



**Cochrane**  
**Library**

**Cochrane** Database of Systematic Reviews

## **Family-based prevention programs for alcohol use in young people (Protocol)**

Gilligan C, Wolfenden L, Foxcroft DR, Kingsland M, Williams AJ, Hodder RK, Small T, Sherker S, Rae J, Tindall J, Stockings E, Wiggers J

Gilligan C, Wolfenden L, Foxcroft DR, Kingsland M, Williams AJ, Hodder RK, Small T, Sherker S, Rae J, Tindall J, Stockings E, Wiggers J.

Family-based prevention programs for alcohol use in young people.

*Cochrane Database of Systematic Reviews* 2016, Issue 8. Art. No.: CD012287.

DOI: 10.1002/14651858.CD012287.

[www.cochranelibrary.com](http://www.cochranelibrary.com)

## TABLE OF CONTENTS

HEADER . . . . .	1
ABSTRACT . . . . .	1
BACKGROUND . . . . .	1
OBJECTIVES . . . . .	3
METHODS . . . . .	3
ACKNOWLEDGEMENTS . . . . .	7
REFERENCES . . . . .	7
APPENDICES . . . . .	9
CONTRIBUTIONS OF AUTHORS . . . . .	13
DECLARATIONS OF INTEREST . . . . .	13
SOURCES OF SUPPORT . . . . .	14

# Family-based prevention programs for alcohol use in young people

Conor Gilligan<sup>1</sup>, Luke Wolfenden<sup>1</sup>, David R Foxcroft<sup>2</sup>, Melanie Kingsland<sup>1</sup>, Amanda J Williams<sup>3</sup>, Rebecca K Hodder<sup>3</sup>, Tameka Small<sup>1</sup>, Shauna Sherker<sup>4</sup>, Julie Rae<sup>4</sup>, Jenny Tindall<sup>3</sup>, Emily Stockings<sup>5</sup>, John Wiggers<sup>3</sup>

<sup>1</sup>School of Medicine and Public Health, University of Newcastle, Callaghan, Australia. <sup>2</sup>Department of Psychology, Social Work and Public Health, Oxford Brookes University, Oxford, UK. <sup>3</sup>Hunter New England Population Health, Hunter New England Local Health District, Wallsend, Australia. <sup>4</sup>Program Development and Evaluation, Alcohol and Drug Foundation, Melbourne, Australia. <sup>5</sup>National Drug and Alcohol Research Centre (NDARC), UNSW, Australia, Randwick, Australia

Contact address: Conor Gilligan, School of Medicine and Public Health, University of Newcastle, Callaghan, NSW, 2308, Australia. [Conor.Gilligan@newcastle.edu.au](mailto:Conor.Gilligan@newcastle.edu.au).

**Editorial group:** Cochrane Drugs and Alcohol Group.

**Publication status and date:** New, published in Issue 8, 2016.

**Citation:** Gilligan C, Wolfenden L, Foxcroft DR, Kingsland M, Williams AJ, Hodder RK, Small T, Sherker S, Rae J, Tindall J, Stockings E, Wiggers J. Family-based prevention programs for alcohol use in young people. *Cochrane Database of Systematic Reviews* 2016, Issue 8. Art. No.: CD012287. DOI: 10.1002/14651858.CD012287.

Copyright © 2016 The Cochrane Collaboration. Published by John Wiley & Sons, Ltd.

## ABSTRACT

This is the protocol for a review and there is no abstract. The objectives are as follows:

To assess the effectiveness of universal, selective and indicated family-based prevention programs in preventing alcohol use, or problem drinking, in school-aged children (up to 18 years of age). Specifically, on these outcomes, the review aims:

1. To assess the effectiveness of universal family-based prevention programs for all children up to 18 years ('universal interventions').
2. To assess the effectiveness of selective family-based prevention programs for children up to 18 years at elevated risk of alcohol use or problem drinking ('selective interventions').
3. To assess the effectiveness of indicated family-based prevention programs for children up to 18 years currently consuming alcohol ('indicated interventions').

## BACKGROUND

### Description of the condition

Alcohol use ranks among the top three risk factors for the global burden of disease, accounting for 5.5% of Disability Adjusted Life Years (DALYs) globally (Lim 2012). A causal relationship has been

established between alcohol and more than 200 chronic and acute diseases, as well as intentional and unintentional injuries (Rehm 2010). Overall, in 2010 alcohol-attributable injuries were responsible for 13.2% of all injury deaths and 12.6% of all injury Potential Years of Life Lost (PYLL) (Rehm 2013). Young people contribute a high proportion of alcohol-related injuries and mortality from alcohol-attributable injury, with 1 in 4 deaths among men

aged 15 to 29 years, and 1 in 10 deaths among women in the European Union, being alcohol-related (Rehm 2005). In the European Union, road traffic accidents are the leading cause of death in children and young adults up to 29 years, and 33% of motor vehicle traffic injuries to males and 11% to females are due to alcohol (WHO 2012). Extensive evidence points to an association between early age of alcohol use (and early intoxication) and an increased frequency of drinking, as well as increased risky drinking and alcohol-related harms later in adolescence and during adulthood (for example: Bonomo 2004; DeWit 2000; Jackson 2015; Kuntsche 2013).

Experimentation with risky behaviours typically begins in adolescence, as part of a natural 'coming of age' process (Room 2004). A dramatic increase is seen in the use of alcohol, tobacco and other substances after the age of 12, with rates gradually increasing throughout adolescence (Currie 2012). This pattern is common globally, with reports from 43 countries included in the Health Behaviour in School-Aged Children Project (Currie 2012) and the European Survey Project on Alcohol and Drugs (ESPAD; (Hibell 2012)) consistent with national surveys conducted in Australia (White 2012) and the United States (US) (Frieden 2014). Any level of alcohol use is potentially harmful for young people, with evidence of an effect upon the developing brain (Bava 2010). Early sipping of alcohol has been associated with increased odds of consuming full drinks, getting drunk, and drinking heavily later in adolescence (Jackson 2015). Even a single occasion of alcohol intoxication can have serious short- and long-term consequences (Courtney 2009; Quinn 2011).

