

Cannabis and schizophrenia: model projections of the impact of the rise in cannabis use on historical and future trends in schizophrenia in England and Wales

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ABSTRACT

Aims To estimate long-term trends in cannabis use and projections of schizophrenia assuming a causal relation between cannabis use and schizophrenia. **Methods** Trends in cannabis use were estimated from a national survey, 2003; and incidence of schizophrenia was derived from surveys in three English cities, 1997–99. A difference equation cohort model was fitted against estimates of schizophrenia incidence, trends in cannabis exposure and assumptions on association between cannabis and schizophrenia. The model projects trends in schizophrenia incidence, prevalence and attributable fraction of cannabis induced schizophrenia. **Results** Between 1970 and 2002 cannabis exposure increased: incidence by fourfold; period prevalence by 10-fold; and use among under 18-year-olds by 18-fold. In 1997–99 incidence and prevalence of schizophrenia were 17 per 100 000 and 0.63% among men and 7.3 per 100 000 and 0.23% among women, respectively. If cannabis use causes schizophrenia, earlier increases in cannabis use would lead to increases in overall schizophrenia incidence and prevalence of 29% and 12% among men between 1990 and 2010. By 2010 model projections which assume an association between schizophrenia and light and heavy users suggest that approximately one-quarter of new schizophrenia cases could be due to cannabis, whereas if the association is twofold and confined to heavy cannabis users, then approximately 10% of schizophrenia cases may be due to cannabis. **Conclusions** If cannabis use causes schizophrenia, and assuming other causes are unchanged, then relatively substantial increases in both prevalence and incidence of schizophrenia should be apparent by 2010. More accurate data on cannabis consumption and future monitoring of schizophrenia are critical.

Keywords Cannabis, epidemiology, modelling, schizophrenia.

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INTRODUCTION

There has long been uncertainty and debate over the health consequences of cannabis use and the related question of the most appropriate regulatory framework to minimize both well-established and possible harms [1]. The clearest health-damaging consequences of use relate to the fact that most users appear to consume cannabis in conjunction with tobacco. However, recent concern, particularly in the United Kingdom, has centred upon whether cannabis use causes schizophrenia, and whether this has implications for the regulatory framework around the drug [2].

Longitudinal studies from outside the United Kingdom show a consistent association between different levels of reported cannabis use and a variety of psychotic symptom phenotypes [3–8]. In one large Swedish study the association extended to an increased risk of hospitalization for schizophrenia [3]. Whether these associations provide sufficient evidence for a causal association or could be explained by other factors is disputed [9–12]. Assuming causality, weekly cannabis use in late adolescence may approximately treble the risk of subsequent schizophrenia, whereas more moderate use may still be associated with a doubling of risk [3].

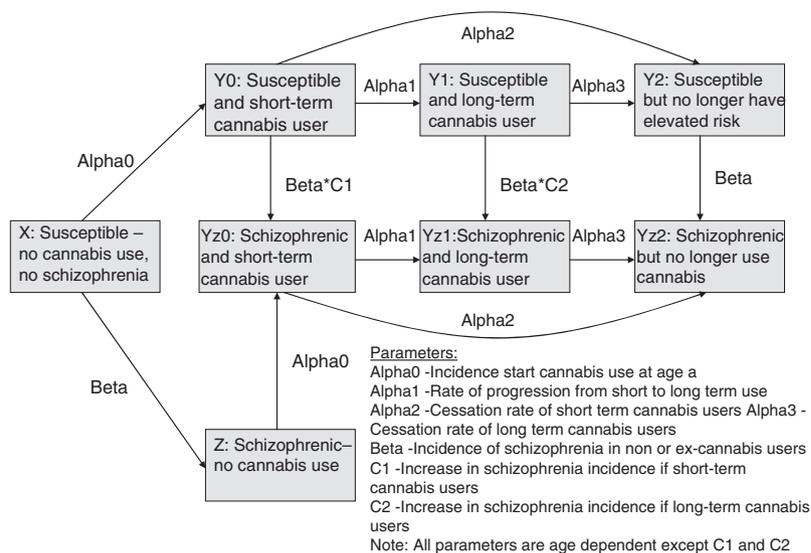


Figure 1 Flow diagram of the model relating cannabis and schizophrenia

The effects of this magnitude on schizophrenia risk should, given the high and apparently increasing proportion of the population exposed, lead to changes in the rates of schizophrenia. In Australia, Degenhardt and colleagues compared projected trends in schizophrenia with trends in cannabis use over the same period and concluded that these were inconsistent with a causal association between cannabis use and schizophrenia [13]. Unfortunately, the United Kingdom has no equivalent schizophrenia register and there is conflicting evidence, with some studies suggesting that clinically diagnosed schizophrenia may have fallen but psychotic syndromes have increased and others suggesting that schizophrenia may have increased in some areas but declined nationally [14–18]. Instead, using national survey data, we estimated patterns in cannabis use among the UK population between 1970 and 2002; estimated incidence of schizophrenia in late 1990s using data from three English cities, and modelled changes in schizophrenia rates that would be expected on the basis of a causal relation between cannabis use and schizophrenia.

