumberger, 2003).

There are several plausible pathways via which school mobility across childhood could be linked to

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psychotic symptoms in late adolescence. First, school mobility could directly impact on the development of psychotic symptoms by triggering psychological (e.g., development of low self-esteem) or physiological (e.g., sensitisation of the mesolimbic dopamine system) stress responses (Cantor-Graae & Selten, 2005; Lodge & Grace, 2011). Second, school mobility could mediate associations (i.e., act as a link in a causal chain) between early risk exposures and psychotic experiences. School mobility and psychotic symptoms share a number of antecedents, that is, bullying, poor friendships (Schreier et al., 2009; Sorin & Iloste, 2006) and residential mobility (Pedersen & Mortensen, 2001; Rumberger, 2003). Thus, it is plausible that school mobility could be one potential mechanism underpinning the negative effects of these prior exposures on subsequent psychotic symptoms (Selten & Cantor-Graae, 2007). Finally, school mobility could be an early risk factor triggering a causal chain of adverse events (Singh et al., 2014). School mobility has a range of negative sequelae. In particular, it has been linked to substance abuse (DeWit, 1998; Gasper, DeLuca, & Estacion, 2010) and antisocial behaviour (Ellickson & McGuigan, 2000; Herbers et al., 2013; Rumberger, 2003). As these two factors are robustly linked to the development of psychosis (Arseneault et al., 2002; Rössler et al., 2007; Vermeiren, 2003; Winsper et al., 2013), one route via which school mobility could increase the risk of psychotic symptoms is by increasing the risk of substance abuse and antisocial behaviour.

Similarly, residential mobility could contribute to the development of psychotic symptoms via indirect pathways. Studies indicate that home moves and psychosis share similar risk factors, including family adversity (Sorin & Iloste, 2006; Stilo et al., 2013), ethnicity (Morgan, Charalambides, Hutchinson, & Murray, 2010; Sorin & Iloste, 2006) and urbanicity (Pedersen & Mortensen, 2001; Sorin & Iloste, 2006). Therefore, a deeper examination of the effects of school mobility on psychotic symptoms should consider both independent and overlapping risk pathways involving residential and school mobility (Pedersen & Mortensen, 2001).

In this study, we aimed to add to the extant literature by testing long-term associations between school mobility in childhood and psychotic symptoms in late adolescence, while incorporating developmentally salient confounders including antisocial behaviour (Winsper et al., 2013) and cannabis use (Arseneault et al., 2002). We utilised path analytical methods to allow us to test several *potential* pathways simultaneously. Path analysis allows for the examination of the direct and indirect (i.e., mediational) effects of multiple independent and dependent variables within one comprehensive model (Stage, Carter, & Nora, 2004; Yanos, Roe, Markus, & Lysaker, 2015). Specifically, we addressed the following questions:

1. Does school mobility independently increase the risk of psychotic symptoms in late adolescence (after adjustment for all other risk factors)?
2. Does school mobility indirectly increase the risk of psychotic symptoms via an increased risk of antisocial behaviour and cannabis use?
3. Are associations between early risk factors (i.e., involvement in bullying, poor friendships and residential mobility) and psychotic symptoms mediated by school mobility?
4. Are associations between early risk factors (i.e., ethnicity, urbanicity and family adversity) and psychotic symptoms mediated by residential mobility?

See Figure 1 for a theoretical representation of the research questions addressed simultaneously within the final model.

Methods

Participants

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a UK birth cohort examining the determinants of development, health and disease during childhood and beyond. The study has been described in detail elsewhere (Boyd et al., 2013). ALSPAC recruited pregnant women in Avon with expected dates of delivery between 1st April 1991 and 31st December 1992. A total of 14,541 pregnant women were initially enrolled in the study and had returned at least one questionnaire or attended a 'Children in Focus' clinic by the 19th July 1999. Of these *initial* pregnancies, there were 14,676 foetuses, resulting in 14,062 live births of which 13,988 children were alive at 1 year of age. When the oldest children were approximately 7 years old, the sample was bolstered with eligible cases who had failed to join the study originally. Consequently, when considering variables collected from the age of 7 onwards, there are data available on 14,701 children (an additional 713 children). The study website contains details of all of the data that are available through a fully searchable data dictionary (http://www.bris.ac.uk/alspac/researchers/data-access/data-dictionary/). The phases of enrolment are described in more detail in Boyd et al. (2013). Ethical approval was obtained from the ALSPAC Law and Ethics committee and the local research ethics committees.