While the use of alcohol is common among young people, some groups can be identified as being at elevated risk of heavy use due to a range of social, peer, and family factors. Livingston and colleagues report that young people who have had their first drink by age 13 are almost twice as likely to engage in very high risk drinking when aged 16 to 24 (Livingston 2008). Parents who allow their children to consume alcohol in adult-supervised settings in early adolescence are more likely to have children who experience harmful alcohol consequences in mid-adolescence (McMorris 2011). Further, parents who themselves have heavy drinking occasions are more likely to have children who report heavy drinking occasions (Hingson 2014), and parental substance use and family history of alcoholism have been identified as predictors of adolescent substance use in longitudinal studies (Chassin 1996; Cranford 2010; White 2000; Wills 2003). Evidence is mixed in relation to the association between socioeconomic disadvantage and risk of adolescent alcohol consumption (Hanson 2007). Some reports show drinking and drunkenness associated with lower levels of disadvantage or higher levels of household income (Reboussin 2010; Richter 2009). Other reports show higher levels of baseline problem drinking among low socioeconomic status communities (Caria 2011; Lowry 1996).

## Description of the intervention

Despite the increasing influence of peers and society during adolescence (Carter 2007; Patton 2004), parenting and home environment factors remain important influencers of development (Steinberg 2001) and predictors of alcohol consumption and other substance use (Carter 2007; Simons-Morton 2009; Turrisi 2010; Wang 2009). Both maternal and paternal knowledge of their child's friends and whereabouts are reported to act as protective factors against substance use and to mediate the variability in substance use by grade and ethnic background (Wang 2009). This protective effect is suggested to act via an influence on peer group selection (Engels 2007; Wang 2009), the transmission of family attitudes and values (White 2010), and parental monitoring (knowledge of their child's whereabouts) (Jimenez-Iglesias 2013). In 1994, the US Institutes of Medicine adopted a framework for the classification of mental health and substance use prevention interventions as universal, selective, or indicated/targeted (Mrazek 1994; Springer 2006). Universal prevention strategies address the entire population, within a particular setting. Selective interventions are delivered to subgroups of individuals based on their membership of a group that has an elevated risk of developing problems. Indicated interventions address vulnerable individuals and help them in dealing and coping with their individual personality traits that make them more vulnerable to escalating drug use (EMCDDA 2015).

While intervention programs are usually classified as belonging to one of these three broad groups, the classification can be regarded as a continuum, with obvious overlap between groups. In the 2010 report 'Fair Society, Healthy Lives', commissioned by the United Kingdom (UK) Government to identify the most evidence-based strategies for reducing health inequalities, a key recommendation was to extend the focus of preventive activities beyond the most disadvantaged, to encompass the full spectrum of the social gradient. It was stated that to "reduce the steepness of the social gradient in health, actions must be universal, but with a scale and intensity that is proportionate to the level of disadvantage" (Marmot 2010). Applied to alcohol prevention efforts, this 'proportionate universalism' can be interpreted as the need to conduct universal prevention programs, but to also include more targeted (selective and indicated) interventions for higher risk groups. Parenting skills are recognised as a key factor in the prevention of adolescent alcohol consumption and other substance use. The proportionate universalism approach maintains that all parents should be given opportunities for support and help to develop appropriate protective parenting skills, and that some parents who demonstrate a particular risk profile or who have particular needs (vulnerable children) should be offered increasingly targeted (and increasingly costly) interventions (Heginbotham 2012; Marmot 2010). For this reason, this review will not be limited to universal interventions, but will incorporate those classified as selective and indicated.

Classification of interventions in the present review will be based on their target population; being all parents or a select group based

on characteristics of the parents or their children. In the context of family-based interventions for alcohol use in young people, universal interventions target parents of all children given the inherent risk of alcohol use among all sectors of the population. These interventions will likely aim to delay the initiation of alcohol use, or reduce the frequency or volume of use among children of participating parents. Selective interventions are those targeting parents whose children have an elevated risk of substance use due to social or family risk factors. Such risk factors include low socioeconomic status or family income, and parental alcohol consumption, alcoholism or other substance use. Similarly, these interventions will likely aim to delay initiation or reduce consumption. Indicated interventions are defined as those that target parents or families whose children are already identified as drinkers. These interventions will more likely aim to reduce levels of consumption or the frequency of binge drinking and/or reduce alcohol-related harms. Parent- and family-based programs for the prevention of alcohol use are often appended to school curricula-based interventions for young people, but may also be stand-alone programs. Such programs frequently focus on parent-child communication and relationship-building. Common elements across many programs include a focus on social competence skills, parental involvement with children, and self-regulation, although the target population, intensity and mode of delivery are highly varied.

### How the intervention might work

The theoretical basis for family-based interventions is that young people whose parents adopt appropriate parenting strategies are likely to develop positive social norms and to resist the negative external influences of peers and society. In this context, positive parenting strategies include rule setting, appropriate communication, monitoring, and conveying positive values and attitudes (Ryan 2010). Family- and parent-based interventions for adolescent substance use operate indirectly, with the mechanism of effect working via parents rather than through a program delivered directly to young people as the target population.

### Why it is important to do this review

Previous Cochrane reviews have covered universal family-based (Foxcroft 2011a), as well as school-based (Foxcroft 2011b) and multi-component (Foxcroft 2011c) interventions for alcohol misuse in young people. The most recent of these reviews was completed with studies published up to July 2010. In the time since that review, several trials have been published, reporting on other family-based preventive programs, and in many cases using innovative approaches including online delivery.

As well as updating the previous review (Foxcroft 2011a), the current review will extend beyond universal interventions to include

those classified as selective and indicated, in keeping with the concept of proportionate universalism.

While parents and families are influential and a key target for intervention, family-based programs are often expensive to run and challenging from a recruitment and engagement perspective. It is important to gather evidence of their effectiveness, and of the differential effectiveness of various components of these programs, to inform policy and funding decisions.

