Cannabis use and adverse outcomes in young people:
A review of the literature

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The Centre for Analysis of Youth Transitions (CAYT) is an independent research centre with funding from the Department for Education. It is a partnership between leading researchers from the Institute of Education, the Institute for Fiscal Studies, and the National Centre for Social Research.
Report objectives

Cannabis is the most widely used illegal drug in Britain, particularly among young people. There is evidence linking cannabis use at a young age to a range of negative social, educational and health outcomes but the nature of these links are not fully understood. This review summarises evidence on the negative outcomes for young cannabis users and focuses on identifying any causal links.
1 Methods

1.1 Aims

This review discusses the available literature on the negative social, educational and health outcomes of young people who use cannabis. As this project is particularly interested in causal links, we focus on studies that used longitudinal and prospective methods to collect data. This means that we can look at how things are related over time, and to see which one came first. By collecting and analysing data in this way, it is more likely that cause and effect can be established.

1.2 Scope

In this review we cover international literature published in peer reviewed journals in the last 10 years\(^1\) and focus on identifying evidence from longitudinal or cohort studies. The literature search was carried out in two stages:

- **Stage one** – a search of systematic reviews about cannabis use and outcomes published in the last 10 years
- **Stage two** – a search of specific key words using electronic databases. This search included literature from 2005. Key words were the word ‘cannabis’ and either ‘young’ or ‘adolescent’ and either ‘cohort’ or ‘longitudinal’ plus one of the key search terms identified (see appendix for these).

We carried also out a Rapid Evidence Assessment based on the research aims. This is a compressed and delineated version of a Systematic Review and it uses the same principles to identify, review and evaluate evidence. However, it is done in a shorter time frame and discusses fewer publications in less detail. Our approach was built on the following principles:

- **The specification of search terms and protocols.** We used online databases\(^2\) to search for the literature reviewed here. We carried out stage one of the process to find systematic reviews and used the results of these to inform us about what search terms to use for the more specific searches carried out in stage 2.
- **The basis for the assessment of study quality.** Empirical studies were assessed based on accepted methodological standards and the transparency presented by authors in presenting results and discussing limitations. Only longitudinal data where a relationship could be considered over time were included.

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\(^{1}\) There is some evidence contained in this review which was carried out more than 10 years ago. This is evidence that was cited or discussed in a paper which was published within the specified timeframe.

\(^{2}\) The sources searched were CINAHL Plus, Criminal Justice Abstracts, International Bibliography of the Social Sciences, PAIS International, PsychInfo, PubMed, SocINDEX and Web of Science.
2 Evidence

Over the last 20 years, a number of international longitudinal datasets have been analysed to explore the relationship between cannabis use and other behaviours. Many of the studies reported here draw on their findings. The value of using longitudinal cohorts is that it allows us to see which factors or behaviours precede or follow others. For example, in young people who have schizophrenia and use cannabis, which came first? By collecting and analysing data in this way, it is more likely that cause and effect between factors can be established (though this is not always clear cut even with longitudinal methods).

When considering if there are adverse outcomes for young people who use cannabis, there are a number of hypotheses about how cannabis use and adverse outcomes may be related. These can be grouped into five overarching hypotheses. If we take the example of how schizophrenia and cannabis use may be related, we can see how each of the hypotheses would approach this:

- **‘Self medication’ hypothesis** – people use cannabis as a response to an adverse situation or condition. This is mostly applied to the link between cannabis use and mental health outcomes. Taking our example of how schizophrenia and cannabis use is related, this hypothesis would suggest that people who have schizophrenia use cannabis as a result of their illness.

- **Other drug use hypothesis** – the relationship between cannabis and an adverse situation or condition is affected by the use of other drugs. Looking at our schizophrenia and cannabis use example, this would suggest that it is use of other drugs, and not cannabis itself, that drives the observed relationship between schizophrenia and cannabis use.

- **Confounding factors hypothesis** – risk factors common to both cannabis use and an adverse situation or condition underlie the relationship seen. Thinking about our example, this hypothesis would suggest that people who have schizophrenia and use cannabis also have other things in common, like things in their environment (for example, being young, disengaged and/or unemployed). It is these other things that they have in common which drive both cannabis use and schizophrenia.

- **Aetiological hypothesis** – cannabis use plays a causal role in the adverse situation or condition. Thinking about our example, this hypothesis would suggest that using cannabis directly causes schizophrenia.

- **Interaction hypothesis** – stronger associations between cannabis use and an adverse situation or condition occur among people with a predisposition for that particular adverse situation or condition. So for our example, this hypothesis would suggest that some people would be more likely to be affected by the use of cannabis because there is something different about them, for example, a difference in the way their body reacts.
to cannabis. This makes this group of people more likely to develop schizophrenia as a response to cannabis.

We will now consider the evidence presented about the negative outcomes for young people who use cannabis. The evidence has been divided into sections, with each section discussing a particular outcome or group of related outcomes. The first section discusses mental health, with particular focus on psychosis and affective disorders. Section 2.2 discusses educational outcomes and section 2.3 discusses other adverse outcomes identified within the literature.

2.1 Mental health

2.1.1 Overview

There is a great deal of literature looking at the relationships between cannabis use in young people and later mental health. Psychosis and affective disorders such as anxiety and depression are often considered separately in the literature and are presented separately within this report.

2.1.2 Psychosis

Definition and measurement of psychosis

Psychosis is something which affects the way that people think, feel and behave. People experiencing psychosis may experience symptoms such as delusions and hallucinations. Psychosis is not a condition in itself, but a symptom of other conditions (most commonly mental health conditions like schizophrenia and bipolar disorder).

Cannabis use has been linked to developing psychosis in many studies and the nature of the link has been much debated. Analysing longitudinal cohorts of young people has allowed researchers to examine this link over time.

Psychosis itself is measured differently in different studies. Most studies measure symptoms based on self reported information and some use medical registers and records to look at the incidence of psychosis. This creates some difficulties when comparing results as they may be measuring different things; self reported symptoms may not always be to the level required for a clinical diagnosis and not all of those with symptoms of psychosis will receive a diagnosis that is then logged in medical records. These issues should be borne in mind when considering the evidence presented in this review; we highlight these issues where appropriate.

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3 It should be noted that different studies have used different terms and outcome measures for psychosis related disorders. In this section, psychosis includes schizophrenia, schizophreniform disorders, psychotic symptoms and psychotic disorders reported in the literature.
Cannabis use and schizophrenia

Two studies looked at the relationship between cannabis use and schizophrenia in a cohort of military conscripts. Both studies concluded that cannabis use was an independent risk factor for later onset of schizophrenia. Andreasson et al (1987, quoted in Degenhardt & Hall, 2006) found that those who reported having tried cannabis by the age of 18 were 2.4 times more likely to receive a schizophrenia diagnosis in the next 15 years. The more a respondent reported having used cannabis, the greater their risk of having a later schizophrenia diagnosis; the risk was three times higher for those who reported using cannabis between 1-50 times and 6 times higher for those who reported using cannabis more than 50 times.