Measures

Psychotic symptoms. Psychotic symptoms at age 18 were assessed by trained psychology graduates with the semistructured Psychosis-Like Symptom Interview (Zammit et al., 2013). The interview comprises 11 core questions to ascertain key psychotic symptoms occurring since age 12, that is, hallucinations (visual and auditory), delusions (spied on, persecution, thoughts read, reference, control and grandiosity) and experiences of thought interference (broadcasting, insertion and withdrawal). Unspecified delusions were also rated. Experiences were rated as not present, suspected or definitely present (if a clear example was provided). Interviewers recorded audio interviews at three time points (approximately 6 months apart) to test for interrater reliability. The average kappa value of psychotic experiences was .83. Test–retest reliability was assessed with 162 adolescents re-interviewed after approximately 47 days (κ = .76, *SE* = .078), 46 of whom were re-interviewed by the same interviewer (κ = .86, *SE* = .136). As responses were highly skewed (i.e., very few

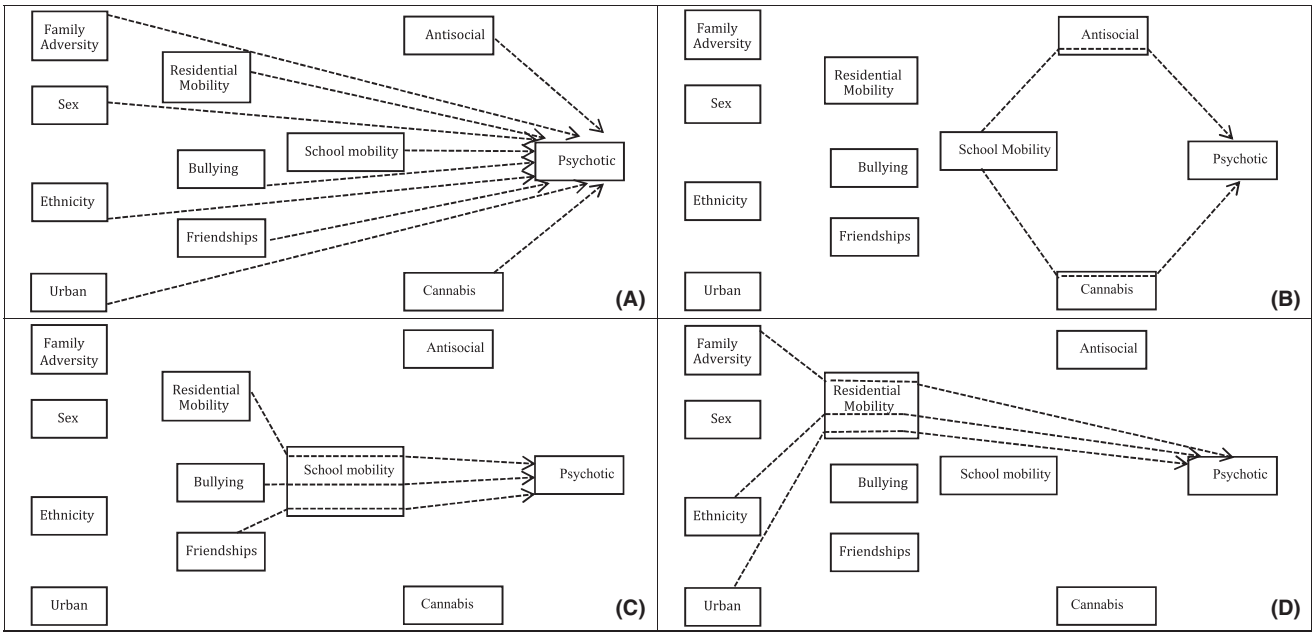

Figure 1 Diagrammatic representation of the four research questions simultaneously tested within the path model. Hypothesis 1 (A) examines direct associations between risk factors and psychotic symptoms. Hypothesis 2 (B) examines indirect associations from school mobility to psychotic symptoms via cannabis use and antisocial behaviour. Hypothesis 3 (C) examines indirect associations to psychotic symptoms from bullying, residential mobility and poor friendship via school moves. Hypothesis 4 (D) examines indirect associations from ethnicity, family adversity and urbanicity to psychotic symptoms via residential mobility

for higher frequencies) and to remain consistent with the extant literature, we constructed our outcome variable to represent the presence of one or more definite psychotic symptoms (Schreier et al., 2009; Zammit et al., 2013).