## OBJECTIVES

To assess the effectiveness of universal, selective and indicated family-based prevention programs in preventing alcohol use, or problem drinking, in school-aged children (up to 18 years of age). Specifically, on these outcomes, the review aims:

1. To assess the effectiveness of universal family-based prevention programs for all children up to 18 years ('universal interventions').
2. To assess the effectiveness of selective family-based prevention programs for children up to 18 years at elevated risk of alcohol use or problem drinking ('selective interventions').
3. To assess the effectiveness of indicated family-based prevention programs for children up to 18 years currently consuming alcohol ('indicated interventions').

## METHODS

### Criteria for considering studies for this review

#### Types of studies

Randomised controlled trials (individual or cluster design).

#### Types of participants

Parents or guardians/carers of young people up to 18 years (of school age). For this review, young people are defined as children and adolescents. Parents of young people who have not previously consumed alcohol, currently consume alcohol or those with heavy or problematic use will be included.

## Types of interventions

Any universal, selective or indicated family-based psychosocial or educational prevention intervention.

Universal prevention strategies are defined as those addressing the entire population without selection of children based on characteristics that may increase their risk of alcohol use or problem drinking; for example, those offered to all parents of children attending a school.

Selective interventions are defined as those delivered to a subgroup of children identified as having socio-demographic characteristics that put them at an elevated risk of alcohol use or problem drinking, for example those delivered to families in which there is a history of substance use among parents, or those living in communities of low socioeconomic status.

Indicated interventions are defined as those targeting a subgroup of children who currently use alcohol and may have alcohol-related problems, or are at elevated risk of heavy drinking and alcohol-related problems.

Psychosocial intervention is defined as one that specifically aims to develop psychological and social attributes and skills in parents and young people (e.g., parental monitoring, behavioural norms, peer resistance) so that young people are less likely to use alcohol. Educational interventions are defined as those that specifically aim to raise awareness amongst parents and/or carers of how to positively influence young people, or of the risks of alcohol consumption, so that young people are less likely to use alcohol.

Comparison: Any alternative prevention program (e.g., school-based, office-based, multi-component, other) or no program.

## Types of outcome measures

### Primary outcomes

Any direct self-reported (by adolescents) measures of alcohol consumption or problem drinking. Prevention programs that focus on alcohol as well as other drugs will be included wherever alcohol outcomes are presented separately. Outcome measures related to psychological perception/attitudes or awareness are deemed to be indirect and therefore are not considered in this review. As an example, the following outcomes are considered to be relevant:

1. Alcohol use (yes/no)
2. Alcohol use (quantity, frequency)
3. 'Binge' drinking, e.g. defined as drinking five or more drinks on any one occasion (yes/no)
4. Incidence of drunkenness

### Secondary outcomes

1. Alcohol initiation (age)
2. Drunkenness initiation (age)
3. Alcohol-related problems (e.g. drink-driving or any physical or social problem self-reported by adolescents as an alcohol-

related consequence; may be measured using scale such as Rutgers Alcohol Problems Index or questions 7 to 10 of the AUDIT; Alcohol Use Disorders Identification Test).

4. Self-reported (by parents and/or children) alcohol-related parenting behaviours (e.g. supply of alcohol, alcohol-specific communication, alcohol-specific rule-setting)

## Search methods for identification of studies

### Electronic searches

We will search the following databases, without restrictions by language or publication status:

- the Cochrane Drugs and Alcohol Group's Specialised Register of Trials;
- the Cochrane Central Register of Controlled Trials (CENTRAL, most recent issue)
- MEDLINE (Ovid) (1966 to present);
- EMBASE (EMBASE.com) (1974 to present);
- ERIC (EBSCOhost) (1966 to present);
- PsycINFO (Ovid) (1806 to present);
- Google Scholar;
- Project CORK (<http://www.projectcork.org>);
- ClinicalTrials.gov ([clinicaltrials.gov](http://clinicaltrials.gov));
- ICTRP ([apps.who.int/trialsearch/](http://apps.who.int/trialsearch/)).

The subject strategies for databases will be modelled on the search strategy designed for CENTRAL (Appendix 1). Where appropriate, these will be combined with subject strategy adaptations of the Cochrane highly sensitive search strategy for identifying randomised controlled trials and controlled clinical trials (as described in the Cochrane Handbook for Systematic Reviews of Interventions, Version 5.1.0, Box 6.4.b. (Higgins 2011)).

### Searching other resources

The references of topic-related systematic reviews and included studies will be handsearched in order to identify potentially-relevant citations. Unpublished reports, abstracts, dissertations, brief and preliminary reports are eligible for inclusion.

## Data collection and analysis

### Selection of studies

Two independent review authors will complete broad screening of titles and abstracts of all identified records (screening level 1). Afterwards, the same two authors will independently assess full-text reports of all potentially-relevant records that pass the initial

screening level. Differences in opinion arising at both screening levels will be resolved through discussion.

### Data extraction and management

Two review authors will extract relevant data independently using an a priori defined data extraction form, and will enter data into Review Manager 5 software ([RevMan 2014](#)). Differences in opinion arising during data extraction will be resolved through discussion. We will extract the following information: number and characteristics of participants, setting, type of experimental and control intervention, length of follow-up, types of outcomes, country of origin.

### Assessment of risk of bias in included studies

For each study included in the review, two authors will independently assess the risk of bias. The risk of bias assessment for RCTs in this review will be performed using the criteria recommended in the Cochrane Handbook ([Higgins 2011](#)). The recommended approach uses a two-part tool, addressing seven specific domains, namely sequence generation and allocation concealment (selection bias), blinding of participants and providers (performance bias), blinding of outcome assessor (detection bias), incomplete outcome data (attrition bias), selective outcome reporting (reporting bias) and other sources of bias. The first part of the tool involves describing what was reported to have happened in the study. The second part of the tool involves assigning a judgement relating to the risk of bias for that entry, in terms of low, high or unclear risk. To make these judgements we will use the criteria indicated by the Handbook adapted to the addiction field. See [Appendix 2](#) for details.

