



# Prevalence of bullying victimisation amongst first-episode psychosis patients and unaffected controls

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## ABSTRACT

**Background:** Despite increasing evidence suggesting that childhood maltreatment is significantly associated with psychosis, the specific role of bullying in the onset of psychotic disorders is still unclear. This study aimed to examine whether bullying was more prevalent amongst individuals presenting to services for the first time with a psychotic disorder than in unaffected community controls.

**Methods:** Data on exposure to bullying, psychotic symptoms, cannabis use and history of conduct disorder were collected cross-sectionally from 222 first-presentation psychosis cases and 215 geographically-matched controls. Bullying victimisation was assessed retrospectively as part of the Brief Life Events schedule. Logistic regression was used to examine associations between exposure to bullying and case-control status, while controlling for potential confounders.

**Results:** Psychosis cases were approximately twice as likely to report bullying victimisation when compared to controls. No significant interactions between bullying and either gender or cannabis use were found. Controls reporting being a victim of bullying were approximately twice as likely to also report at least one psychosis-like symptom.

**Conclusions:** Our results extend previous research by suggesting that bullying victimisation may contribute to vulnerability to develop a psychotic disorder in some individuals.

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## 1. Introduction

In attempting to better understand the aetiology of psychosis, a substantial body of research has focused on the role of psychosocial factors. A quantitative review and meta-analysis of the available empirical literature indicated that exposure to childhood adverse experiences is strongly associated with increased risk for psychosis (Varese et al., 2012). Indeed, large-scale general population studies indicate that exposure to maltreatment in childhood (such as sexual, physical and emotional abuse, and neglect) increases the risk of experiencing psychotic symptoms in adolescence as well as full-blown psychotic disorders in adulthood (Read et al., 2005; Morgan and Fisher, 2007; Schafer and Fisher, 2011).

However, the specific role of bullying in the later development of psychotic disorder is still unclear (Van Dam et al., 2012). A recent survey conducted in the UK reported that approximately 25% of children had

been bullied by peers during their school years (Radford et al., in press), suggesting that bullying is a common form of early victimisation. Being a victim of bullying has been associated with a wide range of mental health problems in adolescence (Arseneault et al., 2010) as well as sub-clinical psychotic symptoms (Lataster et al., 2006; Campbell and Morrison, 2007; Kelleher et al., 2008; Nishida et al., 2008; Schreier et al., 2009; Arseneault et al., 2011; Mackie et al., 2011; Fisher et al., 2012; Kelleher et al., 2013; Mackie et al., 2013). One general population study has also reported that there is a higher prevalence of bullying victimisation in adults considered to meet criteria for probable psychosis when compared to those without such symptoms (Bebbington et al., 2004). A study of adolescent psychiatric inpatients found that victims of bullying had psychotic disorders two to three times more often than the bullies or bully-victims, but the association was not significant (Luukkonen et al., 2010). Sourander et al. (2007) studied predictive associations between bullying victimisation at age 8 years and psychiatric disorders in early adulthood. They also found no significant association between being a pure victim of bullying and psychotic disorder in adulthood.

Therefore, further investigation of the association between bullying victimisation and psychotic disorder is warranted. None of the studies

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to date has explored the association between bullying victimisation and first clinical presentation for psychotic disorders in comparison to a control group. Neither have potential modifiers been investigated. For instance, gender (Fisher et al., 2009) and cannabis use (Houston et al., 2011; Mackie et al., 2013) have been shown to modify associations between other forms of childhood adversity and psychosis, and children who have been bullied are also at risk of engaging in anti-social behaviours (Liang et al., 2007). Additionally, given the strong associations found between bullying victimisation and depression (Hawker and Boulton, 2000), it also seems important to explore whether similar associations will hold for both schizophrenia-spectrum and affective psychosis diagnoses.

Therefore, the aim of our study was to extend the literature on the association between bullying victimisation and psychosis by focusing on clinically-relevant psychotic disorders and exploring a range of possible modifiers. First, we examined whether a history of bullying victimisation was more prevalent amongst individuals presenting to mental health services for the first time with a psychotic disorder than unaffected community controls. Second, we explored the association between bullying and psychosis by gender, conduct disorder, diagnosis and cannabis use.

