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INVITED REVIEWS

# Psychosocial sequelae of cannabis use and implications for policy: findings from the Christchurch Health and Development Study

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**Abstract**
*Background* The Christchurch Health and Development Study is a longitudinal study of a birth cohort of 1265 children who were born in Christchurch, New Zealand, in 1977. This cohort has now been studied from birth to the age of 35.
*Scope of this review* This article examines a series of findings from the CHDS that address a range of issues relating to the use of cannabis amongst the cohort. These issues include: (a) patterns of cannabis use and cannabis dependence; (b) linkages between cannabis use and adverse educational and economic outcomes; (c) cannabis and other illicit drug use; (d) cannabis and psychotic symptoms; (e) other CHDS findings related to cannabis; and (f) the consequences of cannabis use for adults using cannabis regularly.
*Findings* In general, the findings of the CHDS suggest that individuals who use cannabis regularly, or who begin using cannabis at earlier ages, are at increased risk of a range of adverse outcomes, including: lower levels of educational attainment; welfare dependence and unemployment; using other, more dangerous illicit drugs; and psychotic symptomatology. It should also be noted, however, that there is a substantial proportion of regular adult users who do not experience harmful consequences as a result of cannabis use.
*Conclusions* Collectively, these findings suggest that cannabis policy needs to be further developed and evaluated in order to find the best way to regulate a widely-used, and increasingly legal substance.

**Keywords** Cannabis · Cannabis dependence · Education · Unemployment · Welfare dependence · Gateway theory · Psychosis

## Introduction

Over the last two decades there have been ongoing debates about the extent to which the use of cannabis/marijuana has harmful effects upon users [1–4]. These debates have tended to polarize into two groups; first, those who tend of minimize the potential harmful effects of cannabis and argue strongly for the liberalization of cannabis laws and permitting access to legal cannabis [5–7]; and second, those who view cannabis as a harmful drug for which continued prohibition is the correct approach [8, 9].

One of the inevitable features of research into the harmful effects of cannabis is that research has been conducted in different settings, using different research designs and measurement methods. While this heterogeneity has benefits for examining the generality of findings about cannabis, it also has some limitations, as the results from different studies may make it difficult to provide a clear picture of the ways in which cannabis use may influence the health and wellbeing of a particular population.

Against this background the aims of this paper are to provide an overview of the findings of a large longitudinal study in which the use of cannabis has been studied from mid-adolescence (age 14) to mature adulthood (age 35). This study is the Christchurch Heath and Development Study, which is a longitudinal study of a birth 1265 cohort of children born in the Christchurch (New Zealand) area in

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mid-1977. The cohort consisted of 97 % of all live births in the greater Christchurch region during this period, and as such is a representative population. This cohort has been studied on 23 occasions from birth to age 35, with extensive data on issues of health and wellbeing having been gathered. Sample retention in the study has been good and at age 35, a sample of 962 respondents was studied, with this sample representing 79 % of the cohort.

Over the years the CHDS has published 30 articles on the use, misuse and consequences of cannabis over the period from adolescence. In this paper, we provide a summary overview and synthesis of the study findings on the potentially harmful effects of cannabis. We also supplement these findings to describe those adult cannabis users who use the drug regularly and in a non-problematic way. What we seek to show in this review is that:

1. Cannabis is a drug that has harmful effects in the areas of educational achievement, personal adjustment, mental health and related outcomes;
2. Despite this, there is a substantial minority of cannabis users who do not experience adverse consequences of the drug.

## Patterns of cannabis use and cannabis dependence in the CHDS cohort

Figure 1a, b summarizes the history of cannabis use and cannabis dependence in the CHDS cohort from ages 14 to 35 years. Figure 1a shows annual rates of cannabis use and cannabis dependence at ages 15, 18, 21, 25, 30, and 35 years. The following measures of use are reported: any use; regular (at least weekly) use; and dependent use. The measure of dependent use was based on DSM-III-R (at age 15) and DSM-IV (ages 18–35) criteria for cannabis dependence. The figure suggests substantial usage by the cohort with: (a) annual rates of use ranging from 8.7 to 46.8 %; (b) annual rates of regular use ranging from 0.7 to 15.6 %, and; (c) annual rates of dependence ranging from 0 to 6.9 %. These data show a general tendency for the rates of cannabis use to be highest during the mid-20s, with the largest group of cannabis users being occasional (less than monthly) users of the drug at each age.