METHODS

Model description

Crudely, the current incidence of schizophrenia comprises the incidence among the exposed and unexposed population:

$$I_{sch} = I_e \times p_e + I_u \times p_u, \tag{eqn 1}$$

where I_{sch} is schizophrenia incidence, I_e is schizophrenia incidence among cannabis users, p_e is prevalence of cannabis use, I_u is schizophrenia incidence among

non-cannabis users and p_u is prevalence of non-cannabis use ($p_u = 1 - p_e$). If RR is the risk ratio of schizophrenia among the population exposed to cannabis then:

$$I_e = RR \times I_u \tag{eqn 2}$$

If eqn 2 is substituted into eqn 1, the following can be obtained:

$$I_u = \frac{I_{sch}}{p_e[RR - 1] + 1} \tag{eqn 3}$$

Although we do not know I_u or I_e , eqns 2 and 3 can be used to estimate them, given estimates of schizophrenia incidence (I_{sch}), cannabis prevalence (p_e) and assumptions of the RR. Based on this framework we developed a difference equation cohort model to generate projections of schizophrenia incidence and prevalence, and proportion of cases caused by cannabis (i.e. attributable fraction), for different birth cohorts of men and women born in 1945–49 to 1985–89.

The model simulates the flow of individuals into cannabis use and schizophrenia by age. The structure of the model is shown in Fig. 1, with the state variables shown in each compartment. All individuals of a particular birth cohort enter the population at age 10 years, and are assumed to not have schizophrenia or use cannabis. Each year, individuals have an age-specific incidence of starting cannabis use ($\alpha 0$), after which they either become long-term users (at a rate $\alpha 1$) or stop using cannabis (at a rate $\alpha 2$) and become an ex-user. Long-term users also gradually become ‘ex-users’, but at a rate ($\alpha 3$). The model assumes that all individuals can develop schizophrenia, but that cannabis use may increase the risk depending on the duration of use.

The transition equations for the model are shown below, where subscript ‘a’ is the age of the cohort and the

equations define the state at age 'a + 1' in terms of the state at age 'a':

$$\begin{aligned}
 X_{a+1} &= X_a(1 - \alpha 0_a - \beta_a) \\
 Y0_{a+1} &= Y0_a(1 - \alpha 1_a - \alpha 2_a - C1\beta_a) + \alpha 0_a X_a \\
 Y1_{a+1} &= Y1_a(1 - \alpha 3_a - C2\beta_a) + \alpha 1_a Y0_a \\
 Y2_{a+1} &= Y2_a(1 - \beta_a) + \alpha 2_a Y0_a + \alpha 3_a Y1_a \\
 YZ0_{a+1} &= YZ0_a(1 - \alpha 1_a - \alpha 2_a) + C1\beta_a Y0_a + \alpha 0_a Z_a \quad (\text{eqn 4}) \\
 YZ1_{a+1} &= YZ1_a(1 - \alpha 3_a) + C2\beta_a Y1_a + \alpha 1_a YZ0_a \\
 YZ2_{a+1} &= YZ2_a + \alpha 2_a YZ0_a + \alpha 3_a YZ1_a + \beta_a Y2_a \\
 Z_{a+1} &= Z_a(1 - \alpha 0_a) + \beta_a X_a
 \end{aligned}$$

Trends in cannabis use

Our modelling analysis used estimates of prevalence, incidence and trends in cannabis use derived from the Offending, Crime and Justice Survey (OCJS) [19]. OCJS was a general household survey conducted in 2004 with a sample size of approximately 10 000 stratified so that just under half of the sample were aged 10–25 years, plus an additional booster sample of 1883 people from ethnic minority groups. The response rate was 74%, and the estimates have been re-weighted against the England and Wales population and for differences in response rate by age, gender, assuming a design effect of 1.3. Information on drug use was collected using computer-assisted self-interviewing (CASI). Drug use among young people has been published previously [20]. OCJS asked a series of questions on cannabis use (and other drugs) including: ever use, use last year, age at first and age at last use. These data were converted to year at first and last use, and allowed the estimation of the number of new initiates (incidence) by year, the annual number of cannabis users (period prevalence) over time, the length of reported use, the cessation rate and number and proportion of people who report first use at specific ages [21]. Data on consumption or frequency of cannabis use were not collected. Therefore, we defined users according to reported length of cannabis use: none; once only; less than 2 years ('short-term or light use'); and 3 + years ('long-term or heavy use').

