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Review

Risk and protective factors for depression that adolescents can modify: A systematic review and meta-analysis of longitudinal studies

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ABSTRACT

Background: Adolescence is a peak time for the onset of depression, but little is known about what adolescents can do to reduce their own level of risk. To fill this gap, a review was carried out to identify risk and protective factors for depression during adolescence that are modifiable by the young person.
Methods: Employing the PRISMA method, we conducted a systematic review and meta-analysis of longitudinal studies to identify risk and protective factors during the adolescent period (aged 12–18 years) that are potentially modifiable by the young person without professional intervention or assistance. Stouffer's method of combining *p* values was used to determine whether associations between variables were reliable, and meta-analyses were conducted to estimate the mean effect sizes of associations.
Results: We identified 113 publications which met the inclusion criteria. Putative risk factors implicated in the development of depression for which there is a sound evidence base, and which are potentially modifiable during adolescence without professional intervention, are: substance use (alcohol, tobacco, cannabis, other illicit drugs, and polydrug use); dieting; negative coping strategies; and weight. Modifiable protective factors with a sound evidence base are healthy diet and sleep.
Limitations: Limitations include not systematically reviewing moderators and mediators, the lack of generalisability across cultures or to younger children or young adults, and the inability to conduct a meta-analysis on all included studies.
Conclusions: Findings from this review suggest that future health education campaigns or self-help prevention interventions targeting adolescent depression should aim to reduce substance use (alcohol, tobacco, cannabis, other illicit drugs, and polydrug use); dieting; and negative coping strategies; and promote healthy weight; diet; and sleep patterns.

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1. Introduction

1.1. The burden and prevalence of depressive disorders in adolescence

Approximately one in every five adolescents will experience a diagnosable depressive episode by the age of 18 (Costello et al., 2003; Hankin et al., 1998). Given the significance of this period in the life span with respect to physical, social, emotional, educational and vocational development, the onset of mood disorder during adolescence can serve to disrupt healthy developmental trajectories. This disruption can result in persistent, adverse consequences for the adolescent across these domains (Cicchetti and Toth, 1998). Treatment alone is unlikely to be sufficient to turn the tide of depression, due to high incidence rates and the limited effectiveness of current treatments in bringing about a full recovery in affected individuals (Andrews et al., 2004). There is thus a strong imperative for prevention to reduce the burden of these disorders.

1.2. The prevention of adolescent depression

Although there are many opportunities for prevention across the life span, adolescence arguably represents the most promising window for prevention of depression, given: a) the peak in incidence during this period and b) the potential for behavioural patterns established during this period to persist over the life course and influence long-term mental health trajectories (Gladstone et al., 2011). During this period, adolescents take on increasing autonomy for decisions that can potentially influence their mental health status, which further underscores the importance of education about prevention at this time (Crockett and Petersen, 1993).

In recent decades a proliferation of intervention trials have been designed to prevent adolescent depression across the three levels of prevention activity delineated by the Institute of Medicine (1994): universal, selective, and indicated. A recent Cochrane review of psychological and educational interventions for preventing depression in children and adolescents identified pooled effect sizes ranging from small to medium, with larger effect sizes for selective and indicated programs than for universal programs (Merry et al., 2011). This has led some to conclude that universal interventions are not justifiable. However, Merry and colleagues noted that these judgments are likely to be premature due to the early stage of the evidence base, and methodological challenges associated with demonstrating the efficacy of universal approaches. Offord (2000) and Pössel (2005) previously weighed up, in some detail, the advantages and disadvantages of the different levels of prevention. They concluded that the potential disadvantages of universal prevention approaches (e.g. less cost-effective and smaller benefits to individuals) are outweighed by the corresponding advantages (e.g., less stigmatising, greater impact at a population level), particularly within programs which target adolescents. The development of more efficacious universal prevention strategies has been highlighted as a key goal of the prevention research agenda (McLaughlin, 2011).

Depression prevention initiatives, particularly those that are more targeted in focus, have tended to rely on a professional to facilitate the intervention over a brief period of time (Merry et al., 2011). However this approach is highly resource intensive and has an unfavourable ratio in terms of the number of adolescents who are exposed to the intervention messages relative to the number who could potentially benefit from those messages (Pössel, 2005). There is a clear need to develop interventions that are scalable, cost effective, and low intensity which can be delivered by people other than clinicians, given the limited resources available to support prevention (Jacka et al., 2013; McLaughlin, 2011). Whereas there has been considerable attention paid to promoting the physical health literacy of young people over the past several decades through widespread public health campaigns (e.g., targeting tobacco and diet), there has not been an equivalent push to educate and empower young people with respect to preventive actions they can take to decrease their risk for common mental health problems. Public health campaigns in the area of mental health have focused on encouraging early help seeking and reducing stigma, rather than equipping adolescents with the knowledge and skills they need to make informed choices about factors that can potentially influence their mental health outcomes, both contemporaneously and into adulthood (e.g., beyondblue: the national depression initiative, 2011; Mental Health Council of Australia, 2013).

In order to effectively target population health messages for depression prevention during adolescence, there is a need to identify what actions adolescents can undertake in their daily lives, in the absence of formal intervention or assistance from professionals, to lower their risk for depression. This knowledge can subsequently be disseminated to the general public in the form of practical, detailed suggestions that they can act on (Jorm, 2012). In line with a population health approach, these messages should be firmly grounded in evidence derived from quality epidemiologic and clinical research, as well as a thorough understanding of the developmental context within which risk is conferred and resilience acquired (Garber, 2006).

1.3. Aims and scope of this study

There are potent risk and protective factors for adolescent depression which are not amenable to change, either because the exposures occur early in the lifespan, or are systemic in nature (see Shortt and Spence, 2006, for a review). These are worthy of population health intervention in their own right but are beyond the scope of this review and thus are not the subject of further discussion here. However, there remain many mutable factors (e.g., risk behaviours and lifestyle-related factors) that have been implicated in the development of adolescent depression, which can be modified at the individual level during the adolescent period and which could be readily translated into universal preventive intervention strategies.

This review aimed to identify risk and protective factors that are associated with depression adolescents, which in this paper refers to those aged between 12 and 18 years, with a focus on those factors that are potentially modifiable by the adolescent. For the purposes of this review, we follow Kazdin and colleagues (Kazdin et al., 1997) in defining a risk factor as an antecedent condition associated with an increase in the likelihood of the outcome of interest, while a protective factor is used to refer to an antecedent condition associated with a decrease in the likelihood of the outcome. The outcome of interest in the present review is onset of unipolar depressive disorders as they are classified in the DSM-IV-R (American Psychiatric Association, 2000) because unipolar depression, specifically Major Depressive Disorder, is by far the most prevalent form of affective disorder experienced by adolescents.

Prospective cohort studies and systematic reviews of prospective cohort studies were identified as the highest level of evidence to inform the research question, thus studies utilising these designs were considered for inclusion in the review. Studies employing cross-sectional and retrospective designs, including case-control studies, were not considered to provide sufficient information to establish the temporal precedence of the risk or protective factor, and were excluded. The review focused on factors that apply to the universal adolescent population; studies focusing on a subpopulation of adolescents with elevated risk due

to the presence of known risk factors such as parental mental illness or a physical illness or disability (i.e., selective prevention), or showing early manifestations of disorder (i.e., indicated prevention), were excluded.

