Appendix 5: Near Misses

Author (year)	Study description	Summary of findings	Reason excluded
Psychotic outcomes			
Clough (2006)[1]	Adult population based sample, Northern Territory, Australia (*n* = 139)	There were increased reports in health centre records of auditory hallucinations and self-harm attempts in subjects who had used cannabis at both baseline and 3-year follow up compared to those who had never used cannabis at either time point.	No baseline screening for psychotic or affective pathology so cannabis use may have been secondary to these outcomes. Use of medical records for outcome measures introduces large potential for bias
Ferdinand (2005)[2]	Population based cohort study, Netherlands (*n* = 1580)	Cross-sectional study where authors specified a 2-year interval between reported onset of cannabis use and onset of psychosis. Cannabis was associated with increased risk of psychosis that was reduced by 75% (though remained significant) when the 2-year interval enforced. Psychosis was also associated with an increase in cannabis use.	Cross-sectional data, though attempts made to reduce reverse causation effects
Miller (2006)[3]	Edinburgh High Risk Study (*n* = 163)	High-risk subjects had two or more first or second degree relatives with schizophrenia. Subjects who used cannabis frequently had an increased risk of developing schizophrenia. This was substantially attenuated, though remained evident, after adjusting for childhood behavioural measures.	Not population-based sample of 'normal' or 'healthy' individuals. Previous publication from this study indicates that several subjects already had psychotic symptoms at baseline.
Phillips (2002)[4]	Ultra high-risk sample, Australia (*n* = 100)	Cannabis use was not associated with an increase in risk of developing a psychosis outcome over a 1-year follow-up of ultra high-risk subjects	Sample already had psychosis at baseline as subjects with a psychotic illness lasting less than 1 week were included within the high-risk group (outcome was psychotic illness lasting > 1week)
Verdoux (2003)[5]	Experience Sampling Method of students (*n* = 79)	Cannabis use was not associated with a subsequent increase in psychotic-like experiences, though results were adjusted for an end of study measure of psychosis that included symptoms over the course of the study.	Psychosis outcome was measured during the next sample on the same day and the study was therefore almost certainly measuring intoxication effects
Weisser (2003)[6]	Israeli conscript cohort (*n* = 50,413)	Drug use was associated with an increased risk of developing schizophrenia (RR = 2, 95% CI 1.3 to 3.1). Affective disorder admissions were also examined, though these are likely to be highly unrepresentative of affective outcomes (number of cases = 41; RR = 0.75, 95% CI 0.18 to 3.1)	No data on cannabis use specifically but only all drug use, though most of this was likely to have been cannabis

Appendix 5: Near Misses

Author (year)	Study description	Summary of findings	Reason excluded
Affective outcomes			
Angst (1996)[7]	Adult population based sample, Zurich, Switzerland (*n* = 591)	Cannabis use was reported as being associated with depression (OR = 2.3, 95% CI 1.4 to 3.8)	Analysis uses cross-sectional data
Tubman (1990)[8]	New York longitudinal study of children (*n* = 133)	Cannabis use during childhood and young adulthood was not associated with depression or anxiety in young adulthood	Analysis uses cross-sectional data
Repetto (2003)[9]	High-school adolescents, USA (*n* = 579)	Change in cannabis use was not associated with depressive symptoms	Not possible to extract data of use Analysis uses cross-sectional data

RR = risk ratio; OR = odds ratio; 95% CI = 95% confidence intervals

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