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ORIGINAL ARTICLE

# Tobacco smoking is associated with psychotic experiences in the general population of South London

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**Background.** The association between cigarette smoking and psychosis remains unexplained, but could relate to causal effects in both directions, confounding by socioeconomic factors, such as ethnicity, or use of other substances, including cannabis. Few studies have evaluated the association between cigarettes and psychotic experiences (PEs) in diverse, inner-city populations, or relationships with number of cigarettes consumed.

**Methods.** We assessed associations and dose–response relationships between cigarette smoking and PEs in a cross-sectional survey of household residents ($n = 1680$) in South East London, using logistic regression to adjust for cannabis use, other illicit substances, and socioeconomic factors, including ethnicity.

**Results.** We found association between any PEs and daily cigarette smoking, which remained following adjustment for age, gender, ethnicity, cannabis and use of illicit stimulant drugs (fully adjusted odds ratio 1.47, 95% confidence interval 1.01–2.15). Fully adjusted estimates for the association, and with number of PEs, increased with number of cigarettes smoked daily, implying a dose–response effect ($p = 0.001$ and <0.001, respectively). Odds of reporting any PEs in ex-smokers were similar to never-smokers.

**Conclusions.** In this diverse epidemiological sample, association between smoking and PEs was not explained by confounders such as cannabis or illicit drugs. Daily cigarette consumption showed a dose–response relationship with the odds of reporting PEs, and of reporting a greater number of PEs. There was no difference in odds of reporting PEs between ex-smokers and never-smokers, raising the possibility that the increase in PEs associated with smoking may be reversible.

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**Key words**: Confounding, epidemiology, ethnicity, psychosis, tobacco smoking

## Introduction

The search for environmental causes for psychosis (Dean & Murray, 2005) in the past three decades has included factors that can be experienced after childhood, for example, migration and the use of substances (Morgan *et al.* 2010). Investigations into the possible causal effects of cannabis have featured prominently in research into substances, with a meta-analysis estimating that cannabis users experienced nearly three times the odds of having psychosis compared with non-users [odds ratio (OR) 2.93, 95% confidence interval (CI) 2.36–3.64] (Semple *et al.* 2005). More recently, other drugs have been examined, most notably tobacco.

There is a strong positive association between smoking cigarettes and psychotic disorders (de Leon & Diaz, 2005). The most recent meta-analysis of smoking as a risk factor for psychosis estimated the OR for daily smoking to be around 3, based on 11 case–control studies, and the relative risk, from five prospective studies, to be approximately 2 (Gurillo *et al.* 2015). The positive association between tobacco smoking and psychotic illnesses has a number of candidate explanations. These include:

(a) Self-medication (Kumari & Postma, 2005), for example, of psychiatric symptoms (Smith *et al.* 2002), cognitive deficits (George *et al.* 2002; Sacco *et al.* 2005; Barr *et al.* 2008) or adverse effects of psychiatric drugs (Goff *et al.* 1992),

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(b) Shared genetic liability to both smoking and psychoses (Lyons *et al.* 2002; Smith *et al.* 2008; Chen *et al.* 2016),
(c) A causal effect of smoking on schizophrenia (Weiser *et al.* 2004; Kendler *et al.* 2015),
(d) A reverse causal effect – mental health problems could result in people who smoke being less likely to quit, for example, because of more severe nicotine dependence or more limited access to smoking cessation treatment (Szatkowski & McNeill, 2014), and
(e) Confounding by other drug use – people who smoke are more likely to take other drugs, including cannabis and stimulants (Regier *et al.* 1990; Morral *et al.* 2002), which may be causally associated with psychosis (Semple *et al.* 2005; Large *et al.* 2011).

In a recent prospective study, Kendler *et al.* found that smoking was associated with later schizophrenia in two Swedish cohorts, after accounting in the design for shared familial factors between people who developed schizophrenia and those who did not (Kendler *et al.* 2015). Heavy smokers in discordant monozygotic twin pairs were around 1.7 times more likely to develop psychosis compared with the non-smoking twin, suggesting that genetic factors do not completely explain the relationship between smoking and later psychosis. Strengths of association were not affected by specifying different buffer periods between smoking assessment and first diagnosis, implying that the association did not arise as a result of people smoking as part of the psychosis prodrome.