Zammit et al (2002, quoted in Degenhardt & Hall, 2006) found the same relationship by extending the diagnosis period to 27 years post baseline measurement. They also reported a stronger relationship between cannabis use at baseline and diagnosis within the first 5 years.

It is interesting that both studies found the same outcome when looking at different time periods. However, as they are using the same study participants from a specific group, this perhaps isn’t unexpected. Further research is needed to examine if this is a broader phenomena among young people in general and whether this pattern is observed in other jurisdictions.

Cannabis use and non-specific psychosis

Schimmelmann et al (2011) examined the relationship between cannabis use and psychosis by analysing medical records. They used a data audit method and looked at recorded cannabis use disorders and the age of onset of psychotic symptoms.

The authors found the age of onset of psychotic episodes was no different in those who had a cannabis use disorder. However, they did find a small effect between early onset cannabis use (that is, cannabis use before age 14) and earlier onset of psychotic episode. This suggests that using cannabis from an earlier age may bring about a greater risk of developing psychosis. The authors suggested that the mechanism responsible for this finding was the indirect effect of cannabis on brain maturation at a development sensitive time in those sensitive to psychosis, which lowered the age of onset of psychosis.

One limitation of this method of analysis is that it is not usually possible to account for the many confounding factors which may also influence the relationship between cannabis use and psychosis. For example, age and sex will be available in the medical records but it is unlikely that other information that may be important, such as family history or other drug use, will be available. There is also the issue that using medical records only considers those who have been admitted to a care setting, rather than people with self-reported psychosis symptoms. This makes comparing results of this study with others
studies that use self reports difficult. It is well known that there are differences between those who access care and those who do not and this may affect the results seen.

Most studies we looked at did use self-reported psychotic symptoms rather than medical records in their analysis. In general, these studies consistently suggest a relationship between cannabis use and psychosis.

Fergusson et al (2005, quoted in Jenkins, 2005) looked at symptoms of psychosis and cannabis use at ages 18, 21 and 25 years. They found that at all ages, cannabis use was related to self-reported psychotic symptoms, with higher cannabis use being associated with increased symptoms. After controlling for other confounding factors, those reporting a daily use of cannabis had symptom incidences between 1.6 and 1.8 times higher than those who did not use cannabis.

Fergusson et al (2003a, quoted in Degenhardt & Hall, 2006) also found that cannabis dependence at age 18 predicted an increase in the risk of reporting psychotic symptoms at age 21. The authors also reported that the relationship appeared to move from cannabis dependence to psychosis, rather than in the opposite direction.

Wigman et al (2011) took a different approach to self-reported psychotic symptoms and classified patterns of thought problems\(^4\) in their study. They categorised young people into sub-types of thought problems over the course of the study, controlling for many factors, including cannabis use. The results identified four sub-types of thought problems:

- **Low** – low levels of initial thought problems at baseline and shallow decline in these over time
- **Decreasing** – high levels of thought problems at baseline and a steep decline in these over time
- **Increasing** – an average level of thought problems at baseline and a steep increase in these over time
- **Persistent** - high levels of thought problems at baseline and a steep increase in these over time

The authors found using cannabis before the age of 16 significantly predicted group membership for the increasing and decreasing sub-types but did not predict membership to the persistent group. However, the amount of cannabis used did predict membership of the increasing and persistent groups. More frequent use of cannabis was associated more strongly with these groups. This shows that not only is cannabis use potentially

\(^4\) Thought problems were identified using the thought problems subscale from the Youth Self Report (YSR; Achenbach, 1991) questionnaire. This is designed to tap into subclinical psychotic experiences. Wigman et al used 9 of the 12 questions included in the thought problems subscale from the YSR, which includes questions like ‘I deliberately try to hurt myself or kill myself’, ‘I repeat actions over and over’ and ‘I have thoughts that other people would think are strange’.
important in the experience of psychotic symptoms over time, but the amount of cannabis used may also be an important factor. However, it should be noted that the measurement of cannabis in this study was fairly basic and self-reported, ranging from never use, use sometimes to use often.

Wigman’s analysis showed that the four groups had different levels of thought problems at the start of the study. The idea that this might be important in the relationship between cannabis use and psychosis is well documented. Often this is taken into account in analysis by taking some measure of psychotic symptoms at an early age. Those who have higher levels of psychotic symptoms at a young age are sometimes referred to as having a predisposition to psychosis. If someone has a predisposition to psychosis they may be more likely than those who have not to develop psychosis later in life.

Arsenault et al (2002, quoted in Degenhardt & Hall, 2006) specifically examined this issue. They initially reported an increased risk of developing psychotic symptoms by age 26 among early cannabis users. However, once they took into account the presence of psychotic symptoms at age 11, this relationship was no longer evident. This could mean that once consideration of a person’s predisposition to psychosis is taken into account, no significant association with cannabis use is observed. However, the number of respondents within the cohort who had psychotic disorders by age 26 was low, and so it may be that the group size was too small to be able to detect any differences with confidence. Larger sample sizes are needed to examine this fully. Interestingly, Arsenault et al did find an interaction between the risk of psychosis and the age of onset of cannabis use, with earlier onset being associated with increased risk.

Hanquet et al (2005, quoted in Degenhardt & Hall, 2006) provide further support for the importance of age of onset of cannabis use and its relationship with psychosis. Firstly, the authors reported that those who used cannabis were more likely to report psychotic symptoms four years later than those who did not. However, this relationship was stronger for more frequent users and for those who reported using cannabis from an earlier age. They also found that those who were identified at the start of the study as having a predisposition to psychosis were much more likely to report psychotic symptoms four years later if they used cannabis; 51% of cannabis users identified as having a predisposition to psychosis reported psychotic symptoms at follow up, compared to 26% of non users with a predisposition. This suggests that it is not the predisposition alone that is important in the relationship; using cannabis appears to have an interaction, affecting different people in different ways.

**Biological factors, cannabis use and psychosis**

As the studies discussed so far show, there is evidence to suggest that cannabis use is related to psychosis in some way. Some of the studies discussed suggest that the relationship may affect different people in different ways. Those with a predisposition
to psychosis are more likely to report psychotic symptoms later in life if they used cannabis. It has been suggested that this predisposition to psychosis may be biological in nature and there is evidence to suggest that there are biological differences between people who experience difference outcomes after using cannabis.

Minozzi et al (2007) suggests that genes are likely to moderate the relationship between cannabis use and later psychosis by increasing an individual’s susceptibility to psychotic outcomes. They suggest, that the question of whether cannabis use causes or precipitates the development of psychosis remains unanswered and say that more research is needed to address this.