The domains of sequence generation and allocation concealment (avoidance of selection bias) will be addressed in the tool by a single entry for each study.

Blinding of participants, personnel and outcome assessor (avoidance of performance bias and detection bias) will be considered separately for objective outcomes (e.g. drop out) and subjective outcomes (e.g. parent- or adolescent-reported use of alcohol (quantity, frequency, bingeing, drunkenness), self-reported alcohol-related harm, parent- or adolescent-reported parenting behaviours). Blinding of participants and program deliverers is not achievable for these sort of interventions, so all of the studies will be rated as at high risk of performance bias for subjective outcomes. Incomplete outcome data (avoidance of attrition bias) will be considered for all outcomes.

### Grading of evidence

We will assess the overall quality of the evidence for the primary outcome of each study using the GRADE system. The Grading of Recommendation, Assessment, Development and Evaluation Working Group (GRADE) developed a system for grading the quality of evidence ([GRADE 2004](#); [Guyatt 2008](#); [Guyatt 2011](#))

which takes into account issues not only related to internal validity but also to external validity, such as directness, consistency, imprecision of results and publication bias. The 'Summary of findings' tables present the main findings of a review in a transparent and simple tabular format. In particular, they provide key information concerning the quality of evidence, the magnitude of effect of the interventions examined and the sum of available data on the main outcomes.

In this review, we will present 'Summary of findings' tables based on primary outcomes (alcohol use, binge drinking and incidence of drunkenness)

The GRADE system uses the following criteria for assigning grades of evidence:

- High: We are very confident that the true effect lies close to that of the estimate of the effect.
- Moderate: We are moderately confident in the effect estimate: The true effect is likely to be close to the estimate of the effect, but there is a possibility that it is substantially different.
- Low: Our confidence in the effect estimate is limited: The true effect may be substantially different from the estimate of the effect.
- Very low: We have very little confidence in the effect estimate: The true effect is likely to be substantially different from the estimate of effect.

Grading is decreased for the following reasons: Serious (-1) or very serious (-2) limitation to study quality. Important inconsistency (-1). Some (-1) or major (-2) uncertainty about directness. Imprecise or sparse data (-1). High probability of reporting bias (-1).

Grading is increased for the following reasons:

- Strong evidence of association - significant relative risk of  $> 2$  ( $< 0.5$ ) based on consistent evidence from two or more observational studies, with no plausible confounders (+1).
- Very strong evidence of association - significant relative risk of  $> 5$  ( $< 0.2$ ) based on direct evidence with no major threats to validity (+2).
- Evidence of a dose response gradient (+1).
- All plausible confounders would have reduced the effect (+1).

### Measures of treatment effect

We will calculate unadjusted treatment effects using [RevMan 2014](#) where possible.

### Dichotomous outcome data

Dichotomous outcomes will be analysed by calculating the relative risk (RR) for each trial, with the uncertainty in each result expressed using 95% confidence intervals (CIs).



### Continuous outcome data

Continuous outcomes will be analysed by calculating mean differences (MDs) if all studies use the same measurement scale, or standardised mean differences (SMDs) if studies use different measurement scales, each with 95% CIs. If data in small studies are skewed, the implications for outcomes will be assessed on a case by case basis.

### Unit of analysis issues

Additional validity threats will be ascertained regarding appropriate unit of analysis depending on whether the randomisation was implemented at individual or cluster level. Cluster randomised trials are possible in this area of research, as allocation to the intervention group may occur by school or community. Such designs are susceptible to unit of analysis error and P values may be artificially small (Higgins 2011). We anticipate that investigators will have controlled for clustering when presenting their results. Where the clustering effect has not been controlled for, we will contact study authors and request participant data to calculate an estimate of the intracluster correlation coefficient (ICC). If participant data are not available, we will search for external estimates of the ICC from similar studies and from Cochrane resources.

### Dealing with missing data

If important summary data or study level characteristics are missing, we will attempt to contact the authors of included studies. If standard deviations are missing from continuous data, we will scan studies for any other statistics (CIs, standard errors, T values, P values, F values) that allow for their calculation. We will describe missing data and all forms of attrition for each included study in the 'Risk of bias' table, and discuss the extent to which missing data could impact on the conclusions of the review. Missing data will be treated according to whether data are 'missing at random' or 'not missing at random'. In relation to the former, the main option will be to analyse the available data and ignore the missing data.

For data that are not missing at random (e.g. participants who do not experience positive outcomes failing to complete follow-up assessments), imputation will be used to generate replacement values. When imputing missing dichotomous data, we will assume that missing data are negative (e.g. the participant demonstrated high-risk behaviour). When imputing missing continuous data, we will use a 'last observation carried forward' approach. Some relevant studies may fail to provide summary data (e.g. standard deviations). Where this occurs we will, if possible, obtain these data using calculations outlined in the Cochrane Handbook for Systematic Reviews of Interventions (Higgins 2011).

### Assessment of heterogeneity

Assessment of heterogeneity will involve inspecting each included study for variability in the study populations (baseline characteristics), interventions (target/focus, mode of delivery), and outcome measures (tools, instruments, scales and outcome definitions). Methodological heterogeneity will also be considered by inspecting variability in study design and risk of bias. If sufficient homogeneity is found within subgroups (based on age of children, type of intervention or substance targeted), meta-analysis will be considered for subgroups of studies. If any unexpected variability arises, we will discuss this in full in the review. We will assess statistical heterogeneity using the  $\chi^2$  test and its P value, by visual inspection of the forest plots and the  $I^2$  statistic. A P value of the test lower than 0.10 or an  $I^2$  statistic of at least 50% will indicate significant statistical heterogeneity.

### Assessment of reporting biases

We will use funnel plots (plots of the effect estimate from each study against the sample size or effect standard error) to indicate possible publication bias. Asymmetry in the plot could be due to publication bias, but may also reflect a real relationship between trial size and effect. We will use tests for funnel plot asymmetry only when a minimum of 10 studies are included in the meta-analysis, as fewer than 10 studies would render the power of the tests too low to distinguish chance from real asymmetry.

