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, urbanicity) from pregnancy to 4 years; child's involvement in bullying at age 6 to 7 years; residential mobility at 11 years and school mobility at 11 to 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 = 2.17; 95% Confidence Intervals = 1.06, 4.41). Within path analysis, school mobility (β=0.183, p=0.035); involvement in bullying (β=0.133, p=0.013); antisocial behaviour (β=0.052, p=0.004); cannabis use (β= 0.254, p=0.020); and female sex (β=0.420, p<.001) significantly predicted psychotic symptoms. Residential mobility (β=0.375, p<.001), involvement in bullying (β= 0.120, p= 0.022) and poor friendships (β= 0.038, p= 0.014) significantly predicted school mobility. Residential mobility indirectly increased risk of psychotic symptoms via school mobility (β= 0.069, p= 0.041).

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., 2014).

Evidence indicates that psychotic symptoms increase risk of psychotic disorder (Poulton et al.,

2000; Rössler et al., 2007), and that sub-clinical 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., 2014). 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, 2014), 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, non-

promotional 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, financial adversity, etc),

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 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, i.e., 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 the current 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, residential mobility) and psychotic symptoms mediated by school mobility?

4) Are associations between early risk factors (i.e., ethnicity, urbanicity, 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 the 1st April 1991 and 31st December 1992.

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 seven 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 semi-

structured *Psychosis-Like Symptom Interview* (Zammit et al., 2014). The interview comprises 11

core questions to ascertain key psychotic symptoms occurring since age 12, i.e., 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

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kappa value of psychotic experiences was 0.83. Test-retest reliability was assessed with 162

adolescents re-interviewed after approximately 47 days (kappa=0.76, Se=0.078), 46 of whom

were re-interviewed by the same interviewer (kappa=0.86, SE=0.136). As responses were

highly skewed (i.e., very few 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., 2014).

School mobility

Mothers were asked how many different schools their child had attended when children were

approximately 11 to 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. "No school mobility" was coded as 0, 1,

2 or 3 different schools and "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") (Kline, 2013). 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 old.

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 2 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 non-white ethnicity the child was coded as non-white (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, e.g., maternal affective disorder, financial difficulties (see Winsper,

Zanarini, and Wolke (2012) for a full description). The short index has the same items, with the

exception of 3 excluded items: social, practical and financial support. If an adversity item was

reported, it was given one 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 (non-response 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 non-experimental 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 non-experimental, we cannot conclusively ascertain whether associations are causal (Stage

et al., 2004). Path analysis allowed us to control for multiple associations between pre-existing

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 0.053 indicates that each one point increase in the

antisocial behaviour scale resulted in an increase of 0.053 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 co-efficients for

categorical outcomes and linear regression coefficients for continuous outcomes. Missing data

was 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 (see **Figure 1a**).

Second, to test the extent to which school mobility indirectly increased risk of psychotic

symptoms we modelled the indirect pathways from school mobility to psychotic symptoms via

cannabis use and antisocial behaviour (see **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 (see **Figure 1c**). Finally, we

examined the indirect associations from ethnicity; urbanicity; and family adversity to psychotic

symptoms via residential mobility (see **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 behavior on sex (Moffitt, 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 inter-correlations 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 to 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 (OR=1.65, 95% CI = 1.05,

2.59), victim (OR=1.65. 95% CI = 1.22, 2.22), or bully-victim (OR=2.04; 95% CI=1.23, 3.37); 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=0.65, 2.81) subsequent cannabis use

(OR=0.80; 95% CI= 0.25, 2.58) or antisocial behaviour (OR=β = -0.004; 95% CI=-0.43, 0.34).

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

(see **Table 1**).

Path analysis

Fit indices indicated a very good model fit: X^2 = 19.97, p= 0.17; RMSEA=0.01; CFI=0.99. After

controlling for all variables (and inter-correlations between related variables), female sex,

involvement in bullying at 6 to 7 years, school mobility at 11 to 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 (see **Figure 2** which reports the significant direct pathways within the model). Residential mobility was indirectly associated with psychotic symptoms via school mobility (**Table 3**). Family adversity and male sex were indirectly associated with psychotic symptoms via cannabis use and antisocial behaviour (see **Tables 2**).