Incidence of schizophrenia

Until recently there were only a few UK studies that provided any incidence estimates. The estimates used in our modelling analysis were derived from a large population based case-control study on risk-factors for psychotic illness (AESOP) conducted in 1997–99 in three centres Southwark (London), Nottingham and Bristol, which identified all first episodes of schizophrenia and other psychoses in contact with health services. The study revealed clear differences in the incidence of schizophrenia by site, age and gender [22]. In the absence of any other data, or

relevant adjustment factors, we assumed that the combined incidence across the three sites represented incidence in England 1997–99 (see observed lines in Fig. 4). The model was fitted against incidence estimates from these cross-sectional data.

Risk of schizophrenia following cannabis use and model scenarios

Based on the current evidence we modelled multiple scenarios for the risk of schizophrenia following cannabis use. We illustrate three here; two are based principally on the Swedish conscript study, the third is a more conservative scenario:

- 1 Risk is elevated 1.8-fold among 'light or short-term' users and 3.1 among 'heavy or long-term' users; affects only those starting cannabis use under 20 but elevated risk lasts for > 20 years [3].
- 2 As 1 but effects users of all ages [3].
- 3 Risk is elevated twofold only among 'heavy' users; affects only those starting cannabis use under 20 but elevated risk lasts for > 20 years [13].

Fitting the model to cannabis and schizophrenia incidence data

The model was defined for different birth cohorts and run separately for men and women. The model was first fitted to the cannabis use data from the OCJS [19]. These data produced direct estimates for the age-specific cessation rate for short- and long-term cannabis users [$\alpha 2(a)$ and $\alpha 3(a)$], and was then used to fit the model, for different birth cohorts, to cannabis incidence data for different age groups (< 16 years, 16–17 years, 18–19 years and 20 + years) by varying the age-specific cannabis incidence rate for each age band simultaneously [$\alpha 0(a)$]. Following this, the model was fitted to the schizophrenia incidence data for specific age groups (16–19 years, 20–24 years, 25–29 years, 30–34 years, 35–39 years and 40 + years) and birth cohorts by varying the age-specific incidence of schizophrenia among unexposed individuals [$\beta(a)$], while assuming specific risk ratios for the risk of schizophrenia due to cannabis (C1 and C2). However, because the Aesop study had schizophrenia incidence data from only one age group for each birth cohort, the model fitting had to assume that the age-specific incidence of schizophrenia among non-cannabis users was constant across the different birth cohorts [22]. This meant that the model had to be fitted first to schizophrenia incidence data from the youngest age group, and was then fitted to data from successively older age groups while assuming the age-specific unexposed schizophrenia incidence estimates [$\beta(a)$] acquired from previous fits. In all scenarios, the model fitting was undertaken using the Newton optimization method to minimize the squared difference

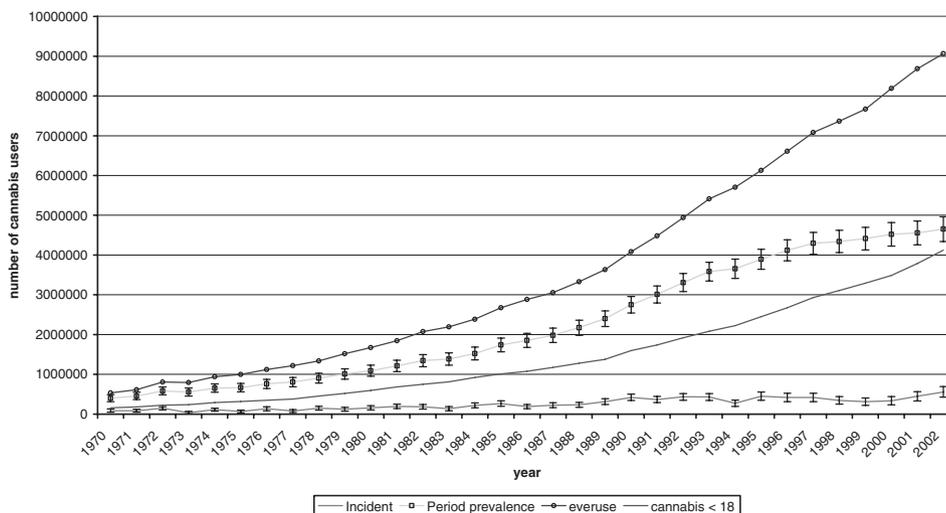


Figure 2 Trends in incidence, period prevalence, ever and under 18 cannabis use in England and Wales 1970–2002; OCJS survey

between the model and data estimates across the parameter space used in the fitting process.