Figure 1b shows estimates of the probability that cohort members would: (a) use cannabis; (b) use cannabis regularly (at least weekly); and (c) become cannabis dependent, by each assessment at ages 15–35 years. All estimates suggest a high rate of cannabis use in the cohort, with approximately 80 % of cohort members using cannabis on at least one occasion, 33.6 % of the cohort being regular users during at some point by age 35, and 15.2 % of the cohort meeting criteria for cannabis dependence by age 35.

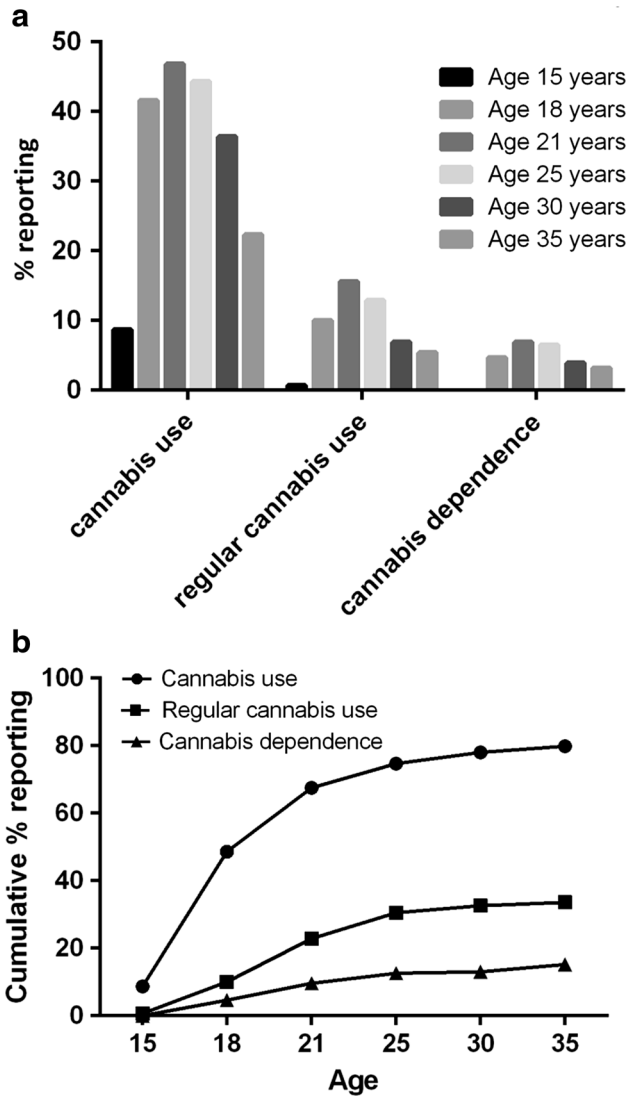


**Fig. 1** **a** Cannabis use, regular (at least weekly) cannabis use, and cannabis dependence at each assessment (ages 15–35). **b** Cumulative rates of cannabis use, regular (at least weekly) cannabis use, and cannabis dependence (ages 15–35)

## Cannabis use and educational achievement, welfare dependence, and unemployment

One area of increasing research interest is the extent to which early and heavier use of cannabis may have adverse effects on educational achievement [10–13], and related outcomes such as welfare dependence [14–16] and unemployment [17–19]. The results of a recent analysis of CHDS data [20] is presented in Fig. 2a, which shows the associations between the age of onset of cannabis use and educational outcomes, after adjustment for a wide range of confounding factors, including: parental education; family socioeconomic status at birth; childhood family living standards; scholastic ability (as measured at age 13);

