Prevention of eating disorders: a systematic review and meta-analysis

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TITLE

Prevention of Eating Disorders: A systematic review and meta-analysis.

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ABSTRACT

Objective To systematically review and quantify the effectiveness of Eating Disorder (ED) prevention interventions.

Methods Electronic databases (including the Cochrane Controlled Trial Register, MEDLINE, PsychInfo, EMBASE, and Scopus) were searched for published randomized controlled trials of ED prevention interventions from 2009 to 2015. Trials prior to 2009 were retrieved from prior reviews.

Results One hundred and twelve articles were included. Fifty-eight percent of trials had high risk of bias. Findings indicated small to moderate effect sizes on reduction of ED risk factors or symptoms which occurred up to three-year post-intervention. For universal prevention, media literacy (ML) interventions significantly reduced shape and weight concerns for both females (-0.69, confidence interval (CI): -1.17 to -0.22) and males (-0.32, 95%CI -0.57 to -0.07). For selective prevention, cognitive dissonance (CD) interventions were superior to control interventions in reducing ED symptoms (-0.32, 95%CI -0.52 to -0.13). Cognitive behavioural therapy (CBT) interventions had the largest effect size (-0.40, 95%CI -0.55 to -0.26) on dieting outcome at 9-month follow-up while the healthy weight intervention reduced ED risk factors and body mass index. No indicated prevention interventions were found to be effective in reducing ED risk factors.

Conclusions There are a number of promising preventive interventions for ED risk factors including CD, CBT and ML. Whether these actually lower ED incidence is, however, uncertain. Combined ED and obesity prevention interventions require further research.
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1. INTRODUCTION

1.1 Why it is important to prevent eating disorders.

Eating disorders (EDs) are serious mental disorders affecting many adolescent females and young women and are associated with significant physical and psychological impairment (Herpertz-Dahlmann, 2009; Hudson, Hiripi, Pope, & Kessler, 2007). The most well-known eating disorders, Anorexia Nervosa (AN) and Bulimia Nervosa (BN), are characterized by extreme eating behaviours and overvaluation of weight and shape. By definition, people with AN are underweight (for age and sex) and people with BN have recurrent binge eating episodes followed by compensatory weight-control behaviours such as self-induced vomiting or fasting (American Psychiatric Association, 2013). The third main ED, binge eating disorder (BED), attained diagnostic status in the fifth revision of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). It is characterised by recurrent binge eating associated with diagnostic specifiers and at least moderate distress without recurrent compensatory weight-control behaviours and without the requirement for weight/shape overvaluation (American Psychiatric Association, 2013). People with BN and BED may be normal weight, overweight or obese. Eating disorders that do not meet diagnostic criteria for AN, BN or BED may be classified under other or unspecified feeding or eating disorders (OS/UFED).

EDs are common in the general population and worldwide prevalence estimates are AN 0.21% (95% confidence interval (CI), 0.11 to 0.38), BN 0.81% (95% CI, 0.59 to 1.09), and BED 2.22% (95% CI, 1.78 to 2.76) with increased prevalence in females compared to males (Qian et al., 2013). There is evidence of an increase in the prevalence of AN in adolescent females, and increases in other EDs since the second half of the twentieth century, although the prevalence of BN may have plateaued (Qian et al., 2013). However, it was noteworthy that the incidence of EDs was stable in mental health facilities from 1970 to the 21st century (Hoek et al. 2016). This suggests that perhaps there has been growth in the incidence of ED not treated...
in mental health settings. The risk of premature death is significantly increased in individuals with EDs (Franko et al., 2014; Preti, Rocchi, Sisti, Camboni, & Miotto, 2011). Mortality is increased in all EDs with AN having the highest mortality rate of any psychiatric illness (Arcelus, Mitchell, Wales, & Nielsen, 2011). A recent review highlighted that the presence of an ED impacts substantially on health related quality of life with the degree of reported impairment increasing with the severity of the ED (Jenkins, Hoste, Meyer, & Blissett, 2011). EDs also have high rates of psychiatric and medical comorbidity, in particular anxiety disorder (Mehler & Brown, 2015; Mehler & Ryhander, 2015; Swinbourne et al., 2012). This is compounded by the current epidemic of obesity as approximately 30% to 80% of individuals with BN, BED or OS/UFED are obese (Hay, Girosi, & Mond, 2015; Hudson et al., 2007; Villarejo et al., 2012).

Furthermore, while the evidence base and options for treatments of EDs have improved in the past three decades, treatment costs are high for AN and other EDs that fail to respond to first-line therapies. Ágh et al. (2016) has reported yearly health care costs internationally of €2993 to €55,270 (equivalent to US$2227 to US$41,121, converted to US$ using purchasing power parities, found at OECD (2014)) for AN, €888 to €18,823 (~US$661 to US$14,004) for BN and €1762 to €2902 (~US$1311 to US$2159) for BED. Although rates are higher for people with AN (around 10%) all people with an ED are at risk of a severe and enduring malignant form of the disorder associated with treatment resistance, very high mortality and morbidity (Hay, Touyz, & Sud, 2012). For various reasons, treatment—and especially expert, multifaceted treatment—is not available to all, and there are unlikely to be sufficient professionals with appropriate advanced training to come close to stemming the tide of EDs using a detect-it/treat-it approach (Cooper & Bailey-Straebler, 2015). Therefore, it is essential to determine whether there are successful interventions to prevent disordered eating problems.
1.2 Aetiology of eating disorders and risk factor research.

The aetiology of EDs is multi-factorial. Genetic, epigenetic and environmental factors all play a role (Mitchison & Hay, 2014). The interplay between temperament, formative relationships and life experiences determine the development of an ED in the individual context. Twin and adoption studies have found the estimated heritability in AN to be between 28% to 74%, BN 54% to 83% and BED 41% to 57% indicating a strong genetic component (Thornton, Mazzeo, & Bulik, 2010). However, to date gene-association studies have not elucidated the genetic architecture of ED disease (Brandys, de Kovel, Kas, van Elburg, & Adan, 2015).

Each ED is likely to have a complex and potentially diverse endophenotype that may overlap with other EDs. Personality characteristics reported as increasing risks of an ED, such as perfectionism, sensitivity to reward, sensitivity to punishment, and obsessionality also have reported heritability estimates between 27% and 71% (Thornton et al., 2010). One promising area of this research is the application of epi-genetics to EDs and the identification of developmental periods where a genetic vulnerability is more likely to result in an ED. For example, in an Australian longitudinal twin study Fairweather-Schmidt and Wade (2015) reported that mid-to late adolescence may be a critical period for increased heritable risk for disordered eating. Mid-to-late adolescence represents a developmental period when non-shared environmental risk factors and weight-related peer teasing have greater impacts in the young person life. Thus prevention may be most effective for certain risk factors and at particular life stages.