It is increasingly argued that psychotic disorders represent the extreme end of a phenomenological continuum of psychotic experiences (PEs), which extend into the general, non-clinical population (Johns & Van Os, 2001; Johns *et al.* 2004; Linscott & van Os, 2013). However, although observational data from a number of sources have indicated high smoking prevalence in people with mental disorders [Royal College of Psychiatrists (RCPSYCH, 2013)], few studies have addressed the question of whether tobacco smoking is associated with PEs in the general population (Van Gastel *et al.* 2013; Gage *et al.* 2014). Furthermore, the extent to which any association is explained by confounding cannabis or socioeconomic factors is unclear.

This study examined the association between cigarette smoking and PEs in a representative population-based sample of South London.

Our objectives were to:

(1) Estimate the association between smoking and PEs and between smoking and the number of PEs reported, taking into account possible confounding by cannabis, stimulant use and ethnicity, and
(2) Test for a linear trend in the odds of reporting PEs with quantity of cigarettes smoked per day.

## Methods

### *Sample*

The South East London Community Health study [SELCoH (Hatch *et al.* 2011)] is a representative household survey of South East London residents collected in 2008–2010. The analytic sample consisted of 1698 people, residing in 1075 households, collected through random sampling of a postcode address file, who were interviewed by lay researchers. Respondents were between 16 and 90 years of age. Of 2359 people eligible within participating households, 1698 (71.9%) participated.

### *Psychotic experiences*

The rating scale used for the assessment of PEs was the Psychosis Screening Questionnaire [PSQ (Bebbington & Nayani, 1995)]. The PSQ is a self-report questionnaire designed to be administered by lay interviewers for use in large-scale epidemiological studies, for the purpose of screening respondents for possible psychotic disorder. It is a five-item questionnaire that assesses different positive psychotic symptom domains experienced in the previous year. These comprise: hypomania, strange experiences, persecutory experiences, auditory hallucinations, and thought interferences. Each domain contains an initial 'probe' item, which is followed by secondary questions. Because the present study was focused on non-affective psychosis, responses to the hypomania item were not examined. Endorsement of PEs was defined as positive response to items in the four remaining domains. This approach was consistent with a previous analysis of PEs originating from these data (Morgan *et al.* 2014). Information on the number of domains endorsed was also available. The PSQ displays good correspondence with psychosis items on the Schedules for Clinical Assessment in Neuropsychiatry (Bebbington & Nayani, 1995) and has seen frequent use in population studies (Johns *et al.* 2002; Bebbington *et al.* 2004; Johns *et al.* 2004).

### *Sociodemographic and clinical measures*

Data on age, gender, employment status (employed, unemployed, student, other), ethnicity (White, Black Caribbean, Black African, Asian, and other), marital status (single, married/cohabiting, divorced/separated, and widowed), social class (measured by the National Statistics Socio-Economic Classification), a composite score of general cognitive ability (details available in

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Mollon *et al.* (2016), and highest educational attainment (no qualifications, General Certificate of Secondary Education, A level, and degree level or above) were available. The presence of symptoms of a common mental disorder in the previous 2 weeks was defined based on responses to the CISR [Clinical Interview Schedule, Revised (Lewis & Pelosi, 1990)], with a cut-off score of 12 (Lewis *et al.* 1992).

### *Measurement of cigarette smoking:*

Information on cigarette smoking analysed in this study was collected from SELCoH participants at four levels: the category of 'never smoked' was based on answering 'no' to the question: 'Have you ever smoked a cigarette?'. Ex-smokers were defined as those answering 'yes' to the question: 'Have you ever smoked a cigarette?' And then answering 'no' to the question: 'Do you smoke cigarettes at all nowadays?'. Sporadic smoking was based on answering 'yes' to the question: 'Have you ever smoked a cigarette?', then 'yes' to the question: 'Do you smoke cigarettes at all nowadays?', and then reporting a zero daily cigarette intake when asked: 'About how many cigarettes a day do you usually smoke?'. Finally, daily smokers were defined by answering positively to both prior questions and providing an estimate of the number of daily cigarettes smoked. All participants defined as daily smokers were therefore current smokers.