2. Method

2.1. Data sources

A systematic computerised literature search of PubMed, Scopus, and PsycINFO databases was performed for studies published up to March 2013 according to the PRISMA statement (Moher et al., 2009; PRISMA checklist provided in Online Supplement 5). These databases were searched using key search terms following the 'PICO' method. Keywords were generated for each of these concepts by examining the terminology used in the literature. Table 1 provides a detailed outline of the terms used for searching the databases. Searches were limited to peer-reviewed articles written in English. No publication date limits were applied. Seventy-seven additional sources were identified between March and September 2013 by hand searching reference lists of studies identified as relevant from the initial search, as well as by screening papers citing these relevant studies in Google Scholar.

2.2. Study selection

Potentially relevant articles were determined by screening of the article's title, abstract and, where needed, article contents. Studies were included in the review if they fulfilled the following criteria: a) employed a prospective study design; b) were subject to peer review; c) included one or more potentially modifiable adolescent risk or protective factors as predictors; d) included depressive symptoms or diagnoses as outcome variables; and e) the mean age of adolescent participants was between 12 and 18 years of age when the risk or protective factor was measured.

Studies were excluded from the review if they: a) presented solely cross-sectional or retrospective analyses; b) were a review, case report, comment, letter, or evaluation of an intervention; c) were published in a language other than English; d) examined a specific subgroup of the population or involved a non-population sample; e) reported a follow-up period of less than 1 year; f) included outcome variables that: i) assessed internalising symptoms, but not depressive symptoms specifically, ii) focused on post-partum depression, transient sadness, or grief iii) were not established measures of depressive affect, or iv) provided insufficient detail on the way depression was assessed; g) mean age or age range of the participants was not specified; and h) the predictor variable included in the analyses was a composite of multiple risk and protective factors, or lacked adequate specificity.

The study selection process is summarised in Fig. 1.

2.3. Data extraction

A standardised, pilot-tested extraction sheet was used to extract and collate the data from the included studies. Data extracted included basic descriptive information about the study (e.g., the design, the sample size, and the length of follow-up), details of the relevant predictor and outcome variables, the effect size measure and corresponding *p* value, and the direction of the effect reported. A set of rules was developed to standardise the *p* value selection process (see Online Supplement 1). Decision hierarchies were also developed to manage articles which reported multiple associations between relevant variables and studies reporting duplicate data. Where possible, gender-specific associations were extracted and analysed separately. Where these were not available, those reported for the total sample were extracted instead.

Data from each study was independently extracted by two researchers. Data extraction was conducted by KC, PP, and one other research assistant. Discrepancies were resolved through discussion between the two coders where possible. Where agreement could not be reached, the issue was resolved through consultation with AJ or MY.

2.3.1. Coding

Risk and protective factors for depression that are modifiable by adolescents were defined as those which could be feasibly altered by the adolescent, in the absence of a professional or other intervention. This precluded the inclusion of variables which: a) relate to personality traits or characteristics of the person as a whole, which are not readily malleable without professional or

Table 1
Outline of search terms used for systematic review of modifiable risk and protective factors for adolescent depression.

Concept	MeSH terms	Alternate search terms
Adolescence	Adolescent	Adolesc[a]
	Student	Youth
	Young adult	Young person
		Teenage[a]
		Student[a]
Depression	Depression	Depress[a]
	Depressive disorders	Dysthym[a]
	Major depressive disorder	Unipolar depression
	Dysthymic disorder	Symptoms of depression
	Mood disorders	Depressed mood
Risk or protective factor	Risk factor	Protective factor
	Precipitating factor	Promotive factor
		Correlate
		Vulnerability
		Predictor
		Resilience

Note: MeSH = Medical Subject Heading search terms. MeSH is the US National Library of Medicine's controlled vocabulary used for indexing articles for MEDLINE/PubMed. MeSH terminology provides a consistent way to retrieve information that may use different terminologies for the same concepts.

[a] Denotes that the search term will look for any word with the given letter combination plus any combination of letters following the original combination.

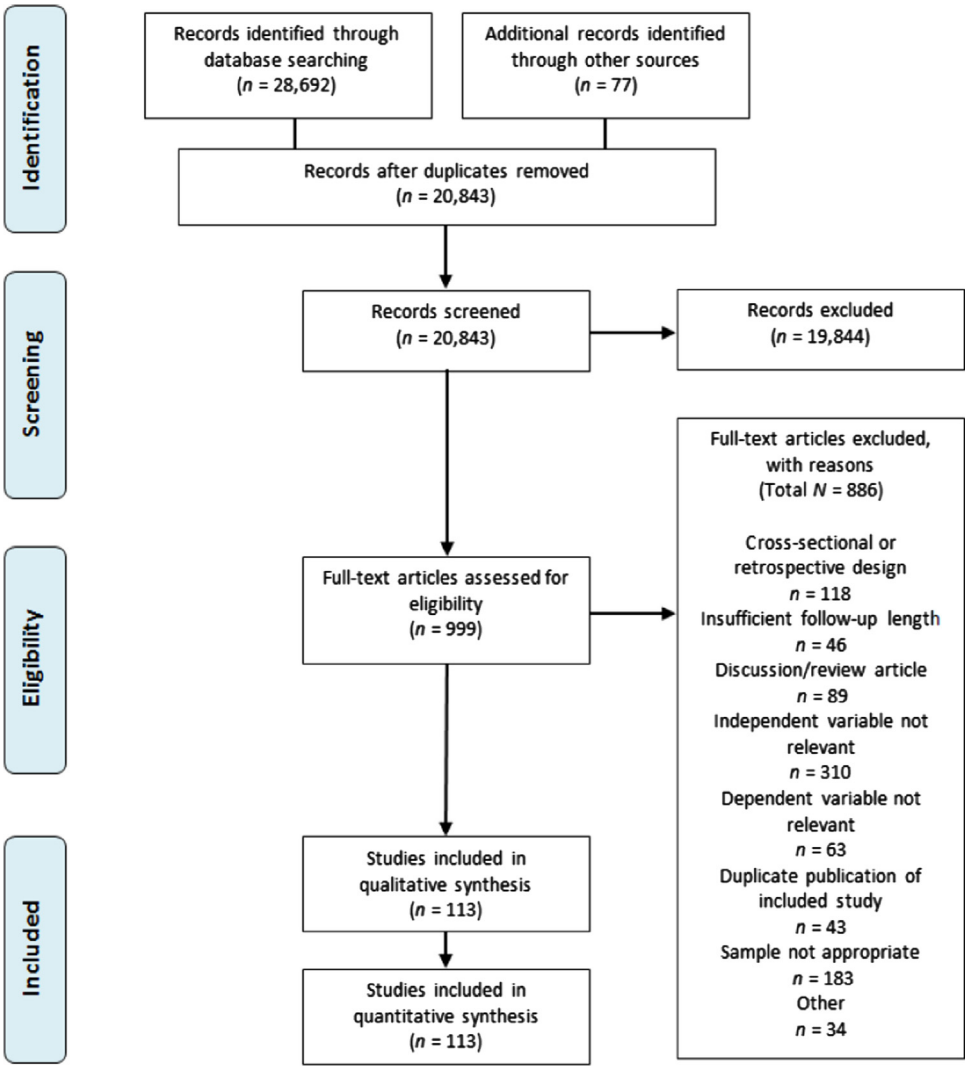

Fig. 1. PRISMA diagram.

other intervention (e.g., cognitive style or information processing); b) may be modifiable in theory, but are framed within the context of a dyad (e.g. parent–child or peer–peer) or wider social system (e.g., the family unit, the classroom) in which action by the young person is necessary but not sufficient; or c) are composite variables (e.g., delinquency) where specific actions (e.g., theft, cigarette smoking) are not specified and/or measured independently.