Capsi et al (2005) looked at the genetic differences of young people and how this may affect the relationship between cannabis use and later psychosis. They found that there were differences in the COMT gene\(^5\) that codes for dopamine and later psychotic symptoms. Young people in the cohort who were homozygous for a functional polymorphism and used cannabis were 10 times more likely to develop psychosis than those who were homozygous and did not use cannabis. Those young people who did not have a polymorphism were not at an increased risk of developing psychosis if they used cannabis. This suggests that there may be a biological basis for the differences seen in the development of psychosis by young people who use cannabis. In short, young people may be more at risk if they have a specific type of genetic make up.

Degenhardt & Hall (2006) agree that there is evidence of a biological basis for the relationship between cannabis and increased risk of psychosis. They argue that cannabinoid receptors\(^6\) in the brain play an important role in schizophrenia and other psychotic disorders. There is case control evidence that the receptor CB1 is found in greater density in the pre frontal cortex of people with schizophrenia, compared with controls; this is the receptor on which THC, the principal psychoactive agent in cannabis, acts upon. McLaren et al (2010) agree that biological evidence seems to support the idea that certain people may be more vulnerable to psychosis and these people may be more sensitive to the effects of cannabis in some way. They suggest that this type of interaction helps to explain why large rises in psychosis, in line with increased prevalence of cannabis use, have not been observed. However, they conclude that much more research is required in order to confirm this.

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\(^5\) Genes are created from codes in a person’s DNA. Often there is more than one version of the same gene. These versions are coded for by different alleles for that gene. If a person has two identical alleles for a particular gene, they are described as homozygous for that gene. If they have a combination of different alleles, they are described as heterozygous. If the different combinations of alleles affect the way that gene functions, this is known as a functional polymorphism. That is, people with different combinations of alleles are different to one another in the way that particular gene works.

\(^6\) Receptors are areas in the brain that react to specific ingredients or chemicals in things. Cannabinoid receptors are the areas in the brain which react to cannabinoids, which are chemical compounds found in cannabis. Tetrahydrocannbinol (THC) is one of these cannabinoids.
One concern when trying to establish causality is that the results may be affected by other things. It is important to consider, specify and measure these other factors so that one can control for their effects in the analysis. These issues are considered in the following section.

Confounding factors

Arsenault et al (2004, quoted in Jenkins, 2005) pooled the results of a number of longitudinal studies and found that people who used cannabis were more than two times more likely to report various psychosis related outcomes. In some studies, at least, a dose response relationship was evident, with greater use of cannabis being related more strongly to psychosis. However, the most prominent and consistent finding was the level of reduction in the observed relationship once other confounding factors were taken into account. These confounding factors were personal, peer and family characteristics. While most of the relationships between cannabis use and later psychosis outcomes remained significant, they were substantially reduced when confounding variables were taken into account. This suggests that many other factors affect the relationship between cannabis and psychosis and highlights the importance of measuring these other factors as well as the complexity of unpicking cause and effect within these associations.

One aspect not often considered when thinking about confounding factors is the impact of where people live and how this may contribute to the outcomes of young people who use cannabis. Kuepper et al (2011) looked at a cohort of young people from Munich to see whether cannabis use and urbanicity (that is, living in an urban area) work together in causing psychosis.

They found that there was an interaction between urbanicity and cannabis use on later psychosis. The association between cannabis use at baseline and later psychotic symptoms was much stronger for individuals from an urban area compared with those from rural areas. This suggests that where a person lives may be another factor involved in the complex relationships between cannabis use and later outcomes.

One way to take account of confounding factors is to match similar people together in analyses. One way to match people is to use siblings as they grow up in the same environment. McGrath et al (2010) used a sibling pair analysis nested within a prospective birth cohort to reduce the influence of unmeasured confounding factors in their analysis. Psychosis measures were self reported and cannabis use was collected by retrospective self report at age 21 (asking about frequency use cannabis in the last month and at what age they started using cannabis). A number of confounding factors were also measured and controlled for in the analysis.

The authors found that individuals with early onset cannabis use (age 15 or younger) had a greater risk of later psychosis related outcomes. The authors concluded from this that early cannabis use is associated with psychosis related outcomes in early adulthood. A greater number of years since first using cannabis were also associated with a higher risk
of psychosis related outcomes. However, they also found that young people with early onset hallucinations were more likely to start using cannabis at a younger age and to use cannabis frequently at age 21. This lends support to the self medication hypothesis - that people use cannabis as a response to an illness or condition.

**Summary**

As we can see, there is a lot of evidence that suggests that cannabis use in young people is related in some way to psychosis. Returning to the five hypotheses discussed in the introduction we can examine how the data best fits each:

‘Self medication’ hypothesis

There does not appear to be a great deal of evidence to support the hypothesis that young people use cannabis as a response to a psychosis. McGrath et al (2010) found evidence that young people with early onset hallucinations were more likely to start using cannabis at a younger age, and use cannabis frequently later in life. However, they warned that the measures used for psychosis outcomes were not currently validated.

Few of the studies reviewed supported the self medication hypothesis, with most results suggesting that this does not explain the relationship between cannabis use and psychosis.

In considering the self medication hypothesis, Degenhardt & Hall (2006) state that it is ‘superficially plausible but the evidence in its favour is not very compelling’. They observe that people with schizophrenia often use cannabis for the same reasons as people without it and that the drugs most commonly used by people with schizophrenia are the same drugs most commonly used by others. However, they do concede that some people with schizophrenia report using cannabis in response to negative symptoms.

They conclude that most of the epidemiological evidence does not support the self medication hypothesis as the relationship over time seems to run from cannabis use to psychosis, rather than the other way. A review by Smit et al (2004) agrees that the evidence suggests that the self medication hypothesis can be discounted.

McLaren et al (2010) also reviewed the evidence on causal links between cannabis use and psychosis and concluded that most evidence does not support the self medication hypothesis as the temporal link is not apparent. That means, cannabis use does not come after the onset of psychotic symptoms. However, like Degenhardt & Hall, they do point out that this does not mean that people with psychosis do not then use cannabis as a means of relieving their symptoms, highlighting the complexity of the associations observed.

**Other drug use hypothesis**

There is little evidence to suggest that other drugs are the cause of the relationship seen between cannabis use and psychosis. Smit et al (2004) agrees that the evidence suggests that this hypothesis can be discounted.
Arsenault et al (2002) looked at the specificity of the association between cannabis use and psychosis. They did this by analysing the effects of other drugs on later psychosis. The results of this analysis found there was no relationship between the use of other drugs and psychotic disorders. This adds weight to the argument that it is cannabis specifically which may be important in the relationship.

**Confounding factors hypothesis**

It is clear that there are many factors that affect the relationship between cannabis use and psychosis. The most prominent finding from the pooled analysis carried out by Arsenault et al (2004, quoted in Jenkins, 2005) was the level of reduction in the strength of the association after controlling for confounding factors. It still remains possible that it is other factors which have not been controlled for in the analysis which underlie the relationship.