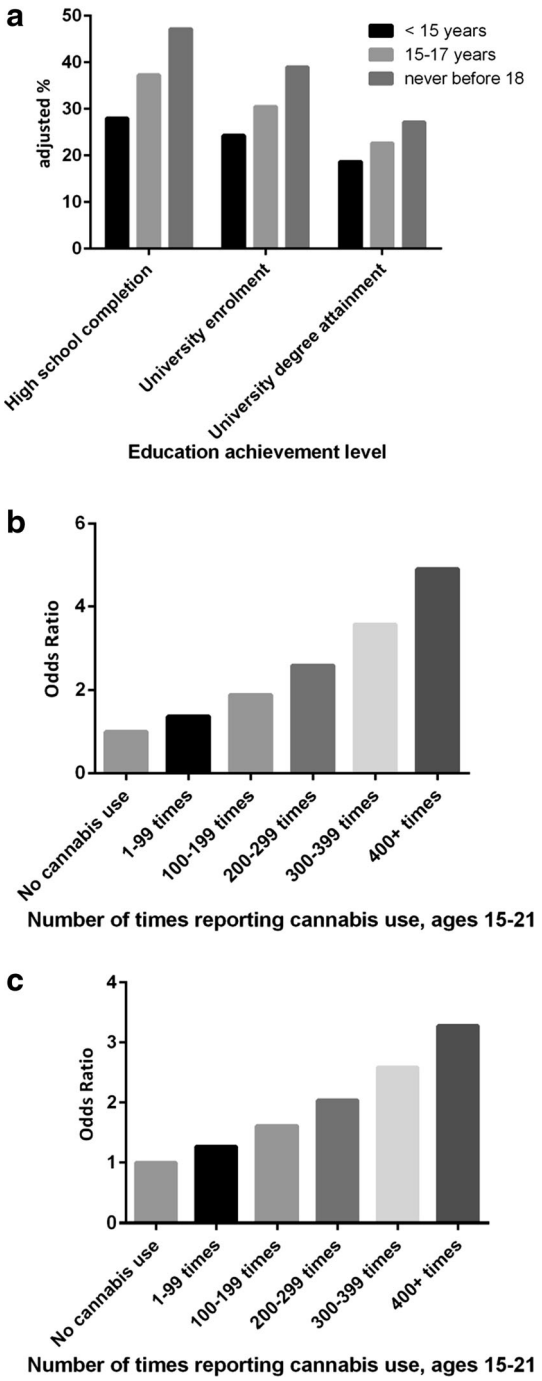


**Fig. 2** **a** Adjusted rates of educational attainment (to age 30) by age of onset of cannabis use; **b** adjusted OR for welfare dependence (ages 21–25) amongst those using cannabis (ages 15–21), compared to non-users; **c** adjusted OR for unemployment (ages 21–25) amongst those using cannabis (ages 15–21), compared to non-users

teacher-rated grade point average (ages 11–13); and childhood conduct problems. The figure shows that for all outcomes, the age of onset of cannabis use was related to lower levels of educational attainment. As a general rule, those using cannabis prior to age 15 had the poorest outcomes, while those who did not use cannabis prior to age 18 had the best outcomes.

Further analyses of CHDS data [21] examined the linkages between cannabis use during the period 15–21 years and: (a) receipt of welfare benefits; and (b) unemployment; during the period 21–25 years. In these analyses, cannabis use was classified using a six-level variable representing an estimate the self-reported total number of times an individual had used cannabis during the period 15–21 years. The findings of these analyses are shown in Fig. 2b, c, which depict the odds ratios for welfare dependence and unemployment, for varying levels of cannabis consumption (relative to non-users), after adjustment for confounding factors (including measures of: the socio-economic background of the family of origin; family functioning and exposure to adversity; exposure to child sexual and physical abuse; childhood and adolescent adjustment; academic achievement in early adolescence; and comorbid mental health disorders and substance use). The figure clearly depicts a dose–response association in which the increasing use of cannabis during the period 15–21 years increases the risk of both welfare dependence and unemployment during the period 21–25 years. Those who reported using cannabis on 400 or more occasions had odds of welfare dependence that were almost five times greater than those who did not use cannabis, and odds of unemployment which were more than three times greater than those who did not use cannabis.

The linkages between cannabis use and educational and economic outcomes have several possible explanations. The first explanation is that the use of cannabis may have consequences for neurophysiological structure and functioning, compromising motivation and cognitive processes [12, 22, 23]. Although speculative at the present time, this explanation does have a growing evidence base related to the neurochemistry of cannabis and the vulnerability of the developing adolescent brain [24, 25].

A second explanation of the linkages between cannabis use and lower levels of educational achievement is that the use of cannabis may introduce young people to social contexts in which anti-conventional behaviour is encouraged, and more normative behaviours related to educational achievement are seen as less attractive [26–28]. It seems likely that the associations between cannabis use and educational attainment and related outcomes reflect the cumulative effects of both biological and social factors that increase the vulnerability of cannabis users to educational underachievement, welfare dependence and unemployment.