There are a number of specific biological, social and psychological/behavioural risk factors associated with the development of EDs which have been identified in the literature (Brandys et al., 2015; Bulik et al., 2007; Bulik, Kleiman, & Yilmaz, 2016). It is well documented that age (adolescents & young adults) and sex (female) stand out as specific risk factors (Rosenvinge & Pettersen, 2015). Although like heritability, these risk factors are immutable.
or invariant, they may inform where and when prevention efforts may be most relevant and
effective (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). In particular, the gender
disparity is large and whilst the reasons for this are not fully understood, socio-cultural effects
and a greater influence of the thin ideal on women at vulnerable developmental periods of life
appear likely to be a major cause. Body dissatisfaction and dieting have, for many years, been
considered predictors for ED onset and mutable risk factors (Rosenvinge & Pettersen, 2014).
A recent epidemiological review (2014) found weight and shape concern, including body
dissatisfaction, to be the most studied and potent predictors for ED onset. Adolescents engaging
in dieting and disordered eating behaviours have been found to have an increased risk for these
behaviours into young adulthood (Neumark-Sztainer, Wall, Larson, Eisenberg, & Loth, 2011).
Negative affect has been found to be a predictor for onset of all types of EDs including AN,
BN, BED and purging disorder since it may decrease appetite, leading to unhealthy weight loss
and increase the likelihood of unhealthy weight control behaviours (Stice, Gau, Rohde, &
Shaw, 2016). Thin-ideal internalization, body dissatisfaction, dieting, overeating, and mental
health care have also been considered as potential predictors for BN or BED onset (Rohde,
Stice, & Marti, 2014; Stice, 2016; Stice et al., 2016). However, limited evidence from
prospective studies has supported these risk factors for AN onset. The most strong evidence to
predict onset of threshold and subthreshold AN has been found to be low body mass index
(Stice, 2016). Other mutable risk factors include sexual and physical abuse and participation in
aesthetic or weight-oriented sports (Mitchison & Hay, 2014).

Further to shared risk factors of EDs and obesity, a personal and familial history of obesity is
itself a risk for an ED and obesity is a very common co-morbidity of both BN and BED
(Campbell, Mill, Uher, & Schmidt, 2011; Hay et al., 2015; Kessler et al., 2013). For example,
research has shown that the proportion of obese females with low levels of body satisfaction
was approximately doubled compared to normal-weight females (Neumark-Sztainer, Story,
Furthermore, body dissatisfaction and dieting have been demonstrated to be shared risk factors for depressive symptoms, overweight and disordered eating (Goldschmidt, Wall, Choo, Becker, & Neumark-Sztainer, 2016). It is, therefore, vital to examine the preventive interventions targeting shared risk for eating-/weight-related issues.

1.3 Overview of prevention research in EDs

Early studies evaluating interventions with largely psycho-educational content found limited effects on reducing ED risk factors and future ED onset (Pearson, Goldklang, & Striegel-Moore, 2002; Rosenvinge & Børresen, 1999). These studies had poor methodology including uncontrolled designs, inappropriate measures and a mismatch between program goals and outcomes (Rosenvinge & Børresen, 1999; Wilksch, 2014). Studies of preventive interventions have since substantially increased during the past two decades from 22 published randomized controlled trials (RCTs) in a 2002 systematic review of preventive interventions (Pratt & Woolfenden, 2002) to 86 controlled trials in the most recent review (Bailey et al., 2014). In the most recent literature, preventive interventions have been found to produce significant effects for ED risk factors and ED symptom reductions. Cognitive behavioural therapy (CBT) based interventions including interactive and internet-based approaches, (e.g. Student Bodies program) have demonstrated reductions in ED risk factors (e.g. weight and shape concerns), the risk of ED onset in some high-risk group and ED symptoms (e.g. frequency of bingeing and purging) in females with subthreshold EDs (Jacobi, Völker, Trockel, & Taylor, 2012; Taylor et al., 2006). Cognitive dissonance (CD) interventions which are programs designed to reduce subscription to the thin-ideal (e.g. the Body Project program) have produced a 60% statistically significant and clinically meaningful reduction in Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV) ED onset over a 3-year follow-up compared to an assessment only control (Stice, Marti, Spoor, Presnell, & Shaw, 2008). However, this effect was not replicated in a follow-up high school/college effectiveness trial (Stice, Rohde, Butryn,
Shaw, & Marti, 2015; Stice, Rohde, Shaw, & Gau, 2011). Another intervention, promoting participant-driven improvements to dietary intake and physical activity (e.g. Healthy Weight program), has demonstrated a reduction of eating disorder onset by 60% over a 2-year to 3 year follow-up compared to an assessment only or educational brochure control (Stice et al., 2008; Stice, Rohde, Shaw, & Marti, 2013). Given the important association between ED and obesity, obesity prevention interventions have been recently tested in ED prevention and appeared to be effective and cost-effective in preventing disordered weight control behaviours and obesity (Austin, Field, Wiecha, Peterson, & Gortmaker, 2005; Austin et al., 2007; Wang, Nichols, & Austin, 2011).

A meta-analysis by Stice et al. (2007) found that in 51 ED prevention programs, 51% of the interventions significantly reduced at least one established risk factor for EDs and 29% significantly reduced current or future EDs (Stice, Shaw, & Marti, 2007). This meta-analysis also highlighted the programs that appeared to be most effective, were interactive, utilized multi-session formats, and were targeted to high-risk individuals (e.g. females and individuals over 15 years of age) and delivered by professionals. Additionally, the preventive interventions evaluated in trials using validated measures and a shorter follow up period had larger effects in two of six risk factor outcomes but no actual prevention of the onset of new cases were investigated. Watson et al. (2010) further investigated evidence-based interventions for ED prevention including a range of interventions from the commonly used prevention nomenclature of Mrazek and Haggerty (1994). These include universal preventive strategies targeting whole populations, selected preventive strategies targeting specific high risk populations and indicated preventive strategies targeting people with symptoms of disorders but not yet at diagnostic level. This review found that universal prevention using media literacy (ML) and selective and indicated prevention using CBT and CD interventions were effective in reducing several ED risk factors (e.g. body dissatisfaction, weight and shape concern,
negative affect…) and symptoms at post-intervention and follow-up. However, the quality of
the evidence (in terms of study design) was judged to be moderate. Importantly, a quantitative
meta-analysis and systematic quality appraisal of the included studies were not undertaken in
this review. The most recently published systematic review (conducted at the same time as the
current review), has suggested that the strongest evidence is for selective preventive
interventions (Watson et al., 2016). In particular, dissonance-based interventions demonstrated
moderate-large effects on several ED risk factors and pathology outcomes (eg. dieting, thin-
ideal internalization, negative affect, eating pathology) at post-intervention and small effects
at 6- to 18- month compared to waitlist or non-specific control. CBT and the healthy weight
program also showed small effects on these outcomes compared to waitlist and non-specific
control at both post-intervention and average of 6- to 18-month follow-up. Other interventions
including ML, psychoeducation and the multicomponent interventions showed small effects
on examined outcomes at post-intervention and under 12-month follow up compared to waitlist
controls. This review also stated that ML for universal prevention and dissonance-based
intervention and CBT for indicated prevention were effective interventions, however, this
conclusion was based on a narrative synthesis of individual studies’ results instead of a
qualitative meta-analysis (Watson et al., 2016). Furthermore, this review did not include
obesity prevention intervention studies which may be effective for EDs as well as obesity.
Methodologically, it used a random effect model for meta-analysis of selective prevention
outcomes which may substantially underestimate the statistical error. (Doi, Barendregt, Khan,
Thalib, & Williams, 2015a, 2015b; Noma, 2011).