RESULTS

Cannabis trends

Table 1 shows estimates of cannabis use in England and Wales by birth cohort, age at first use and frequency based on the Offending and Criminal Justice Survey and used for the modelling. Figure 2 illustrates the considerable increase in reported cannabis use over time, suggesting that on average, since the early 1970s to 2002, there has been a fourfold increase in incidence to approximately 425 000–690 000, a 10-fold increase in period prevalence to approximately 4.3–5 million people, a 13-fold increase in people who have ever used cannabis to over 9 million and an 18-fold increase in the number of people exposed under 18 years. Cannabis use is higher

among men than women—although more recently the rate of increase has been similar (Table 1 and Fig. 3). Figure 4 shows that the proportion reporting cannabis use more than once increased from less than 5% among men and women born in 1940–45 to 35% and nearly 50% among women and men, respectively, born in 1980–84.

The model was able to fit accurately the OCJS trends in cannabis incidence in different birth cohorts and age bands, with the relative difference between the model and data estimate almost always being less than 5%, and usually less than 1% (see Fig. 3).

Model output—incidence, prevalence and attributable fraction

Figure 4 shows the observed (and modelled) age distribution of schizophrenia incidence for men and women from the Aesop study [22], and the projected contribution due

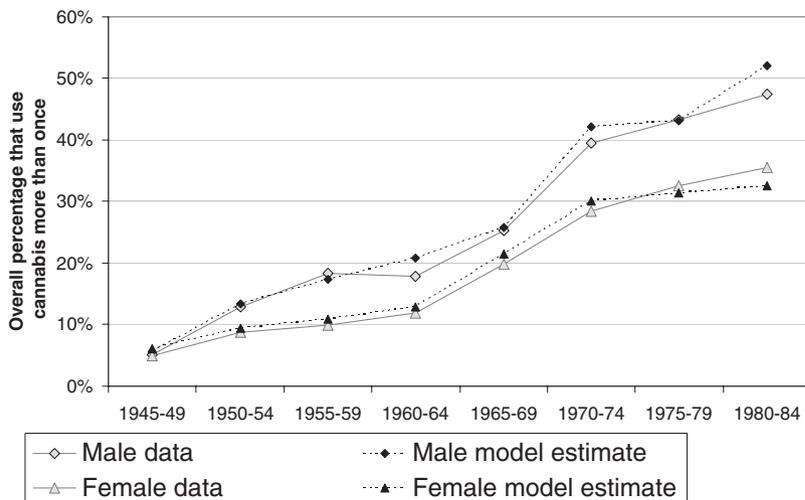
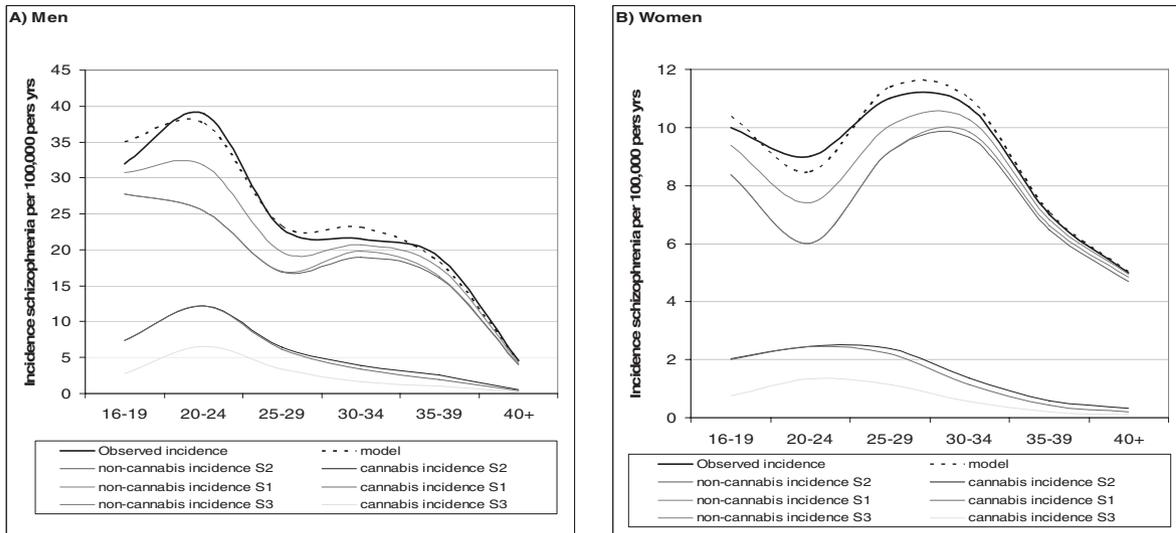


Figure 3 Observed and model estimates of proportion of men and women who use cannabis more than once by birth cohort

Table 1 Cannabis use by birth cohort, age first use and frequency: Offending and Criminal Justice Survey, 2003.