Smit et al (2004) agree that based on the current evidence, the hypothesis that the relationship may be the results of risk factors common to both cannabis use and psychosis can not yet be discounted. Moore et al (2007) following a systematic review of the evidence, agreed with this. They stated that the possibility that the unobserved association between cannabis use and psychosis may be the result of confounding factors not accounted for, can not be ruled out. They also argued that these uncertainties are unlikely to be clarified in the near future. Despite this, the authors concluded that there is now sufficient evidence to warn young people that using cannabis may increase the risk of them developing a psychotic illness in the future.

**Aetiological hypothesis**

There is much evidence that cannabis use does play a causal role in the development of psychosis. Jenkins (2005) reported that, despite the limitations of the available evidence, cannabis plays some causal role in the development of psychosis. Cannabis users are more likely than non users to report psychotic symptoms and this link appears to be stronger for those who use the drug more frequently and for those who use it from a younger age. Crucially, this link remains significant after controlling for various family, individual and social factors.

The question remains as to whether this is the case for all people, or whether the risk exists only for a certain group of people.

**Interaction hypothesis**

There is compelling evidence suggesting that there may be an interaction between having a predisposition to psychosis and using cannabis. Those who are identified as having a predisposition to psychosis and who use cannabis appear to be at greater risk of future psychosis. It seems that this explanation best fits the current data about cannabis use in young people and risk of psychosis.
In conclusion to their review, Degenhardt & Hall (2006) suggest that there is good epidemiological evidence from several countries which supports the theory that cannabis use predicts an increased risk of psychosis. They say that the evidence suggests it is most likely that cannabis use precipitates schizophrenia in those who are vulnerable to it because of personal or family history. A review by Hall (2006) agrees that the relationship is stronger for those with personal or family history of schizophrenia which points to an interaction between cannabis use and individuals with a genetic vulnerability to psychosis.

Similarly, McLaren et al (2010) say that ‘it seems most likely that cannabis produces psychotic disorders in individuals who possess an underlying vulnerability to psychosis’. They suggest three explanations of how this relationship may work:

- People who go on to develop a psychotic disorder after using cannabis would have eventually developed it without using cannabis
- The same factors which underlie using cannabis also underlies a person’s vulnerability to psychosis
- People who are vulnerable to psychosis may be more sensitive to cannabis than people who are not vulnerable to psychosis

McLaren et al feel that in light of the evidence, the latter of these (those vulnerable to psychosis are more sensitive to cannabis) is the most plausible explanation and state that biological evidence seems to support this.

Both Degenhardt & Hall (2006) and McLaren et al (2010) point out that an interaction effect of this type helps to explain why there has not been large increases in psychosis in line with the rise in the prevalence of cannabis use by young people. McLaren et al (2010) do, however, point out that while overall psychosis prevalence has not increased, age of onset of psychosis has fallen in the last three decades (DiMaggio et al, 2001) and that people who use cannabis are more likely to be younger at their first psychotic episode than non cannabis users (Veen et al, 2004). This supports findings from Schimmelmann et al (2011) who found that using cannabis from an earlier age may have greater risks around developing psychosis early. The authors conclude that it may be an indirect effect of cannabis on brain maturation at a sensitive development time among those sensitive to psychosis that lowers the age of onset. As this study was done using a data audit of diagnosed cases, we are unable to say whether this effect of early use acts on everyone or just those vulnerable to psychosis.

Minozzi et al (2010) suggest in their review that genes are likely to moderate the relationship between cannabis use and later psychosis by increasing an individual’s susceptibility to psychotic outcomes. However, they also note that no study has yet verified any interaction between particular genes and cannabis use and more research is needed to address this.
Conclusions

It seems clear from the evidence that cannabis use in young people is associated with an increased risk of later psychosis. What remains unclear is how this relationship operates.

- There is most support for the hypothesis that individuals with a specific vulnerability may be most at risk, and that this vulnerability may be genetically determined.
- There is also support for the idea that there is a period during adolescence when individuals may be particularly affected by the use of cannabis. The suggestion is that this is at a time when the adolescent brain is in a state of development.
- It also appears likely that there is a dose-related effect, with greater levels of cannabis use being related to a greater risk of later psychosis.

However, it is also clear that confounding factors play a large role in the relationships and that these need to be adequately addressed in future studies to be able to draw robust conclusions about the roles these play. McLaren et al (2010) state that ultimately the current data can not definitively answer whether cannabis use causes serious psychotic disorders that would not have occurred if an individual had not used the drug. They suggest further, rigorous cohort studies are needed to inform this.

Despite this level of uncertainty about the precise nature of the relationships, as advocated by Moore et al (2007), there does now seem to be sufficient evidence to warn young people that using cannabis could increase their risk of developing a psychotic illness later in life.

2.1.3 Affective disorders

The relationship between cannabis use in young people and affective disorders, like depression and anxiety, has been extensively studied using longitudinal data. The evidence tends to focus on two broad areas:

- Whether cannabis use is associated with onset of later affective disorders
- Whether affective disorders are associated with later cannabis use

Most studies examine the former and tend to demonstrate that there is a relationship between cannabis use and onset of later disorders but note that relationship is complex and may vary at different stages of the life course. Fewer studies have examined the latter, but they are an important component of the association between affective disorders and cannabis use. This also relates directly to the self-medication hypothesis. These data are summarised below.

Finally, the range of affective disorders considered in the literature focuses on
depression, suicidal thoughts and behaviour, and anxiety. Only these issues are considered here.

**Cannabis use and later onset of affective disorders**

Jenkins’ review (2005) identified four studies that assessed the relationship between cannabis use at baseline and onset of depression at subsequent interview. Of these studies, two showed evidence that prior use of cannabis was associated with later depression among some young people. For example, Paton et al (2002) found that, compared with non-cannabis users, young women who had used cannabis on a weekly basis during adolescence were 2.5 times more likely to meet the criteria for self-reported anxiety and depression at follow up. This analysis also took into account anxiety and depression at baseline measurement. This means that this observation is not an artefact of younger women who use cannabis being more likely to experience affective disorders to start with. However, this finding only applied to young women and similar patterns were not observed for young men.

This single example highlights that the relationship between cannabis use and affective disorders is not necessarily clear cut and other research serves to reiterate this. Further evidence of this complexity is provided by McGee *et al* (2000). They found that early cannabis use (by age 15) had no association with an increased risk of a mental disorder by age 18. However, among boys, cannabis use at a young age did predict a greater likelihood of indication of a mental disorder at age 21. In this research, this relationship was not apparent for girls. Furthermore, Degenhardt *et al* (2003; quoted in Jenkins) stated that their evidence showed that the observed relationship between adolescent cannabis use and later depression was explained by confounding factors (such as family, peer or individual characteristics). The importance of other factors in influencing the onset of affective disorders was supported by Ferguson & Horwood (1997) who found that the observed association between early cannabis use and later onset of depression or suicide attempts was largely explained by these confounding factors.