## 2. Methods

### 2.1. Participants

The sample was drawn from patients who participated in the Genes and Psychosis (GAP) study from the Lambeth, Southwark and Croydon adult in-patient units of the South London & Maudsley (SLAM) Mental Health National Health Service (NHS) Foundation Trust. Inclusion criteria for cases were: age 16–65 years, presenting to psychiatric services for the first time with a psychotic disorder (codes F20–29 and F30–33 from the International Classification of Diseases [ICD-10]; WHO, 1992) and resident within tightly defined catchment areas in Southeast London, UK. Exclusion criteria were: organic psychosis; intelligence quotient (IQ) under 50; previous contact with services for psychosis, and transient psychotic symptoms resulting from acute intoxication (ICD-10; WHO, 1992). ICD-10 diagnoses were determined using data from the Schedules for Clinical Assessment in Neuropsychiatry (SCAN; WHO, 1994).

Controls were aged 16–65 years and recruited from the local population living in the area served by the Trust, by means of internet and newspaper advertisements, and distribution of leaflets at train stations, shops and job centres. Considerable efforts were made to obtain a control sample that was representative of the general population in age, gender, ethnicity, educational qualifications and employment status. The Psychosis Screening Questionnaire (PSQ; Bebbington and Nayani, 1995) was administered to all potential control group participants; individuals were excluded if they met criteria for a psychotic disorder.

Ethical permission was obtained from the SLAM and the Institute of Psychiatry Research Ethics Committee. All participants provided written consent after reading a detailed information sheet.

### 2.2. Measures

A range of socio-demographic information was obtained including age at interview, gender, current level of education and self-ascribed ethnicity using the UK 2001 census categories. Symptom data were collected on patients during face-to-face interviews with the SCAN (WHO, 1994). This information, supplemented by clinical records, was used to estimate lifetime DSM-IV diagnoses using the OPCRIT diagnostic system (McGuffin et al., 1991).

Data on sub-clinical psychosis-like symptoms in the past year were obtained from controls using the PSQ (Bebbington and Nayani, 1995). Endorsement of one or more symptoms (hypomania, thought

insertion, paranoia, strange experiences, hallucinations) using the criteria outlined by Morgan et al. (2009) was considered to indicate the presence of psychosis-like experiences (PLEs).

Family history of psychotic and affective disorders was obtained from patients and controls for their first degree relatives using the Family Interview for Genetic Studies (FIGS; <https://www.nimhgenetics.org/interviews/figs>).

Bullying was assessed as part of the Brief Life Events schedule adapted from Bebbington et al. (2004). Patients and controls were shown a card listing 10 adverse events (serious injury or assault to yourself, bullying, violence at work, violence in the home, sexual abuse, being expelled from school, running away from home, being homeless, taken into local authority care, and time in children's institution) and asked whether they had ever experienced any of them during their lifetime. If a positive response was obtained, then participants were asked to point out which events they had experienced and whether each one had occurred in the last six months, one year previously, or more than 5 years previously. Only positive responses concerning bullying 5 or more years previously were taken as evidence of having been a victim of bullying in order to minimise the likelihood of psychotic symptoms occurring prior to the bullying exposure. Indeed none of the cases were deemed to have an onset of psychosis more than 5 years prior to interview. An additional life events variable was also created to indicate the presence of any of the other life events (excluding bullying).

Conduct disorder prior to 15 years of age was assessed using the Antisocial Personality/Conduct Disorder module of the Structured Clinical Interview for DSM-IV (SCID-CD; First et al., 1996). This comprises 15 items rated as present, sub-threshold or absent by the interviewer and the presence of 3 or more items was taken to indicate a history of conduct disorder (Malcolm et al., 2011).

Lifetime cannabis use was assessed with the Cannabis Experience Questionnaire modified version (Di Forti et al., 2009). This provides a detailed assessment of lifetime patterns of cannabis and other substance use, including type, age at first use, frequency and duration of use of each substance reported by the respondent. This detailed self-report questionnaire was read out to participants. Participants who responded positively to the item "Have you ever smoked/used cannabis" were subsequently asked about the frequency of use (coded as "everyday" or "once a week or less").

### 2.3. Statistical analysis

Binary logistic regression was used to examine associations between exposure to bullying and psychosis case status, while controlling for potential confounders (age, gender, ethnicity, level of education and family psychiatric history). This was done first with the sample unstratified and then stratified by gender, conduct disorder, diagnosis and cannabis use. Associations are expressed as odds ratios (OR) with 95% confidence intervals (CI). Statistical interactions were assessed using likelihood ratio tests. All analyses were conducted using Stata version 10.1 for Windows (StataCorp, 2009).