The aim of the current study is to systematically review the recent literature of preventive
interventions for EDs across age spectrums in both general and at-risk populations. In contrast
to previous reviews, we aim to use a quality effect model to pool the effect sizes for the meta-
analysis. This approach results in a decreased mean squared error of the estimator while
maintaining the nominal level of coverage probability of the confidence interval (Doi, Barendregt, Khan, Thalib, & Williams, 2015c). This approach has also been demonstrated to be superior to the random effect model in terms of handling heterogeneity when the quality of studies is available (Doi et al., 2015c). Additionally, in opposition to the most recently published study by Watson et al. (2016), the current meta-analysis aims to quantitatively evaluate all included preventive interventions (universal, selective and indicated) in order to contribute to the growing body of literature supporting the effectiveness of preventive interventions for EDs.

2. METHODS

The methods undertaken in this review adhered to the guidelines in the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) statement 2009 (Moher, Liberati, Tetzlaff, & Altman, 2009). The original method was registered on International Prospective Register of Ongoing Systematic Reviews (PROSPERO) databases (Le, Carter, & Mihalopoulos, 2014).

2.1. Identification and selection of studies

A literature review of existing ED preventive intervention systematic/literature reviews was initially conducted to extract any reviews published between 2004 and 2014 in the Cochrane Controlled Trial Register, MEDLINE and PsychInfo. Hand-searching of the Australian National Eating Disorder Collaboration Report and International Journal of Eating Disorders was also undertaken. The key words used in all these searches included “review or systematic review or meta-analysis or meta-analytic” and “eating disorder* or anorexia or bulimia”.

This initial search yielded 109 reviews. After screening and removal of duplicates, the 2010 systematic review by Watson et al. was used as the current starting point (Watson, Elphick, Dreher, Steele, & Wilksch, 2010). The advantage of this review is that it was a high quality
review of systematic reviews and randomized controlled trials (RCTs) for prevention of ED from 2004 to 2009 and categorized the interventions according to intervention strategy. However, a quantitative meta-analysis to pool effects was not undertaken. Importantly, in order to ensure that any RCTs prior to 2009 were included in the current review (and possibly excluded from Watson et al. 2010), other reviews were screened to retrieve any RCTs which were not included in Watson et al. (2010). A systematic review by Stice et al. (2007) provided 19 more RCTs for preventive interventions which were published prior to 2006.

To ensure all relevant studies were included for the current review, an update of both Watson et al (2010) and Stice et al (2007) was undertaken. The same search term strategy as reported in Watson et al. (2010) was used. The electronic databases including the Cochrane Controlled Trial Register, MEDLINE, PsychInfo, EMBASE, and Scopus were searched until December 2015. Hand-searching was conducted in the table of contents for journals which commonly publish eating disorders studies (e.g. International Journal of Eating Disorders, Journal of Eating Disorders and European Eating Disorders Review). The reference sections of all articles (e.g. review papers, review chapters or review books) were also examined. The trials were selected for inclusion if they were RCTs or quasi-RCTs (e.g. randomised by school), published in English, and evaluated a preventive intervention for ED compared to another control preventive intervention or wait list/ no intervention or minimal intervention control. Controlled trials, which did not state “random assignment”, were excluded.

2.2. Data extraction

All citations were imported into an electronic database (Endnote® version X2). Included RCTs were extracted by the first author (LKDL) and information from all studies was extracted including: recruitment method and target group (females or males); outline of interventions evaluated (e.g., format, duration of intervention, number of sessions); and a description of main outcome measures. Preventive interventions were classified into the three main types using the
nomenclature of Mrazek and Haggerty (1994). A preventive intervention was considered universal if it targeted a whole population (e.g. school based prevention for females and males). Selective prevention interventions targeted a subgroup of the population at risk of developing an eating disorder (e.g. females with/without elevated scores on risk indicators). Indicated prevention interventions targeted people who have some symptoms of a disorder without meeting full diagnostic criteria (e.g. sub-threshold EDs). The approach of each intervention program was classified into 10 sub-types by the review authors, based upon the information provided in the published studies. The subtype classification reported in Watson et al. (2010) was used in this classification procedure (e.g., CBT) with the addition of mindfulness based interventions which applied key aspects of mindfulness and acceptance-based practice specifically to body image. Interventions which contained only one session were classified as “one-shot” interventions regardless of the length of single session (e.g. 2-hour workshop). The data or information regarding features of any of the studies, which were not included in the published articles were obtained by contacting the first author via e-mail. In the Wilksch et al. (2015) study, the number of females and males in each arm was provided by the first author. In the series of studies evaluating CD and healthy weight programs, the leading author explained the different versions of CD and healthy weight interventions used in these studies (Professor Eric Stice, personal communication).

2.3. Meta-analysis methods

A quantitative meta-analysis was undertaken to pool effects across the RCTs using an EXCEL add-in package, MetaXL (Barendregt & Doi, 2015).

2.3.1. Quality assessment

The Cochrane Collaboration criteria were used to evaluate the risk of bias (RoB) for the included RCTs. The RoB criteria include quality ratings related to: random sequence generation; allocation concealment; blinding of participants; outcome assessment; incomplete
outcome data; and, selective outcome reporting (Higgins, 2011). The RoB of each trial was assessed on each criterion and categorized as 'low', 'high', or 'unclear’. The criterion relating to blinding of participants was not used, as blinding is not possible in the types of interventions included in this review. Complete outcome data were defined as 'low risk’ when the percentage of participants lost to follow-up was low (arbitrarily set at values lower than 20%) and when numbers and causes of loss to follow-up were balanced between arms. In order to observe how the quality of RCTs impacted the effect size of interventions a quality score was determined by assigning 3 points to ‘low risk’, 2 to ‘unclear’, and 1 to ‘high risk’. The derived quality score of each individual study was normalized by dividing the maximum possible quality score (called the quality index (Qi) range: 0–1). Subsequently, Qi values were entered in the quality effects model as described by Doi et al. (2011; 2008).

2.3.2. Data analysis

An effect size was calculated to determine the difference between the intervention group and the comparison group at post-treatment or follow-up if available. If both intention-to-treat and per-protocol data were presented, the former estimate was used in the meta-analysis. A Hedges’ g standardized mean difference (SMD) was used where trials measured the same outcome using different scales. A weighted mean difference (WMD) was used for trials which used the same continuous outcome measured on the same scale. For dichotomous data, a relative risk was used to estimate the effect of the intervention. Confidence intervals (CIs) were obtained for all estimates. Effect sizes of around 0.2, 0.5 and 0.8 were respectively considered small, moderate and large (Cohen, 1988). The quality-effect (QE) model was used to pool the data, with the random-effect (RE) model used for standardized comparison. The advantage of the QE meta-analysis is that it allows for redistribution of individual studies’ inverse variance weights based on assessed study deficiencies rather than the usual random redistribution of weights seen with the RE model (Doi et al., 2011; Thalib & Doi, 2011). Results from the quality-effect model are
presented except in the case where the random-effect model showed a different significant trend. In this instance both results from the random effects and the quality effects models are presented.

Statistical heterogeneity was assessed using the $I^2$ and Cochran's Q test. Heterogeneity was regarded as substantial when the $I^2$ statistic exceeded 40% and/or the Q statistic was significant at a $P < 0.10$, as per the Cochrane collaboration recommendations (Higgins, 2011). Pooled effect sizes were examined to identify whether they were driven by outliers, defined as studies in which the effect sizes and 95% CIs were outside the 95% CIs of the pooled studies (on both sides of the CI) (Cristea, Kok, & Cuijpers, 2015).