### *Ascertainment of cannabis use*

Participants were asked about cannabis use frequency and categorised into the following groups: never used, use less frequently than once a week, use more than once a week but less than daily, and use daily.

### *Evaluation of stimulant substance use*

Participants reported use of amphetamines, ecstasy, cocaine, and crack use; all were combined into a single variable with three levels – never used, use but not in the previous year, and use in the previous year. All models which adjusted for substance misuse included this three-level variable.

### *Analysis*

All analyses were carried out in STATA 14 (StataCorp, 2014) and took account of non-response weights and clustering of responses by household. Inverse probability weights (Pickles *et al.* 1995) were calculated from logistic regression models for non-response of an eligible individual within households. Predictor variables for non-response were selected for inclusion in the final weights model based on strength of statistical evidence ($p$ values of <0.05) and whether the selected weighting scheme reproduced the means and prevalences of participants with complete data. The final prediction model contained effects of age and gender. Categorical descriptions of the sample by PEs were inspected. Univariate associations between PE status and cigarette smoking, stimulants, and sociodemographic variables (age, gender, and ethnicity) were evaluated and presented. Multivariate models were used to assess and account for confounding. Age and gender were included in all models. Covariates whose inclusion in the model did not deviate the association between PEs and daily cigarette smoking by more than 10% of the unadjusted OR were discarded (Greenland *et al.* 2016). This left age, gender, and ethnic group as covariates in modelling, alongside stimulant and cannabis use as potential confounders of primary interest. In particular, neither the inclusion of general cognitive ability, marital status, employment status, social class, nor educational attainment altered estimates sufficient for their inclusion. Having identified evidence of strong negative confounding by ethnicity, we explored the association between ethnic group and smoking, presented in online Supplementary Table S7. Descriptive data on the overlap between cigarette smoking and use of cannabis and stimulants are also presented as supplementary material. Modification of the association between current smoking and reporting any PEs by age, cannabis use, and common mental disorder was tested by fitting multiplicative interaction terms for smoking status by age, cannabis use, and common mental disorder in fully adjusted models. Ordinal logistic regressions were used to assess the association between smoking status and number of PEs (range from 0 to 4). Finally, we examined the possibility of a dose–response relationship by assessing linear trends in the association between the number of cigarettes smoked and (a) the odds of reporting any PEs (from logistic regression models), and (b) the odds of reporting one further PE (from ordinal logistic regression models).

## Results

After excluding participants with missing data on the modelled variables, 1680 survey participants remained for analysis. Sociodemographic and substance use associations with PEs are shown in Table 1. PEs were more frequently reported by younger participants, and those with Black Caribbean and Black African ethnic status. Cannabis, ecstasy, cocaine, and other stimulants were associated with PEs. The estimate for crack cocaine, while indicating a possible strong association, was imprecise and not statistically significant, as its use was seldom reported. Cannabis use frequency was strongly associated with use of stimulant drugs (see

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**Table 1.** *Counts and survey-weighted univariate associations between PEs and each variable used in this study, based on the analytic sample of 1680*