### Data synthesis

The outcome measures from the individual trials will be pooled through meta-analysis where possible. We plan to synthesise results from studies where the interventions are similar with regard to study populations (e.g., baseline characteristics, gender), interventions (e.g., type, differences in target/focus, universal/targeted), target groups (age of children, well/at risk groups) and outcome measures (e.g., different tools, instruments, scales, alcohol/other drugs) as well as the methodology of conduct (e.g., units of randomisation and analysis, cluster versus individual trials). We will perform a random-effects meta-analysis using an inverse variance weighting method using RevMan 2014 as we expect a certain level of heterogeneity among the included studies. If some primary studies report an outcome as a dichotomous measure and others use a continuous measure of the same construct, we will convert results for the former from an odds ratio to an SMD, provided that we can assume that the underlying continuous measure has approximately a normal or logistic distribution (otherwise we will carry out two separate analyses). If meta-analysis is not appropriate, we will report results from individual studies.

### Subgroup analysis and investigation of heterogeneity

The extent of heterogeneity will be investigated through examination of forest plots ( $\chi^2$  statistic and P value;  $I^2$  statistic).



Where there is evidence of heterogeneity ( $I^2$  statistic > 50%), the source of heterogeneity will be investigated through subgroup analyses. Specifically, subgroup analyses will be conducted based on the characteristics of participants, interventions, and comparison groups of included studies.

### Sensitivity analysis

We will perform sensitivity analysis of the main review outcomes,

removing trials judged to be at high risk of bias (graded as high on three or more 'Risk of bias' measures).

## ACKNOWLEDGEMENTS

We acknowledge the work of previous Cochrane review authors, whose efforts have contributed to the present protocol and review; in particular, David Foxcroft and Alexander Tsertsvadze.

## REFERENCES

### Additional references

#### Bava 2010

Bava S, Tapert SF. Adolescent brain development and the risk for alcohol and other drug problems. *Neuropsychological Reviews* 2010;**20**:398–413.

#### Bonomo 2004

Bonomo YA, Bowes G, Coffey C, Carlin JB, Patton GC. Teenage drinking and the onset of alcohol dependence: a cohort study over seven years. *Addiction* 2004;**99**:1520–8.

#### Caria 2011

Caria MP, Faggiano F, Bellocco R, Galanti R. The influence of socioeconomic environment on the effectiveness of alcohol prevention among European students: a cluster randomised controlled trial. *BMC Public Health* 2011;**11** (312). [DOI: 10.1186/1471-2458-11-312]

#### Carter 2007

Carter M, McGee R, Taylor B, Williams S. Health outcomes in adolescence: associations with family, friends and school engagement. *Journal of Adolescence* 2007;**30**:51–62.

#### Chassin 1996

Chassin L, Curran PJ, Hussong AM, Colder CR. The relation of parent alcoholism to adolescent substance use: A longitudinal follow-up study. *Journal of Abnormal Psychology* 1996;**105**:70–80.

#### Courtney 2009

Courtney KE, Polich J. Binge drinking in young adults: Data, definitions, and determinants. *Psychological Bulletin* 2009;**135**:142–56.

#### Cranford 2010

Cranford JA, Zucker RA, Jester JM, Puttler LI, Fitzgerald HE. Parental alcohol involvement and adolescent alcohol expectancies predict alcohol involvement in male adolescents. *Psychology of Addictive Behaviours* 2010;**24**(3): 386–96.

#### Currie 2012

Currie C, Zanotti C, Morgan A, Currie D, de Looze M, Roberts C, et al. *Social determinants of health and well-being among young people. Health Behaviour in School-aged Children (HBSC) study: international report from the 2009/*

*2010 survey, Health Policy for Children and Adolescents, No. 6.* Copenhagen: WHO Regional Office for Europe, 2012.

#### DeWit 2000

DeWit DJ, Adlaf EM, Offord DR, Ogborne AC. Age at first alcohol use: a risk factor for the development of alcohol disorders. *American Journal of Psychiatry* 2000;**157**:745–50.

#### EMCDDA 2015

EMCDDA. Prevention of Drug Use. European Monitoring Centre for Drugs and Drug Addiction, 2015.

#### Engels 2007

Engels RC, De Leeuw RNH, Poelen EP. The impact of parents on adolescent drinking and friendship selection process. In: Jarvinen M, Room R editor(s). *Youth Drinking Cultures: European Experiences.* Hampshire, England: Ashgate Publishing, 2007.

#### Foxcroft 2011b

Foxcroft DR, Tsertsvadze A. Universal school-based prevention programs for alcohol misuse in young people. *Cochrane Database of Systematic Reviews* 2011, Issue 5. [DOI: 10.1002/14651858.CD009113]

#### Foxcroft 2011c

Foxcroft DR, Tsertsvadze A. Universal multi-component prevention programs for alcohol misuse in young people. *Cochrane Database of Systematic Reviews* 2011, Issue 9. [DOI: 10.1002/14651858.CD009307]

#### Frieden 2014

Frieden T, Jaffe HW, Cono J, Richards C, Iademarco MF. Youth Risk Behaviour Surveillance - United States 2013. Surveillance Summaries. Atlanta, GA: Centers for Disease Control and Prevention, US Department of Human Services, 2014; Vol. 63:1–168.

#### GRADE 2004

The GRADE working group. Grading quality of evidence and strength of recommendations. *BMJ* 2004;**328**(7454): 1490.

#### Guyatt 2008

Guyatt GH, Oxman AD, Vist GE, Kunz R, Falck-Ytter Y, Alonso-Coello P, et al. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. *BMJ* 2008;**336**(7560):924–6.

**Guyatt 2011**

Guyatt G, Oxman AD, Akl EA, Kunz R, Vist G, Brozek J, et al. GRADE guidelines 1. Introduction-GRADE evidence profiles and summary of findings tables. *Journal of Clinical Epidemiology* 2011;**64**:383–94.