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 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 drop-out (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 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 pre-existing 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, e.g.,

mesolimbic alterations (Selten & Cantor-Graae, 2007) and psychological, e.g., 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 there 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, Oesterle, 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 the current 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, e.g., externalised locus of control (Thompson et al., 2011) and resilience factors, e.g., 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 versus 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 mis-classification 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 non-promotional school moves, or definitively determine whether school moves were attributable to home moves or school problems. However, our assumption that 4 or more school moves represent non-promotional 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

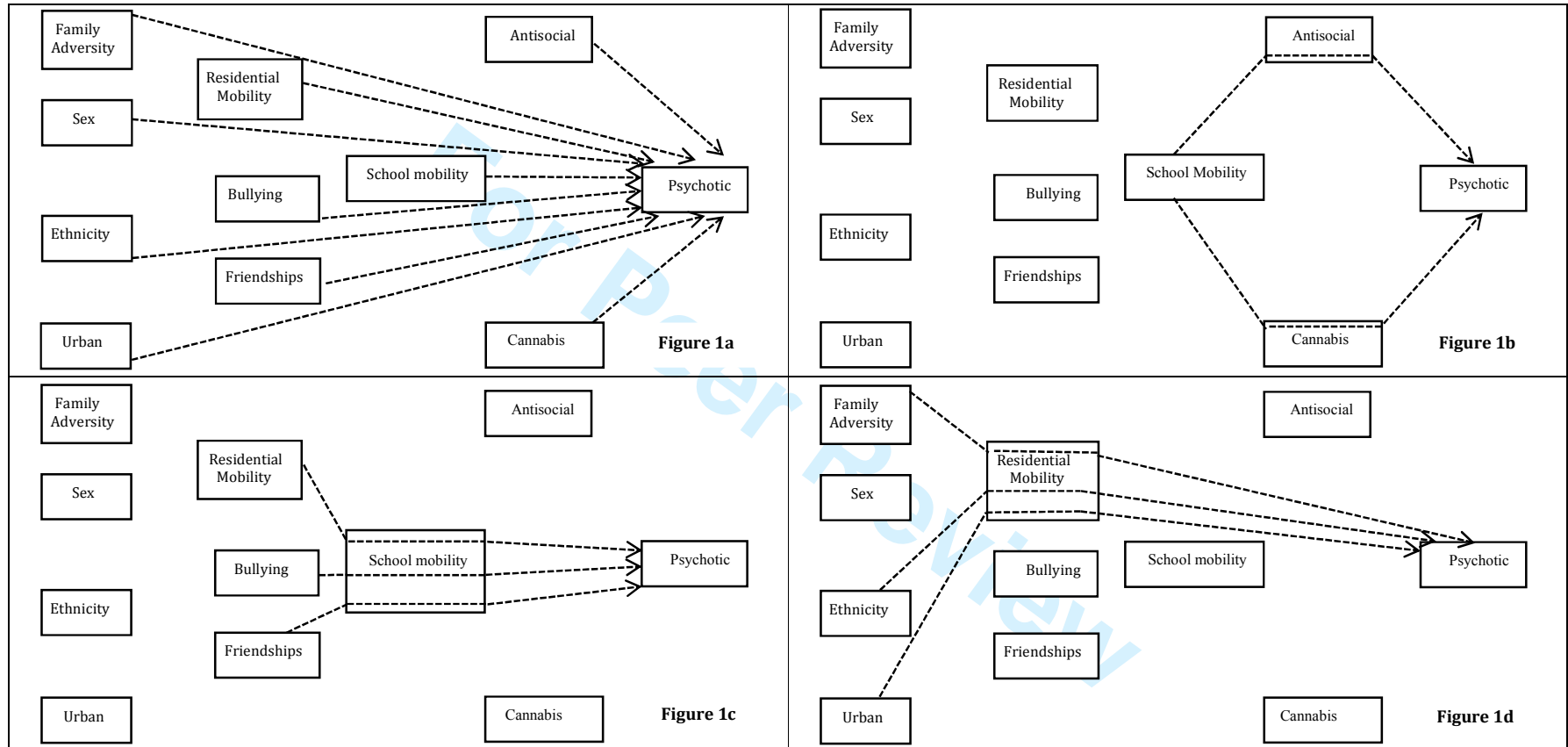which school mobility may increase the risk of psychosis.