| | Number in each category | Number reporting PEs in category (%) | PE odds ratio | 95% CI |
|---|---|---|---|---|
| Age | | | | |
| 16–24 | 356 | 85 (23.88) | Reference | |
| 25–34 | 401 | 69 (17.21) | 0.69 | 0.48–0.99 |
| 35–44 | 334 | 64 (19.16) | 0.76 | 0.52–1.11 |
| 45–54 | 259 | 57 (22.01) | 0.91 | 0.62–1.34 |
| 55–64 | 157 | 25 (15.92) | 0.61 | 0.37–1.00 |
| 65+ | 173 | 19 (10.40) | 0.37 | 0.21–0.66 |
| Female | 950 | 171 (18.00) | 0.86 | 0.68–1.09 |
| Ethnicity | | | | |
| White | 1045 | 170 (16.27) | Reference | |
| Black Caribbean | 143 | 45 (31.47) | 2.28 | 1.51–3.47 |
| Black African | 229 | 51 (22.27) | 1.46 | 1.01–2.10 |
| Asian | 62 | 8 (12.90) | 0.71 | 0.35–1.44 |
| Other | 201 | 44 (21.89) | 1.43 | 0.98–2.06 |
| Smoking pattern | | | | |
| Never-smoker | 513 | 84 (16.37) | Reference | |
| Ex-smoker | 448 | 77 (17.19) | 1.06 | 0.75–1.49 |
| Sporadic smoker | 297 | 47 (15.82) | 0.97 | 0.65–1.44 |
| Daily smoker | 422 | 110 (26.07) | 1.76 | 1.27–2.42 |
| Crack use | | | | |
| Never | 1642 | 300 (18.27) | Reference | |
| Yes, not in the last year | 34 | 16 (47.06) | 3.82 | 1.93–7.61 |
| Yes, in the last year | 4 | 2 (50.00) | 4.04 | 0.56–28.91 |
| Ecstasy use | | | | |
| Never | 1383 | 245 (17.72) | Reference | |
| Yes, not in the last year | 215 | 50 (23.26) | 1.42 | 1.00–2.02 |
| Yes, in the last year | 82 | 23 (28.05) | 1.78 | 1.05–3.02 |
| Amphetamine use | | | | |
| Never | 1409 | 247 (17.53) | Reference | |
| Yes, not in the last year | 241 | 61 (25.31) | 1.59 | 1.16–2.18 |
| Yes, in the last year | 30 | 10 (33.33) | 2.19 | 1.04–4.63 |
| Cocaine use | | | | |
| Never | 1308 | 225 (17.20) | Reference | |
| Yes, not in the last year | 238 | 55 (23.11) | 1.45 | 1.03–2.04 |
| Yes, in the last year | 134 | 38 (28.36) | 1.87 | 1.22–2.84 |
| Any stimulant use | | | | |
| Never | 1234 | 204 (16.53) | Reference | |
| Yes, not in the last year | 287 | 69 (24.04) | 1.80 | 1.25–2.61 |
| Yes, in the last year | 159 | 45 (28.30) | 1.94 | 1.31–2.88 |
| Cannabis use | | | | |
| Never | 1502 | 255 (16.98) | Reference | |
| Less than once a week | 74 | 21 (28.38) | 1.89 | 1.10–3.22 |
| More than once a week but less than daily | 57 | 22 (38.60) | 3.00 | 1.76–5.16 |
| Daily | 47 | 20 (42.55) | 3.49 | 1.89–6.45 |
| Total | 1680 | 318 (18.93) | – | – |

PE, psychotic experience; CI, confidence interval.

online Supplementary Table S6). There was an association between PEs and daily, but not sporadic or past, cigarette smoking. Multivariate models for the odds of reporting any PEs are show in Table 2: when sociodemographic variables were included in the model, the estimate increased, indicating positive confounding by age, gender, and ethnicity. Further adjustment for cannabis frequency attenuated the OR

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**Table 2.** *OR estimates for smoking pattern on PEs from survey weighted logistic regression*

| | Model I: unadjusted | | Model II: Model I adjusted for age, gender, and ethnicity | | Model III: Model II further adjusted for frequency of cannabis use | | Model IV: Model III further adjusted for stimulant use | |
|---|---|---|---|---|---|---|---|---|
| | OR | 95% CI | OR | 95% CI | OR | 95% CI | OR | 95% CI |
| Never-smoker | Reference | | Reference | | Reference | | Reference | |
| Ex-smoker | 1.06 | 0.75–1.49 | 1.40 | 0.96–2.05 | 1.30 | 0.89–1.90 | 1.25 | 0.78–1.82 |
| Sporadic smoker | 0.97 | 0.65–1.44 | 1.09 | 0.72–1.66 | 1.05 | 0.69–1.59 | 1.03 | 0.68–1.58 |
| Daily smoker | 1.76 | 1.27–2.42 | 2.05 | 1.44–2.92 | 1.66 | 1.15–2.4 | 1.47 | 1.01–2.15 |