2.3.3. Sensitivity analysis and publication bias

To assess the robustness of the meta-analyses, three main sensitivity analyses were undertaken. Firstly, the quality of studies was tested. This sensitivity analysis evaluated the impact of study quality by removing low quality trials (e.g. quasi-RCTs) from the meta-analysis. Secondly, one-way sensitivity analyses which excluded individual studies sequentially from the meta-analyses were performed. While this form of sensitivity analyses is not traditionally undertaken it is a very useful as it examines which study is the prime determinant of the pooled results (Barendregt & Doi, 2015). The final sensitivity analysis considered the pooled results of selective and indicated prevention interventions based on broader classifications of selective and indicated prevention for EDs which have been used in previous reviews (Levine & Piran, 2004; Rodgers & Paxton, 2014; Yager, Diedrichs, Ricciardelli, & Halliwell, 2013). For this broader classification, we still classified preventive intervention studies as selective if they allowed participants to self-select as having high level of ED attitudes/behaviours (e.g. high body dissatisfaction). The main difference is that we allowed interventions which have formally screened participants for both ED symptoms and/or behaviours (but may or may not formally meet the criteria for sub-threshold disorder) to be classified as indicated prevention.
Therefore in this sensitivity analysis there are more interventions classified as indicated since our inclusion criteria is less strict for this classification. The outcomes of the meta-analysis can be considered robust when sensitivity analyses in subsets of studies show similar pooled effect sizes.

The analysis of the effect of potential unpublished studies or missed studies with negative results was undertaken to evaluate the robustness of the meta-analysis. Funnel plots and Doi plots were used to evaluate the presence of publication bias. Interpretation of Doi plots is similar to the funnel plot in that a symmetrical plot suggests no or little publication bias while an asymmetrical plot suggests bias (Barendregt & Doi, 2015). However, the Doi plot is easier to interpret than the funnel plot. The Doi plot displays the Luis Furuya-Kanamori (LFK) index of asymmetry, including an assessment of severity (‘No’, ‘Minor’, ‘Major’) asymmetry (Barendregt & Doi, 2015).

3. RESULTS

3.1. Selection and inclusion of studies

The systematic searches resulted in a total of 2,187 abstracts after removal of 1,603 duplicates. A total of 411 papers were retrieved, of which 299 were excluded because they did not meet the inclusion criteria. Figure 1 presents a PRISMA flowchart describing the inclusion process, and includes an overview of the reasons for exclusion of 299 studies. A total of 112 articles (describing 101 trials) met all inclusion criteria. Of these, 78 articles have been reported in previous systematic reviews (Stice et al., 2007; Watson et al., 2010).

[INSERT FIGURE 1 HERE]

In terms of preventive strategies, there were 18 trials which evaluated universal prevention interventions, 79 trials which evaluated selective prevention interventions and 4 trials which
evaluated indicated prevention. The studies were carried out in the U.S., Australia, UK, Spain, Italy, Mexico, Sweden, Norway, Canada, Israel, Germany and the Netherlands. The smallest study had 24 participants and the largest 1451.

3.2. Study characteristics

There were seven, twelve and two types of interventions evaluated under the umbrellas of universal, selective and indicated prevention, respectively. ML was the most frequently evaluated universal prevention intervention (five trials) followed by self-esteem enhancement, obesity prevention and multicomponent interventions (all were evaluated in four trials). Cognitive dissonance and CBT were the most commonly evaluated selective prevention interventions followed by the one-shot intervention (12 trials). Other commonly evaluated selective interventions included multi-component programs (nine trials) and ML and healthy weight program (both had seven trials). Only CBT and psychoeducation were evaluated as indicated preventive interventions.

Most of the trials assessed preventive interventions aimed at children, adolescents and youth (defined as aged up to 24 years). In particular, universal prevention was evaluated in 18 trials which included children or adolescents with mean ages of 11 to 14 years while selective prevention and indicated prevention mostly included participants with mean ages of 16 to 24 years. Only two studies from selective prevention included adult participants aged 18 up to 68 years.

Preventive interventions for ED were mainly group-based delivered via classes, face-to-face or online. Most universal interventions where group-based delivered in classes. For selective prevention, the ML, multicomponent, one-shot, perfectionism and obesity prevention interventions also used group formats delivered by school classes while the rest were group-based delivered face-to-face or online. The majority of control groups were waitlist or delayed
treatment or non-specified control. A small number of studies also used active interventions or minimal treatments as control groups.

More generally, the ED preventive interventions consisted of multiple sessions and interactive formats which helped participants engage in the interventions. Notably, the intensity and content of the interventions varied considerably. For universal prevention, the number of sessions ranged from four sessions to many sessions spanning two-years (the obesity prevention). Selective preventive interventions mostly ranged from four to eight sessions.

Twelve studies evaluated single session group format interventions of CBT, CD, acceptance and commitment therapy, obesity prevention and psychoeducation. These sessions varied from 45 minutes to one-day workshop. CBT interventions ranged from two sessions to sessions over a 16-week duration however the Student Bodies program (eight weekly sessions) was the most commonly evaluated (9/22 trials). The Body Project program, which is a CD interventions consisted of four 1-hour sessions or six 45-minute sessions or two 2-hour sessions. Of the indicated preventive interventions, CBT ranged from 8 to 16 sessions while psycho-education was only three sessions.

Further details of interventions may be found in APPENDIX A.1.

3.3. Risk of bias assessment

In general, most trials (58% of trials) had high risk of bias. Risk of bias was mostly evident through lack of blinded outcome assessments (in 37% of studies) and incomplete outcome data (in 30% of trials). In particular, 83% of universal prevention trials did not include blinded outcome assessment compared to 27% of selective prevention trials and 25% of indicated prevention trials. The majority of trials (50%) did not report random sequence generation or allocation concealment in the randomization process. Therefore, the level of bias across studies was unclear or high risk. It is noted that some of the trials included in this meta-analysis were
effectiveness trials which used “routine-care” type of providers and often relaxed
inclusion/exclusion criteria so that results are more generalizable to real world settings.
Findings are displayed in Figure 2 and further details can be found in SUPPLEMENTARY
APPENDIX A.1.

3.4. Summary of results of meta-analysis.
There were 112 pooled comparisons between the active interventions versus no or minimal
controls and 12 pooled comparisons amongst two active interventions. Results of pooled
comparison are summarized in SUPPLEMENTARY APPENDIX A.2.

3.4.1. Universal Prevention for Eating Disorders
3.4.1.1. Active interventions versus no/minimal treatment controls
There were 38 pooled comparisons of CBT, ML, multicomponent, obesity prevention, and self-
esteeem enhancement interventions. No pooled comparisons were possible for physical activity
and psycho-education interventions due to only one available study. However, the physical
activity intervention had a negative effect on drive for thinness for females and no effect on
body dissatisfaction (Zabinski, Calfas, Gehrman, Wilfley, & Sallis, 2001). The psycho-
education improved knowledge about EDs but whether this reduced ED risk factors for the
target groups was unclear (McVey, Gusella, Tweed, & Ferrari, 2008).