A power calculation using the program QUANTO Version 1.2.4 software (<http://hydra.usc.edu/gxe/>) indicated over 90% statistical power (0.92) at a significance level of 0.05, 2-sided, for unmatched case-control analyses to obtain an OR of 2.0 with the total sample size in this study based on estimates of exposure to bullying victimisation amongst the UK general population (25%; Radford et al., in press). In addition, we calculated power for multivariate logistic regression with 7 variables in the regression model using the 'powerlog' function in Stata version 10. For 90% statistical power at a significance level of 0.05, we would require 112 or 150 unmatched cases and controls assuming 0.2 or 0.4 collinearity between the variables, respectively.

### 3. Results

#### 3.1. Sample characteristics

A total of 222 people with psychosis and 215 controls provided information on exposure to bullying. There were no significant differences between psychosis cases and controls with versus without bullying data in terms of demographic characteristics (results, not shown, are available from the authors). The psychosis cases comprised 129 (58.11%) diagnosed with schizophrenia-spectrum disorders and 45 (20.27%) with affective psychosis.

Sociodemographic data by case and control status is presented in Table 1. There was no significant difference between psychosis cases and unaffected controls in terms of age and gender, but controls were more likely to have at least GCSE-level qualifications than cases and be from a White British or White Other ethnic background. Sociodemographic characteristics were therefore controlled for in the subsequent analysis.

In terms of lifetime and frequency of cannabis use, controls were more likely to have never smoked cannabis or to have smoked cannabis infrequently while psychosis cases were more likely to have used cannabis every day. Additionally, around a quarter of psychosis cases had a history of conduct problems.

#### 3.2. Prevalence of bullying victimisation by demographic characteristics

Prevalence of bullying victimisation amongst first-episode psychosis cases and healthy controls, stratified also by gender and cannabis use, is provided in Table 2. Compared with controls, psychosis cases were approximately twice as likely to report experiences of bullying ( $p < 0.001$ ). This association held when adjustment was made for other life events (Adj. OR 2.28, 95% CI 1.49–3.49,  $p < 0.001$ ). Stratifying by gender, the association between bullying victimisation and being a psychosis case held for both men and women and no

statistical interaction by gender was found. Furthermore, significant associations were found between bullying victimisation and having a psychotic disorder regardless of whether individuals had or had not used cannabis in their lifetime (Table 2). The numbers of individuals with different frequencies of cannabis use were too small to permit a more fine-grained stratified analysis.

#### 3.3. Bullying victimisation and psychosis-like experiences

Table 3 presents the prevalence of bullying amongst unaffected controls by presence and absence of PLEs. Previous studies estimated the prevalence of PLEs in the general population within a range from 8% to 28% (Morgan et al., 2009; van Os et al., 2009). In our sample, the prevalence of PLEs was 15.3%, which falls within the range reported by these previous studies. Amongst controls, those that reported at least one PLE were approximately twice as likely to report exposure to bullying as those without such symptoms, though this association just fell short of statistical significance ( $p = 0.051$ ). The strength of the association was similar for men and women, when analysed separately, but also failed to reach conventional levels of statistical significance.

#### 3.4. Bullying victimisation and psychiatric comorbidity/diagnosis

Table 4 presents the prevalence of bullying amongst first-episode psychosis cases by psychiatric comorbidity and diagnosis. Amongst the psychosis cases, those presenting with a history of conduct problems were over two times more likely to report experiences of bullying victimisation ( $p = 0.036$ ). In terms of psychosis diagnosis, a higher prevalence of bullying victimisation was found amongst patients with both schizophrenia-spectrum disorders ( $p < 0.001$ ) and those with affective psychosis ( $p = 0.031$ ) when compared to controls. When adjusted for age, gender, ethnicity, level of education and family psychiatric history, the association between being a victim of bullying and history of conduct problems ( $p = 0.020$ ) and diagnosis of schizophrenia-spectrum disorders ( $p < 0.001$ ) held. However, the strength of the association between bullying victimisation and diagnosis of affective psychosis was attenuated (Adj. OR 1.50) and fell short of statistical significance following adjustment for these confounders ( $p = 0.281$ ).