All models are based on 1680 participants. Age was adjusted for as a continuous variable.
OR, odds ratio; CI, confidence interval.

for daily smoking on PEs. Finally, adjustment for stimulant use (recent and in the lifetime) modestly reduced the association. No statistical evidence was found for differences in the association between current smoking and the odds of reporting PE within different age groups, or at different levels of cannabis use.

We found strong statistical evidence for a dose–response relationship between the number of cigarettes smoked and the odds of reporting any PEs, and the reporting of a greater number of PEs, in adjusted models. On average, an increase in daily cigarette consumption from 0 to 1–9, from 1–9 to 10–19, or 10–19 to 20 or more was accompanied by a 1.04 increase in the overall relative odds of reporting any PEs (95% CI 1.02–1.07; Table 3) and a 1.58-fold increase in the relative odds of reporting one further PE (95% CI 1.32–1.90; Table 3).

Daily smoking was associated not only with an increased odds of reporting PEs, but also with increasing number of PEs, although this estimate lost precision after adjusting for stimulant use (fully adjusted OR 1.55, 95% CI 0.98–2.47, Table 4). The most common PE was strange experiences (6.05%), followed by auditory hallucinations (3.87%), then persecutory experiences (3.27%), with thought interferences the least common PE (1.32%). Individual types of PE were associated with daily smoking, with precise estimates for strange experiences, but not for the other symptoms. In fully adjusted models, associations remained for each symptom, but lost precision. On account of the association between PEs and other symptoms of mental disorder, we estimated associations of PEs with smoking pattern by common mental disorder, as shown in online Supplementary Table S5. No statistical evidence was found for variation in effect estimates by common mental disorder, although this test lacked power. Because of the association between ethnicity and PEs, and the attenuation in estimates observed when it was included in regression models, we described the association between ethnicity and smoking, as reported in online Supplementary Table S7: all non-White ethnic groups had lower proportions of reported daily, ex-, and sporadic smoking compared with the White reference group ($p < 0.001$).

## Discussion

### *Summary of findings*

We found evidence of a cross-sectional association between daily cigarette smoking, but not ex-smoking, and PEs in a sample of household residents in South East London. The association was not explained completely by cannabis use frequency, or by use of stimulant drugs, or by ethnicity (ethnicity was strongly associated with daily smoking, see online Supplementary Table S7). There was an increasing strength of association observed by number of cigarettes smoked, and increased cigarette consumption predicted a greater number of PEs. We did not find statistical evidence for interaction of smoking with age, cannabis use, or with symptoms of common mental disorder.

### *Previous literature*

Smoking is a crucial, potent, and modifiable cause of morbidity and mortality in the UK (Matcham *et al.* 2017). Although the number of people who smoke in the UK is falling (Action on Smoking and Health, 2015), this decline is not reflected in people with mental illness (McManus et al. 2010); and data from the Health Survey for England suggest that smoking may be declining more slowly in people with mental health problems compared with those without

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**Table 3.** *OR estimates for the association between (a) any PEs (upper panel) and (b) the number of PEs (lower panel, reflecting the increase in relative odds for one more PE) with quantity of cigarettes smoked per day*