Studies reporting modifiable risk and protective factors were subsequently grouped thematically into one or more of the themes provided in Table 2, which provides examples of assessment measures and items for each theme. Themes were generated based on identifying studies that included similar variables or measures, noting any distinctions commonly applied in the literature. However, where there was a compelling theoretical reason, certain factors employing similar measures were investigated separately to allow for an examination of their unique contributions to risk of depression (e.g., sport vs. physical activity).

2.4. Data analysis

Due to the heterogeneity of the included studies with respect to the statistical approaches and methodologies employed, the extraction of standard effect sizes was not possible for all included studies, which meant that a meta-analysis could only be conducted on a subset of studies. It was not feasible to contact all study authors for missing or unadjusted data, given the size of the literature. In lieu of this, statistical procedures were utilised to handle a lack of usable data in a published study. Following previous reviews of risk factors for common mental health problems in adolescence which examined similarly heterogeneous literature (Ryan et al., 2010; Yap et al., 2014), we attempted to compensate for this limitation by using Stouffer's method of combining p values (Stouffer, 1949). This method provided a means of synthesising the findings of all studies included in this review, to determine the reliability of associations between variables. The meta-analysis conducted on a subset of studies was intended to supplement the findings from the Stouffer's p synthesis by providing an estimate of the magnitude of the observed effects.

2.4.1. Stouffer's p

Stouffer's p is a method of synthesising studies by testing the combined significance level rather than assessing effect magnitude. Stouffer's z was calculated by dividing the sum of the $z(p_i)$ values by the square root of k, where k is the number of studies. The p values for each association were converted to one-tailed values in order to test directional hypotheses specified a priori. Stouffer's zs were calculated to determine the overall p value of the associations reviewed for each theme and depression.

Table 2
Theme definitions and example measures and items.

Theme	Definition	Example measures	Example items
Alcohol use (frequency)	The frequency with which the adolescent engages in alcohol consumption.	• Behavioural self-report • Customary Drinking and Drug Use Inventory (CDDR)	• How many times did you consume beer, wine, wine coolers, or distilled spirits within the past month? • Over the past 12 months, on how many days did you drink [5 or more or drinks in a row?
Alcohol use (quantity)[a]	The quantity of alcoholic beverages the adolescent consumes during single drinking episodes.	• Behavioural self-report	• On average, how much alcohol do you consume in a single drinking episode?
Cannabis use	The extent to which the adolescent engages in use of cannabis (in any form).	• Behavioural self-report • Diagnostic Interview Schedule for Children and Adolescents (DISC)	• During your life, how many times have you used marijuana? • In the past 12 months, how often did you use marijuana?
Dating during adolescence	The extent to which the adolescent is involved (current or historic) in romantic activities or relationships.	• Behavioural self-report	• Have you ever had a boyfriend or girlfriend? [If YES] How old were you when you had your first boyfriend or girlfriend?
Dieting	The extent to which the adolescent engages in deliberate attempts to restrict food intake for the purposes of weight reduction.	• Children's Eating Attitudes Test • The Dutch Eating Behavior Questionnaire	• How often do you refuse food or drink offered because you are concerned about your weight? • I eat less when I have put on weight.
Early moving out of home	Moving out of the family home earlier than is typical (i.e., during the high-school attending years).	• Behavioural self-report	• At what age did you move out of home to live in a separate house, apartment, trailer home, or group quarters?
Early sex	The extent to which the adolescent engages in sexual activities or is involved in relationships of a sexual nature (current or historic).	• Behavioural self-report	• At what age did you first have sexual intercourse? • How often have you engaged in (a) sexual intercourse with a date or romantic partner and (b) other sexual relations with a date or romantic partner (more than kissing, but not intercourse)?
Extracurricular activities	The extent to which the adolescent is involved in activities that occur outside or parallel to the traditional school curriculum (e.g., clubs, teams, volunteering etc.)	• Behavioural self-report	• How many clubs are you involved in? (Example: drama, debate, foreign language, computer, band) • How many hours/day do you spend participating in school-based extracurricular activities?
Healthy diet	The extent to which the adolescent engages in healthy habits in relation to food, and to which their usual diet includes a range of nutritious foods.	• Behavioural self-report • Food Frequency Questionnaire	• About how many lots of vegetables do you usually eat in a day? • During the past 7 days, how many times did you eat green salad?
Media use	The extent to which the adolescent uses various forms of electronic media for recreational or communication purposes.	• Behavioural self-report	• How much time do you spend surfing the Web on an average day? • Outside of school hours, about how much time do you spend watching television or video cassettes on an average school day?
Negative coping strategies	The extent to which the adolescent uses negative or maladaptive strategies or skills to offset disadvantages or stress in daily life. This encompasses any dysfunctional coping strategies such as avoidant or withdrawal coping, using substances or drinking, distraction or disengagement from the problem, and emotion-focused coping.	• Adolescent Coping Orientation for Problem Experiences • Behavioural Inventory of Strategic Control • Coping Actions Scale	• When you face difficulties or feel tense, how often do you get angry and yell at people? • When making decisions, you usually go with your "gut feeling" without thinking too much about the consequences of each alternative. • I try not to think about the problem.
Negative emotion regulation strategies	The extent to which the adolescent engages in negative or maladaptive processes pertaining to the monitoring, evaluation, and modification of emotional reactions in response to emotional stimuli.	• Emotion Regulation Questionnaire (ERQ)	• I control my emotions by not expressing them.
Other illicit drug use[b]	The extent to which the adolescent engages in use of any named illicit substances (historic or current), not including cannabis.	• Behavioural self-report • CIDI	• Have you ever used ecstasy (MDMA)? • In the past 12 months, how often did you use the following substances? (e.g., cocaine)
Part-time employment	The adolescent's involvement in part-time or casual paid employment (i.e., not including school leavers in full-time employment).	• Behavioural self-report	• How many hours a week do you work on average in paid employment?
Physical activity	The extent to which the adolescent engages in non-specific body movement, organised or otherwise, that works the muscles and requires more energy than resting.	• Behavioural self-report • Accelerometer	• Outside school/work hours, how often do you do sports or exercise until you are out of breath or sweating? • How many days in an average week do you take part in physical activities?
Polydrug use[c]	The extent to which the adolescent engages in concurrent use of different substances (licit and illicit).	• Behavioural self-report	• How often in the past six months did you consume beer/wine/wine coolers, hard liquor, cigarettes, marijuana, stimulants, sedatives, inhalants, and hallucinogens? • How often in the last year did you smoke cigarettes, drink alcohol, or use marijuana or hashish?
Positive coping strategies	The extent to which the adolescent uses positive or productive strategies or skills that are employed to offset disadvantages or stress in daily life. This encompasses functional and proactive coping strategies, such as active/approach-oriented coping, problem solving, and seeking support or talking about problems with others.	• Adolescent Coping Orientation for Problem Experiences • Behavioural Inventory of Strategic Control • Coping Actions Scale	• When you have a problem to solve, one of the first things you do is to get as many facts about the problem as possible. • When you face difficulties or feel tense, how often do you talk to a teacher or counsellor at school about what bothers you? • I talk about problems when they appear and do not worry about them later.
	The extent to which the adolescent engages in positive or adaptive processes pertaining to the	• Trait Meta-Mood Scale (TMMS)	• No matter how badly I feel, I try to think about pleasant things

Table 2 (*continued*)