#### 3.5. Childhood bullying victimisation and psychosis

A total of 133 people were aged 22 or below at the time of interview and thus we can be more certain that their reports of bullying victimisation that occurred 5 or more years previously, were experienced during childhood (prior to 18 years of age). Of these, 64 were cases and 69 were controls. There were significant differences between cases and controls in terms of demographic characteristics (gender, ethnicity and level of education). Controls were more likely to be women ( $\chi^2 = 3.98$ ,  $p = 0.046$ ), to have at least GCSE-level qualifications ( $\chi^2 = 45.84$ ,  $p < 0.001$ ) and be from a White British or White other ethnic background ( $\chi^2 = 14.12$ ,  $p = 0.028$ ) than cases.

We found similar results to those obtained for the overall sample (Table 5). Compared with controls, psychosis cases were approximately twice as likely to report experiences of childhood bullying victimisation ( $p = 0.034$ ). This association held when adjustment was made for confounders ( $p = 0.007$ ). Stratifying by gender, a non-significant association between bullying victimisation and being a psychosis case was observed for men ( $p = 0.085$ ) and a significant association for women ( $p = 0.023$ ). Despite a stronger association being evident amongst women, no statistical interaction by gender was found (Likelihood ratio  $\chi^2 = 0.52$ ,  $p = 0.469$ ). Unfortunately, the small size of this sub-sample did not permit extension of the analysis to testing the

**Table 1**  
Sociodemographic characteristics of first-episode psychosis patients and unaffected controls.

Demographic variable	Patients (N = 222) n (%)	Controls (N = 215) n (%)	Chi-square	df	p Value
Gender			1.99	1	0.159
Men	134 (60.4)	115 (53.5)			
Women	88 (39.6)	100 (46.5)			
Ethnicity			40.21	6	<0.001
White British	51 (23.3)	83 (38.6)			
Black Caribbean	31 (14.2)	32 (14.9)			
Black African	59 (26.9)	29 (13.5)			
White other	23 (10.5)	47 (21.9)			
Asian (all)	17 (7.8)	11 (5.1)			
Mixed background	21 (9.6)	4 (1.9)			
Other	17 (7.8)	9 (4.2)			
Level of education			66.14	4	<0.001
No qualification	35 (15.9)	5 (2.3)			
GCSE/O level	53 (23.6)	20 (9.3)			
A level	33 (15.0)	52 (24.3)			
Vocational/college	54 (24.5)	35 (16.4)			
University or professional qualifications	46 (20.9)	102 (47.7)			
Age, years			t = -0.656	433	0.512
Mean (S.D.)	28.4 (8.8)	27.8 (9.1)			
Cannabis use			6.72	1	0.010
Never	63 (28.4)	88 (40.4)			
At least once	154 (69.4)	127 (59.1)			
History of conduct problems					
Yes	27 (26.0)	–			
No	77 (74.0)	–			

df, degrees of freedom; S.D., standard deviation.

**Table 2**

Prevalence of bullying amongst first-episode psychosis cases and unaffected controls as well as by gender and cannabis use.

Demographic characteristic	Patients n (%)	Controls n (%)	Unadjusted OR	95% CI	p Value	Adjusted <sup>a</sup> OR	95% CI	p Value
Full sample	106 (47.7)	60 (27.9)	2.36	1.59–3.51	<0.001	2.66	1.66–4.26	<0.001
Gender								
Men	56 (41.3)	30 (26.1)	1.99	1.16–3.43	0.012	1.90	0.99–3.67	0.055
Women	50 (56.8)	30 (30.0)	3.07	1.68–5.60	<0.001	3.66	1.80–7.44	<0.001
Likelihood ratio chi-squared test = 1.09, $p = 0.297$ (Adjusted LR $\chi^2 = 1.33$ , $p = 0.249$ )								
Cannabis use								
Never	28 (44.4)	23 (26.1)	2.26	1.14–4.5	0.020	3.40	1.39–8.31	0.007
At least once	76 (49.3)	37 (29.1)	2.37	1.44–3.89	0.001	2.51	1.40–4.52	0.002
Likelihood ratio chi-squared test = 0.01, $p = 0.913$ (Adjusted LR $\chi^2 = 0.02$ , $p = 0.895$ )								

CI, confidence interval; LR, likelihood ratio test; OR, odds ratio.

<sup>a</sup> Adjusted for gender (where applicable), age at interview, ethnicity, educational level and family psychiatric history.

potential interaction with cannabis use, comorbidity with conduct disorder or associations for diagnostic sub-categories.