## Cannabis use and other illicit drug use: the cannabis gateway hypothesis

Another area of long-standing interest in research on the consequences of cannabis use is the possible link between the use of cannabis and an increased risk of using other illicit drugs. Known as the "stepping stone" or "gateway" hypothesis, this theory suggests that exposure to cannabis increases the likelihood that an individual will use illicit drugs other than cannabis at some later point [29–35].

A critical issue in evaluating the gateway hypothesis concerns the extent to which the associations between cannabis use and other illicit drug use can be explained by third or confounding factors. It has been argued, for example, that the association may arise because of common factors that predispose young people to use both cannabis and other illicit drugs [36–38], and that controlling for these factors may account for the linkages between cannabis use and other illicit drug use. However, other studies employing often extensive control for confounding factors reported that associations between cannabis use and other illicit drug use could not be explained by confounding factors [27, 39, 40].

A potential criticism of this research is that studies had only controlled observed covariates, and that any remaining association between cannabis use and other illicit drug use could be attributed to residual confounding [41]. This raises important issues about methods for controlling both observed and non-observed confounding. Conventionally, issues of confounding have been addressed in epidemiological research by adjusting associations between outcomes and potentially causative factors for observed confounders such as age, race, and social, family and childhood background. The difficulty with such analyses is that it is always possible to suggest the presence of non-observed confounders which explain the observed association. The conditional fixed effects regression model provides a technique for addressing the issue of omitted confounders. The logic of this approach can be illustrated by a study in which the same sample is assessed on two occasions ($t1$; $t2$) with assessments of illicit drug use ($Y$t) and a time varying predictor $X$ being collected. We assume that the associations between $Y$t and $X$t are described by the model:

$$Y1 = B \times X1 + U + E1 \quad (1)$$

$$Y2 = B \times X2 + U + E2, \quad (2)$$

where U denotes non-observed factors that have fixed and enduring consequences on the measures of illicit drug use, $Y1$ and $Y2$, and Et is a random error term that is uncorrelated with $X$ and uncorrelated between time periods. The fixed effects factor U is permitted to be correlated with $X1$, $X2$ and thus potentially confounds the relations between $Y$t and $X$t. The effects of the non-observed fixed factor U can be taken into account by subtracting Eq. 2 from Eq. 1:

$$(Y1 - Y2) = B1(X1 - X2) + (E1 - E2). \quad (3)$$

It is evident that the model in Eq. 3 provides an estimate of the parameter of interest B1 in a way that excludes the influence of the fixed effects factor $U$.

The principles illustrated above can be generalized to develop fixed effect adjusted estimates for a wide range of statistical models [42, 43]. In addition, the models may be extended to include observed time-dynamic covariate factors that may also account for the associations between exposure and outcome. Indeed, all analyses of CHDS data employed fixed effects regression models have also included a range of time-dynamic covariate factors to address the potential issue of omitted variables.

The fixed effects model can be used for any situation in which there is interest in the causal effects of a time-dynamic predictor and time-dynamic measures of outcome. This approach has been used in a number CHDS publications [30, 44–52]. One specific application of this approach is to examine the associations between cannabis use and other illicit drug use, taking into account non-observed sources of confounding.

Data from the CHDS have been used in two papers examining the linkages between cannabis use and other illicit drug use [30, 40]. By age 25, three-quarters of the remaining cohort had reported using cannabis on at least one occasion, and approximately 40 % had reported using illicit drugs other than cannabis at least once. Amongst those using both cannabis and other illicit drugs, all but a single individual reported using cannabis at an earlier point in time than using other illicit drugs. These drugs included: cocaine; heroin or other opiates; hallucinogens such as LSD or psilocybin mushrooms; party drugs including MDMA; stimulants including amphetamine; sedatives; solvents; prescription medications taken for recreational purposes; or other substances including nitrous oxide.

Figure 3a shows the unadjusted associations between frequency of cannabis use at different ages (14–15; 17–18; 20–21; and 24–25 years) and rates of use of other illicit drugs. The figure clearly shows a dose–response relationship such that increasing frequency of cannabis use at each age is linked to increasing rates of other illicit drugs.