| | Model I: unadjusted | | Model II: Model I adjusted for age, gender, and ethnicity | | Model III: Model II further adjusted for frequency of cannabis use | | Model IV: Model III further adjusted for stimulant use | |
|---|---|---|---|---|---|---|---|---|
| Daily cigarettes smoked | | | | | | | | |
| Any PE | OR | 95% CI | OR | 95% CI | OR | 95% CI | | OR 95% CI |
| 0 | Reference | | Reference | | Reference | | Reference | |
| 1–9 | 1.51 | 1.06–2.15 | 1.38 | 0.96–2.00 | 1.15 | 0.78–1.69 | 1.05 | 0.71–1.56 |
| 10–19 | 2.26 | 1.54–3.31 | 2.36 | 1.59–3.49 | 1.98 | 1.31–2.98 | 1.88 | 1.24–2.84 |
| 20 or more | 2.13 | 1.24–3.67 | 2.54 | 1.46–4.43 | 2.20 | 1.25–3.89 | 2.04 | 1.15–3.62 |
| | *T for linear trend = 4.19 p < 0.001* | | *T for linear trend = 4.76 p < 0.001* | | *T for linear trend = 3.66 p < 0.001* | | *T for linear trend = 3.32 p = 0.001* | |
| Number of PEs | | | | | | | | |
| 0 | Reference | | Reference | | Reference | | Reference | |
| 1–9 | 1.65 | 1.03–2.64 | 1.62 | 1.00–2.64 | 1.39 | 0.85–2.29 | 1.36 | 0.83–2.22 |
| 10–19 | 2.52 | 1.61–3.94 | 2.97 | 1.85–4.76 | 2.50 | 1.52–4.11 | 2.37 | 1.43–3.91 |
| 20 or more | 4.17 | 2.29–7.62 | 5.53 | 3.03–10.09 | 4.70 | 2.50–8.83 | 4.32 | 2.29–8.15 |
| | *T for linear trend = 3.32 p = 0.001* | | *T for linear trend = 6.52 p < 0.001* | | *T for linear trend = 5.30 p < 0.001* | | *T for linear trend = 4.98 p < 0.001* | |

Based on overall analytic sample of 1680. Test statistics (T) are from survey-weighted logistic regression models.
PE, psychotic experience; OR, odds ratio; CI, confidence interval.

(Szatkowski & McNeill, 2014). Therefore, identifying the mechanisms by which smoking and mental illness are associated could be beneficial for public health.

Our findings that PEs and daily smoking are associated, are consistent with a small body of literature suggesting that smoking is more common in people with sub-clinical PEs than the rest of the general population. Firstly, van Gastel *et al.* (2013) reported an analysis of an internet survey, finding that the cross-sectional association between scores on the community assessment of PEs and daily smoking for the past month remained apparent despite accounting for cannabis use and for a group of other confounders. Secondly, smokers were 1.3 times more likely to report PEs in the World Health Surveys compared with non-smokers, after adjustments, suggesting the association is consistent across national settings (Koyanagi *et al.* 2016). Thirdly, Wiles *et al.* (2006) reported association between smoking and PEs in the 2007 UK Adult Psychiatric Morbidity Surveys, but found that the crude association was strongly attenuated by adjustment for cannabis, general cognitive ability and marital status. Fourthly, Saha *et al.* (2011) found that daily smoking was associated with reporting delusion-like experiences in an Australian household survey (2011), after adjusting for a broad range of confounders. Fifth, in an analysis of prospective data from the Avon Longitudinal Study of Parents and Children, Gage *et al.* (2014) reported that smoking at age 16 was predictive of PEs at 18, after accounting for cannabis use frequency and a range of early and mid-life confounders. Overall, few previous studies have assessed dose–response relationship with number of cigarettes smoked or by number of PEs reported, and few studies have adjusted for cannabis use in detail, for example, by including cannabis use frequency in statistical models.

### *How our results fit in*

Our results, from a highly socioeconomically and ethnically diverse sample, are consistent with the previous literature suggesting the cross-sectional association between cigarette smoking and PEs is not fully explained by cannabis use, the use of stimulant drugs, or confounding by demographic or socioeconomic status, particularly by ethnic group. Furthermore, we present evidence that the relationship between odds of reporting any PEs, and a greater number of PEs is related to the number of cigarettes smoked per day. Finally, we extend previous literature by presenting evidence that daily smoking predicts the reporting of more PEs on a continuous scale. We also found no evidence of association between PEs and being an ex-smoker, implying that our analysis did