Theme	Definition	Example measures	Example items
Positive emotion regulation strategies	monitoring, evaluation, and modification of emotional reactions in response to emotional stimuli.		
Relationships with positive peers	The extent to which the adolescent affiliates with positive peers (who are deemed to be so by way of the activities they engage in).	• Self-report	• How many of your friends engage in positive behaviours such as doing well in school and volunteering? • How many of your friends break the law by stealing, hurting someone, or damaging property? (reversed)
Religious observation (private)	The extent to which the adolescent engages in private observation of religion or individual forms of worship, in accordance with their particular belief system.	• Behavioural self-report	• How often do you pray?
Religious observation (public)[d]	The extent to which the adolescent engages in communal religious services or activities in accordance with their particular belief system.	• Behavioural self-report	• Many churches, synagogues, and other places of worship have special activities for teenagers such as youth groups, Bible classes, or choir. In the past 12months, how often did you attend such youth activities?
Self-disclosure to parents	The extent to which the adolescent spontaneously tells their parent or caregiver about their lives, experiences and feelings.	• Adolescent Disclosure Scale (ADS)	• Do you spontaneously tell your parents about your friends such as which friends you hang out with and how you think and feel about various things?
Sleep	The sleeping habits of the adolescent. This theme does not include severe sleeping difficulties or disorders, which were deemed not modifiable by the adolescent in the absence of professional assistance or intervention.	• Behavioural self-report	• Do you usually get enough sleep? • How many hours of sleep on average did you get on weeknights during the past four weeks?
Sport[e]	Involvement in organised sport (individual or team-based). Where feasible, this was coded separately from physical activity and extracurricular activities.	• Behavioural self-report • Adolescent Physical Activity Recall Questionnaire	• On how many sports teams did you play during the past 12 months, including those run by the school or a community group? • Frequency and duration of organised sports and games involvement during a typical week, and total time spent engaging in them (minutes/week).
Tobacco	The extent to which the adolescent engages in use of tobacco (in any form).	• Behavioural self-report • Schedule for Affective Disorders and Schizophrenia (K-SADS)	• Have you ever smoked? [If YES] On how many days did you smoke during the past 30 days? • Have you ever tried or experimented with cigarette smoking, even a few puffs?
Weight	The adolescent's actual body weight (as opposed to perceived body weight or weight status)	• Self-report • Observed	• What is your body weight (in kg) and your height (in m)? • BMI, calculated from objectively measured height and weight.

[a] Quantity of alcohol consumption was examined independently from frequency of consumption, to explore any differential impact of heavy (i.e., binge-drinking) versus frequent alcohol use (which may not necessarily be heavy use) on depression risk.

[b] Other illicit drug use and cannabis use were examined independently, as several of the larger prospective studies separated these measures out, possibly due to the higher prevalence of cannabis use amongst adolescent populations relative to other illicit drugs.

[c] Polydrug use was examined independently from other substance use themes, as the literature suggests this form of substance use is associated with a unique set of risks (e.g., Collins et al., 1998).

[d] Public forms of religious observation, such as attendance at services or youth groups, were examined independently of private forms of observation, to explore any differential impact of social support derived from faith communities, relative to the act of religious observance in and of itself.

[e] Sport was examined independently from physical activity to explore any differential impact of physical activity within a collective context (i.e., derived from social support or connection.

2.4.2. Effect size computation

Meta-analysis was conducted for a subset of included studies using Comprehensive Meta-Analysis (CMA) Version 2.2.064. Studies were included in the meta-analysis if they: (1) examined a population sample; (2) reported an effect size that had not been adjusted for any covariates; and (3) reported an effect size measure that could be converted to *r*. Here, a population sample was defined as a sample drawn from the general population (rather than a clinical sample, for instance). Given the focus of this paper on universal, modifiable risk and protective factors to facilitate prevention efforts in the community, non-population samples were excluded from the meta-analyses. The correlation coefficient *r* was used as the measure of effect size, as this was the most commonly reported effect measure. Where studies did not report a correlation coefficient, we used alternative effect size measures that were accepted by CMA, e.g., odds ratios with confidence intervals, *t* values for correlations, and *t*-tests or analyses of variance comparing groups. When a study compared more than two groups based on cut-offs on a continuous variable, we used the means, standard deviations and sample sizes of the two most-extreme groups. CMA converts these alternative measures into *r* using formulas outlined by Borenstein et al. (2011). In some instances, a correlation coefficient or alternative effect size measure could not be extracted (e.g., if the study simply reported that an association was non-significant without providing the corresponding *r*). In these instances, where feasible, we calculated $r_{equivalent}$ (Rosenthal and Rubin, 2003) based on the sample size and the *p* value for the association.

For each analysis, only one effect size per included article was used, with extraction guided by pre-specified decision rules (see Online Supplement 1). Confounders differed considerably across included studies, therefore a decision was made to analyse primarily unadjusted data in the meta-analyses to increase homogeneity of results.

2.4.3. Meta-analysis procedures

A meta-analysis was conducted for each risk/protective factor theme, where there were at least two independent studies reporting effect size measures accepted by CMA. As we anticipated considerable heterogeneity between studies, a random-effects model was used for all analyses. We tested for between-study

heterogeneity using Cochran's Q. The I^2 statistic gives an indicator of heterogeneity in percentages, with a value of 0% indicating no observed heterogeneity between studies and increasing values indicating higher levels of heterogeneity (25%=low, 50%=moderate, 75%=high). Post-hoc sensitivity analyses were conducted to explore whether age (senior adolescents aged 16–18 years, junior adolescents aged 12–15 years, or both), gender (male, female or mixed sample), follow-up interval (1–3, 4–6, or 6+years), or choice of outcome measure (symptom or disorder based) could explain observed heterogeneity in results. The presence of publication bias was assessed by visual inspection of the funnel plot of the meta-analyses and by using Egger's test (Egger et al., 1997). The presence of publication bias was only tested for where there were three or more studies for the relevant theme.

2.4.4. Interpretation of Stouffer's p and effect sizes

When interpreting the findings of this review, we focus on the Stouffer's p and corresponding mean effect size r of included associations. When interpreting the Stouffer's p, if the resultant Stouffer's z corresponded to a probability level less than 0.01, the null hypothesis of no effect was rejected. When interpreting mean effect sizes, we used Cohen's (1992) guidelines whereby r of at least 0.1=small, 0.3=medium, and 0.5=large.

3. Results

3.1. Characteristics of included studies

Fig. 1 summarises the results of different stages of the systematic literature search. We identified 113 publications reporting 209 associations. The studies were published between 1986 and 2013 and the number of participants per study ranged from 68 to 13,500. The study populations were relatively heterogeneous. The majority of studies involved samples from North America (n=81), with the remainder from Europe (n=19), Australia (n=10) and Asia (n=3). The median follow-up period for studies was 3 years (IQR 1.25–6 years). Of the 113 articles included in the review, 69 studies containing 126 associations were included in the meta-analyses. Of the 83 associations that were excluded from the meta-analyses, the reasons for exclusion were as follows: 71 reported only an effect size that had been adjusted for covariates; six could not be included because there were fewer than two studies for the relevant theme containing associations that could be converted to r; and four reported an effect size measure that could not be converted to r. Further information on the studies included and excluded from the meta-analyses is presented in Online supplement 2.

3.2. Quality assessment

All of the 113 articles included in the review utilised a prospective cohort design and involved population samples. Some articles reported on confounding factors, such as baseline symptoms, gender, age, ethnicity, socioeconomic status and family history of mental illness. Several studies used multivariable logistic regression to adjust for these confounding factors, whereas others only assessed the bivariate correlation. Due to considerable heterogeneity in the number and nature of the covariates included in analyses, only unadjusted estimates were included in the meta-analyses.