While these confounding factors are clearly important, the pattern observed by Ferguson & Horwood (1997) is not universal. Some studies showed a significant relationship between cannabis use and affective disorder even after these other factors had been taken into account. For example, a second study by Ferguson *et al* (2002) of the same cohort at an older age showed significant associations between cannabis use and affective disorders. Those who had reported at least weekly cannabis use at any age between the age of 14 and 21 were twice as likely to be diagnosed with depression. In this study, cannabis use also predicted increased risk of suicidal thoughts and suicide attempts, though the pattern varied among different cohorts. People who used cannabis at least weekly at a younger age were also more likely to report suicidal thoughts and suicide attempts. People using cannabis at least weekly by the age of 21 were at no increased risk of this. Confounding factors did explain some of the variance in these relationships but not all. Despite this, the
possibility remains that some other unmeasured, and therefore, unaccountable, factors may be driving this association.

This last example is also illustrative of a broader phenomenon whereby when researchers follow-up cohorts over several time points, they often observe different results at different ages. Ferguson *et al* did not see significant results when their cohort was aged 18, but in a later study when participants were 21 differences emerged. Furthermore, Pederson’s (2008) initial examination of a cohort of youth in Norway found no associations between cannabis use in adolescence and depression. However, when they extended their research to follow-up cohort members at later ages, they found that cannabis use at 21 predicted suicide attempts at age 27. It is worth noting the earlier example given of McGee *et al*’s work whereby cannabis use at age 15 was not associated with depression at age 18 but was at age 21.

Since Jenkins’ review further studies examined this issue and by and large, they identify similar issues. Richardson *et al* (2010) conducted a review of the relationship between cannabis use and affective disorders. This highlighted Hayatbakhsh *et al*’s (2007) work in Australia. They found that cannabis use in young adulthood predicted an increased risk of depressive disorders in young adulthood, which was significant after taking into account a range of other factors. They also found that those who reported using cannabis at an earlier age were over three times more likely to report anxiety and depression than non-users (though there may be some questions about the reliability of this data as age of onset for cannabis was based on retrospective self-reports).

Richardson concludes his review by suggesting that there is some evidence of a causal mechanism between cannabis use and affective disorders. He points to emerging evidence from neurobiology, including a study from Medina *et al* (2007) which compared brain images from cannabis users aged 16-18 with a matched control. This demonstrates that there were physical differences between the brains of the cannabis users and their matched controls. The brains of the cannabis users had smaller volumes of white matter. This suggests that there may be some underlying biological explanation, similar to that proposed by the evidence around psychosis, which mediates the effects of cannabis. However, it is important to note that the evidence this is based on is not longitudinal and so it is not possible to say which came first – the reduced white matter volume or the onset of cannabis use. Richardson also suggests that evidence of the relationship between age of onset of cannabis use and later depression may be related to the age at which cannabis is consumed, similarly to psychosis risk, with adolescence being a high risk time period in brain development and therefore this group may be more sensitive to the effects of cannabis.

This is an interesting area for further development and mirrors investigation and research questions in other risk taking behaviour (such as gambling behaviour). However, further research is needed to explore the neurobiological impact of cannabis.
use among adolescents as the evidence reviewed within this study is not a sufficient basis for firm conclusions.

**Affective disorders and later cannabis use**

Only two studies reviewed looked at whether affective disorders were associated with later cannabis use. This is, however, an important component in assessing the validity of the self-medication hypothesis. In Jenkin’s review he noted that Paton *et al.* (1977) identified that depressive mood at baseline interview was associated with later use of cannabis when compared with those who were not using cannabis at first interview. This may provide some support for the self-medication hypothesis. However, the relationship was more complex than this as depressive mood among cannabis users was also associated with stopping using cannabis at a later date. McGee *et al.* (2000) also showed a small but significant association between indication of mental disorder at age 15 and increase risk of cannabis use at age 18.

The pathways, behaviours and relationship between depressive mood and subsequent cannabis use are not clear cut and may vary among different sub-groups. The evidence base is also very thin. For some, there may be a self-medication explanation to some behaviour, though this is likely to be related to a range of other issues also. What is lacking from this literature is a focus on understanding the motivations that underpin behaviour. Longitudinal research is powerful in tracing onset and sequence but broad gaps in data collection can miss potentially nuanced changes in social or personal circumstances which may be inter-related with motivations for behaviour. A more holistic perspective is needed to untangle these varying influences, over an above accounting for them in regression models.

**Methodological issues**

The evidence reviewed about cannabis use and affective disorders raises some interesting methodological and substantive issues.

Firstly, as Richardson *et al.* (2010) point out, there are a range of other factors that may influence and shape this relationship. However, comparing across studies is difficult because different studies measure and analyse these contextual factors in different ways, which may explain some of the variation in findings between studies.

Pederson (2008) also pointed out the need to carefully consider cultural differences and practices when making cross national comparisons. They attributed the lack of observed relationship between Norwegian cannabis user and affective disorder to lower prevalence rates of cannabis use among youth (i.e. cannabis use tends to develop later in Norway than in some other countries).

Finally, this evidence highlights that to fully unpick the association between cannabis use and affective disorders, cohorts need to be followed throughout adolescence and early adulthood to monitor developments at different stages of the life course.
Summary
It is clear from the evidence we have reviewed that there is a relationship between cannabis use in young people and affective disorders. As with the evidence about cannabis use and psychosis, what remains uncertain is the nature of this relationship. Whilst there is less evidence to support the self medication hypothesis, largely because motivations for cannabis use have not been explored, confounding factors have been shown to play a large part in the relationship between cannabis use and later depression and anxiety. As with the evidence about psychosis, some reviews suggest that there maybe a causal relationship, specifically relating to cannabis use and subsequent affective disorders. However, this may be mediated by a vulnerability which could be genetic in origin and this needs further consideration. We have also seen that there appears to be an increased risk for those who use cannabis either more frequently or over longer periods of time with development of affective disorders. Further evidence from well controlled longitudinal studies is required to fully understand the nature of this relationship, the range of other factors that may be associated with it and how it develops for different groups across the life course.

2.2 Educational attainment

2.2.1 Overview

Some studies have considered the effect that cannabis use in adolescence has on educational attainment. Most of these studies have examined the relationship between frequency of cannabis use and educational attainment, with some demonstrating evidence of a dose-response relationship. Some studies have also examined age of onset of cannabis use and its associations with educational attainment. Few studies have looked at the interaction between onset and frequency, though one notable example is provided below. Finally, a few studies have also considered cannabis use and educational attainment among different minority ethnic groups. Data relating to all of these issues are summarised below:

2.2.2 Frequency of cannabis use and educational attainment

Jenkins’ review (2005) includes a section on cannabis use in young people and educational attainment. He cites studies from Fergusson and Horwood (1997) and Fergusson et al (2003). Both of these demonstrated a clear association between cannabis use and poor educational attainment even after other confounding factors had been taken into account. In the earlier of the two studies, those who had used cannabis when aged 15-16 were significantly more likely to leave school without and formal qualifications at age 18. The relationship had a dose response dimension: 54% of those who had used cannabis ten or more times gained no formal qualifications, compared with 30% of those who had used cannabis between one and nine times and 14% of those who had not used cannabis. In the later study, Fergusson and
colleagues further confirmed this dose-response relationship. High frequency cannabis users (those who had used the drug 100 times or more before the age of 16) were 3.5 times more likely to leave school without any formal qualifications. This analysis also took into account confounding individual, family and peer level factors.