#### 4. Discussion

This study found that first-episode psychosis patients were significantly more likely to report having been victims of bullying than community controls. This association was also present when experiences of bullying occurred in childhood (prior to 18 years of age). Although the small sample size limits the generalisability of the results, this finding extends previous studies that reported elevated risk for psychotic symptoms in adolescence (Kelleher et al., 2008; Nishida et al., 2008; Schreier et al., 2009; Arseneault et al., 2011; Kelleher et al., 2013) and adulthood (Bebbington et al., 2004) amongst victims of bullying by demonstrating consistent results in patients with clinically-relevant psychotic disorders.

Compared to men (41.3%), a larger proportion of women (56.8%) in the FEP group had been bullied, and the effect of bullying was estimated to be stronger (OR = 3.07 versus 1.99) in women. These results indicate that there is a trend towards effect modification by gender — or that this specific pathway may be more prevalent amongst women. However, in our sample no statistical interaction by gender was found for the association between bullying victimisation and psychosis, but this was probably due to a lack of power. Although bullying victimisation has been shown to be more prevalent amongst boys than girls, possibly because they are more exposed to a range of individual and social risk factors compared to girls (Liang et al., 2007; Arseneault et al., 2010), our previous study found that other forms of early victimisation, such as severe childhood physical or sexual abuse, were associated with psychosis in women but not in men (Fisher et al., 2009). One possible explanation for a differential gender outcome following the experience of childhood victimisation is that girls are more prone to develop internalising difficulties, whereas boys tend to respond by exhibiting externalising behaviour (McFadyen-Ketchum et al., 1996; Fisher et al., 2009). Furthermore, internalising problems have been found to mediate the association between bullying exposure and psychotic symptoms (Fisher et al., 2012) which may therefore put bullied girls at greater risk of developing psychosis. Further research is required to fully elucidate these pathways.

We also failed to find significant differences between cannabis users and non-users in terms of associations between bullying victimisation and being a psychosis case. This result contradicts previous studies that demonstrated an interaction between bullying or other forms of childhood adversity and cannabis use in predicting psychotic experiences in adolescence as well as full-blown psychosis in adulthood (Houston et al., 2011; Mackie et al., 2013). However, Mackie et al. (2013) found that only bullying by peers three or more times a month was associated with cannabis use early in adolescence and cannabis use more than twice predicted a subsequent change in psychotic experiences over time in adolescents. Unfortunately, no information about the frequency and intensity of bullying victimisation and the timing of cannabis use were collected in the current study and these factors might explain the differential results obtained in our study. In fact, only a small proportion of the individuals who use cannabis and experience bullying behaviour go on to develop psychosis, suggesting that certain individuals might present a genetic vulnerability or different patterns of cannabis use and bullying experiences that lead to the development of psychotic experiences (Henquet et al., 2005).

However, we did find a strong association between experiences of bullying and history of conduct problems before age 15 amongst psychosis cases. This result is in line with existing literature that suggests conduct problems or anti-social behaviour are psychiatric correlates of bullying (Liang et al., 2007; Cruzeiro et al., 2008) and that a proportion of victims will also be bullies (Arseneault et al., 2010). Unfortunately, we had insufficient data to explore associations for individuals who had been both a bully and a victim although a previous study suggested that these individuals were at greatest risk of psychotic disorder (Sourander et al., 2007).

Significant associations were also found between bullying victimisation and a diagnosis of both schizophrenia-spectrum disorders and affective psychosis, before adjusting for confounders. Studies assessing clinical diagnoses in psychiatric patients with a history of childhood trauma, specifically neglect, physical or sexual abuse, have found a lack of specificity for psychosis and depression (Livingston, 1987; Friedman et al., 2002). Moreover, a recent meta-analysis concluded that there was no significant difference in rates

**Table 3**

Prevalence of bullying amongst unaffected controls by psychosis-like experiences.

Demographic characteristic	PLE present (N = 26) n (%)	PLE absent (N = 144) n (%)	Unadjusted OR	95% CI	p Value	Adjusted <sup>a</sup> OR	95% CI	p Value
Full sample	11 (42.3)	34 (23.6)	2.37	0.99–5.65	0.051	2.14	0.77–5.93	0.142
Men	6 (40.0)	18 (23.1)	2.22	0.69–7.08	0.177	1.24	0.30–5.02	0.768
Women	5 (45.5)	16 (24.2)	2.06	0.70–9.68	0.153	3.96	0.85–18.45	0.080
Likelihood ratio chi-squared test = 0.03, $p = 0.859$ (Adjusted LR $\chi^2 = 0.67$ , $p = 0.412$ )								

CI, confidence interval; LR, likelihood ratio test; OR, odds ratio; PLE, psychosis-like experiences.