Birth cohort	Once only					Total					3 + years					Total
	No use	< 16	16-17	18-19	20+	Once only	< 2 years				3 + years					
							< 16	16-17	18-19	20+	< 16	16-17	18-19	20+		
Males																
1945-49	88.2%	0.0%	0.0%	0.4%	6.2%	6.6%	0.0%	0.0%	0.0%	1.1%	0.9%	0.0%	0.0%	0.4%	2.9%	3.2%
1950-54	79.7%	0.0%	0.0%	3.6%	3.8%	7.4%	0.0%	0.0%	0.0%	2.0%	1.5%	0.0%	0.0%	2.5%	5.7%	9.4%
1955-59	70.2%	0.3%	1.2%	2.9%	7.0%	11.4%	0.0%	0.0%	0.0%	1.5%	2.2%	1.4%	2.5%	6.1%	4.6%	14.6%
1960-64	72.6%	0.0%	0.8%	1.5%	7.3%	9.6%	0.0%	1.3%	0.1%	0.1%	2.1%	1.3%	3.1%	3.1%	6.8%	14.3%
1965-69	63.2%	0.6%	1.1%	2.9%	6.9%	11.5%	0.5%	0.0%	0.0%	0.0%	2.3%	2.8%	5.6%	7.6%	6.5%	22.6%
1970-74	47.9%	0.0%	1.4%	3.4%	7.7%	12.5%	0.0%	0.7%	0.6%	0.6%	3.2%	5.1%	7.2%	9.4%	13.5%	35.2%
1975-79	43.5%	1.7%	3.1%	3.8%	4.5%	13.1%	1.1%	2.1%	0.8%	2.3%	2.3%	4.6%	9.4%	13.3%	9.8%	37.0%
1980-84	44.4%	1.7%	2.7%	3.0%	0.8%	8.2%	0.4%	3.0%	5.2%	1.4%	1.4%	16.8%	8.9%	7.6%	4.1%	37.4%
Total	67.7%	0.9%	1.3%	2.0%	4.2%	8.4%	1.1%	1.2%	1.0%	1.5%	4.8%	5.0%	4.4%	4.8%	5.0%	19.1%
Females																
1945-49	89.3%	0.2%	0.0%	0.2%	5.3%	5.7%	0.0%	0.0%	0.0%	0.0%	0.3%	0.0%	0.2%	0.9%	3.6%	4.6%
1950-54	86.6%	0.2%	0.3%	2.1%	2.2%	4.7%	0.0%	0.5%	0.3%	1.2%	1.2%	0.8%	0.9%	2.0%	3.1%	6.8%
1955-59	79.8%	0.3%	1.5%	1.5%	7.1%	10.4%	0.0%	1.1%	0.0%	1.0%	1.0%	0.7%	2.7%	1.7%	2.7%	7.8%
1960-64	76.5%	0.0%	1.8%	3.5%	6.2%	11.6%	0.0%	0.2%	0.9%	1.5%	1.5%	0.6%	1.4%	2.5%	4.9%	9.4%
1965-69	71.3%	0.3%	1.4%	1.0%	6.2%	9.0%	0.2%	1.2%	0.6%	1.6%	1.6%	1.7%	3.6%	4.5%	6.4%	16.2%
1970-74	61.8%	0.7%	0.8%	3.1%	5.1%	9.7%	0.1%	0.0%	1.0%	2.5%	2.5%	3.2%	6.9%	6.6%	8.1%	24.7%
1975-79	55.8%	1.4%	2.1%	4.4%	3.8%	11.6%	0.5%	1.0%	0.9%	1.1%	1.1%	8.0%	6.4%	11.9%	2.7%	29.0%
1980-84	49.6%	3.8%	4.9%	4.0%	2.2%	14.9%	1.9%	5.3%	4.1%	1.7%	1.7%	11.2%	8.8%	2.5%	0.0%	22.4%
Total	76.2%	1.1%	1.5%	1.8%	3.8%	8.2%	0.8%	1.2%	0.7%	1.1%	3.8%	2.7%	2.8%	3.1%	3.2%	11.8%



Footnote: S1 Risk lasts for > 20 years and is elevated 1.8 fold amongst "light" users and 3.1 amongst "heavy" users. Only affects those starting cannabis use under 20; S2 As 1 but effects users of all ages; S3 Risk lasts for > 20 years and is elevated 2-fold amongst "heavy" users. Only affects those starting cannabis use under 20

Figure 4 Overall observed and projected incidence of schizophrenia and model projections of schizophrenia incidence due to cannabis and non-cannabis exposure. S1: risk lasts for > 20 years and is elevated 1.8-fold among 'light' users and 3.1 among 'heavy' users; affects only those starting cannabis use under 20 years. S2: as S1 but affects users of all ages. S3: risk lasts for > 20 years and is elevated twofold among 'heavy' users; affects only those starting cannabis use under 20 years

to and not due to cannabis for the three scenarios. Thus, the model accurately projects and fits the observed data on schizophrenia incidence. Pooling the data suggests an incidence and prevalence of schizophrenia of approximately 11 per 100 000 and 0.43% overall; and 17 per 100 000 and 0.63% among men and 7.3 per 100 000 and 0.23% among women, respectively.