School mobility. Mothers were asked how many different schools their child had attended when children were approximately 11–12 years. Most children experienced one, two or three school changes. This reflects normal progression through the English school system, typically beginning with reception class at 5 years (American equivalent: kindergarten), primary school from 6 to 11 years (American equivalent: elementary school) and secondary school from 11 to 16/18 years (American equivalent: high school). We constructed a dichotomous school mobility variable, as the distribution of responses was highly skewed, and we wanted to specifically test associations with school moves outside of normal school progression. 'Normative school mobility' was coded as 0, 1, 2 or 3 different schools and 'repeated school mobility' as four or more different schools. Consistent with previous research, the threshold of four was selected (DeVylder et al., 2015).

Involvement in bullying. Involvement in bullying was mother-reported when children were approximately 6.8 years of age. Mothers responded to the following statements: 'she/he often fights with other children or bullies them', and 'she/he is picked on or bullied by other children'. Responses were coded as 'not true' = 0; 'somewhat true' or 'certainly true' = 1. In line with previous research (Winsper, Lereya, Zanarini, & Wolke, 2012), we combined these variables to create an involvement in bullying indices: 0 = no involvement in bullying; 1 = involvement as a bully; 2 = involvement as a victim and 3 = involvement as both a bully and victim.

Poor friendships. Friendship quality was self-reported during clinic sessions when children were approximately 8 years old. Questions were based on the Cambridge Hormones and Moods Project Friendship Questionnaire (Goodyer, Wright, & Altham, 1990). Children were asked five questions: 'Are you happy with the number of friends you've got (0 = very happy; 1 = quite happy; 2 = quite unhappy; 3 = unhappy)'; 'How often do you see your friends outside of school (0 = almost every day; 1 = >once a week; 2 = <once a week; 3 = hardly ever)'; 'Do your friends understand you (0 = most of time; 1 = sometimes; 2 = not often; 3 = not at all)'; 'Do you talk to your friends about problems (0 = most of time; 1 = sometimes; 2 = not often; 3 = not at all)'; 'Overall how happy are you with your friends (0 = very happy; 1 = quite happy; 2 = quite unhappy; 3 = unhappy)'. Responses were summed to create a continuous friendship scale from 0 to 15, with 0 denoting the most positive friendship score and 15 the poorest (Singh et al., 2014).

Antisocial behaviour. Antisocial behaviour was self-reported during clinics at approximately 15 years. Adolescents were presented with 22 items (e.g., 'Frequency young person has written things or sprayed paint on property that did not belong to them'). A full list of items is presented (see Table S1 available online). Responses to each item were coded as 0 ('not at all' or 'just once') and 1 ('2–5 times' or '6+ times'). Item responses were summed, creating a continuous scale ranging from 0 to 22, with higher scores denoting more antisocial behaviour.

Cannabis use. Cannabis use was self-reported during clinics at approximately 15 years. Young people were asked if they had ever taken cannabis and if yes, how often. We constructed a variable representing regular cannabis use coded as 0 = no use (i.e., not applicable, once twice ever, used to take sometimes never now; sometimes, less than once a week) and 1 = at least weekly use (i.e., 1–6 times a week; >6 times a week, not every day; to every day). We constructed a dichotomous cannabis variable with a threshold of at least weekly, as weekly use has been indicated as a strong predictor of psychosis risk (Henquet et al., 2004).

Residential mobility. Home moves were mother reported when the child was 5, 6, 7, 8 and 11 years. Responses at each

time point were summed to derive the total number of home moves. Because of the skewed distribution of responses (very few responses for higher frequencies), we constructed a dichotomous variable consistent with previous reports. Unlike school progression changes (e.g., nursery to reception), home moves are not normative; therefore, we chose a lower threshold of two or more to indicate residential mobility (Singh et al., 2014).

Ethnicity. Child's ethnicity was based on the ethnicity of the mother and her partner. If the mother and/or her partner reported nonwhite ethnicity, the child was coded as nonwhite (Singh et al., 2014).

Family adversity. Multiple family risk factors were assessed using the Family Adversity Index (FAI) during pregnancy ('long index'), 2 years ('long index') and 4 years ('short index'). The FAI 'long index' has 18 items, for example, maternal affective disorder and financial difficulties [see Winsper, Zanarini, and Wolke (2012) for a full description]. The short index has the same items, with the exception of three excluded items: social, practical and financial support. If an adversity item was reported, it was given 1 point. Points were summed for a total FAI score at each time point. Consistent with previous research (Wolke, Schreier, Zanarini, & Winsper, 2012), we summed the three FAI indices.