Third, there was selective attrition in the cohort, reducing statistical power and

potentially biasing results. However, previous simulations indicate that selective drop out 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, drop-out 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, e.g., 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.

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Figure 1 Diagrammatic representation of the four research questions simultaneously tested within the path model

Hypothesis 1 (1a) examines direct associations between risk factors and psychotic symptoms; Hypothesis 2 (1b) examines indirect associations from school mobility to psychotic symptoms via cannabis use and antisocial behaviour; Hypothesis 3 (1c) examines indirect associations to psychotic symptoms from bullying, residential mobility and poor friendship via school moves; Hypothesis 4 (1d) examines indirect associations from ethnicity, family adversity and urbanicity to psychotic symptoms via residential mobility

Figure 2 Main significant direct pathways between early risk factors and psychotic experiences at 18 years
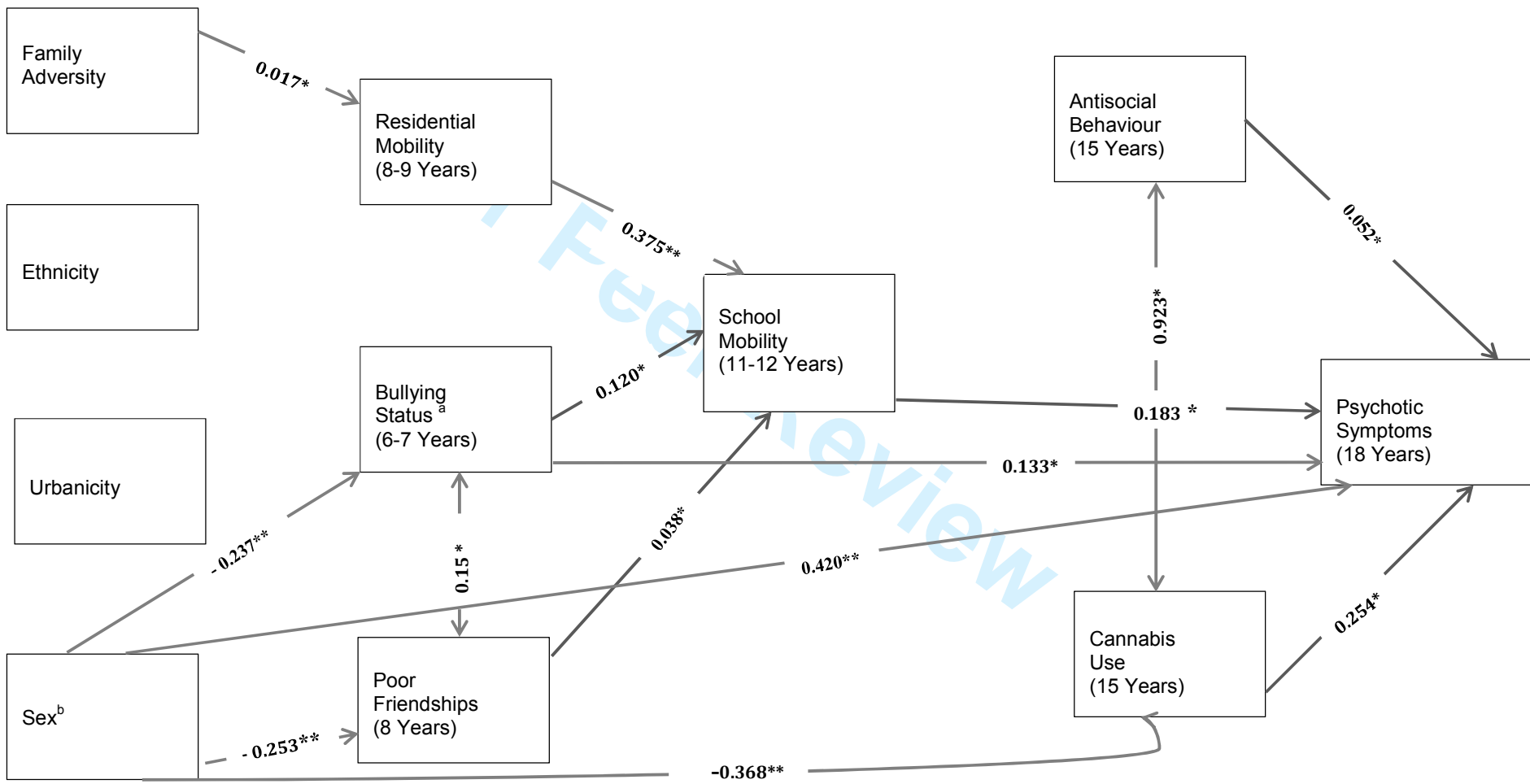
Figures represent probit co-efficients; * indicates significant associations at the p<0.05 level; * *indicates significant associations at the p<0.01 level [a]Bullying status was entered as an ordinal variable in line with the dose response relationships observed in the logistic regressions. In Mplus an ordinal variable is treated as a continuous variable and interpreted accordingly, i.e., one co-efficient per variable is produced; negative sign for sex indicates association with male sex (i.e., male sex was reference group). Results corresponds to research question 1

