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Review

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

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HIGHLIGHTS

- The review described 13 ED preventive interventions spanning universal, selective and indicated preventive interventions.
- A bias adjusted meta-analysis is undertaken of 112 articles that evaluated eating disorder prevention interventions.
- Promising preventive interventions for ED risk factors/ behaviours included cognitive dissonance, cognitive behavioural therapy and media literacy.
- Combined ED and obesity prevention interventions require further research.
- Insufficient evidence supported the effect of ED prevention interventions on pre-adolescent children and adults.

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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.

Contents

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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 characterized 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, 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