Table 3 presents the Stouffer's p and effect size r (with 95% confidence interval), as well as the Egger's test two-tailed p value as an index of publication bias, for each theme. The results of the Stouffer's p analyses are only reported in text where these differed from those derived from the meta-analysis. Subgroup analyses and tests for publication bias are reported in text only where these were significant. Stouffer's p values for each included study are presented in Online supplement 2, and forest plot diagrams for each meta-analysis are presented in Online supplement 3. References for all included studies are included in Online supplement 4.

3.3. Modifiable risk and protective factors

3.3.1. Alcohol use (frequency)

Based on 14 studies encompassing 12 associations, more frequent engagement in alcohol use in adolescence was associated with higher levels of depression, with a small but significant mean effect size emerging from these 12 associations. There was evidence of substantial heterogeneity between studies (I^2=70.0%). Subgroup analysis by age demonstrated a significant interaction that could potentially explain heterogeneity (p=0.009), with a greater mean effect size for senior adolescents (16–18 years) relative to junior adolescents (12–15 years).

3.3.2. Alcohol use (quantity)

Based on four studies, each contributing one association, the consumption of greater quantities of alcohol during drinking episodes (i.e., bingeing) was associated with higher levels of depression, with a small but significant mean effect size, but substantial heterogeneity (I^2=89.2%).

3.3.3. Cannabis use

Based on eight associations from six studies, cannabis use in adolescence was found to be associated with higher levels of depression. A small but significant effect size emerged, with substantial heterogeneity evident (I^2=81%). A subgroup analysis by gender revealed a significant interaction, with larger effect size estimates for those studies (n=2) with female samples (p=0.003).

3.3.4. Dating during adolescence

Based on three associations from two studies, the evidence linking dating during adolescence with higher levels of depression was mixed, with a small, non-significant mean effect size and high heterogeneity between studies (I^2=91%). The Stouffer's p, based on five associations from four studies, was, however, significant (p=0.000). A subgroup analysis by gender revealed a significant interaction (p=0.000), whereby a greater effect size was associated with the study with a mixed gender sample.

3.3.5. Dieting

Based on two associations from two studies, the evidence suggests that dieting during adolescence is associated with higher levels of depression, with a small, significant mean effect size, and zero heterogeneity.

3.3.6. Early moving out of home

There were insufficient studies to conduct a meta-analysis for this theme. The Stouffer's p analysis, based on two associations from two studies, suggests that the evidence linking early moving out of home and depression is weak (p=0.500).

3.3.7. Early sex

Based on two associations from two studies, there is no clear evidence linking early sex with higher levels of depression, with a small, non-significant mean effect size and substantial heterogeneity between studies (I^2=75%).

3.3.8. Extracurricular activities

Eight associations from eight studies failed to find a significant association between engagement in extracurricular activities and lower levels of depression, with a small, non-significant mean effect size with high heterogeneity (I^2=97%). A subgroup analysis

Table 3
Summary of findings for Stouffer's *p* and effect sizes.

Theme	*N* of studies	*N* of associations in Stouffer's *p*	*N* of associations in meta-analysis	Stouffer's *p*	*r* (95% CI)	*p* value for *r*	I^2	Eggers test two-tailed *p*
Alcohol (frequency)	17	17	12	0.002*	0.059 (0.024, 0.094)	0.001*	70.0	0.71
Alcohol (quantity)	4	4	4	0.001*	0.119 (0.040, 0.197)	0.003*	89.2	0.75
Cannabis	15	15	8	0.000*	0.118 (0.068, 0.168)	0.000*	81.22	0.950
Dating during adolescence	5	5	4	0.000*	0.176 (−0.013, 0.354)	0.068	90.62	0.447
Dieting	4	4	2	0.000*	0.187 (0.133, 0.240)	0.000*	0	n/a
Early moving out of home	2	2	0	0.500	n/a	n/a	n/a	n/a
Early sex	3	3	2	0.129	0.110 (−0.168, 0.371)	0.440	74.70	n/a
Extracurricular activities	9	9	8	0.250	−0.026 (−0.122, 0.070)	0.597	97.35	0.966
Healthy diet	5	5	3	0.001*	−0.064 (−0.105, −0.022)	0.003*	0	0.731
Media use	5	5	3	0.001*	0.006 (−0.076, 0.087)	0.892	93.37	0.431
Negative coping strategies	15	15	8	0.000*	0.106 (0.072, 0.140)	0.000*	97.249	0.171
Negative emotion regulation strategies	2	2	0	0.003*	n/a	n/a	n/a	n/a
Other illicit drugs	8	8	2	0.000*	0.167 (0.055, 0.275)	0.004*	77.983	n/a
Part-time employment	2	2	2	0.500	0.001 (−0.049, 0.051)	0.971	59.718	n/a
Physical activity	16	16	7	0.010*	−0.066 (−0.138, 0.007)	0.078	90.478	0.933
Polydrug use	4	4	4	0.010*	0.075 (−0.022, 0.171)	0.010	79.87	0.671
Positive coping strategies	14	14	9	0.000*	−0.011 (−0.022, −0.000)	0.042	83.492	0.58869
Positive emotion regulation strategies	1	0	0	n/a	n/a	n/a	n/a	n/a
Relationships with positive peers	6	6	6	0.001*	−0.130 (−0.247, −0.009)	0.036	92.22	0.579
Religious observance (private)	1	0	0	n/a	n/a	n/a	n/a	n/a
Religious observance (public)	2	2	0	0.083	n/a	n/a	n/a	n/a
Self-disclosure to parents	3	3	3	0.001*	−0.110 (−0.235, 0.019)	0.095	91.637	n/a
Sleep	3	3	2	0.000*	−0.210 (−0.287, −0.129)	0.000*	58.09	n/a
Sport	11	11	5	0.000*	−0.046 (−0.083, −0.008)	0.017	53.10	0.388
Tobacco	36	36	23	0.000*	0.093 (0.064, 0.122)	0.000*	84.02	0.787
Weight	16	16	11	0.003*	0.054 (0.019, 0.090)	0.003*	71.769	0.76561

Note. A meta-analysis was not conducted if the total number of associations available <2. *N*=Total number; *r*=mean effect size; CI=confidence interval; I^2=indicator of heterogeneity in percentages.

by follow-up interval demonstrated a significant interaction ($p=0.000$). This appeared to be due to one larger study with a follow-up period of 4–6 years finding a small to medium protective effect, while those studies with alternative follow-up periods (1–3 and 6+years) reported more modest effects.

3.3.9. Healthy diet

Three associations from three separate studies revealed an association between healthy diet and lower levels of depression, with a small but significant mean effect size, and zero heterogeneity.

3.3.10. Media use

Based on three associations from three studies, the evidence for an association between media use and higher levels of depression is equivocal, with a small, non-significant mean effect size with high heterogeneity between studies ($I^2=93\%$). The Stouffer's *p*, based on five associations from five studies, was however significant ($p=0.001$). A subgroup analysis by age revealed a significant interaction to explain heterogeneity ($p=0.000$), which appeared to be due to one study with senior adolescents reporting a protective effect of media use, while the two other studies, which sampled junior adolescents and a mixed sample, reported a risk effect.