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=2054)	71 (3.5%)	[reference]	[reference]
Female (n=2666)	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=4082)	201 (4.9%)	1.09 (0.63, 1.90)	1.25 (0.56, 2.78)
Family adversity to 4 yrs			[reference]
Mean score	3.58 (3.81) vs 4.70 (4.07)	**1.07 (1.03, 1.10)**	1.04 (0.99, 1.10)
Ethnicity			
White (n=4048)	195 (4.8%)	[reference]	[reference]
Non-white (n=180)	11 (6.1%)	1.29 (0.69, 2.41)	0.64 (0.22, 1.92)
Residential mobility at 11 yrs			
<2 moves (n=3676)	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 yrs			
<4 moves (n=3557)	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 yrs			
None (n=2992)	117 (3.9%)	[reference]	[reference]
Bully (n=161)	8 (5.0%)	1.29 (0.62, 2.68)	1.16 (0.48, 2.78)
Victim (n=504)	33 (6.5%)	**1.72 (1.16, 2.56)**	1.37 (0.76, 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 yrs			
Mean score	3.49 (2.41) vs 3.42 (2.54)	0.99 (0.93, 1.06)	1.00 (0.92, 1.08)
Weekly cannabis use at 15 yrs			
No (n=3672)	156 (4.2%)	[reference]	[reference]
≥Once a week (n=85)	12 (14.1%)	**3.71 (1.97, 6.97)**	1.48 (0.56, 3.90)
Antisocial behaviour at 15 yrs			
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

Table 2. Unstandardised probit coefficients (β) for the indirect pathways to psychotic symptoms with cannabis use and antisocial behaviour as mediators

Predictor Variables	Via Cannabis Use			Via Antisocial Behaviour		
	B	SE	P	B	SE	P
Sex	**-0.093**	**0.047**	**0.047**	**-0.031**	**0.012**	**0.008**
Family Adversity	**0.010**	**0.005**	**0.047**	**0.003**	**0.001**	**0.018**
Bullying	-0.004	0.018	0.841	0.002	0.003	0.639
Negative Friendships	-0.002	0.005	0.642	0.000	0.001	0.686
School Mobility	-0.030	0.036	0.408	-0.003	0.006	0.588
Residential Mobility	0.017	0.024	0.491	0.006	0.005	0.224