Urbanicity. Urbanicity was ascertained at birth and coded as in previous studies: 0 = village/hamlet; 1 = urban/town (Zammit et al., 2009).

Analysis

Missing data. As a substantial proportion of the original sample was lost to follow-up, we conducted logistic regressions to identify significant predictors of attrition. Adolescents lost to attrition were more often boys, of ethnic minority and low birth weight. They more often lived in rented properties and were born to single mothers of lower educational level (see Table S2 available online). Using the variables associated with selective dropout as the predictors, we fitted a logistic regression model (nonresponse vs. response outcome) to determine weights for each individual using the inverse probability of response (Kinner, Alati, Najman, & Williams, 2007). We then compared results from the weighted and unweighted analysis.

Logistic regressions. Using *SPSS version 22*, we conducted unadjusted and adjusted (forced entry method) logistic regressions to examine which risk factors were associated with psychotic symptoms at 18 years. We conducted regressions (logistic and linear) to examine the characteristics of children who had moved school often. Results are reported as odds ratios (OR) with 95% confidence intervals (CIs) for dichotomous outcomes and β coefficients with 95% CIs for continuous outcomes.

Path analysis. We conducted path analysis using *Mplus version 6*. Path analysis is a method that can be used to determine whether a set of nonexperimental data fit well with an a priori causal model. Modelled associations are unidirectional and based on the temporal ordering of assessments (i.e., earlier risk factors are hypothesised to predict later outcomes). However, because the data are nonexperimental, we cannot conclusively ascertain whether associations are causal (Stage et al., 2004). Path analysis allowed us to control for multiple associations between preexisting risk factors, school mobility, subsequent risk factors and psychotic symptoms and to examine direct and indirect (i.e., mediational) associations between risk factors and psychotic symptoms (Lleras, 2005). We used probit estimation as recommended for path models with both categorical and continuous variables (Winship & Mare, 1983). Probit regression is a log-linear approach analogous to logistic regression, producing similar chi-square statistics, p values and conclusions to logit models (Allison, 2012). Coefficients indicate the strength of the relationship between the predictor variable and the probability of group membership, representing the change in the probability of 'caseness' associated with a unit change in the independent variable. Thus, it is important to keep the scale of the predictor in mind when interpreting results. For example, a probit coefficient of .053 indicates that each one-point increase in the antisocial behaviour scale resulted in an increase of .053 standard deviations in the predicted Z score of psychotic symptoms. The WLSMV estimator (weighted least squares with robust standard errors, mean and variance adjusted) was used yielding probit coefficients for categorical outcomes and linear regression coefficients for continuous outcomes. Missing data were accommodated using the reliable Full Information Maximum Likelihood method (Wiggins & Sacker, 2002).

We modelled several simultaneous pathways to test our four a priori research questions. First, to confirm whether school mobility was independently associated with subsequent psychotic symptoms (Singh et al., 2014), we incorporated direct associations between all risk factors in the model and the psychotic symptoms outcome (Figure 1A). Second, to test the extent to which school mobility indirectly increased the risk of psychotic symptoms, we modelled the indirect pathways from school mobility to psychotic symptoms via cannabis use and antisocial behaviour (Figure 1B). Third, to test the role of school mobility as a mediator, we modelled indirect pathways linking involvement in bullying, poor friendships and residential mobility to psychotic symptoms via school mobility (Figure 1C). Finally, we examined the indirect associations from ethnicity, urbanicity and family adversity to psychotic symptoms via residential mobility (Figure 1D).

In order to test the robustness of our main hypothesised pathways, we also adjusted for other associations between variables included within the model. We regressed cannabis use and antisocial behaviour on sex (Moffitt, Caspi, Rutter, & Silva, 2001), family adversity (Thapar, van den Bree, Fowler, Langley, & Whittinger, 2006), bullying (Bender & Lösel, 2011), poor friendships (Sutton, Smith, & Swettenham, 1999) and residential mobility (Simpson & Fowler, 1994). We also controlled for intercorrelations between highly related risk factors assessed in close temporal proximity [e.g., bullying involvement with poor friendships (Wei & Jonson-Reid, 2011) and antisocial behaviour with cannabis use (López & Emler, 2011)].