3.3.11. Negative coping strategies

Based on eight associations from seven studies reporting on negative coping strategies, the evidence suggests that there is an association between negative coping and higher levels of depression, with a small, significant mean effect size, but substantial heterogeneity ($I^2=97\%$).

3.3.12. Negative emotion regulation strategies

There were insufficient studies to conduct a meta-analysis for this theme. The Stouffer's *p* analysis, based on two associations from one study, suggests that negative emotion regulation is associated with higher levels of depression ($p=0.003$).

3.3.13. Other illicit drug use

Based on two associations from two studies reporting on use of illicit drugs other than cannabis, there is evidence of an association between use of illicit drugs and higher levels of depression, with a small but significant mean effect size, but with considerable heterogeneity between studies ($I^2=78\%$).

3.3.14. Part-time employment

Two studies, encompassing two associations, that reported on part-time employment and subsequent depression did not find evidence of an association, with a small, non-significant mean effect size, and moderate heterogeneity ($I^2=60\%$).

3.3.15. Physical activity

Based on seven associations from six studies that reported on physical activity, there is no clear evidence of an association with lower levels of depression, with a small but non-significant mean effect size and high heterogeneity ($I^2=90\%$). The Stouffer's *p*, based on 16 associations from 12 studies, was however significant ($p=0.010$).

3.3.16. Polydrug use

Based on four associations from four studies reporting on concurrent substance use, a small, significant mean effect size emerged between polydrug use and higher levels of depression, with high heterogeneity between studies ($I^2=80\%$).

3.3.17. Positive coping strategies

Based on nine associations from eight studies, there was no clear association between positive coping strategies and lower levels of depression, with a small, non-significant mean effect size and high heterogeneity between studies ($I^2=83\%$). A significant association did emerge from the Stouffer's *p* analysis however ($p=0.005$).

3.3.18. Positive emotion regulation strategies

There were insufficient studies to conduct a meta-analysis or Stouffer's *p* analysis for this theme.

3.3.19. Relationships with positive peers

Based on six associations from four studies, the evidence linking relationships with positive peers with lower levels of depression is mixed, with a small but non-significant mean effect size from these associations, with high heterogeneity ($I^2=92\%$). A significant association did emerge from the Stouffer's *p* analysis, however, based on the same associations ($p=0.001$). Subgroup analyses revealed a significant interaction for age group ($p=0.001$) and outcome measure ($p=0.001$), which appeared to be due to one large study involving senior adolescents and a disorder-based outcome measure finding a small-to-moderate protective effect, while the remainder of the studies involving junior adolescents, with smaller samples and symptom-based outcome measures, reported more modest effects.

3.3.20. Religious observation (private)

There were insufficient studies available to conduct a meta-analysis or a Stouffer's *p* analysis for this theme.

3.3.21. Religious observation (public)

There were insufficient studies available to conduct a meta-analysis for this theme. The Stouffer's *p* analysis, based on two associations from two studies, indicates that there is no clear evidence that public forms of religious observation (e.g., church attendance) are protective against higher levels of depression ($p=0.083$).

3.3.22. Self-disclosure to parents

The evidence linking self-disclosure to parents with lower levels of depression, based on three associations from two studies, is equivocal, with a small but non-significant mean effect size from these associations, and high heterogeneity ($I^2=92\%$). A significant association emerged from the Stouffer's *p* analysis, however, based on the same associations ($p=0.001$). A subgroup analysis by gender revealed a significant interaction effect ($p=0.000$), with one study that reported gender-specific associations finding greater mean effect sizes (for both males and females) relative to the other mixed gender study.

3.3.23. Sleep

Based on three associations from three studies, the evidence suggests that getting enough sleep during adolescence is associated with lower levels of depression, with a small but significant mean effect size emerging from these associations, which showed moderate heterogeneity ($I^2=58\%$).

3.3.24. Sport

The evidence linking participation in sport during adolescence with lower levels of depression, based on five associations from five studies, is equivocal, with a small but non-significant mean effect size with moderate heterogeneity ($I^2=53\%$). The Stouffer's *p* was significant however ($p=0.000$).

3.3.25. Tobacco use

Based on 23 associations from 17 studies with mostly consistent findings, the evidence suggests that tobacco use is associated with higher levels of depression, with a small but significant mean effect size, but with substantial heterogeneity between studies ($I^2=84\%$).

3.3.26. Weight

Based on 11 associations from six studies, the evidence suggests that increasing weight, most commonly conceptualised as body mass index (BMI), is associated with higher levels of depression, with a small but significant mean effect size. Substantial heterogeneity was evident between studies ($I^2=72\%$). There was a significant interaction by outcome measure, with a larger effect size evident for one study that utilised a disorder-based outcome measure, relative to the remaining studies that used symptom-based measures.

4. Discussion

This systematic review and meta-analysis of prospective studies investigating modifiable risk and protective factors for adolescent depression has identified several factors for which there is a sound or emerging evidence base, albeit with significant effect sizes that were modest in magnitude. These are summarised in turn, followed by those factors for which there is minimal evidence for a relationship.

4.1. Modifiable risk and protective factors with a sound evidence base

4.1.1. Alcohol use (frequency and quantity)

Both the frequency and quantity (i.e., bingeing) of alcohol use emerged as significant predictors of higher levels of depression. There are both biologically plausible and common sense explanations that could support the role of alcohol as a risk factor for depression. Alcohol use is known to have neurotoxic effects during this developmentally sensitive period, and can also have various deleterious social and academic consequences for the adolescent which could indirectly increase their risk for depression (Powell et al., 2007). However, due to limitations of the data available, we cannot rule out the possibility of reverse causality (i.e., that young people with a tendency towards depression engage in alcohol use as a form of self-medication), or that some third variable increases risk for both outcomes.

4.1.2. Cannabis use

This review found a small but significant association between cannabis use in adolescence and higher levels of depression. This finding is consistent with other reviews (e.g., Degenhardt et al., 2003; Moore et al., 2007). Degenhardt et al. (2003) outlined several possible pathways that could explain the observed association between the cannabis use and depression. Firstly, there may be a neurobiological link, by which cannabis impacts on serotonin and other neurotransmitters causing an increase in depressive symptoms. Secondly, the presence of either cannabis use or depression can result in life circumstances that make the other more likely (e.g., cannabis use leads to academic difficulties or family conflict which subsequently elevates depressive symptoms). Thirdly, there may be some unknown factor that predisposes people to cannabis use and is also associated with depression, independent of its association with cannabis. There is also a possibility that the relationship is the inverse: elevated depression symptoms lead to cannabis use. It would be useful for future studies to investigate further the role of gender as a moderator given the finding in this review that gender accounted for a significant proportion of the variance across studies, suggesting that cannabis use may be a more potent risk factor for adolescent girls relative to boys.

4.1.3. Dieting

A small but significant mean effect size was observed for the relationship between dieting and higher levels of depression. While this was derived from a relatively small number of studies, the results from these studies were highly consistent. It is plausible that this association in part reflects a shared comorbidity between eating disorder pathology and depressive disorders. In an attempt to avoid this, this review only included associations that precisely focused on the behaviour of dietary restraint, rather than on body dissatisfaction and other eating disorder symptoms. Dietary restraint behaviours might be considered somewhat normative during adolescence for girls, and increasingly for boys (Neumark-Sztainer et al., 2011). This is in contrast to other facets of eating disorder symptomatology, which are less normative and more difficult to modify in the absence of professional intervention. Whether the role of dieting in relation to the onset of depression is as a precursor to a comorbid eating disorder, or whether it has a unique causal role in the development of depressive disorders is worthy of further investigation.