Figure 4 suggests that the age distribution of schizophrenia incidence, if cannabis was causal, should have changed over time—as shown by comparing the line depicting non-cannabis related schizophrenia incidence with the observed and model projections for schizophrenia due to cannabis. In addition, the higher schizophrenia incidence observed among young men could be attributed mainly to cannabis, and that otherwise (in the absence of cannabis exposure) schizophrenia incidence would have been similar for all men aged 16–40 years, or may even have fallen with age. Thus, between 1965 and 2000 the incidence of schizophrenia among young men aged 16–24 would have increased by 39% under scenarios 1 and 2 and by 20% under scenario 3. Indeed, by 2000 the attributable fraction of schizophrenia incidence due to cannabis would be between 15% and 30% among young men aged 16–24 years. In contrast, the model projections suggest that cannabis exposure may have minimized age differences in schizophrenia incidence among women aged 16–35 years.

Model estimates of schizophrenia incidence were projected to rise steadily by birth cohort (Fig. 5). In older cohorts the absolute number of additional cases attributable to cannabis is likely to be small, given the relatively low proportion of the population exposed to cannabis. Greater rates of increase are projected for: men than women; for scenario 2, which hypothesizes an increased risk for all cannabis users at any age; and for people born from 1970 onwards. Thus, under scenario 2 schizophrenia incidence was projected to increase by 17% comparing men born 1945–49 with those born 1965–69, and a further 19% comparing men born 1965–69 with those born 1980–84. In contrast, increases of 8% and 9%, respectively, were projected under scenario 3. The corresponding increases for women, comparing those born 1945–49 with those born 1965–69 were below 10% for all scenarios, whereas comparing women born 1965–69 with those born 1980–84 were 23% and 11% under scenario 2 and 3, respectively.

Figure 6 shows the projected incidence and prevalence of schizophrenia from 1990 to 2010 for men and women. Thus, under all scenarios the proportion of new or prevalent cases due to cannabis was projected to be small by 1990 (approximately 5% or less overall). However, by 2000–10, for most scenarios the impact of cannabis on schizophrenia prevalence and incidence should be observable, at least for men. Thus, under

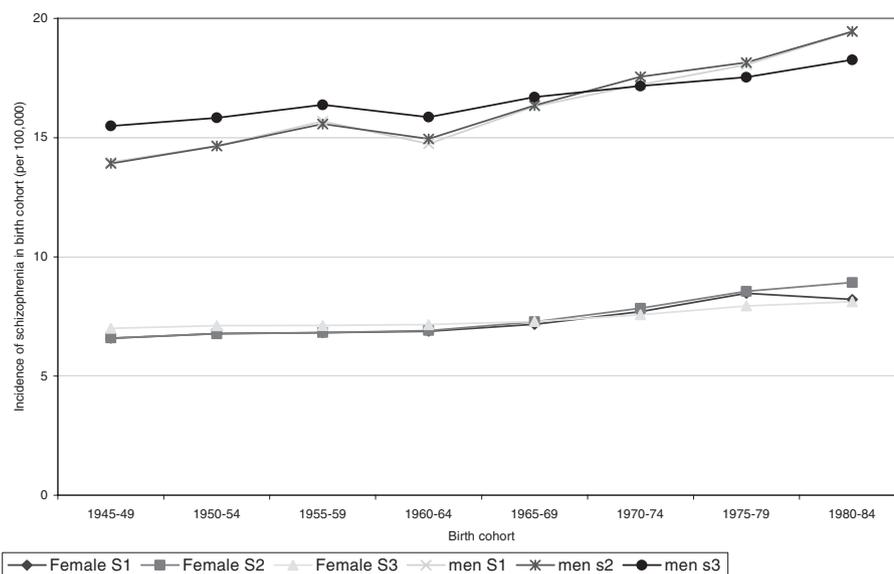


Figure 5 Projected incidence of schizophrenia among men and women in England and Wales by birth cohort; model projections assuming causal association between cannabis exposure and schizophrenia. S1: risk lasts for >20 years and is elevated 1.8-fold among 'light' users and 3.1 among 'heavy' users; affects only those starting cannabis use under 20 years. S2: as S1 but affects users of all ages. S3: risk lasts for >20 years and is elevated twofold among 'heavy' users; affects only those starting cannabis use under 20 years

scenario 2 (which assumes an elevated risk for light and heavy cannabis users of all ages) the prevalence of schizophrenia among men was projected to increase from 1990 to 2000 by 10% and 19% by 2010. Similarly, since 1990 the incidence of schizophrenia among men was projected to have increased by 15% in 2000 and 29% in 2010; and by 2010 cannabis would have caused over 25% of incident and 20% of prevalent cases of schizophrenia among men. Scenario 1 (which follows scenario 2 but restricts the risk to those starting under 20) yields similar projections. Scenario 3 (which assumes a twofold risk among heavy users that start using cannabis under 20) projects smaller increases in the prevalence and incidence of schizophrenia among men: 5–7% by 2000 and 8–12% by 2010 compared to 1990; and an attributable fraction of new and prevalent schizophrenia cases in men due to cannabis in 2010 of 9% and 13%, respectively. Among women the prevalence of schizophrenia was projected to increase from 1990 by 5% in 2010 under scenario 3, and 14% under scenario 2. The attributable fraction of incident schizophrenia due to cannabis among women in 2010 was projected to be 11% and 23% under scenario 3 and 2, respectively.