Additional support for the dose-response relationship was provided by Lynskey et al (2003; cited in Jenkins) who looked at how frequency of cannabis use during adolescence and early school leaving may be related. They found that weekly cannabis use was associated with an increased risk of early school leaving. Specifically, once other confounding factors had been taken into account, weekly cannabis users who had been frequent users from a young age had a significantly increased risk of early school drop out. This highlights an interesting interaction between age of onset and frequency of use. It suggests that more nuanced analysis is needed to try to tease out which parts of the relationship are important and, if and how, these factors combine to affect the level of risk observed for young people. Some studies have highlighted that the relationship between cannabis use (and frequency of use) and educational attainment differs among certain minority ethnic groups.

Ellickson et al (1998; cited in Jenkins) examined cannabis use and school drop out rates among individual ethnic groups in California. The cohort separated ethnic group into four classifications; Asian, black, white and Latino. Results showed that after controlling for confounding factors, marijuana use among Latino students was related to a significant increase in risk of early school dropout. Interestingly this was not the case for members of the other ethnic groups; for these groups cigarette smoking significantly predicted early school drop out.

Marie et al (2008) also considered ethnic identity and the relationship it may have with cannabis use and educational attainment among Maori cohorts. They found that there were higher rates of cannabis use and dependence in Maori cohort members compared with non Maori members and concluded that this was largely to do with socio economic disadvantage and greater exposure to negative environmental factors known to influence the risk of using cannabis. However, when these factors were taken into account, a significant relationship was still observed between cannabis use and poorer educational outcomes among the Maori cohort.

Together, these two studies suggest that ethnicity, identity and socio-economic circumstances may be important factors to consider when looking at the relationships between cannabis use and adverse educational outcomes for young people.

### 2.2.3 Age of onset and educational attainment

Three studies specifically examined age of onset of cannabis use and subsequent educational attainment. Each study used slightly different measures of educational attainment but broadly looked at early school drop-out rates and confirmed a
relationship between early cannabis use and early school leaving.

Horwood et al (2006) reported a robust association between age of onset of cannabis use and subsequent educational achievement in their review of three Australasian cohort studies. Educational achievement was examined using three outcomes – high school completion, university enrolment and university degree attainment. They found a significant association between the age of onset of cannabis use and all educational outcomes measured. Educational attainment was higher for those who had never used cannabis before the age of 18 and lowest for those who started using cannabis before the age of 15. Many confounding factors were taken into account in the analysis and the relationships remained significant.

Fergusson et al (1996) found that before adjusting for confounding factors, early cannabis users had significantly higher rates of truancy and early school leaving than non-users. After controlling for confounding factors, including family background and previous educational attainment, early cannabis users did not have significantly higher rates of truancy than non-users. However, the risk of early school leaving remained significant, with early cannabis users being over three times more likely to drop out of school than non users.

What is particularly interesting is the different interpretations authors have assigned to these results. Horwood et al (2010) suggested that their findings point to a cause and effect relationship in which early cannabis use leads to an increase risk of underachievement in education (although they note that this relationship may well be mediated by various pathways and that some of these are not well understood). They recommend further research into both the neuro-physiological and social processes involved.

Hall (2006), who noted in his review that early cannabis use predicts early school leaving, takes a more circumspect approach. He suggests that pre-existing poor performance coupled with acute cognitive impairment from intoxication, an affiliation with peers that reject school and early transitions into adulthood without appropriate preparation may all play a part in the relationship. However, Hall also acknowledges that cannabis use has been shown to play a part in poor educational performance even after controlling for a number of confounding factors.

2.2.4 Summary

The review of longitudinal studies by Lynskey and Hall (2000) concluded that there is evidence that cannabis use may play some causal role in poor educational performance as the associations observed remain significant after controlling for a number of other possible influences. However, they exercise caution and say that it remains possible that the association is in fact non-causal and that the observed findings are the result of some other unmeasured factors that has not been controlled.
for in the analysis. This echoes some of the main findings relating to the relationship between cannabis use and psychosis or affective disorders.

Others have suggested (Jenkins, 2005) that the most plausible argument for how cannabis use may affect educational attainment is that cannabis use itself occurs within peer groups who encourage adolescents to take on other non-conventional behaviours and more adult roles. This, in turn, increases the likelihood of individuals in those groups leaving school early. Jenkins suggests that this argument is consistent with other work that identified associations between cannabis use and higher levels of other non-conventional behaviours such as early sexual activity, unplanned parenthood, unemployment and leaving home early. He notes that more research is needed to explore and understand these (potential) transitional pathways.

The evidence examining the relationship between cannabis use in young people and subsequent educational attainment highlights the complex nature of these associations. The range of potential confounding factors which may influence educational attainment is large and for this reason we would be cautious about assuming cause and effect based on the evidence reviewed. It is also possible that educational attainment is more strongly tied with the immediate effects of cannabis use; if young people are intoxicated then they may be less able to perform. Furthermore, the relationship seems to be stronger among younger users and more frequent users and interactions between onset and frequency needs further exploration.

It has been suggested that many factors, including ethnicity and peer group may be important in the pathways which contribute to educational attainment. Therefore research examining this issue needs to be rigorously designed and able to account for many confounding factors that may augment the relationship between cannabis use and poorer educational outcomes.

### 2.3 Others outcomes

A number of other adverse outcomes have also been examined using longitudinal data. These are summarised below.

#### 2.3.1 Life satisfaction

Some studies have used longitudinal cohort data to examine the relationship between cannabis use and later life satisfaction. One such study was carried out by Fergusson & Boden (2008) which used the Christchurch cohort study to examine cannabis use in adolescence and later life outcomes. They found that an increasing frequency of cannabis use at a young age (between ages 14-18) was significantly associated with lower levels of education attainment, lower income, higher welfare dependence, higher unemployment, lower levels of relationship satisfaction and lower levels of overall life satisfaction. These relationships all remained significant after controlling for a range of
confounding factors at the individual and family level.

A study looking at outcomes in young adults who use cannabis and tobacco by Georgiades and Boyle (2007) using a longitudinal cohort from Ontario, Canada also found associations between adolescent cannabis use and decreased life satisfaction. While this association reduced in strength after controlling for confounding variable, it remained significant. Interestingly, they concluded that cannabis use in adolescence had fewer or weaker ill effects in young adulthood than tobacco use in adolescence.