In order to control for non-observed sources of confounding, conditional fixed effects regression models were fitted to the data for cannabis use and other illicit drug use over the period 14–25 years [30]. These models were augmented with a series of time-dynamic covariate factors observed during the period 14–25 years, which included measures of: mental health disorders; alcohol use

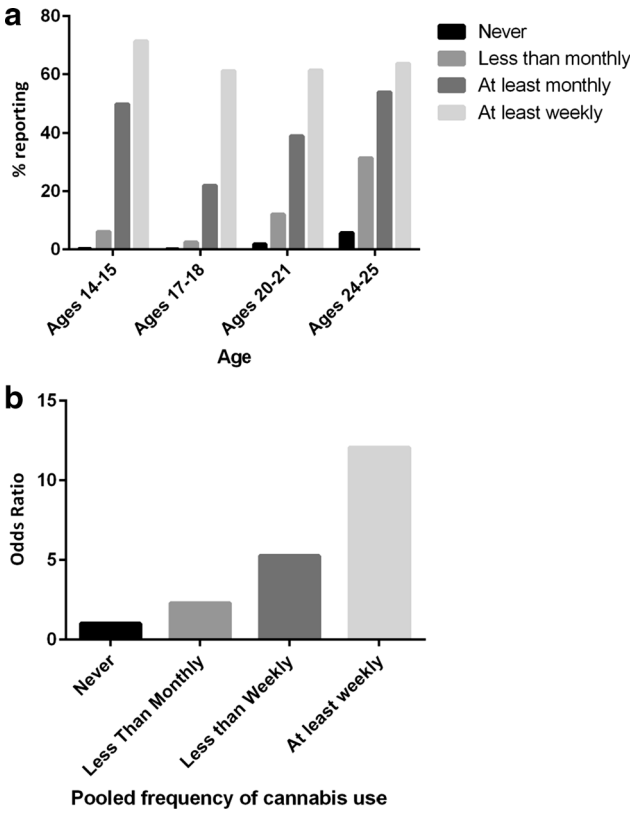


**Fig. 3** **a** % Reporting other illicit drug use by frequency of cannabis use, at selected ages, **b** adjusted OR for other illicit drug use, by frequency of cannabis use (pooled over 14–25 years), compared to non-users

disorders; nicotine dependence; life stress; unemployment; deviant peer affiliations; partner deviant behaviour; and several other lifestyle-related factors. Estimates of the adjusted associations between varying levels of cannabis use and other illicit drug use derived from the fitted models are presented in Fig. 3b, which shows estimates of the adjusted OR for other illicit drug use pooled over the period 14–25 years. The figure shows that after adjustment there remained a strong (and statistically significant) association between cannabis use and other illicit drug use.

It is also important to note that the fixed effects regression models also contained a term representing an age by frequency of cannabis use interaction, which was statistically significant ($p < 0.05$). The negative slope parameter of the interaction term indicated that the associations between cannabis use and other illicit drug use were stronger at younger ages. For example, estimates from the fitted models indicated that at ages 14–15 those who used cannabis at least weekly had odds of other illicit drug use that were approximately 66 times higher than non-users. By ages 24–25, the odds of other illicit drug use amongst weekly cannabis users had declined to 3.9 times higher than non-users.

The results of these analyses clearly suggest the presence of a causal association between the use of cannabis and the use of other illicit drugs. Possible causal mechanisms include:

- Neurobiological effects of cannabis which may encourage illicit drug use [53–55];
- The effects of the illegality of cannabis use in increasing the contact of cannabis users with the illicit drug market [35, 56, 57];
- The effects of peer support and influence in encouraging the use of cannabis and other illicit drugs [58–60];
- Social learning processes by which cannabis users learn to experiment with other illicit drugs [60–62].

## Cannabis use and psychotic symptomatology

A further area of research interest in the sequelae of cannabis use has been the linkages between cannabis use and psychotic symptoms. A number of longitudinal studies [51, 63–66] have found increased rates of psychosis/psychotic symptoms in those using cannabis. However, it could be argued that these findings may have arisen due to: (a) sources of non-observed confounding that increased the risk of both cannabis use and psychotic symptoms; of (b) reverse causal processes in which the experience of psychotic symptoms increased the risk of cannabis use.