DISCUSSION

Cannabis exposure seems to have increased substantially in the UK population. Around 90% of men and women born between 1945 and 1949 report never using cannabis, whereas among men and women born 1980–84

more than 50% report cannabis use. Further, patterns of cannabis use appear to be changing. Levels of one-off and short-term use have fluctuated across successive birth cohorts; however, earlier onset and longer-term use has shown steady increases.

The strength of evidence supporting a causal basis for the association between cannabis use and psychosis observed in several studies has been debated [9–13]. This debate has focused upon the possibility of non-causal explanations for the association—in particular the possibility of confounding by factors independently associated both with increased risk of cannabis use and increased risk of psychosis [10]. If cannabis use causes schizophrenia then the substantial increases in use suggested by the data described above should have led to increases in schizophrenia incidence and prevalence—assuming other causes of schizophrenia to be constant.

The incidence of schizophrenia peaks in early adulthood and is higher among men. We directly measured age and sex-specific schizophrenia prevalence and incidence in the late 1990s [22]. Historical trends in cannabis exposure were derived from the Offending and Criminal Justice Survey conducted in 2003 [19], which was consistent with estimates of the prevalence use generated by the British Crime Survey (Home Office, personal communication). We developed a model that fitted accurately both observed schizophrenia incidence and trends in cannabis use. Model projections provided estimates of the proportion of schizophrenia cases attributable to earlier increases in cannabis use and estimates of how incidence might change by 2010 assuming different