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**Table 4.** *Models comparing daily smokers to never-smokers for an increase in number of PEs, and for separate types of psychotic experience*

| | OR (95% CI) for an increase of one psychotic experience | OR (95% CI) for auditory hallucinations | OR (95% CI) for thought interferences | OR (95% CI) for persecutory experiences | OR (95% CI) for strange experiences |
|---|---|---|---|---|---|
| Number reporting this symptom (% of sample) | | 65 (3.88) | 22 (1.32) | 55 (3.30) | 100 (6.05) |
| Model I: unadjusted | 1.76 (1.19–2.59) | 1.68 (0.91–3.08) | 2.17 (0.72–6.57) | 1.45 (0.76–2.77) | 2.02 (1.19–3.42) |
| Model II: Model I further adjusted age, gender, and ethnicity | 2.12 (1.37–3.26) | 2.14 (1.05–4.37) | 4.65 (1.53–14.09) | 1.62 (0.76–3.46) | 2.39 (1.37–4.18) |
| Model III: Model II further adjusted for cannabis use | 1.70 (1.09–2.66) | 1.78 (0.85–3.72) | 3.37 (1.04–10.91) | 1.32 (0.59–2.96) | 1.89 (1.05–3.4) |
| Model IV: Model III further adjusted for stimulant use | 1.55 (0.98–2.47) | 1.56 (0.72–3.39) | 3.25 (0.97–10.87) | 1.14 (0.51–2.58) | 1.74 (0.95–3.18) |

All models are based on 1680 participants. Estimates for ex-smokers and sporadic smokers are not presented.
PE, psychotic experience; OR, odds ratio; CI, confidence interval.

not suffer from confounding by non-time-varying characteristics, such as unadjusted sociodemographic factors. The finding of no association between PEs and ex-smoking is consistent with other literature suggesting that mental health improves following smoking cessation (Taylor *et al.* 2014) raising the possibility that the increase in PEs associated with smoking may be reversible. Our results are also consistent with smoking being a more persistent behaviour in people with PEs compared with those without, and fit with some evidence that people with psychosis who smoke tend to have more severe positive symptoms and more limited social adjustment (Barnes *et al.* 2006; Krishnadas *et al.* 2012).

### *Strengths and limitations*

This was a cross-sectional study, and these associations could be explained by smoking occurring after PEs. Measurement of smoking, PEs, and cannabis use were by self-report in the same survey, and some way of confirming this information in independent data would have improved the validity of the study. Strong collinearity between exposure and a confounder limits the ability of regression methods to correctly adjust for confounding – in this study, the close overlap between cannabis use and cigarette smoking (Amos *et al.* 2004) might not have fully allowed for the identification of separate effects for these two factors (Gage *et al.* 2014; Greenland *et al.* 2016). There were no data on the persistence or timeframe of PEs, further limiting inference. Although we were able to adjust estimates for stimulant use, this was in the form of an aggregated variable across four different stimulants, leaving open the possibility of residual confounding by the use of individual stimulants. However, despite these limitations to the data, the study did allow the assessment of this association in an urban, diverse population with relatively high levels of cannabis and stimulant use, in contrast to previous studies. The generalisability of our results to the rest of the UK population could be limited. However, a previous study based on these data suggested similarity in the distributions of age, gender, economic activity, and ethnicity to the overall English population recorded in the UK Census (Hatch *et al.* 2011). We found evidence that ethnic group was strongly related to the probability of daily smoking, in accordance with other studies (Best *et al.* 2001; Wanigaratne *et al.* 2003; McCambridge & Strang, 2005), and adjusted for it as a possible confounder (see online Supplementary Table S7).

## Conclusions

The association between PEs and smoking is apparent in a highly diverse population with relatively prevalent use of cannabis and stimulant drugs. The linear relationship between cigarette consumption and odds of reporting PEs requires urgent explanation in longitudinal studies and diverse populations.

## Supplementary Material

The supplementary material for this article can be found at https://doi.org/10.1017/S0033291717001556.

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