**Hanson 2007**

Hanson MD, Chen E. Socioeconomic status and health behaviours in adolescence: a review of the literature. *Journal of Behavioural Medicine* 2007;**30**:263–85.

**Heginbotham 2012**

Heginbotham C. *Values-Based Commissioning of Health and Social Care*. Cambridge, UK: Cambridge University Press, 2012.

**Hibell 2012**

Hibell B, Ulf Guttormsson U, Ahlström S, Balakireva O, Bjarnason T, Kokkevi A, et al. The 2011 ESPAD Report: Substance Use Among Students in 36 European Countries. <http://www.espad.org/en/references--literature/the-2011-espad-report---substal/>. Stockholm: The Swedish Council for Information on Alcohol and other Drugs (CAN), The European Monitoring Centre for Drugs and Drug Addiction (EMCDDA), Council of Europe, Co-operation Group to Combat Drug Abuse and Illicit Trafficking in Drugs (Pompidou Group), 2012.

**Higgins 2011**

Higgins JPT, Green S (editors). *Cochrane Handbook for Systematic Reviews of Interventions* Version 5.1.0. The Cochrane Collaboration 2011:Available from [www.cochrane-handbook.org](http://www.cochrane-handbook.org).

**Hingson 2014**

Hingson R, White A. New research findings since the 2007 Surgeon General's Call to Action to Prevent and Reduce Underage Drinking: a review. *Journal of Studies on Alcohol and Drugs* 2014;**75**(1):158–69.

**Jackson 2015**

Jackson KM, Barnett NP, Colby SM, Rogers ML. The prospective association between sipping alcohol by the sixth grade and later substance use. *Journal of Studies on Alcohol and Drugs* 2015;**76**:212–21.

**Jimenez-Iglesias 2013**

Jimenez-Iglesias A, Moreno C, Rivera F, García-Moya I. The role of the family in promoting responsible substance use in adolescence. *Journal of Child and Family Studies* 2013;**22**:585–602.

**Kuntsche 2013**

Kuntsche E, Rossow I, Simons-Morton B, Bogt TT, Kokkevi A, Godeau E. Not early drinking but early drunkenness is a risk factor for problem behaviors among adolescents from 38 European and North American countries. *Alcoholism, Clinical and Experimental Research* 2013;**37**:308–14.

**Lim 2012**

Lim SS. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for

the Global Burden of Disease Study 2010. *Lancet* 2012;**380**(9859):2224–60.

**Livingston 2008**

Livingston M, Laslett AM, Dietze P. Individual and community correlates of young people's high-risk drinking in Victoria, Australia. *Drug and Alcohol Dependence* 2008;**98**(3):241–8.

**Lowry 1996**

Lowry R, Kann L, Collins JL, Kolbe LJ. The effect of socioeconomic status on chronic disease risk behaviours among US adolescents. *JAMA* 1996;**276**:792–7.

**Marmot 2010**

Marmot M. Fair Society, Healthy Lives. The Marmot Review. [www.ucl.ac.uk/marmotreview](http://www.ucl.ac.uk/marmotreview), 2010.

**McMorris 2011**

McMorris BJ, Catalano RF, Kim MJ, Toumbourou JW, Hemphill SA. Influence of family factors and supervised alcohol use on adolescent alcohol use and harms: similarities between youth in different alcohol policy contexts. *Journal of Studies on Alcohol and Drugs* 2011;**72**(3):418–28.

**Mrazek 1994**

Mrazek PJ, Haggerty RJ (editors). *Reducing Risks for Mental Disorders: Frontiers for Preventive Intervention Research*. Washington DC: Institute of Medicine, 1994.

**Patton 2004**

Patton GC, McMorris BJ, Toumbourou JW, Hemphill SA, Donath S, Catalano RF. Puberty and the onset of substance use and abuse. *Pediatrics* 2004;**114**:e300–6.

**Quinn 2011**

Quinn PD, Fromme K. Predictors and outcomes of variability in subjective alcohol intoxication among college students: an event-level analysis across 4 years. *Alcoholism, Clinical and Experimental Research* 2011;**35**(3):484–95.

**Reboussin 2010**

Reboussin BA, Preisser JS, Song EY, Wolfson M. Geographic clustering of underage drinking and the influence of community characteristics. *Drug and Alcohol Dependence* 2010;**106**(1):38–47.

**Rehm 2005**

Rehm J, Room R, van den Brink W, Jacobi F. Alcohol use disorders in EU countries and Norway: an overview of the epidemiology. *European Neuropsychopharmacology* 2005;**15**(4):377–88.

**Rehm 2010**

Rehm J, Baliunas D, Borges GL, Graham K, Irving H, Kehoe T, et al. The relation between different dimensions of alcohol consumption and burden of disease: an overview. *Addiction* 2010;**105**(5):817–43.

**Rehm 2013**

Rehm J, Shield KD. Global alcohol-attributable deaths from cancer, liver cirrhosis, and injury in 2010. *Alcohol Research: Current Reviews* 2013;**35**(2):174–83.

**RevMan 2014 [Computer program]**

Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration. Review Manager (RevMan). Version 5.3.

Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014.

**Richter 2009**

Richter M, Vereecken CA, Boyce W, Maes L, Gabhainn SN, Currie CE. Parental occupation, family affluence and adolescent health behaviour in 28 countries. *International Journal of Public Health* 2009;**54**(4):203–12.

**Room 2004**

Room R. Drinking and coming of age in a cross-cultural perspective. In: Bonnie RJ, O'Connor ME editor(s). *Reducing Underage Drinking: A Collective Responsibility*. Washington DC: National Academy Press, 2004.

**Ryan 2010**

Ryan SM, Jorm AF, Lubman DI. Parenting factors associated with reduced adolescent alcohol use: a systematic review of longitudinal studies. *The Australian and New Zealand Journal of Psychiatry* 2010;**44**(9):774–83.