Results

Data were available on 4,720 adolescents who completed the Psychosis-Like Symptoms Interview at 18. A total of 4.9% of adolescents reported at least one psychotic symptom. A total of 4.9% of children had moved school 4 or more times by the age of 11–12. The pattern of results from the weighted (using the inverse probability of response) and unweighted analyses was very similar; therefore, we report the unweighted analysis here.

Logistic regressions

Adolescents who moved school often were significantly more likely to have moved home (OR = 4.90; 95% CI = 3.63, 6.64); been involved in bullying as a bully victim (OR = 2.89; 95% CI = 1.50, 5.57); and

experienced poor friendships (OR = 1.07; 95% CI = 1.00, 1.14). School mobility was not significantly associated with ethnicity (OR = 1.35, 95% CI = .65, 2.81), subsequent cannabis use (OR = .80; 95% CI = .25, 2.58) or antisocial behaviour (OR = β = −.004; 95% CI = −.43, .34).

In unadjusted logistic regressions, female sex, family adversity, residential mobility, school mobility, being a victim only and a bully victim, weekly cannabis use and antisocial behaviour were all associated with psychotic symptoms. Ethnicity and urbanicity were not significantly associated with psychotic symptoms. In adjusted logistic regressions simultaneously controlling for all other risks, female sex, residential mobility, school mobility, being a bully victim and antisocial behaviour independently predicted psychotic symptoms (Table 1).

Path analysis

Fit indices indicated a very good model fit: $\chi^2 = 19.97$, $p = .17$; RMSEA = .01; CFI = .99. After controlling for all variables (and intercorrelations between related variables), female sex, involvement in bullying at 6–7 years, school mobility at 11–12 years, and weekly cannabis use and antisocial behaviour at 15 years all predicted psychotic symptoms at 18 years. Involvement in bullying, poor friendships and residential mobility significantly predicted school mobility. Family adversity significantly predicted residential mobility (Figure 2 reports the significant direct pathways within the model). Residential mobility was indirectly associated with psychotic symptoms via school mobility (Table 2). Family adversity and male sex were indirectly associated with psychotic symptoms via cannabis use and antisocial behaviour (Table 3).

Discussion

Using a large community cohort, we examined whether school mobility increases risk of psychotic symptoms in late adolescence, and the pathways via which this increased risk may manifest. Our findings extend the literature in two ways. First, we found that school mobility was independently associated with increased risk of psychotic symptoms in late adolescence following adjustment for a number of salient confounders. Second, we found that the association between residential mobility and psychotic symptoms was significantly mediated by school moves. This supports the previously untested hypothesis that there may be something about school moves, in particular, rather than residential

Table 1 Unadjusted and adjusted associations between psychosocial risk factors, school mobility and definite psychotic symptoms at 18 years

Risk factor	Number (%) of psychotic symptoms	Psychotic symptoms unadjusted OR (95% CI)	Psychotic symptoms adjusted[a] OR (95% CI)
Sex			
Male (n = 2,054)	71 (3.5%)	[Reference]	[Reference]
Female (n = 2,666)	158 (5.9%)	**1.76 (1.32, 2.34)**	**2.14 (1.38, 3.31)**
Urbanicity			
Rural (n = 309)	14 (4.5%)	[Reference]	[Reference]
Urban (n = 4,082)	201 (4.9%)	1.09 (.63, 1.90)	1.25 (.56, 2.78)
Family adversity to 4 years			[Reference]
Mean score	3.58 (3.81) vs. 4.70 (4.07)	**1.07 (1.03, 1.10)**	1.04 (.99, 1.10)
Ethnicity			
White (n = 4,048)	195 (4.8%)	[Reference]	[Reference]
Nonwhite (n = 180)	11 (6.1%)	1.29 (.69, 2.41)	.64 (.22, 1.92)
Residential mobility at 11 years			
<2 moves (n = 3,676)	149 (4.1%)	[Reference]	[Reference]
≥2 moves (n = 753)	53 (7.0%)	**1.79 (1.29, 2.48)**	**1.65 (1.05, 2.61)**
School mobility at 11–12 years			
<4 moves (n = 3,557)	138 (3.9%)	[Reference]	[Reference]
≥4 moves (n = 185)	18 (9.7%)	**2.67 (1.60, 4.47)**	**2.15 (1.06, 4.40)**
Bullying status at 6–7 years			
None (n = 2,992)	117 (3.9%)	[Reference]	[Reference]
Bully (n = 161)	8 (5.0%)	1.29 (.62, 2.68)	1.16 (.48, 2.78)
Victim (n = 504)	33 (6.5%)	**1.72 (1.16, 2.56)**	1.37 (.76, 2.45)
Bully and victim (n = 115)	10 (8.7%)	**2.34 (1.19, 4.59)**	**2.84 (1.26, 6.43)**
Poor friendships at 8 years			
Mean score	3.49 (2.41) vs. 3.42 (2.54)	.99 (.93, 1.06)	1.00 (.92, 1.08)
Weekly cannabis use at 15 years			
No (n = 3,672)	156 (4.2%)	[Reference]	[Reference]
≥Once a week (n = 85)	12 (14.1%)	**3.71 (1.97, 6.97)**	1.48 (.56, 3.90)
Antisocial behaviour at 15 years			
Mean score	2.00 (2.41) vs. 3.45 (3.39)	**1.17 (1.12, 1.23)**	**1.17 (1.10, 1.26)**