Another study considering life satisfaction was carried out by Swain et al. (2012) who also used the Christchurch cohort study to look at life outcomes up to the age of 30. Initial analysis showed that previous cannabis abuse and dependence was related to lower levels of life satisfaction at age 30. This relationship remained significant after controlling for the effects of other factors such as parental and family background, child behaviour and academic ability. But once time dynamic factors, like recent life events, employment and current living factors were controlled for the relationship was no longer significant. Swain et al. concluded that the findings suggest that the relationship noted between cannabis abuse and dependence and later life satisfaction can be explained by these time dynamic factors. This suggests that it is important to control not just for fixed characteristics measured at baseline but to include future characteristics which change over time in any longitudinal research into the later effects of cannabis use in young people. This finding reflects the complexity of the trajectories that young people take and the huge range of changing factors in their lives as they get older.

2.3.2 Social welfare assistance

Fergusson & Boden (2008) show that cannabis use is related to higher levels of social welfare dependence in New Zealand. Pederson’s (2011) Norwegian study also found this to be the case. He found that there was an association between recent cannabis use and the receipt of social welfare assistance in young adults that remained when controlling for confounding factors. However, his results did not show that early onset of cannabis use was associated with social welfare assistance once other factors were taken into account. These results suggest that while there is some association between cannabis use and later social welfare assistance, the relationship is by no means clear and is likely to be heavily influenced by both fixed and time dynamic factors. Much more research is required to examine if the associations are causal or if they are the result of other unmeasured factors. It would also be helpful to consider factors such as labour market changes over time within the analysis.

2.3.3 Problem behaviours

There is some evidence linking cannabis use among young people with problematic behaviour. This includes antisocial behaviour and offending behaviour. Jenkins (2005)
covers a range of evidence in his review about how cannabis use at a young age has been linked with these types of behaviours.

Derzon & Lipsey (1999; cited in Jenkins, 2005) conducted a meta-analysis of longitudinal and cross sectional data on cannabis use and delinquent behaviour. Most of the studies included in the analysis were carried out in the USA. They found that relationship between marijuana use and delinquent behaviour was stronger in cross sectional studies than in longitudinal ones. They went on to conclude that it is likely that the association between the two is co-occurring in nature rather than causal.

Macleod et al (2004) looked at a series of studies using the Christchurch cohort and found that younger people who used cannabis were more likely than non-users to be juvenile offenders. Early cannabis users were more likely to have repeat offences and had greater contacts with the police than non users. They also reported that more frequent cannabis users had greater risks associated with juvenile offence.

Finally, Pederson & Skardhamar’s (2009) cohort study of young Norwegians examined cannabis use in adolescence and young adulthood and later criminal charges. The study made use of administrative data on crime statistics and information from the cohort study. They found that there was a significant association between cannabis use before the age of 16 and later criminal charges. However, when drug-related crimes were excluded from the analysis, this relationship became non-significant. That is, the results showed no increased risk of crimes such as criminal gain or violence when comparing adolescent cannabis users with non users. Cannabis use before the age of 16 did, however, significantly predict an increase in the risk of being arrested for a drug specific crime. This includes the charge of possession of a drug.

This study also showed that adolescents who used cannabis were more likely to have a greater number of contacts with the police. The authors conclude that adolescent cannabis use is related to an increased likelihood of young people coming into contact with the penal system and that this may have a detrimental impact on their lives in the future. The authors noted that the levels of adolescent cannabis users in the sample was low and also that attrition analysis showed that the study lost some of those who had the most serious criminal records at follow up. Therefore, these results should be interpreted cautiously.

2.4 Other substance use

There is a large volume of data considering whether cannabis use leads to other substance use – the so called ‘gateway effect’. This aspect of the literature has not been extensively reviewed here but there were some discussion in the various papers included which are presented below.
A study by Patton et al (2006) looked at the use of adolescent cannabis and alcohol and the different trajectories they had into adulthood. The authors used an Australian cohort study to examine this and found that adolescents who had used cannabis at about once a week had a seven fold higher rate of risk of progressing to high risk use (daily or almost daily) in early adulthood. They also showed a two fold increase in transition to high risk alcohol use in early adulthood among this group. Weekly cannabis users who did so persistently in adolescence (i.e. were classified as weekly users at more than one time point during cohort data collection period) had higher risks of poorer education and training outcomes, less likelihood of being in a relationship and clearly elevated levels of later illicit substance use and drug and alcohol service consultation. Interestingly, the authors noted that although there was some association between alcohol use and cannabis use over the course of the data collection, high risk level users tended to use one substance predominantly. This has potentially important implications for targeted health interventions in this age group.

Degenhardt et al (2010) looked at alcohol and nicotine dependence in early adulthood and found that both were more likely among adolescent cannabis users than non users. This also had a dose response relationship, with the risk of being alcohol and nicotine dependent in early adulthood being highest for frequent cannabis users and lowest for non users. Occasional cannabis user’s risk of being dependent was between the levels observed for frequent and non users. Degenhardt et al did note though that while the outcomes remained significant after controlling for a range of confounding variables, one variable in particular reduced the association hugely. This was adolescent smoking. This suggests that there may be common factors at play which underlie both behaviours.

In Hall’s 2006 review of mental health outcomes for young people and cannabis use, he evaluates evidence of the gateway theory in that cannabis use leads to further illicit drug use. He concludes that there is strong evidence of an association between cannabis use and other illicit drug use. He adds, though, that the theory that the relationship is not causal and is in fact the result of other common factors can not be discounted. As with other areas of the literature, further well controlled research is needed to investigate the complex trajectories which exist between cannabis use in young people and future illicit drug use.
3 Limitations

In terms of investigating causality, many studies have used longitudinal data to investigate the relationship between cannabis use and adverse life outcomes. However, there are a number of limitations to these studies which makes comparing the results and reaching a conclusion difficult. Some of the limitations noted during this review are:

- **Definition of concepts** – cannabis use and outcome measures are defined differently between studies. Some define cannabis use in terms of frequency, others in terms of lifetime use and different ages at which cannabis use is measured. Most use is retrospectively self reported, which can be problematic. In regards to outcome measures, these are also defined differently. With mental health outcomes, some studies measure self reported symptoms, some measure clinical diagnosis and some measure hospital admissions. These are clearly very different and this makes comparing results difficult. This adds to the difficulty of reaching a firm conclusion about the impact of cannabis use on the outcome measure.

- **Age of cohort members** – there are differences in the mean age of cohort members at the time points measured. As there is evidence to suggest that some periods in adolescence may be more important than others in regards to the effects of cannabis on later psychosis - this is another problem when comparing results between studies. However, individuals mature at different times and so the theoretical ‘critical period’ when cannabis use may be more risk prone may also differ from individual to individual.