**Simons-Morton 2009**

Simons-Morton BG, Farhat T, Ter Bogt TF, Hublet A, Kuntsche E, Nic Gabhainn S, et al. Gender specific trends in alcohol use: cross-cultural comparisons from 1998 to 2006 in 24 countries and regions. *International Journal of Public Health* 2009;**54**(Suppl 2):199–208.

**Springer 2006**

Springer F, Phillips JL. The IOM model: a tool for prevention planning and implementation. *Prevention Tactics* 2006;**8**.

**Steinberg 2001**

Steinberg L. We know some things: parent-adolescent relationships in retrospect and prospect. *Journal of Research on Adolescence* 2001;**11**(1):1–19.

**Turrisi 2010**

Turrisi R, Ray AE. Sustained parenting and college drinking in first-year students. *Developmental Psychobiology* 2010;**52**(3):286–94.

**Wang 2009**

Wang J, Simons-Morton BG, Farhat T, Luk JW. Socio-demographic variability in adolescent substance use: mediation by parents and peers. *Prevention Science* 2009;**10**(4):387–96.

**White 2000**

White HR, Johnson V, Buyske S. Parental modeling and parenting behavior effects on offspring alcohol and cigarette use: a growth curve analysis. *Journal of Substance Abuse* 2000;**12**(3):287–310.

**White 2010**

White J, Halliwell E. Alcohol and tobacco use during adolescence: the importance of the family mealtime environment. *Journal of Health Psychology* 2010;**15**(4): 526–32.

**White 2012**

White V, Bariola E. Australian secondary school students' use of tobacco, alcohol, and over-the-counter and illicit substances in 2011: report. National Drug Strategy, Department of Health and Ageing 2012.

**WHO 2012**

WHO. Alcohol in the European Union. Consumption, harm, and policy approaches. Copenhagen, Denmark: World Health Organisation, Regional Office for Europe, 2012.

**Wills 2003**

Wills TA, Yaeger AM. Family factors and adolescent substance use: models and mechanisms. *Current Directions in Psychological Science* 2003;**12**:222–6.

**References to other published versions of this review**

**Foxcroft 2011a**

Foxcroft DR, Tsertsvadze A. Universal family-based prevention programs for alcohol misuse in young people. *Cochrane Database of Systematic Reviews* 2011, Issue 9. [DOI: 10.1002/14651858.CD009308]

\* Indicates the major publication for the study

## APPENDICES

### Appendix 1. CENTRAL search strategy

#1 MeSH descriptor: [Alcohol-Related Disorders] explode all trees  
 #2 MeSH descriptor: [Alcohol Drinking] explode all trees  
 #3 (alcohol near (drink\* or intoxicat\* or abus\* or use or misus\* or risk\* or consum\* or excess\* or reduc\* or intervention\*)):ti,ab,kw  
 #4 (drink\* near (excess or heavy or heavily or harm or harmful or hazard\* or binge or harmful or problem\*)):ti,ab,kw  
 #5 drunk\*  
 #6 alcoholic\*:ti,ab,kw  
 #7 #1 or #2 or #3 or #4 or #5 or #6  
 #8 (parent\* or father\* or mother\* or mum or dad or maternal or paternal or family or families or daughter or son or home):ti,ab,kw  
 #9 MeSH descriptor: [Family] explode all trees  
 #10 MeSH descriptor: [Parents] explode all trees  
 #11 MeSH descriptor: [Parent-Child Relations] explode all trees  
 #12 #8 or #9 or #10 or #11  
 #13 #7 and #12  
 #14 (youth\* or juvenile\* or adolescen\* or teen\* or schoolchild\* or girl\* or boy\* or minor\* or student\* or child\* or pupil\* or pupil\* or kid or kids or underage)  
 #15 (young\* near/2 (adult\* or people or person\* or male\* or female\*))  
 #16 early near/2 adult\*  
 #17 #14 or #15 or #16  
 #18 #13 and #17

### Appendix 2. Criteria for risk of bias assessment

Item	Judgement	Description
1. Random sequence generation (selection bias)	Low risk	The investigators describe a random component in the sequence generation process such as: random number table; computer random number generator; coin tossing; shuffling cards or envelopes; throwing dice; drawing of lots; minimisation
	High risk	The investigators describe a non-random component in the sequence generation process such as: odd or even date of birth; date (or day) of admission; hospital or clinic record number; alternation; judgement of the clinician; results of a laboratory test or a series of tests; availability of the intervention
	Unclear risk	Insufficient information about the sequence generation process to permit judgement of low or high risk
2. Allocation concealment (selection bias)	Low risk	Investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, and web-based randomisation); sequentially- numbered, opaque, sealed envelopes

(Continued)

	High risk	Investigators enrolling participants could possibly foresee assignments because one of the following method was used: open random allocation schedule (e.g. a list of random numbers); assignment envelopes without appropriate safeguards (e.g. if envelopes were unsealed or nonopaque or not sequentially numbered); alternation or rotation; date of birth; any other explicitly unconcealed procedure
	Unclear risk	Insufficient information to permit judgement of low or high risk This is usually the case if the method of concealment is not described or not described in sufficient detail to allow a definite judgement
3. Blinding of participants and providers (performance bias) Objective outcomes	Low risk	No blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding; Blinding of participants and key study personnel ensured, and unlikely that the blinding could have been broken
	High risk	No blinding or incomplete blinding, and the outcome is likely to be influenced by lack of blinding; Blinding of key study participants and personnel attempted, but likely that the blinding could have been broken, and the outcome is likely to be influenced by lack of blinding
	Unclear risk	Insufficient information to permit judgement of low or high risk
4. Blinding of participants and providers (performance bias) Subjective outcomes	Low risk	Blinding of participants and providers ensured and unlikely that the blinding could have been broken
	High risk	No blinding or incomplete blinding, and the outcome is likely to be influenced by lack of blinding; Blinding of key study participants and personnel attempted, but likely that the blinding could have been broken, and the outcome is likely to be influenced by lack of blinding
	Unclear risk	Insufficient information to permit judgement of low or high risk
5. Blinding of outcome assessor (detection bias) Objective outcomes	Low risk	No blinding of outcome assessment, but the review authors judge that the outcome measurement is not likely to be influenced by lack of blinding; Blinding of outcome assessment ensured, and unlikely that the blinding could have been broken
	High risk	No blinding of outcome assessment, and the outcome measurement is likely to be influenced by lack of blinding; Blinding of outcome assessment, but likely that the blinding could have been broken, and the outcome measurement is likely to be influenced by lack of blinding
	Unclear risk	Insufficient information to permit judgement of low or high risk