4.1.4. Healthy diet

While there is a considerable evidence base derived from cross-sectional research linking a healthy diet with lower levels of depression (Sanchez-Villegas and Martínez-González, 2013), there is a relative dearth of prospective studies that have explored this association. However, we did find evidence for a small but significant relationship between a healthy diet in adolescence and reduced risk for depression. Other studies have explored the links between intake of specific nutrients or foods and risk for depression during adolescence, such as omega-3 fatty acids (e.g., Oddy et al., 2011). Given the small number of included studies, we were unable to explore in any depth the specific dietary components that are prospectively linked with depression outcomes, but rather focused on measures of diet quality more generally.

4.1.5. Coping strategies (negative and positive)

The included studies which investigated the association between negative coping and higher levels of depression revealed a modest but significant relationship between the two. This is consistent with findings from previous studies which have found that coping styles that are characterised by a self-destructive, avoidant or impulsive response to a given problem or stressor increase risk for subsequent depression (Compas et al., 2001, 1993). The evidence for a relationship with positive coping strategies such as active coping and support-seeking, however, was more equivocal, with a significant association emerging from the Stouffer's p analysis, but a small, non-significant mean effect size revealed in the meta-analysis. Taken together with findings from previous reviews, the findings suggest that educating adolescents about constructive responses to problems or stressors may help them more effectively navigate the physical, social, mental and emotional transitions which are characteristic of adolescence, and in so doing contribute to a decreased risk for depression.

4.1.6. Other illicit drug use

A relatively small number of studies focused on illicit drug use outside of cannabis, but they were consistent in suggesting that use of illicit drugs leads to higher levels of depression, as indicated by a small but significant combined mean effect size. This is unsurprising given the documented neurotoxic effects of illicit substances such as ecstasy and methamphetamines, giving rise to elevations in depressive symptomatology in past and/or present users (Marshall and Werb, 2010; Sumnall and Cole, 2005; Zweben et al., 2004). There is, however, a paucity of research investigating these effects on adolescent users. Given that adolescence marks a period of rapid neurological and cognitive development, this area warrants further research attention.

4.1.7. Polydrug use

A small, significant mean effect size emerged for the association between polydrug use and higher levels of depression. From the research conducted to date, it cannot be clearly elucidated to what extent a unique risk process is associated with concurrent use of substances, which is distinct from the use of these same substances in isolation. Further, it is unclear whether particular combinations of substances are more harmful than others. Confounding effects are likely to have been problematic for this theme in particular, and will be important for future studies to address.

4.1.8. Sleep

The relationship between sleep and depression is well documented, and appears to be reciprocal in nature; one of the symptoms of depression is a disruption in normal sleep patterns, and insufficient sleep can contribute to the development and worsening of depressive symptoms. Concurring with previous reviews (e.g., Clarke and Harvey, 2012), we found a consistent relationship between getting an adequate amount of sleep during adolescence, and reduced risk for subsequent depression. There is a growing evidence base to suggest that sleep is vital to support cognitive and emotional development during adolescence (Gregory and Sadeh, 2012). The education of adolescents about the importance of high quality sleep for good mental health, and communication of corresponding sleep hygiene messages, should therefore be a priority for universal prevention programs.

4.1.9. Tobacco use

The relationship between tobacco use and depression is well established, however the causal nature of this relationship has been less clearly understood (Munafò and Araya, 2010). The findings from this review are consistent with others (e.g., Chaiton et al., 2009) in finding a small but significant mean effect size between tobacco use and increased risk for depression. Due to the limitations of the available data referred to previously, we too are unable to elucidate with certainty the direction of the association. It is plausible that the relationship observed reflects either the reverse interpretation (that is, depression leads to tobacco use), or that the relationship is bidirectional; a finding which has been supported in a previous review (Chaiton et al., 2009). Further empirical attention is warranted to elucidate the underlying risk mechanisms and processes which contribute to the co-occurrence of tobacco use and depression.

4.1.10. Weight

Concurring with an earlier review (Luppino et al., 2010), we found evidence of a relationship between increasing weight in adolescence and higher levels of depression, based on a small but significant mean effect size. However, an understanding of the precise mechanisms that underlie the relationship remains elusive; as Luppino et al. (2010) note, it could be that being overweight has a negative impact on self-image or has somatic repercussions which elevate risk for depression. Alternatively, depressed persons may lead a less healthy lifestyle, and/or may suffer from dysregulation in the stress response system, which may contribute to weight gain (Atlantis and Baker, 2008; Luppino et al., 2010). Given the previous finding that dieting is associated with risk for depression, the framing of any messages around weight which are communicated to adolescents must be carefully considered. Such messages should emphasise healthy methods of maintaining a healthy weight, such that they do not inadvertently

contribute to extreme weight loss behaviours or eating disorder pathology.

4.2. Modifiable risk and protective factors with an emerging evidence base

4.2.1. Dating during adolescence

Studies examining dating during adolescence were somewhat limited and the evidence was equivocal. The meta-analysis revealed a small, non-significant mean effect size, however there was a non-significant trend towards increasing risk with romantic involvement during adolescence, and the Stouffer's *p* was significant. This finding is consistent with the notion that romantic relationships may represent a source of considerable interpersonal stress, which adolescents have not yet developed the relevant emotion regulation and coping skills to contend with (Davila, 2008). However romantic involvement during this developmental stage is both normative and salient (Collins, 2003); further, there are many positive corollaries of romantic involvement at this time, such as supporting the development of personal identity and interpersonal skills (Furman and Shaffer, 2003). It would be helpful to explore in further detail the nature of the relationship between romantic involvement and depression, as presumably there are aspects of romantic involvement which may be considered protective (e.g., confers social support), while other aspects would be expected to elevate risk (e.g., relationship stress or break-up). Finally, the included associations focused on heterosexual relationships. Future studies should explore the relation between depression and dating within non-heterosexual contexts, particularly given the elevated risk for depressive disorders among same-sex attracted and gender diverse adolescents (Marshal et al., 2011).

4.2.2. Media use

Several putative mechanisms by which media use could contribute to risk for depression have been proposed, for example through exposure to media content that is harmful (e.g., that promotes a negative body image, or models risky behaviours), by affecting the quantity and quality of adolescent sleep, or by displacing or influencing the nature of social activities and interactions (Primack et al., 2009). The findings from the present review were mixed, with the meta-analysis revealing a small, non-significant mean effect size, but a significant association emerging from the Stouffer's *p* synthesis. This may be an artefact of the exposure conceptualisation and measurement in this review which was necessarily broad, encompassing television, internet use, gaming, telephone and radio. There were insufficient studies to explore the unique effects of these forms of media use on subsequent depression outcomes. It will be important for future cohort studies to disentangle the differential effects of various forms of media use.

4.2.3. Physical activity

We found mixed evidence of an association between physical activity and lower levels of depression, with a small but non-significant mean effect size derived from the meta-analysis, but a significant association emerging in the Stouffer's *p* analysis. Taken together with the findings from other reviews (Biddle and Asare, 2011; Johnson and Taliaferro, 2011; Mammen and Faulkner, 2013), it would appear that there may be some benefit associated with physical activity during adolescence with respect to depression prevention, however these benefits may be modest in magnitude, and the evidence to date does not allow for a clear determination of the causal nature of this relationship.