<sup>a</sup> Adjusted for gender (where applicable), age at interview, ethnicity, educational level and family psychiatric history.



**Table 4**

Prevalence of bullying amongst first-episode psychosis cases by psychiatric comorbidity and diagnosis.

Psychiatric comorbidity/diagnosis	N (%)	Unadjusted OR	95% CI	p Value	Adjusted <sup>a</sup> OR	95% CI	p Value
History of conduct problems	18 (66.7)	2.67	1.06–6.68	0.036	3.98	1.25–12.68	0.020
Schizophrenia-spectrum disorders	62 (48.1)	2.39	1.51–3.77	<0.001	3.39	1.88–6.09	<0.001
Affective psychosis	20 (44.4)	2.07	1.07–3.99	0.031	1.50	0.72–3.16	0.281

CI, confidence interval; OR, odds ratio.

<sup>a</sup> Adjusted for gender, age at interview, ethnicity, educational level and family psychiatric history.

of childhood adversity between individuals diagnosed with schizophrenia and those with affective psychosis (Matheson et al., 2012), which is consistent with the findings presented here.

#### 4.1. Bullying victimisation and psychosis-like experiences

We found an association between bullying victimisation and presence of psychosis-like experiences amongst controls, but due to the small size of the sample the generalisability of these results is limited. Nevertheless, this finding is consistent with prospective studies that reported bullying victimisation was a moderate to strong predictor of sub-clinical delusional ideation and hallucinatory experiences in early adolescence (Schreier et al., 2009; Arseneault et al., 2011; Mackie et al., 2011). Therefore, our finding, along with those of previous studies, is in keeping with the hypothesised existence of an aetiological continuum underlying psychotic phenomena in the general population and clinical psychotic disorder (Johns and Van Os, 2001; Johns et al., 2004; Binbay et al., 2011).

#### 4.2. Strengths and weaknesses

This study has several strengths. First of all, we utilised a sample of patients that had recently presented to mental health services with a psychotic disorder, thus extending previous reports that only examined psychotic symptoms or probable psychosis in the general population. Secondly, we utilised a control group to compare the rates of bullying victimisation against, unlike a previous study involving psychiatric patients (Luukkonen et al., 2010). Thirdly, we found prevalence rates of bullying exposure in our controls similar to those reported in studies of the UK general population (Van Dam et al., 2012; Radford et al., in press). Additionally, we were able to control for the potentially confounding effects of other adverse life events, examine associations by diagnostic sub-type, and also explore interactions with gender and cannabis use.

However, this study needs also to be considered in light of a number of limitations. One is the reliance on the retrospective reporting of experiences of bullying. Although several studies have shown some bias in retrospective reports (Cohen and Cohen, 1984), such bias is not considered sufficiently great to invalidate retrospective case-control studies of childhood experiences (Hardt and Rutter, 2004). Moreover, previous studies have demonstrated that the effect of childhood adversity on psychosis remains significant regardless of study design (Varese et al., 2012) and histories of childhood adversity obtained by psychosis patients are reasonably reliable over time and

unaffected by current symptoms (Fisher et al., 2011). Nevertheless, clearly large cohort studies with prospective assessments of bullying victimisation which were followed over time to assess associations with the later development of psychosis would be ideal in order to avoid potential recall bias. However, given the very low prevalence of psychotic disorders in the general population (3% - van Os et al., 2009) and a period of risk extending to on average 40 years of age (Hafner et al., 1993), this is unlikely to be feasible in practice.

As only bullying by peers was investigated in this study, it is possible that other traumas in childhood or adulthood might demonstrate stronger associations with psychotic disorder (Bebbington et al., 2004; Fisher et al., 2010; Beards et al., 2013) or confound this relationship. However, controlling for the presence of other adverse events did not substantially alter the association between bullying victimisation and psychosis in this sample. No information about frequency, intensity and forms of bullying were collected though and these factors might influence the relationship between bullying victimisation and psychosis.