(Continued)

6. Blinding of outcome assessor (detection bias) Subjective outcomes	Low risk	Blinding of outcome assessment ensured, and unlikely that the blinding could have been broken
	High risk	No blinding of outcome assessment, and the outcome measurement is likely to be influenced by lack of blinding; Blinding of outcome assessment, but likely that the blinding could have been broken, and the outcome measurement is likely to be influenced by lack of blinding
	Unclear risk	Insufficient information to permit judgement of low or high risk
7. Incomplete outcome data (attrition bias) For all outcomes except retention in treatment or drop out	Low risk	No missing outcome data; Reasons for missing outcome data unlikely to be related to true outcome (for survival data, censoring unlikely to be introducing bias); Missing outcome data balanced in numbers across intervention groups, with similar reasons for missing data across groups; For dichotomous outcome data, the proportion of missing outcomes compared with observed event risk not enough to have a clinically relevant impact on the intervention effect estimate; For continuous outcome data, plausible effect size (difference in means or standardised difference in means) among missing outcomes not enough to have a clinically relevant impact on observed effect size; Missing data have been imputed using appropriate methods All randomised patients are reported/analysed in the group they were allocated to by randomisation irrespective of non-compliance and co-interventions (intention to treat)
	High risk	Reason for missing outcome data likely to be related to true outcome, with either imbalance in numbers or reasons for missing data across intervention groups; For dichotomous outcome data, the proportion of missing outcomes compared with observed event risk enough to induce clinically relevant bias in intervention effect estimate; For continuous outcome data, plausible effect size (difference in means or standardised difference in means) among missing outcomes enough to induce clinically relevant bias in observed effect size; 'As-treated' analysis done with substantial departure of the intervention received from that assigned at randomisation
	Unclear risk	Insufficient information to permit judgement of low or high risk (e.g. number randomised not stated, no reasons for missing data provided; number of drop outs not reported for each group)
8 Selective reporting (reporting bias)	Low risk	The study protocol is available and all of the study's pre-specified (primary and secondary) outcomes that are of interest in the review have been reported in the pre-specified way; The study protocol is not available but it is clear that the published reports include all expected outcomes, including those that were pre-specified (convincing text of this nature may be uncommon)

(Continued)

	High risk	Not all of the study's pre-specified primary outcomes have been reported; One or more primary outcomes is reported using measurements, analysis methods or subsets of the data (e.g. subscales) that were not pre-specified; One or more reported primary outcomes were not pre-specified (unless clear justification for their reporting is provided, such as an unexpected adverse effect); One or more outcomes of interest in the review are reported incompletely so that they cannot be entered in a meta-analysis; The study report fails to include results for a key outcome that would be expected to have been reported for such a study
	Unclear risk	Insufficient information to permit judgement of low or high risk
9. Other bias	Low risk	No difference in important covariates (e.g. gender or alcohol use) between study groups at baseline; No risk of contamination of program effects (e.g. randomisation at school level of geographically- dispersed schools)
	High risk	Baseline between study group imbalance in important covariate/s such as gender or alcohol use; Contamination of program effects (e.g. clusters of students randomised to experimental or control program within one school)
	Unclear risk	Insufficient information to permit judgement of low or high risk for confounding or contamination

## CONTRIBUTIONS OF AUTHORS

Dr Conor Gilligan will be the guarantor of the review, and is responsible for designing and coordinating the review including interpreting results and writing the review. As author of the previous review, Professor Foxcroft has been responsible for conceiving and designing the review, and will provide general advice on the present review. Tameka-Rae Small and Amanda Williams will be involved with data collection for the review including screening search results, writing to authors for additional information, obtaining and screening data on unpublished studies, and entering data into RevMan. Rebecca Hodder and Dr Shauna Sherker will be involved with data extraction from included papers, as well as assisting with writing the review. Melanie Kingsland will be involved with analysis of data and assistance with writing. Professor John Wiggers, Dr Luke Wolfenden, and Ms Julie Rae will be involved in data interpretation, writing, and providing general advice about the review.



## DECLARATIONS OF INTEREST

This work is being conducted in a partnership between researchers from the University of Newcastle (Australia), Oxford Brookes University (UK), and the Australian Drug Foundation (ADF). The Australian Drug Foundation (ADF) is a not-for-profit organisation. Its vision is Healthy People, Stronger Communities. Its mission is working together to prevent alcohol and other drug problems in communities. The ADF has a 50 year history of supporting communities to prevent alcohol and other drug (AOD) misuse. The national office is in Melbourne and the organisation is active across Australia, specialising in averting AOD harm rather than providing treatment services like most organisations in this field. The ADF works in consultation and collaboration with communities and through partnerships and alliances with like-minded organisations. The Other Talk, assisting parents to talk to their children about alcohol and other drugs, is a major community program.

DFs Department has received funding from the alcohol industry for adapting and evaluating a family based prevention program, the ISFP. The adapted version was evaluated in large scale randomised controlled trials in Wales, funded by the UK Medical Research Council, and Poland, funded by the Polish National Bureau for Drug Prevention.

## SOURCES OF SUPPORT

### Internal sources

- University of Newcastle, Australia.  
In kind, author time
- Oxford Brookes University, UK.  
In kind, author time
- Australian Drug Foundation, Australia.  
Financial support for review assistance
- Hunter New England Population Health, Australia.  
Financial support for review assistance

### External sources

- No sources of support supplied