OR, odds ratio; CI, confidence interval; boldface indicates significant associations.
[a]Adjusted for all other risk factors in model.

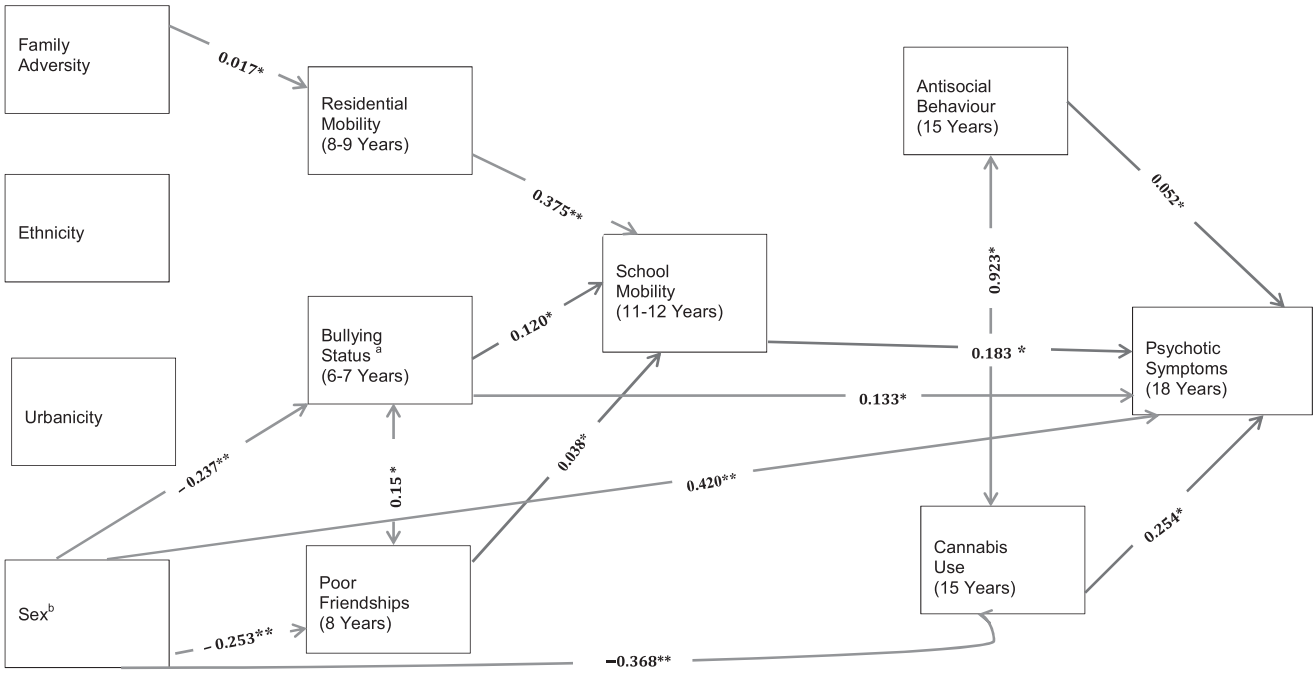

Figure 2 Main significant direct pathways between early risk factors and psychotic experiences at 18 years. Figures represent probit coefficients; *significant associations at the $p < .05$ level; **significant associations at the $p < .01$ level; [a]bullying status was entered as an ordinal variable in line with the dose–response relationships observed in the logistic regressions. In Mplus, an ordinal variable is treated as a continuous variable and interpreted accordingly, that is, one coefficient per variable is produced; [b]negative sign for sex indicates association with male sex (i.e., male sex was reference group). Results corresponds to research question 1