- **Measurement of cannabis use** – as with the definition of use, this is measured differently across studies. Some measure any use, some measure at least a certain number of uses and some having different increments of usage. Some studies measure frequency of use in terms of average weekly or monthly consumption and some measure total consumption to a certain date. Again, comparing results across studies accurately is challenging because of these differences. Furthermore, as most cannabis use is self reported it is impossible to know about the levels of cannabis actually consumed. There is the assumption that, for example, daily use of cannabis is the same across cohort member and time point. However, as there is no accurate way to measure dose per use or strength of active agent, this is uncertain and may confound results.

- **Confounding factors** – the literature shows clearly that there are a range of confounding factors involved in the relationship between cannabis use and psychosis. However, different studies measure and control for different factors. As all studies which control for confounding factors find that there is a reduction in the effect of cannabis use on psychosis once these factors are accounted for, it is clear that the effects reported may need to be interpreted with care as some studies include some confounding factors and others include different ones. It is
partly for this reason that the confounding factors hypothesis can not be discounted.

- **Time and place of study** – there is evidence to suggest that cannabis as a drug has changed over time. Nowadays there are higher levels of THC (the main psychoactive agent of cannabis) found in cannabis compared with 30 years ago. The time of data collection and the country in which it is collected may lead to differences in the strength of cannabis used and therefore might account for differences observed between countries and/or studies.
4 Conclusions

This review shows clearly that the level of certainty with which conclusions can be drawn is limited. There is a large body of evidence that strongly suggests that cannabis use in young people is linked to an increased risk of a range of negative social, educational and health outcomes. These risks appear to be elevated in those who use cannabis at a younger age and those who use cannabis more frequently.

The way in which this risk operates remains unclear and further well controlled longitudinal research is needed to examine these. Neuro-physiological research would help to examine further the idea that there is a genetic pre-disposition to certain outcomes with which cannabis use interacts. This could also be used to examine whether there are biological reasons that use at a younger age appears to carry greater risks of the negative outcomes.

- Returning to the five original hypotheses, in the light of the evidence presented in this report it seems that
- There is less support for the self medication hypothesis and other drugs hypothesis in the current literature.
- The aetiological hypothesis has some support but outcomes are different for people who use cannabis; not all young people who use cannabis go on to develop or experience the negative outcomes.
- Both the confounding factors and the interaction hypotheses have strong evidence in their favour and so these must be considered. It may be that different theories are best applied to different types of outcomes.
- Much stronger evidence for the interaction hypothesis can be seen in the literature looking at mental health outcomes than for negative social or educational outcomes, where confounding factors appear to be more important.
References


* These papers have not been fully reviewed. They have been referenced in other works included in the review.
cohort effect of age at onset of schizophrenia. *American Journal of Psychiatry, 158, 489-492*


Dependence, 110, 247-253


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health outcomes: a systematic review. *The Lancet*, 370, 319-328


Appendix 1

Key search terms used in stage two of the literature search were:

- (psychosis or psychotic or schizophren* or delusion* or hallucination)
- (depression or anxiety or mood)
- (school or performance or exam or (education + achiev*) or (education + attain*)
- (antisocial or delinquen* or offen* or crim* or conviction or violent or truan*)
- (circulat* or blood or respirat* or lung or reproduct* or sperm or ovulation or fertility)
- (other drug or illicit drug or gateway or cocaine or heroin or ecstasy)
## Appendix 2

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<td>Fergusson, D. M., Horwood, L. J. &amp; Swain-Campbell, N. R. (2002)</td>
<td>Cannabis use and psychosocial adjustment in adolescence young adulthood. <em>Addiction, 97, 1123-1135</em></td>
<td>Analysis of Christchurch Health and Development Study, New Zealand, Birth Cohort of 1265 children born in 1977. Self reported cannabis use for each year obtained at follow ups between ages 14-21 years. Self reported psychosocial measures included: criminal offending using self report of early delinquency scale (SRED), depression was classified according to DSM-III-R/DSM-IV (depending on year), suicidal ideation and attempts, other illicit drug use, adverse or stressful life events (based on feeling bad scale; FBS), deviant peer affiliations, age of leaving school, age of leaving home, and alcohol abuse/dependence.</td>
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<td><strong>Georgiades, K. &amp; Boyle, M.H. (2007)</strong></td>
<td>Adolescent tobacco and cannabis use: young adult outcomes from the Ontario Child Health Study. <em>Journal of Child Psychology and Psychiatry, 48, 724-731</em></td>
<td>Analysis of data from the Ontario Child Health Study (OCHS) which began in 1983 with follow ups in 1987 and 2001. Self reported marijuana/hashish use in the last 6 months Self reported outcomes included number of years in education, personal income in last year, physical health, life satisfaction, 12 month prevalence of major depressive disorder, daily use of cigarettes and use of marijuana/hashish in last year</td>
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- Maternity University Study of Pregnancy, which recruited 7223 women and their baby born between 1981 and 1984, in Brisbane, Australia. 3801 children remained in sample at 21 year follow up.  
- Victoria Adolescent Health Cohort Study (VAHCS) of 2032 students recruited at year 9, born in 1977-1978. Self reported cannabis use. Self reported educational attainment. |
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<td>Kuepper, R., van Os, J., Lieb, R., Wittchen, H. U. &amp; Hanquet, C. (2011)</td>
<td>Do cannabis and urbanicity coparticipate in causing psychosis? Evidence from a 10 year follow up cohort study.</td>
<td>Psychological Medicine, 41, 2121-2129</td>
<td>10 year follow up of Early Developmental Stages of Psychopathology (EDSP) study of German adolescent and young adults aged between 14 and 24 years at baseline in 1995. 1923 respondents included at 10 year follow up. Self reported cannabis use reported at 3 times points in the 10 year period. Self reported psychotic symptoms collected via Munich version of Composite International Diagnostic Interview (CIDI) at 3 times points in the 10 year period.</td>
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Analysis from New Zealand Birth Cohort started 1972/1973 – Dunedin Multidisciplinary Health and Development Study; small sample size of 759 children (with full data) which may limit analysis

Self reported cannabis use analysed for frequency and dependence

Diagnostic interview for range of mental health problems

A sibling pair analysis nested within the Maternity University Study of Pregnancy, which recruited 7223 women and their baby born between 1981 and 1984, in Brisbane, Australia. 3801 children remained in sample at 21 year follow up.

Self reported retrospective cannabis use collected at 21 year follow up.

Self reported psychosis related outcomes (nonaffective psychosis, hallucinations and Delusions Inventory score)

A review of 10 studies using 7 general population cohorts looking at the evidence for a causal link between cannabis and psychosis.

Sample of 32; 16 marijuana users and 16 matched controls.

MRI scanning to measure white matter volume

Self reported substance use via Customary Drinking and Drug Use Record (CDDR), later used to calculate dependence using DSM-IV criteria

Self reported psychosocial functioning using Structured Clinical Interview (SCI) and Computerised NIMH Diagnostic Interview Schedule for children (C-DISC-4.0)
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Self reported thought problems taken from Youth Self Report (YSR) and Child Behavior Checklist (CBCL)

Self reported cannabis use |