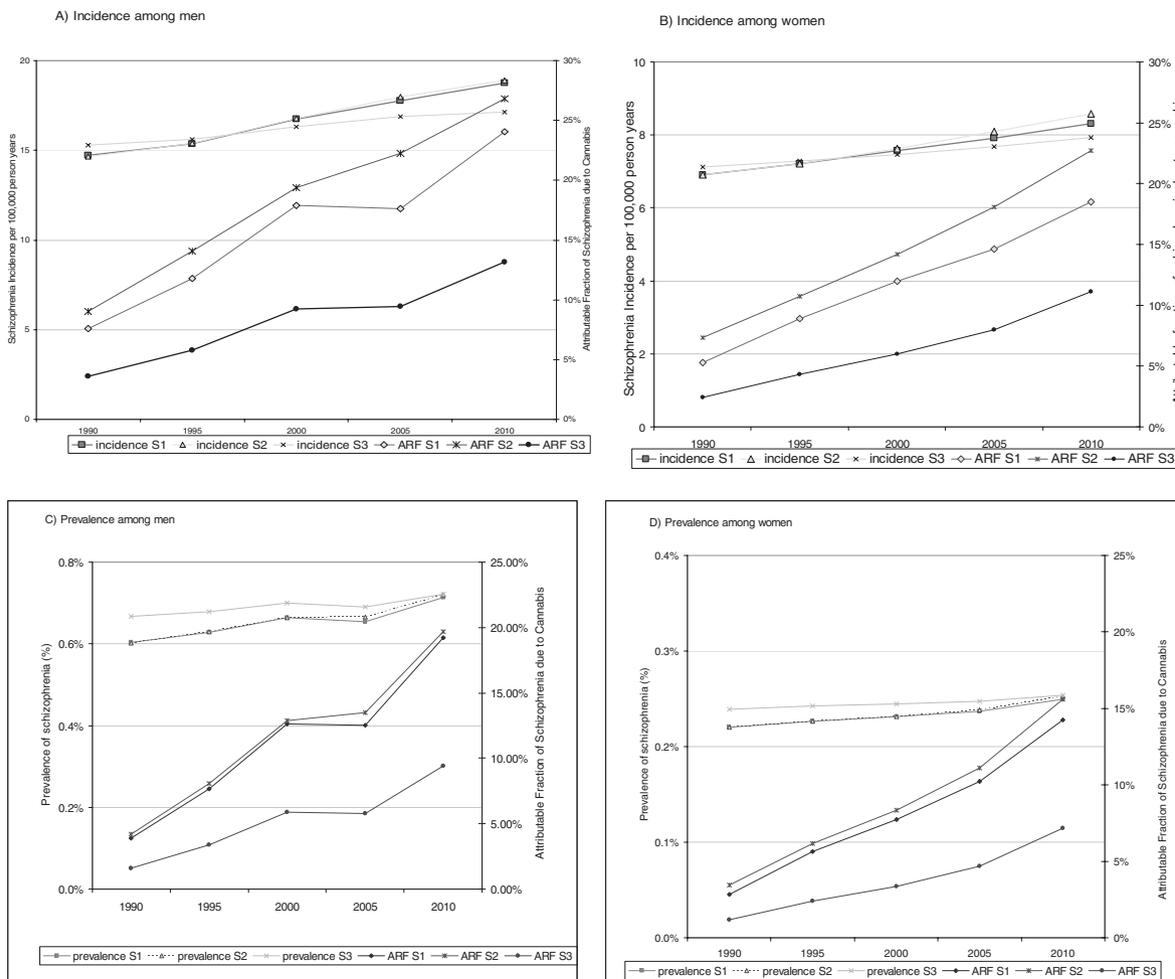


Figure 6 Projected incidence and prevalence of schizophrenia and attributable fraction of schizophrenia due to cannabis, 1990–2010. S1: risk lasts for >20 years and is elevated 1.8-fold among 'light' users and 3.1 among 'heavy' users; affects only those starting cannabis use under 20 years. S2: as S1 but affects users of all ages. S3: risk lasts for >20 years and is elevated twofold among 'heavy' users; affects only those starting cannabis use under 20 years. ARF: attributable risk fraction

kinds of causal relation between cannabis use and schizophrenia. Our projections suggest that among men around 7% of incident schizophrenia cases observed in the late 1990s were due possibly to cannabis use—by 2010 the proportion is projected to increase to 25%.

The proportion of schizophrenia cases currently attributable to cannabis use may be lower than expected intuitively, given the apparently large recent increases in cannabis use. This reflects mainly the fact that increased use has been most marked in the youngest cohort of users who have yet to experience their full life-time risk of schizophrenia. However, the increases in schizophrenia projected by 2010 are more substantial.

A recent review of the epidemiology of schizophrenia concluded that, overall, the contemporary incidence of the disease was equivocal, but probably either stable or falling [15]. To date, however, even within studies that report increases in schizophrenia or psychoses, no evidence has been found of a greater increase

in young men or a change in the age distribution of schizophrenia over time [14–16]. If cannabis were a cause of schizophrenia such changes would be expected, given patterns of cannabis use. It is also important to note that we used a narrow, operational definition of schizophrenia for our estimate of incidence, similar to that used in the Swedish conscript study. This represents a more stringent set of model assumptions than would have been generated had we used a more prevalent, broad definition of disorder such as has been used in other studies (e.g. [4,6]).

One limitation to our analysis is that we did not vary any other potential risk or protective factors associated with schizophrenia, such as maternal nutrition, paternal age or environment [23,24]. It is conceivable that a change in other factors could have masked or diminished the recent and future projected increases in schizophrenia occurrence due to cannabis. This could explain further why no change in schizophrenia trends has been

observed. However, such factors would also need to explain how potentially larger and earlier increases in schizophrenia incidence among young men may have been missed.

Other limitations to the projections are due principally to data uncertainty and availability. First, evidence of the incidence and prevalence of schizophrenia is not as strong as it should be, either now or historically, given the seriousness of the condition [15]. The models were fitted against a single cross-sectional incidence estimate [22], although it is corroborated by age and sex distribution patterns shown elsewhere [25]. Individuals at higher risk of schizophrenia (for example, young black men) may be under-represented in census estimates of denominator data, leading to over-estimates of incidence. This, however, should not influence the overall patterns of changing incidence that our models project. Secondly, historical trends in cannabis use were estimated from a single survey, which may suffer greater recall bias among older participants, and possibly exaggerate the increase in trends over time. However, such a bias would imply that any projected increases in schizophrenia due to cannabis should have been seen earlier. Thirdly, cannabis consumption or frequency of use was not measured directly by the Offending and Criminal Justice Survey—and our estimates of exposure level were crude and not directly comparable with the Swedish conscript or other longitudinal studies.

CONCLUSIONS

Our data provide no direct evidence on whether cannabis use causes schizophrenia. They appear to confirm assumptions of substantial increases in cannabis use in the UK population over the last 30 years, and suggest a shift to more prolonged use initiated at younger ages. This shift is relatively recent and its full impact on population levels of schizophrenia, if cannabis use does cause schizophrenia, may not yet be apparent. However, this impact should become apparent within the next 5 years. Consideration of these questions is constrained significantly by the lack of reliable data on trends in both cannabis use and schizophrenia, emphasizing the importance that these data be collected in future through robust surveillance systems.

Acknowledgements

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and J. K. and P. J. provided expertise and data on schizophrenia epidemiology. Matthew Hickman and John Macleod are funded by NHS Career Scientist Grants.

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