Table 2 Unstandardised probit coefficients (β) for the indirect pathways to psychotic symptoms with residential and school mobility as mediators

	Via school mobility			Via residential mobility		
Predictor variables	β	*SE*	*p*	β	*SE*	*p*
Family adversity				.002	.001	.184
Urbanicity				−.009	.012	.432
Ethnicity				.003	.008	.750
Bullying	.022	.014	.127			
Negative friendships	.007	.004	.109			
Residential mobility	**.069**	**.034**	**.041**			

Boldface indicates significant associations; β, probit coefficient; *p*, probability; *SE*, standard error. Results correspond to research questions 3 and 4.

Table 3 Unstandardised probit coefficients (β) for the indirect pathways to psychotic symptoms with cannabis use and antisocial behaviour as mediators

	Via cannabis use			Via antisocial behaviour		
Predictor variables	β	*SE*	*p*	β	*SE*	*p*
Sex	**−.093**	**.047**	**.047**	**−.031**	**.012**	**.008**
Family adversity	**.010**	**.005**	**.047**	**.003**	**.001**	**.018**
Bullying	−.004	.018	.841	.002	.003	.639
Negative friendships	−.002	.005	.642	.000	.001	.686
School mobility	−.030	.036	.408	−.003	.006	.588
Residential mobility	.017	.024	.491	.006	.005	.224

Boldface indicates significant associations; β, probit coefficient; *p*, probability; *SE*, standard error; negative sign for sex indicates association with male sex; results correspond to research question 2.

moves *per se* that contributes to the development of psychosis (Pedersen & Mortensen, 2001).

It is striking that school mobility across childhood remained a significant predictor of psychotic symptoms even after controlling for subsequent cannabis use and antisocial behaviour. Furthermore, this association was not significantly attenuated following adjustment for a range of early psychosocial risk factors, suggesting that school mobility may have long-term effects on the development of psychotic symptoms reaching into late adolescence. While many studies have reported on the detrimental effects of school mobility on academic performance, behaviour problems, and high school dropout (Herbers et al., 2013), few have focused on the impact on mental health. School mobility is stressful for children and adolescents (Pollari & Bullock, 1988; Rumberger, 2003). Mobile students have to cope with new peers and social expectations and to negotiate new academic standards and expected classroom behaviours (Rumberger & Larson, 1998). These tasks may prove particularly difficult for those with a history of peer problems [as observed in this, and other (Wolke, Woods, & Samara, 2009) cohorts]. Once children become involved in bullying, the pattern tends to persist for months or years even when the child changes school (Sapouna et al.,

2011). Within this context, repeated school moves, especially for those with preexisting experiences of exclusion (i.e., peer difficulties or recurrent home moves), may induce or exacerbate feelings of 'social defeat' (Selten & Cantor-Graae, 2007). Social defeat, especially if chronic, may lead to physiological, for example, mesolimbic alterations (Selten & Cantor-Graae, 2007) and psychological, for example, external locus of control (South, Haynie, & Bose, 2007) alterations, both of which could increase risk of psychosis (Fisher et al., 2013; Thompson et al., 2011).

It is somewhat surprising that antisocial behaviour and cannabis use did not mediate the association between school mobility and psychotic symptoms. Unlike previous reports (Gasper et al., 2010; Herbers et al., 2013), we did not find a significant association between school mobility and antisocial behaviour or cannabis use. As hypothesised, these risk factors were associated with psychotic symptoms. Furthermore, they significantly mediated associations between male sex and family adversity and subsequent psychotic symptoms.

It may be that school mobility in this general population cohort is associated with antisocial behaviours in the realm of peer relationships (Ellickson & McGuigan, 2000) rather than criminal acts and drug taking, which may be more characteristic of high-risk populations (Herbers et al., 2013). Alternatively, links with antisocial behaviour may become apparent later in adolescence when their tends to be a peak in such behaviour (Monahan, Steinberg, Cauffman, & Mulvey, 2009). In view of the strong associations between delinquency, substance abuse and psychosis, this area merits further attention (Catalano, Oesterele, Fleming, & Hawkins, 2004).