4.2.4. Relationships with positive peers

The evidence linking relationships with positive peers with decreased risk for depression is mixed, with a small but non-significant mean effect size derived from the meta-analysis, but a significant association emerging from the Stouffer's *p* analysis. It is unclear from the extant evidence whether this association is causal, or reflects confounding, or both. Future research should explore the process through which affiliation with positive peers confers protection, for example, investigating whether the observed positive consequences are directly related to the quality of the relationship (i.e., a causal relation), or whether they are indirect and relate to the kinds of activities and experiences adolescents are exposed to as a consequence of their peer group (i.e., the relation is confounded; Fergusson et al., 2003).

4.2.5. Self-disclosure to parents

We identified only a small number of studies which prospectively investigated the impact of self-disclosure to parents on depression, and the findings from this review were equivocal. A small but non-significant mean effect size emerged in the meta-analytic synthesis of these associations (with more disclosure associated with less depression), but a significant association emerged from the Stouffer's *p* analysis. More prospective research is required to confirm and extend these findings, and in particular to explore the potential for confounding. For example, it may be that these associations reflect, to some extent, supportive relations with parents more generally, which may have additional protective value because they are associated with adolescents' perceived self-efficacy and high self-esteem (Shortt and Spence, 2006).

4.2.6. Sport

The evidence which links participation in sports during adolescence with lower levels of depression is equivocal, with a small but non-significant mean effect size emerging from the meta-analysis, but a significant association revealed by the Stouffer's *p* analysis. Taken together, the findings concur with those of recent reviews (Eime et al., 2013; Johnson and Taliaferro, 2011), which reported less depressive symptoms among adolescents who engaged in sport. The research in this area comprises predominantly cross-sectional studies, and those longitudinal studies that have been conducted have not always been of high quality (Eime et al., 2013). Given the physical, social, academic and emotional benefits of sport so often touted (e.g., Bailey, 2006), it is surprising that there has not been more rigorous research to clearly establish the direction of effects and the underlying causal mechanisms (e.g., increases in self-esteem, social interaction etc.).

4.3. Modifiable risk and protective factors with minimal evidence base

4.3.1. Factors for which there is no evidence of a relationship, based on the extant evidence

There were a number of studies available which examined extracurricular activities, but no association with depression was found in the meta-analytic synthesis of this evidence. This conflicts with findings from a recent review (Farb and Matjasko, 2012) which found that participation was related to psychological adjustment outcomes; this discrepancy may reflect our focus on prospective studies, whereas the majority of the published literature in this area is cross-sectional.

4.3.2. Factors for which there were limited studies available for synthesis

A number of factors had a limited number of studies available for synthesis, and for those we failed to find a significant

association with subsequent depression. These factors were: early moving out, early sex, emotion regulation strategies (positive and negative), part-time employment and religious observance (public and private). There is a clear need for greater empirical attention to these factors, using robust research methodologies which allow for a clearer determination of the causal pathways to depression.

4.4. Strengths and limitations

Strengths of the present review include the use of a clear, systematic search and evaluation protocol following the PRISMA statement (Moher et al., 2009), the inclusion of the highest level of evidence for the research question in the form of prospective cohort designs with predominantly population samples, and the supplemental use of a meta-analysis to determine pooled effect sizes where possible. Further, we systematically conducted analyses designed to detect publication bias, the results of which suggest that this was not a significant issue for this review.

There are, however, limitations associated with meta-analyses of observational studies which are not unique to this review (Egger et al., 2008). We are cognisant of the limitations of statistical combination of data from observational studies in the absence of important contextual information at the individual study level.

A significant limitation of this review is that only a subset (60%) of the associations from the included studies could be included in the meta-analysis. The primary reason for this was the way in which the data was reported in original publications. Articles commonly reported an effect size that: a) had been adjusted for various covariates, thus precluding their meaningful combination in a meta-analysis with studies using different control variables, or b) could not be converted to our chosen effect size metric. We echo the call from Yap et al. (2014) for the introduction of standardised reporting requirements for longitudinal studies in this field, specifically for the reporting of crude estimates, and estimates with adjustment for baseline symptoms only, to facilitate the integration of data from multiple datasets in meta-analytic work.

For this review, prospective studies were employed to clearly establish temporal precedence of the risk factor. However, inconsistent reporting of effect sizes and variation in the variables controlled for in analytic models meant that we had to preference unadjusted associations. Consequently, we could not control for baseline depression symptoms, and thus reverse causality cannot be ruled out. The results of our meta-analysis should thus be interpreted with caution, and in the context of the limitations of the data that was available from the included studies.

The search strategy generated such a large article yield that, for pragmatic reasons, certain exclusion criteria were applied. As the authors do not speak languages other than English, non-English language articles were excluded from the review. The search was also limited to studies subjected to peer-review, which included published dissertations and manuscripts. While this strategy may result in significant findings being oversampled, subsequent analyses for publication bias suggest this was not a significant source of bias in the present review. Longitudinal studies with a follow-up period of less than one year were excluded, as this was not considered a sufficient time frame for risk and protective factors to exert a causal influence on depression symptom levels.

4.5. Implications for research translation

This review has highlighted some key areas around which evidence-based population health messages could be developed and communicated to adolescents with a view to universal depression prevention. There is some evidence from community mental health literacy surveys that adolescents respond positively to the idea of preventive action for mental health problems, which is commensurate with the generally favourable response that is reported in the literature to prevention interventions targeting adolescent mental health (e.g., Pössel et al., 2005). These surveys have found that adolescents endorse a range of preventive strategies that may be helpful for mental health problems, some of which are congruent with the evidence base and beliefs of professionals; however, there is also some misinformation evident in adolescents' beliefs that may be counterproductive (e.g., Jorm et al., 2010; Schomerus et al., 2008; Yap et al., 2012). These studies highlight the importance of a programme of education about effective strategies for prevention during adolescence. While the results of these studies are encouraging in suggesting that preventive action for mental health may be acceptable to adolescents, more research is needed to establish to what extent these hypothetical endorsements of preventive strategies translate into a willingness to implement changes in their own lifestyles and behaviours.

4.6. Conclusions

This review adds to our understanding of the complex 'web of causation' (McMahon et al., 1960) that leads to depression, by delineating a broad set of modifiable risk and protective factors that contribute to the development of these disorders during adolescence. The findings provide support for recent claims about the importance of lifestyle for adult mental health outcomes (e.g., Jacka et al., 2012; Walsh, 2011), and suggest that these may be equally pertinent during adolescence. Previous reviews have tended to emphasise non-modifiable risk factors in conceptualising depression vulnerability early in the lifespan, such as parental psychopathology, trauma, or poverty. The findings from this and other recent reviews (Yap et al., 2014), however, suggest that there are modifiable, lifestyle-related practices that can be promoted to young people and their families to effectively reduce their level of risk for depressive disorders.

It seems likely that the risk and protective factors that emerged in this review could feasibly influence other common emotional, behavioural and physical health problems, given the known overlap in risk factors for these various disorders (O'Connell et al., 2009). Given the finite resources available to support prevention efforts, it would be useful to examine the extent to which these factors are specific to depression. The promotion of prevention messages targeting non-specific risk and protective factors, may be most efficient in reducing the collective impact of multiple problems that can co-occur during adolescence (Angold et al., 1999).

The output from this review will be instrumental in the development of evidence-based guidelines containing key actions that adolescents can take to reduce their risk for depression. The findings can also support the development of evidence-based population health messages more broadly, which could form the basis of widespread community health promotion and education strategies. Given the scale of the problem of depressive disorders, policy responses are critical to minimise the associated burden, and this review may be of further use in informing decisions in the prevention policy-making space.

Conflict of interest

None declared

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.jad.2014.08.006.

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