Moreover, as multiple comparisons were made in this study, it is possible that false positives were obtained, thus the results should be interpreted with caution. Although efforts were made to obtain a control sample that was representative of the local community population, it was not randomly selected and thus it is possible that this may have led to erroneous findings. Nonetheless, as mentioned previously, the rates of bullying within the control sample were similar to those found in surveys of the UK general population (Radford et al., in press), suggesting that this aspect of the control sample is unlikely to have affected the results.

From these cross-sectional data, we cannot determine the direction of the relationship between bullying and psychosis. However, we purposely only included bullying victimisation experiences that reportedly occurred 5 or more years before presentation to minimise the likelihood that they occurred after the onset of psychosis. Indeed, none of our cases reported that their psychotic symptoms started more than 5 years prior to the assessment. Nevertheless, we cannot rule out the possibility that people predisposed to psychosis or with prodromal symptoms may appear odd and threatening to peers and so attract bullying (Sideli et al., 2012). However, Kelleher et al. (2013) recently found that bullying victimisation still predicted psychosis-like experiences even when a bidirectional relationship was taken into account. Nonetheless, replication of our findings is warranted in large well-characterised samples of psychosis patients and screened community controls, ideally for whom early behavioural characteristics are known.

**Table 5**

Prevalence of childhood bullying amongst first-episode psychosis cases and unaffected controls by gender.

Demographic characteristics	Patients n (%)	Controls n (%)	Unadjusted OR	95% CI	p Value	Adjusted <sup>a</sup> OR	95% CI	p Value
Full sample	33 (53.1)	24 (34.8)	2.13	1.06–4.27	0.034	3.43	1.40–8.38	0.007
Gender								
Men	19 (46.3)	11 (33.3)	1.73	0.67–4.46	0.259	2.96	0.86–10.14	0.085
Women	14 (63.6)	13 (36.1)	3.10	1.03–9.33	0.045	5.41	1.26–23.25	0.023
Likelihood ratio chi-squared test = 0.62, p = 0.431 (Adjusted LR $\chi^2$ = 0.52, p = 0.469)								

CI, confidence interval; LR, likelihood ratio test; OR, odds ratio.

<sup>a</sup> Adjusted for gender (where applicable), age at interview, ethnicity, educational level and family psychiatric history.

### 4.3. Theoretical mechanisms

The aetiology of psychosis is complex and requires explanatory models that include gene by environment interactions (Shah et al., 2011). Monocausal models have been replaced by multidisciplinary perspectives which integrate psychosocial interactions as well as neurobiological predispositions. In terms of a vulnerability-stress model (Nuechterlein et al., 1994), genetic and developmental vulnerabilities, such as FKBP5 and NOS1AP genotypes, could make individuals more susceptible to psychosocial adversity, such as bullying, increasing stress sensitivity through dysregulation of the hypothalamic–pituitary–adrenal axis and potentially leading to the development of stress-related disorders, such as psychosis and PTSD (Husted et al., 2010; Xie et al., 2010; Appel et al., 2011; Kramer et al., 2011; Boscarino et al., 2012; Klengel et al., 2013). Moreover, it has been demonstrated that the activity of mesolimbic dopamine neurons of the ventral tegmental area is a key determinant of behavioural susceptibility vs resilience to chronic social defeat stress (Cao et al., 2010).

From a cognitive perspective, dysfunctional appraisals about the self and the world that might develop following bullying victimisation, such as hostile attributions of others' intentions, negative self-perceptions and lack of personal control over events, could be related to the onset and maintenance of psychotic phenomena (Campbell and Morrison, 2007; Fowler et al., 2012). Indeed, both low self-esteem and an external locus of control have been reported to form an indirect pathway between bullying victimisation in childhood and psychotic-like symptoms in early adolescence in a large prospectively assessed UK birth cohort (Fisher et al., 2012).

### 4.4. Conclusion

This paper adds to the evidence that bullying may be part of a pathway during adolescence and young adulthood that leads to the development of psychotic disorders (Morgan et al., 2010). The present results tentatively suggest that bullying may contribute towards vulnerability to a psychotic disorder in some individuals, though clearly replication in other large first-presentation psychosis samples is required.

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### Contributors

Authors AT, MDF, VM, PD, CP, TD, RMM and HLF designed the study and wrote the protocol. AT, AM, LF, IF, RMM and HLF conducted the literature searches and statistical analyses. AT, RMM, HLF wrote the first draft of the manuscript. All authors have contributed to and have approved the final manuscript.

### Conflict of interest

All authors declare they have no conflicts of interest.

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