CBT interventions. Pooled comparison for ED behaviour revealed no significant differences
between CBT interventions compared with control at post-test and 6- to 12-month follow up
(WMD 0.44, 95% CI: -7.00 to 7.89; I²=92%, P=12.86, P=0.00).

ML interventions. There were significant reductions in weight and shape concern of the
population at post-test and at follow up (6 to 30 months) (g for females -0.69, 95% CI: -1.17
to -0.22, $I^2=39\%$, $Q=1.64$, $P=0.20$ and for males -0.32, 95% CI: -0.57 to -0.07, $I^2=0\%$, $Q=0.95$, $P=0.33$). Furthermore, pooled effects showed significant differences for media internalization outcomes for males at both examined times and females at post-test (g -0.21, 95% CI: -0.34 to -0.07). No significant differences were found on dieting, self-esteem, and body dissatisfaction when the studies examining ML interventions were pooled.

**Multicomponent interventions.** Two comparisons at post-intervention and at 6- to 30-month follow up of eating behaviours for females revealed significant differences (follow up: -0.59, 95% CI: -0.77 to -0.42; $I^2=0\%$, $Q=0.49$, $P=0.48$).

**Obesity prevention interventions** including traditional obesity prevention targets and/or modified versions plus targeting psychological risk factors associated with healthy eating were found to significantly reduce the thin-ideal internalization for males at post-test (WMD -1.75, 95% CI: -3.10 to -0.40; $I^2=0\%$, $Q=0.11$, $p=0.74$).

**Self-esteem enhancement interventions** did not significantly reduce dieting at post-test and 3- to 12-month follow up for whole population. However, subgroup analysis revealed the significant reductions of dieting in females but not males at post-test and follow up (g = -0.99, 95% CI: -1.39 to -0.58; $I^2=10\%$, $Q= 1.12$, $p=0.29$).

### 3.4.1.2. Active interventions versus other preventive intervention controls

Three trials evaluated ML interventions vs. other preventive interventions including obesity prevention, self-esteem enhancement and multicomponent interventions (González, Penelo, Gutiérrez, & Raich, 2011; Wade, Davidson, & O'Dea, 2003; Wilksch et al., 2015). There was a significant reduction on media internalization for male adolescents at post-test (g = -0.35, 95% CI -0.52 to -0.18; $I^2 = 0\%$, $Q = 0.20$, $p =0.65$) and 12- to 30-month follow up (g = -0.20, 95% CI -0.37 to -0.03; $I^2= 0\%$, $Q=0.19$, $p=0.67$). No significant differences among these
interventions was found for other outcomes including weight concern, shape concern and dieting.

**Summary findings for universal prevention**

ML was the only intervention that had small to moderate effect sizes of risk factor reductions including weight and shape concerns and media internalization for both females and males at post-intervention and up to 30-month follow up compared to non-intervention controls. Furthermore, this intervention was superior to other active intervention controls in reducing media internalization of males at post-intervention and up to 30-month follow up. The multicomponent intervention had moderate effect sizes on reduction of eating behaviours and media internalization while self-esteem enhancement interventions demonstrated large effect sizes on dieting reduction. However, these effects were found only for females. No evidence of efficacy for CBT or obesity prevention interventions were found for ED prevention.

**3.4.2. Selective Prevention for Eating Disorders**

**3.4.2.1. Active interventions versus no/minimal treatment control**

Eighty-three trials, reporting 12 different types of interventions of selective prevention were retrieved. In three studies examining obesity prevention interventions, no pooled comparisons were possible due to different outcomes although some of these studies reported significant differences between evaluated groups. The Planet Health program reduced the number of females who reported purging or using diet pills by 50% relative to a non-specified control (Austin et al., 2005) while the low-calorie diet program reported a reduction in bulimic symptoms (Presnell & Stice, 2003). The New Moves program also reduced the proportion of females using unhealthy weight control behaviours by 13.7% and significantly improved body dissatisfaction (Neumark-Sztainer et al., 2010). Similarly, no pooled comparisons were possible for self-esteem enhancement and perfectionism due to only one available study. Both
interventions reported little effects on ED risk factors and these were not maintained at follow up (Weiss & Wertheim, 2005; Wilksch, Durbridge, & Wade, 2008).

**CBT interventions.** In the meta-analysis of 18 trials, where data were available, significant differences were observed between active interventions vs. controls at both post-test and 4-week to 9-month follow up for dieting (at follow up $g = -0.40$, 95% CI: -0.55 to -0.26; $I^2=0\%$, $Q=7.83$, $p=0.45$) and bulimic behaviours (at follow up $g = -0.20$, 95% CI -0.35 to -0.05; $I^2=0\%$, $Q=2.71$, $p=0.74$). CBT interventions also reduced body dissatisfaction at up to 9-months post-intervention ($g = -0.23$, 95% CI: -0.42 to -0.04; $I^2=0\%$, $Q=5.35$, $p=0.62$). No significant differences were found for other outcomes including shape concern, weight concern, eating concern, BMI and self-esteem outcomes at both post-test and follow up.

**Media literacy interventions** had a significant reduction in internalization (or acceptance) of societal ideals relating to appearance with substantial heterogeneity compared to no treatment controls at 3-month follow up ($g = -1.53$, 95% CI: -2.90 to -0.15; $I^2 = 99\%$, $Q = 198.58$, $p = 0.00$) but not at post-test ($g = -1.20$, 95% CI: -2.46 to 0.06; $I^2 = 99\%$, $Q = 276.40$, $p = 0.00$). With the exclusion of one outlier (Sharpe, Schober, Treasure, & Schmidt, 2013), the effect size at post-test decreased to a $g$ of -0.45 (95% CI -1.02 to 0.12) and the heterogeneity remained significant ($I^2 = 90\%$, $Q = 30.50$, $p = 0.00$).

**One-shot interventions.** Four pooled comparisons of one-session interventions on thin-ideal internalization, dieting, body dissatisfaction, and self-esteem outcome were possible with no significant differences observed between the intervention and control groups.

**Cognitive dissonance interventions.** Given the various points of follow-up across the studies examining CD interventions, results were pooled at post-intervention, 3- to 12- month follow-up and over 12-month follow-up. The pooled analysis indicated that there were statistically significant differences at both post-test and 3- to 12-month follow up in body dissatisfaction ($g$}
= -0.20, 95% CI -0.39 to -0.02); dieting (g = -0.28, 95% CI -0.43 to -0.12); and negative affect (g = -0.23, 95% CI -0.35 to -0.10). However, these effects were not maintained at the over 12-month follow up. Furthermore, CD interventions were superior to control in reducing thin-ideal internalization at post intervention (g = -0.71, 95% CI (-1.14 to -0.27) with substantial heterogeneity ($I^2 = 92\%$)) and at the 3-year follow up (g = -0.16, 95% CI -0.27 to -0.05), heterogeneity was moderate ($I^2 = 40\%$). Removal of one outlier (Serdar et al., 2014) at post-test increased the effect size to $g = -0.61$, 95% CI (-0.77 to -0.45) and heterogeneity was no longer significant. Importantly, CD interventions significantly reduced ED symptoms (measured by the Eating Disorder Diagnostic Interview/ Survey) at post intervention (g = -0.30, 95% CI -0.47 to -0.13 with substantial heterogeneity $I^2 = 51\%$, $Q = 16.49$, $p = 0.04$).