Despite finding an association between school mobility and psychotic symptoms, we did not identify any significant mediators of this association. In a previous study, the association between school mobility and psychotic symptoms in early adolescence was significantly mediated by involvement in bullying (Singh et al., 2014). Considering the significant associations between peer problems and subsequent school mobility in this study, bullying involvement may likely mediate the school mobility-psychotic symptoms link later in the developmental trajectory. Future studies may consider further potential mediators such as cognitive dysfunction, for example, externalised locus of control (Thompson et al., 2011), and resilience factors, for example, academic performance (Keefe et al., 2006; Temple & Reynolds, 2000).

Contrary to previous studies, we did not find significant associations between ethnicity and residential/school mobility (Sorin & Iloste, 2006) or psychotic symptoms (Morgan et al., 2010). This is somewhat surprising and may be partly attributable to the data available, which did not allow for a fine-grained analysis of ethnic type. Previous studies (Singh et al., 2015) have indicated variations in psychosis (and associated correlates) according to ethnic type (e.g., Black vs. Asian) and migrant status (Cantor-Graae, Pedersen, McNeil, & Mortensen, 2003).

Our study had limitations. First, although the prospective design of our study enabled us to model predictive pathways based on the temporal ordering of the assessments, we cannot conclusively establish the direction of causality (e.g., that bullying led to school mobility) as risk factors were assessed at just one time point. We cannot rule out reverse causality for some of the associations or that another unexplored variable had effects on the outcome (Stage et al., 2004). Nevertheless, we carefully planned our analysis grounded in the extant literature to reduce the likelihood of spurious results. As a related point, some risk factors such as bullying were only assessed fairly early on in the developmental trajectory, which may have resulted in misclassification bias.

Second, due to the epidemiological nature of the cohort, school mobility was assessed with a single question. Therefore, the data did not allow us to differentiate between promotional (i.e., standard progression) and nonpromotional school moves or definitively determine whether school moves were attributable to home moves or school problems. However, our assumption that four or more school moves represent nonpromotional school changes has face validity in view of the English educational system (i.e., reception/primary school/secondary school) (DeVylder et al., 2015). Furthermore, we examined several potential pathways to psychosis to delineate developmental routes according to home- (e.g., family adversity) and school-related moves (e.g., peer problems), respectively. Future studies may collect more detailed information on reasons for school moves to further elucidate the mechanisms via which school mobility may increase the risk of psychosis.

Third, there was selective attrition in the cohort, reducing statistical power and potentially biasing results. However, previous simulations indicate that selective dropout may lead to an underestimation of psychiatric disorders but only have a small impact on predictor and outcome relationships (Wolke, Waylen, et al., 2009). Indeed, weighted analysis taking into account factors associated with selective attrition did not substantially alter the results. Nevertheless, dropout may still limit the generalisability of the results.

Fourth, as our study focused on environmental determinants, we did not incorporate endogenous factors such as neurodevelopmental impairment or genetic vulnerability into our analyses. Future studies may explore the complex relationships between environmental and biological processes and examine associations between mobility-related risk factors and specific types of psychotic symptoms, for example, peer victimisation and paranoid beliefs (Bentall & Fernyhough, 2008).

We found that school mobility is independently associated with psychotic symptoms in late adolescence and that it may mediate the association

between residential mobility and subsequent psychotic symptoms. While school moves as a consequence of moving home may be unavoidable, our findings suggest that reducing school-related mobility and its associated antecedents (e.g., peer problems) may help alter risk trajectories to psychosis. As poor friendships and bullying were found to predict school mobility, interventions aimed at improving school connectedness, in terms of improving relationships and increasing commitment to school performance (Catalano et al., 2004), may help prevent a cycle of peer/discipline problems and subsequent school moves. Equally important are programmes designed to help mobile students successfully establish themselves within new school environments (MacArthur & Higgins, 2007). Our findings highlight that teachers and healthcare professionals should be aware of mobile students as a high-risk population.

Supporting information

Additional Supporting Information may be found in the online version of this article:

Table S1. Antisocial behaviour questionnaire at 15 years.

Table S2. Dropout analysis comparing those not available with those who completed the psychotic symptoms interview.

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Key points

- We found that school mobility was an independent risk factor for psychotic symptoms and that bullying and poor friendships increased risk of school mobility.
- School mobility mediated the association between home moves and psychotic symptoms, indicating school moves, in particular, contribute to the risk of psychosis.
- Our findings lend further weight to the hypothesis that repeated experiences of marginalisation and exclusion (social defeat) increase the risk of developing psychosis.
- Our findings suggest that interventions to reduce peer problems and school-related mobility may help alter risk trajectories to psychosis. Teachers and clinicians should be aware of mobile students as a potential high-risk population.

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