Boldface indicates significant associations; B=probit coefficient; P=probability; SE=standard error; negative sign for sex indicates association with male sex; results correspond to research question 2

Table 3. Unstandardised probit coefficients (β) for the indirect pathways to psychotic symptoms with residential and school mobility as mediators

Predictor Variables	Via School Mobility			Via Residential Mobility		
	B	SE	P	B	SE	P
Family Adversity				0.002	0.001	0.184
Urbanicity				-0.009	0.012	0.432
Ethnicity				0.003	0.008	0.750
Bullying	0.022	0.014	0.127			
Negative Friendships	0.007	0.004	0.109			
Residential Mobility	**0.069**	**0.034**	**0.041**			

Boldface indicates significant associations; B=probit coefficient; P=probability; SE=standard error. Results correspond to research questions 3 and 4

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Supplementary Table 1. Antisocial behaviour questionnaire at 15 years

Frequency YP has travelled on a bus/train without paying enough money or using someone else's pass, in the last year

Frequency YP has written things or sprayed paint on property that did not belong to them, in the last year

Frequency YP has stolen something from a shop or store, in the last year

Frequency YP has sold an illegal drug to someone, in the last year

Frequency YP has ridden in a stolen car/van/motorbike, in the last year

Frequency YP has broken into a car/van to try and steal something out of it, in the last year

Frequency YP has ignored someone they know on purpose or left them out of things, in the last year

Frequency YP has said nasty things to someone they know, or slagged them off or called them names, in the last year

Frequency YP has threatened to hurt someone they know, in the last year

Frequency YP has hit, spat or thrown stones at someone they know, in the last year

Frequency YP has got other people to do these things to someone they know, in the last year

Frequency YP has broken into a house or building to try and steal something, in the last year

Frequency YP has hit/kicked/punched a brother or sister on purpose, in the last year

Frequency YP has hit/kicked/punched someone else on purpose with the intention of really hurting them, in the last year

Frequency YP has deliberately damaged or destroyed property that did not belong to them, in the last year

Frequency YP has sold something that didn't belong to them or that they knew was stolen, in the last year

Frequency YP has stolen money or property that someone was holding/carrying/wearing, in the last year

Frequency YP has hit or picked on someone because of their race or skin colour, in the last year

Frequency YP has hurt or injured animals or birds on purpose, in the last year

Frequency YP has set fire or tried to set fire to something on purpose, in the last year

Frequency YP has carried a knife or other weapon with them for protection or in case it was needed in a fight, in the last year

Frequency YP has been rowdy or rude in a public place such that people complained or they got in trouble, in the last year

Computer task completed by YP at clinic; YP= Young Person

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Supplementary Table 2. Drop-out analysis comparing those not available to those who completed the psychotic symptoms interview

	Interview available	Interview not available	Interview not available versus available
	N (%)	N (%)	OR (95% CI)
Gender			
Female	2054 (43.5%)	5585 (55.1%)	[reference]
Male	2666 (56.5%)	4556 (44.9%)	**0.63 (0.59, 0.67)**
Ethnicity			
White	4048 (95.7%)	7495 (94.5%)	[reference]
Black	180 (4.3%)	433 (5.5%)	**0.77 (0.64, 0.92)**
Birthweight			
>2499g	4172 (7.0%)	8917 (4.8%)	[reference]
<2500g	214 (93.0%)	598 (95.2%)	**0.77 (0.65, 0.90)**
Home ownership			
Mortgage	3649 (84.6%)	6241 (67.9%)	[reference]
Rent	664 (15.4%)	2952 (32.1%)	**0.39 (0.35, 0.42)**
Marital status			
Married	3551 (81.7%)	6598 (71.6%)	[reference]
Single	797 (18.3%)	2618 (28.4%)	**0.56 (0.52, 0.62)**
Mothers education			
O level or above	3484 (81.2%)	5259 (64.1%)	[reference]
Below O level	809 (18.8%)	2947 (35.9%)	**0.41 (0.38, 0.45)**

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