However with the exclusion of one outlier (Serdar et al., 2014), the effect size decreased to $g = -0.25$ (95% CI -0.37 to -0.14) and heterogeneity became insignificant but this effect was not maintained at 3- to 12-month ($g = -0.06$, 95% CI -0.19 to 0.06) and over 12-month follow up (Hedges’ $g = -0.08$, 95% CI -0.22 to 0.07).

**Healthy weight intervention.** There were four trials which compared a healthy weight intervention versus minimal/non-treatment control (Smith & Petrie, 2008; Stice, Rohde, Shaw, & Marti, 2012; Stice, Shaw, Burton, & Wade, 2006; Stice, Trost, & Chase, 2003). At follow-up, difference in dieting ($g = -0.30$, 95%CI -0.57 to -0.04) and thin-ideal internalization scores ($g = -0.27$, 95% CI: -0.49 to -0.05) were observed. Differences at post-test of body dissatisfaction (Hedges’ $g = -0.28$, 95% CI -0.45 to -0.12) were also observed. Importantly, use of the RE model resulted in significant differences in level of the body dissatisfaction ($g = -0.35$, 95% CI: -0.61 to -0.08) at follow up. In contrast, use of the QE model did not result in significant differences. Participants in the healthy weight intervention had a lower increase in BMI compared to controls at 2- and 3-year follow up (WMD -0.90, 95% CI -1.61 to -0.18), which indicated that this intervention might be effective for obesity prevention as well. No
significant differences were found for other outcome measures including negative affect and bulimic behaviours.

**Psychoeducation interventions.** No significant difference between active interventions compared to controls was found for body dissatisfaction (g = -0.25, 95% CI: -0.96 to 0.45; I²=79%, Q=9.66, p=0.01).

**Mindfulness based interventions.** Pooled comparison of studies investigating mindfulness based interventions revealed no significant differences in dieting outcome (g = 0.38, 95% CI: -0.04 to 0.80) between active intervention and control.

**Multicomponent interventions.** Pooled comparisons of three trials resulted in significant Hedges’ g of -0.15 (95% CI -0.28 to -0.02) favouring the active intervention for eating behaviour outcomes. This intervention was also superior to non-intervention control in reducing media internalization at post-intervention and 6-month follow up.

3.4.2.2. Active interventions versus other preventive intervention control.

There were four trials comparing ML interventions vs. other preventive interventions including CD, multi-component and perfectionism interventions (Becker, Bull, Schaumberg, Cauble, & Franco, 2008; Becker, Smith, & Ciao, 2006; López-Guimerà, Sánchez-Carracedo, Fauquet, Portell, & Raich, 2011; Wilksch et al., 2008). No significant differences between these interventions were found for thin-ideal internalization and dieting outcome at both post-test and at follow up.

In the meta-analysis of nine trials comparing CD interventions to other preventive interventions including healthy weight interventions and ML interventions. Significant differences were found for thin-ideal internalization at post-test (g = -0.19, 95% CI -0.32 to -0.06; I² = 0%, Q= 6.89, p= 0.44) but this effect was not maintained at 4-week to 3-year follow up (g= -0.05, 95%
CI -0.25 to 0.15; $I^2 = 54\%$, $Q= 13.15$, $p= 0.04$). Subgroup analysis revealed no significant difference between CD and ML. No differences were found for other outcomes including body dissatisfaction, dieting, and bulimic behaviours.

**Summary findings for selective prevention**

CD interventions demonstrated small to moderate effect size with substantial heterogeneity on a number of outcomes including dieting, thin ideal internalization, body dissatisfaction, and negative affect at post-intervention and 12-month follow up. The effect of CD on reducing thin ideal internalization was maintained up to 3-year follow up. The healthy weight program had small effect on ED risk factors at post-intervention as well as 12-month follow up and large effect size with zero heterogeneity on BMI at 12-month follow up. CBT was found to be effective in reducing ED risk factors including body dissatisfaction, dieting, and bulimic symptoms with small to moderate effects at post-intervention and up to 9-month follow up. The effects were small for other interventions including multicomponent and media literacy. Across active interventions, CD was superior to healthy weight program in reducing media internalization at post-intervention but it was not maintained at follow up. No significant differences were found between CD and ML or ML and other interventions.

**3.4.3. Indicated eating disorder prevention**

**3.4.3.1. Active interventions versus no/minimal treatment control**

Of the four studies which were classified as indicated prevention, three including Jacobi et al. (2012), Jones et al. (2008) and Paxton, McLean, Gollings, Faulkner, and Wertheim (2007) evaluated CBT interventions. In the meta-analysis of these trials, where data were available, no significant differences of dieting, weight and shape concern, body dissatisfaction, BMI and bulimic behaviour outcomes between active interventions compared with controls were observed. Another study by Buddeberg-Fischer, Klaghofer, Gnam, and Buddeberg (1998)
evaluated a psychoeducation intervention which did not demonstrate any significant differences between the intervention and non-specified control.

3.4.3.2. Active interventions versus other preventive intervention control.

No pooled comparisons were possible between the different preventive intervention controls.

3.4.4. Sensitivity analysis

In the first sensitivity analysis to assess the “quality of the studies”, the effect of the multicomponent intervention in universal prevention relied on three quasi-RCTs. Therefore, no pooled comparison were possible when these studies were removed. For the self-esteem enhancement intervention (universal preventive intervention), when low quality studies were removed, there were no changes to the significance of results and no pooled comparisons for subgroup analysis (e.g. females/males) were possible. In the meta-analysis of the five studies which evaluated a multicomponent intervention (selective prevention), when three quasi-RCTs were removed, the pooled effect of eating disturbance became insignificant (Hedges’ g = -0.14, 95% CI: -0.41 to 0.12) and no pooled comparison were possible for other examined outcomes. When two of the four studies evaluating the ML intervention (selective prevention) were removed, there were no changes to the significance or direction of any results.

One-way sensitivity analyses for the QE models were undertaken by excluding each study in turn. For universal prevention, the ML intervention became insignificant for reduction of media internalization in boy adolescents at post-test and at follow up when the trial by González et al. (2011) or Wilksch et al. (2015) were excluded. In contrast, when the trial by Wilksch (2015) was excluded the effect size of ML interventions on dieting and body dissatisfaction outcomes in males at post-test became significant and the heterogeneity remained insignificant. For selective prevention, the multicomponent interventions became significant in reducing ED behaviours at post-test when the trial by McVey and Davis (2002) was excluded but lost
significance at follow up when the trials of López-Guimerà et al. (2011) or Franko et al. (2005) were excluded. Excluding the Wilksch et al. (2008) trial significantly increased the effect size of ML interventions in media internalization outcome at post-test ($g= -1.39, 95\% CI -2.75$ to $-0.03; I^2=99\%, Q=227, p<0.000$) while excluding the Sharpe et al. (2013) or Neumark-Sztainer, Sherwood, Coller, and Hannan (2000) trials led to an insignificant difference in the effect size at follow up. The impact on body dissatisfaction of the healthy weight interventions became insignificant when the Stice et al. (2006) trial was excluded at post-test but this effect at follow up became significant and the heterogeneity was no longer insignificant when the Stice et al. (2003) trial was excluded. The reduction of negative affect at post-test for CD interventions became insignificant when the Stice et al. (2006) or the Stice, Rohde, Durant, Shaw, and Wade (2013) trials were excluded. Similarly, body dissatisfaction at 1-year follow up became insignificant when any trials, except the Stice et al. (2011) or Rohde, Auslander, et al. (2014) trials, were excluded; and thin-ideal internalization at 2- to 3-year follow up became insignificant when the Stice et al. (2011) or Stice et al. (2015) trials were excluded. CBT lost significance in reduction of bulimic behaviours at post-test in comparison to controls when excluding any of trials by Taylor et al. (2006); Celio et al. (2000); Jacobi et al. (2007); Bearman, Stice, and Chase (2003); Heinicke, Paxton, McLean, and Wertheim (2007); or, Graff Low et al. (2006). In contrast, with the exclusion of the Winzelberg et al. (1998) or Doyle et al. (2008) studies, the effect size for weight concern at follow up became significant. Excluding Butters and Cash (1987) or Nicolino, Martz, and Curtin (2002) trials resulted in significant difference for body dissatisfaction of CBT interventions at post-test compared to controls.