In order to examine these issues, data from the CHDS on cannabis use and psychotic symptomatology during the period 18–25 years were used to fit conditional fixed effects models of cannabis use and symptoms of psychosis, net of non-observed sources of confounding and time-dynamic covariate factors [51]. The results of these analyses are shown in Fig. 4, which depicts the incidence rate ratio (IRR) for psychotic symptomatology, for four levels of cannabis use, after controlling for both non-observed fixed effects and time-dynamic covariate factors. The figure shows a dose–response association between cannabis use and psychotic symptomatology, in which increasing levels of cannabis use are associated with increased risk of symptoms of psychosis. After adjustment for both non-observed fixed effects and time-dynamic covariate factors, those who used cannabis on a daily basis had rates of psychotic symptoms that were 1.75 times higher than non-users ($p < 0.05$). These data were consistent with a causal explanation of the role of cannabis in increasing the risk of psychotic symptoms.

Although the analyses above established that there was an association between cannabis use and psychotic symptoms net of non-observed sources of confounding and time-dynamic covariate factors, the evidence does not establish the direction of causation. In particular, there are potentially two causal pathways that link

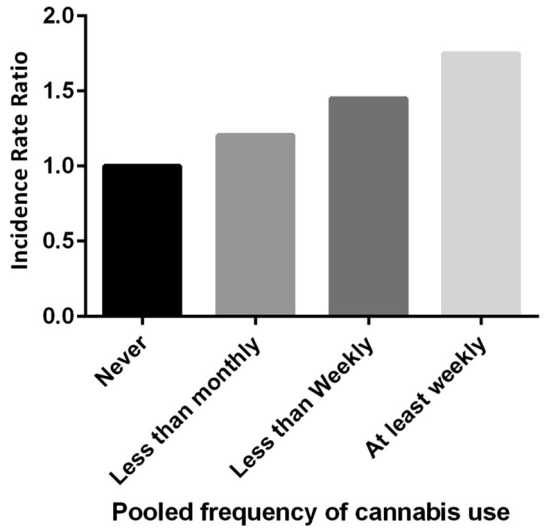


**Fig. 4** Adjusted IRR for rate of symptoms of psychosis by frequency of cannabis use (pooled over the period 18–25 years), compared to non-users

cannabis use and psychosis. First, cannabis use may lead (via changes in neurophysiological functioning) to increased susceptibility to psychotic symptoms. Alternatively, those developing psychosis may have an increased susceptibility to using cannabis as a consequence of their psychological state. Addressing this issue proves to be difficult and even with well-collected longitudinal data, establishing which factor is antecedent and which factor is consequent not straightforward [67, 68]. Furthermore, there is a possibility that cannabis use and psychosis are related to each other reciprocally by a feedback loop in which the use of cannabis increases risks of psychosis while at the same time the onset of psychosis leads to an increased consumption of cannabis. Structural equation models provide one means of addressing such a complex issue by devising statistical models that permit reciprocal relationships between cannabis use and psychosis and using these models to guide probable patterns of causation. This approach to determining likely patterns of causality has been used in a number of CHDS publications [48, 51, 69, 70].

In order to examine these issues, a structural equation model was fitted to the data which: (a) allowed the estimation of pathways from cannabis to psychosis, and vice versa; (b) accounted for auto-regressive effects for cannabis and psychosis over time; and (c) accounted for non-observed fixed effects. The results of this modelling clearly showed a statistically significant pathway from cannabis to psychotic symptoms ($B = 0.352$, SE $= 0.087$, $p < 0.001$); but a statistically non-significant and weaker pathway from psychotic symptoms to cannabis use ($B = -0.045$, SE $= 0.043$, $p > 0.25$).

While the mechanisms linking cannabis use to increased risks of psychotic symptomatology are not entirely clear, it is likely that the linkage involves the activation of the dopamine and serotonin systems in the brain [71]. Both of these neurotransmitters are known to be involved in the maintenance of psychotic mental states [72]. In addition, there has been some evidence to suggest that the associations between cannabis use and psychotic symptomatology may be influenced by gene × environment interactions. Caspi and colleagues found evidence that carriers of the COMT valine 158 allele were at greater risk of psychotic symptomatology after using cannabis [73].