With regard to the last sensitivity analysis (where studied were re-classified according to broad classification as specified above) there were 60 studies which were classified as selective prevention and 23 studies classified as indicated prevention. No significant changes were observed in the results for CD, media literacy, healthy weight, and one shot interventions
regardless of how they were classified. In particular, the Atkinson and Wade (2014) study from CD interventions, and the Shafran, Farrell, Lee, and Fairburn (2009) study from one shot interventions was categorised as indicated prevention. The psychoeducation and mindfulness-based interventions were classified as indicated prevention according to the broader classifications. Furthermore, while only three CBT studies were classified as indicated in the primary analyses, under the broader classification 16 studies were classified as indicated. This obviously then meant that few studies were classified as selective prevention in this sensitivity analysis. The CBT interventions classified as selective prevention significantly reduced dieting (at follow up g = -0.46, 95% CI: -0.73 to -0.19) and bulimic behaviours (at follow up g = -0.33, 95% CI -0.56 to -0.09) at both post-test and 9-month follow up. In contrast to the primary analyses which used the narrower classification, the indicated CBT interventions significantly reduced ED pathology (EDE-Q global score at follow up WMD = -0.40, 95% CI -0.59 to -0.22), dieting (at follow up g = -0.38, 95% CI: -0.54 to -0.22) and shape and weight concerns (at follow up g = -0.34, 95% CI -0.53 to -0.15) at both post-test and 4-week to 12-month follow up.

3.4.5. Publication bias

Risk of publication bias was examined by visual inspection together with LFK index. Most of the pooled effect results (34 of 38) of interventions from the universal prevention comparisons exhibited minor and major asymmetrical Doi plots, indicating a possibility of publication bias. Similarly, asymmetrical Doi plots were found for 37 of 65 pooled effect results of interventions from selective prevention and six of seven pooled effect results of interventions from indicated prevention. More details (e.g. LFK index) of risk of publication bias are presented in SUPPLEMENTARY APPENDIX A.3.
4. DISCUSSION

4.1. Main findings

In the current study, 101 RCTs were extracted from 112 articles and described 13 types of ED preventive interventions spanning universal, selective and indicated interventions. One hundred and twelve pooled comparisons between active interventions and no/minimal intervention controls and twelve pooled comparisons among the active interventions were conducted. The current review included 34 more articles than previous reviews, which represented a 43% increase. To our knowledge, this is the first systematic review followed by a qualitative meta-analysis using the updated quality effect estimator to pool the effect size for all interventions including obesity prevention interventions regardless preventive strategy. Previous meta-analysis of high quality trials (including only RCTs) focused on only adolescents and children and did not identify prevention strategies (Pratt & Woolfenden, 2002). The meta-analytic review published by Stice et al. (2007) identified moderators for ED preventive intervention that produced the largest intervention effects based on RCTs and non-randomized controlled trials but it did not determine which type of preventive interventions were the most effective. A parallel review from Watson et al. (2016) did not conduct a qualitative meta-analysis for universal and indicated prevention.

The current review found, for universal prevention, only the ML interventions, targeting adolescents, were effective for reducing ED risk factors up to 30 months after intervention for both females and males. This finding is consistent with previous systematic reviews (Pratt & Woolfenden, 2002; Watson et al., 2010; Watson et al., 2016). It is noteworthy that the pooled results of some examined outcomes (media internalization, dieting, and body dissatisfaction for males) of ML intervention were not robust after excluding one single study from the sensitivity analysis. This may not validate the statistically significant outcomes of the current results. We also found that other universal preventive interventions including multicomponent
and self-esteem enhancement interventions were effective in only females. However, these results should be interpreted with some caution because of the limited quantity and quality of trials (e.g. 2-3 trials included in pooled comparisons) and/or different scales to measure the same outcomes. In particular, Gonzalez et al. (2011) used EAT-40 while McVey et al. (2007) used EAT-children version). Similarly, the pooled comparison for dieting outcomes related to self-esteem enhancement interventions came from two studies in which one study used school randomization (Martinsen et al., 2014). Given that baseline scores of risk factors are low in universal prevention, statistically significant results are difficult to detect particularly where there is insufficient long-term follow-up (Wilksch, 2014). A more accurate test of a “prevention effect” would require larger, more representative samples with sufficiently long follow up time period to detect changes in ED and their associated risk factors (Muñoz, Beardslee, & Leykin, 2012).

For selective prevention, the CD intervention was the only intervention found to be superior to controls in reducing ED behaviours and symptoms up to 3 years post intervention and was superior to other preventive interventions in reducing thin-ideal internalization at post-test. It is important to note that the ED symptoms were assessed by diagnostic interviews and surveys which are used to reflect the broader array of disordered eating and may not map directly onto the DSM diagnostic criteria (Stice, Ng, & Shaw, 2010). Healthy weight interventions improved both eating disorder risk factors/behaviours and body mass index. However, these findings should be interpreted with caution because the CD and healthy weight programs were different across the trials and the results of the meta-analysis were not robust in the sensitivity analysis. Other selective prevention interventions including CBT and ML interventions appeared to significantly reduce ED behaviours/ risk factors such as dieting, bulimic behaviours, body dissatisfaction and media internalization at 3- to 8-month post intervention. Multicomponent interventions were superior to control in reduction in ED behaviours at up to 1 year post.
intervention but this effect became insignificant when low quality trials were removed. These results were consistent with the results from the review conducted at the same time as the current review by an independent team (Watson et al., 2016). It is noted that in the sensitivity analyses where individual studies were excluded, the pooled effects of selected examined outcomes (i.e. ED behaviours, body dissatisfaction, bulimic symptoms) varied substantially. These results were consistent with the results from the review conducted at the same time as the current review by an independent team (Watson et al., 2016). Further research is required to determine the most effective versions of the selective prevention based interventions for reducing ED risk factors and symptoms in high-risk population. As there is reasonably strong evidence for selective prevention, it is important to also undertake “real-world” effectiveness trials which will determine whether any gains are maintained within routine settings (Shaw & Stice, 2016; Stice, Becker, & Yokum, 2013).