## Additional findings on cannabis from the CHDS

In addition to the findings described above, data from the CHDS has been used in a number of other investigations to examine the sequelae of cannabis use for a range of psychosocial and other outcomes. Some of these additional findings include:

1. *Cannabis and major depression* Examination of the CHDS data [74], and a meta-analysis of data from several longitudinal cohorts including the CHDS [75] suggest that there is a modest association between cannabis use and increased risk of major depression. It is unclear, however, to what extent these findings may be explained by reverse causal processes in which depression increases the risk of cannabis use;
2. *Cannabis and suicidal ideation* A recent investigation [76] suggests that, amongst males in the CHDS cohort, earlier and heavier use of cannabis strongly increases the risk of suicidal thoughts. These findings are consistent with earlier studies using CHDS data examining suicidal ideation and suicide attempt [74];
3. *Cannabis and tobacco use* An additional recent examination of CHDS data [70] showed evidence of a reciprocal causal association between cannabis use and tobacco use, such that increasing levels of cannabis use increased the risk of tobacco use, and vice versa. This evidence is consistent with both "gateway" and "reverse gateway" [77] explanations of the association between cannabis use and tobacco use;
4. *Cannabis and risk of motor vehicle accidents* An examination of CHDS data at age 25 [78] found evidence that, after adjustment for confounding, self-reported driving under the influence of cannabis was associated with a marginally ($p < 0.10$) increased risk of motor vehicle accidents in which the cohort member (driver) was at fault. However, the linkage between self-reported driving under the influence of alcohol and

motor vehicle accidents was not statistically significant ($p > 0.70$) after adjustment for confounding.

5. *Cannabis use in adolescence and criminal offending* CHDS data were also used to analyse the linkages between the frequency of cannabis use during mid-adolescence (ages 15–16) and several measures of criminal offending, including self-reported violent and property offending, arrest and conviction [26]. The analyses showed an association between the frequency of cannabis use and each of the offending measures, after controlling for social disadvantage, family adversity, early-onset behavioural problems, and affiliation with deviant peers.

## The consequences of cannabis use for regular adult users

Most cannabis research has focused on documenting the harms of cannabis use. A topic that has not been addressed concerns the proportion of adult users who use cannabis in a non-problematic way. It is likely that this group of users, quite reasonably, provides the major critical commentary on the adverse effects of the prohibition of cannabis [5].

To examine this issue, for the purposes of the present investigation, CHDS data were used to classify cohort members according to their frequency of cannabis use during the period 30–35 years, ranging from "no cannabis use" to "at least weekly cannabis use". For each level of cannabis use frequency, the risk of having at least one of several psychosocial problems described previously (major depression; suicidal ideation; psychotic symptomatology; welfare dependence; being unemployed for at least 6 months) during the period 30–35 years was computed. The results these analyses are presented in Fig. 5, which shows the percentage of individuals for each level of cannabis use frequency that reported at least one psychosocial problem. The figure shows that the risk of psychosocial problems amongst cannabis users ranged from 41.9 % (occasional users) to 49.3 % (at least weekly use), as compared with 27.5 % for non-users. It should be noted that these comparisons do not address the psychosocial issues likely faced by long-term chronic users of cannabis [79–82]; however, these comparisons do highlight the fact that while regular users of cannabis have elevated rates of problems, by no means do all users experience these difficulties.

## Cannabis and public policy

An ongoing debate in the area of cannabis has focused upon: (a) the extent to which cannabis has harmful consequences; and (b) the legal and societal response to these problems [1, 3]. These debates have led to a polarization of views with pro-cannabis advocates arguing that cannabis has minimal harms [5–7], and that any harmful effects can be attributed to prohibition and its adverse consequences. Alternatively, there has been a growing body of empirical evidence clearly suggesting that cannabis has adverse effects in a number of areas of psychosocial functioning, including reduced educational achievement; increased risk of welfare dependence and unemployment; increased risk of the use of other illicit drugs; and increased risk of psychotic symptomatology [1, 3, 4, 83].