For indicated prevention, there is insufficient evidence, when using the narrower classification system for indicated prevention, to suggest a positive effect of CBT interventions in preventing risk factors and behaviours of EDs. The number of studies classified as indicated prevention in the primary analyses in our review is lower compared to those in the review by Watson et al. 2016. This is partly because our classification allowed each study to be only classified under one category while the Watson et al. 2016 review classified some studies as both selective and indicative prevention (Franko et al., 2005; Green, Scott, Diyankova, & Gasser, 2005). It is worth noting that in the mental health prevention intervention spectrum, there might be a shading of selective into indicated prevention, with the distinction between the two sometimes not clear (Levine, 2016). It is often not immediately apparent whether interventions are selective or indicated since high risk populations (i.e. selective interventions) may also be considered as showing early signs of illness (indicated). The sensitivity analysis demonstrated
no significant changes were found in the results for CD, media literacy, healthy weight, and one shot interventions regardless of how they were classified. The most important changes in the results were observed for the CBT interventions based indicated prevention. In particular, these interventions became effective in reducing ED behaviours, dieting and shape and weight concerns at post-test and up to 12-month follow up when the broad category was used. Therefore, it is very important that further research examines effective indicated prevention interventions in order to develop and refine them by identifying those who are at greatest risk of ED onset (Stice et al., 2010).

“Challenging the thin-ideal” is not only a part of many psychological therapies in eating disorders (e.g., CBT) but is important in the broader public health arena. “Implementing prevention programs at an early age when behaviour is more amenable to change may produce better outcomes than treatment delivered when rigid patterns of cognition and behaviour have already been established and are engrained” (Werner-Seidler, Perry, Calear, Newby, & Christensen, 2017). Additionally, clinicians should have a good understanding of the evidence for prevention programs as it is a part of psychoeducation and a common inquiry from people with the lived experience, either during clinical consultations or when the clinician is contributing to a community educational seminar or similar. It is also important where the clinician is joining with people with the lived experience and others in advocating for the introduction and wider use of effective and cost-effective programs. In this instance, with regards to eating disorders, advocacy is needed for support, fiscal and otherwise, of more research into the development and implementation of prevention programs.

Therapists and education staffs have become increasingly aware of whether there might be any harms caused by preventive interventions for EDs (Pratt & Woolfenden, 2002). The current systematic review found that there is insufficient evidence indicating that there are any harms
associated with the existing preventive interventions for EDs. Further research certainly is needed to verify whether there are harmful impacts of the evaluated interventions.

Importantly, this review provides support for the reproducibility of findings. The review presents average effect sizes for various ED prevention interventions, and thus found that preventive interventions have produced effects that replicate.

### 4.2. Recommendation and future directions.

The evidence to determine whether ED preventive interventions can reduce the onset of EDs is still scarce. Only eight of 101 trials investigated this outcome. Although some studies reported the number of participants which met criteria for clinical ED behaviour (not diagnosed criteria for ED), most studies focused on continuous measures of ED risk factors or behaviours without reporting outcomes for actual cases who met ED diagnostic criteria (Wilksch, 2014). This is partly because very large numbers of subjects are needed to provide sufficient statistical power in such preventive studies (Cuijpers, 2014). This is a common problem for preventive intervention studies for mental disorders more generally, particularly for low prevalence disorders. Indicated preventive strategies are therefore easier to evaluate since the target populations for these interventions are at greater risk for the development of the disorder compared to universal or selective interventions where the risk of developing the disorder is more diffuse (Cuijpers, 2014). Our findings also revealed that the majority of the ED prevention interventions were classified as selective prevention (79 selective vs. 18 universal vs. 4 indicated). Future research needs to evaluate true indicated prevention interventions as these have been shown to be effective and cost-effective in the prevention of other mental disorders (Mihalopoulos et al., 2011).

There is insufficient evidence to support the efficacy of universal prevention based interventions. Only ML interventions demonstrated evidence of efficacy in preventing ED risk.
factors of both females and males. Furthermore, it is very important to improve the methodology for universal intervention given that most of studies were quasi-RCT with sample sizes likely to be underpowered to detect significant differences. As noted above, further research, with longer follow-up periods, is required to detect evidence of reduced incidence.

With regards to effects on obesity, this review found that only healthy weight interventions significantly reduced both eating disorder risk factors and body mass index. The obesity prevention interventions failed to demonstrate superiority to control interventions in preventing ED onset. However, positive results were reported in some studies (Austin et al., 2005; Austin et al., 2007) which were not examined in the meta-analyses as the relevant data were unavailable. The Planet Health program prevented over half of expected new cases of disordered weight-control behaviour among females (Austin et al., 2007). Future research should evaluate potential secondary benefits of ED and obesity prevention interventions in a broad range of eating- and weight-related problems.

The current review also found that the majority of ED prevention studies focused on adolescent and young females who are considered at high-risk of developing an ED (Rohde, Stice, et al., 2014; Stice, Marti, Shaw, & Jaconis, 2009). There is insufficient evidence to support the effect of ED prevention interventions on pre-adolescent children and adults. A recent review found that poor body image and disordered eating are quite prevalent in middle-aged women (Slevec & Tiggemann, 2011). Further research needs to examine these particular sub-groups.

Limited research has been undertaken on the implementation of evidence-based preventive interventions for ED (Stice, Becker, et al., 2013). Furthermore, given the lack of evidence of cost-effectiveness of these interventions (Stuhldreher et al., 2012), research into cost-effectiveness is needed.
4.3. Study limitations

Several limitations of this review need to be considered. First, this review only used published data and data was solely extracted by the first author, although there was substantial discussions of the studies and classification systems with all the co-authors. Second, a full search of studies published post 2009 was undertaken for the current study, with reliance on existing high quality reviews for studies published prior to 2009. There may be unpublished studies that were inadvertently not included. Third, we used end point data rather than change scores which is more sensitive to baseline imbalance. This may have impacted the accuracy of the results (Higgins, 2011). Fourth, the outcomes considered in most studies are only risk factors and not final outcomes of actual cases of averted disorders. Relatedly, the outcome instruments employed in the studies did not, in most instances, map directly onto DSM diagnoses although elements of the diagnosis are included in most measures (e.g. heavy preoccupation with eating).

5. CONCLUSION

The public health burden of eating disorders is well documented, and the considerable progress that has been made in the prevention of eating disorders over the past several decades (Austin, 2016; Levine, 2016). This systematic review and meta-analysis found that for selective prevention, CD interventions were effective for late adolescent and young adult females in reducing risk factors and symptoms of ED. Research is, however, needed into use of this intervention in younger adolescents (15 years and under) and whether it can be used by non-professionals (e.g with Becker and Stice’s “train the trainers” approach (Kilpela et al., 2014)). Healthy weight programs were promising in reducing both ED risk factors and obesity onset risks but this result requires further research. Other selective prevention based interventions including CBT and ML are effective in preventing risk factors and behaviours of ED. The universal prevention intervention for which there was the best evidence of efficacy in preventing ED risk factors was ML, but future research is also required to confirm this result.
Although some positive findings were identified, the value of other preventive interventions should be viewed as uncertain. Further research, with improved methodologies, is necessary to determine definitively whether such interventions reduce the onset of clinical EDs. Furthermore research regarding how to improve uptake of such interventions is required. Such research will help to better establish the effectiveness and cost-effectiveness of preventive interventions in EDs and thus provide vital information to decision-makers, including clinicians and funders of health services, as to which interventions are worth delivering.
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