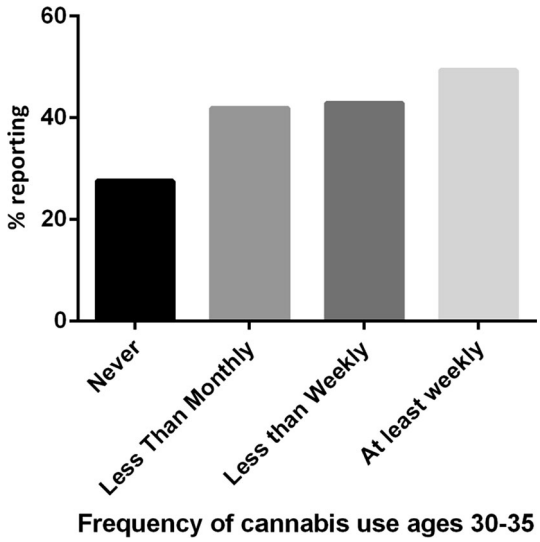


**Fig. 5** % Reporting at least one psychosocial problem, by frequency of cannabis use (ages 30–35)

At the same time, as we have shown above, by no means all cannabis users, or even heavy users, suffer adverse consequences of cannabis use. In turn, these findings lead to consideration of the most appropriate social and legal methods for regulating a moderate-risk drug which is widely used. There are a variety of responses to this issue, ranging from strict prohibition to recent US policy changes whereby cannabis has been either fully legalized, or legalized for medical purposes [83]. Which of these models provides the best approach to addressing issues relating to cannabis remains a matter of debate. It is our view that the best approach to addressing this issue is through a systematic quasi-experimental approach, in which legislation relating to the supply and consumption of cannabis is progressively liberalized, with these innovations being assessed to determine their positive and negative consequences. Regrettably, this approach has not been widely adopted, with the result that changes in cannabis policy have often been large and seldom adequately evaluated. A possible exception to this was the experiment conducted in Portugal, where not only cannabis but all illicit drugs were decriminalized in 2001. Several years later, a study

evaluating the effect of the law change found that there had been no increase in drug use amongst the population, and in fact the prevalence of drug use in the 15–19 year age group had declined for all drugs including cannabis [84, 85]. While the interpretation of the evidence following the law change in Portugal has been subject to some controversy [86], it is clear that any legal or policy changes in regard to cannabis must be subject to rigorous evaluation.

## Summary and conclusions

In summary, it is our conclusion that the accumulated findings of the CHDS suggest that cannabis use, and in particular heavy use and use at younger ages, is associated with increased risks in a number of areas of functioning, including: (a) educational achievement; (b) welfare dependence; (c) unemployment; (d) other illicit drug use; and (e) psychotic symptomatology. These findings have contributed to a growing body of evidence that suggests that cannabis is a drug that does confer some degree of risk [4, 83]. However, it is also clear that not all cannabis users experience the problems associated with cannabis. The reviewed findings highlight the importance of developing well-evaluated policies to minimize the harmful consequences of cannabis use, whilst at the same time protecting the rights of the large number of users who experience no harmful consequences.

While most of the CHDS findings point to the adverse effects of cannabis use and particularly heavy use on a number of outcomes (e.g. educational achievement; welfare dependence; unemployment; other illicit drug use; psychotic symptomatology), this evidence remains contentious. Specifically pro-cannabis groups [5–7] and others [2, 87] have consistently argued that the apparent associations between cannabis and adverse outcomes is due to faulty research design and particularly failure to control confounding factors. These comments are reminiscent of the claims of the tobacco industry in its defense of cigarette smoking. The position taken is, in effect, that since research cannot eliminate all possibility that the relationships between cannabis and adverse outcomes are confounded by non-observed factors and processes, any evidence suggesting harmful effects of cannabis can be discounted and ignored. It is our view that this logic is deeply flawed. In particular, in situations in which research produces consistent evidence that a given practice may have harmful consequences, the precautionary principle [88, 89] requires that these findings should be taken seriously and not discounted on the basis of non-observed processes and findings. This approach does not imply that an uncritical attitude should be taken to evidence of cannabis and adverse consequences, but it does require that this evidence should not be rejected on the basis of claims, conjectures and hypotheses that have not been subject to empirical test. As the history of research into the adverse consequences of tobacco smoking shows [90], what is required is an accumulation of evidence from different sources and different methods that supports a common conclusion. Looked at from this perspective, the findings of the CHDS can be seen as part of a growing body of evidence documenting the adverse consequences of heavy cannabis use.

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**Conflict of interest** The authors declare no conflicts of interest.

**Ethical standard** All data described herein were collected with the informed consent of participants. All aspects of study design and conduct have been approved by the Canterbury (New Zealand) Ethics Committee.

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