2017

Online prevention of disordered eating in at-risk young-adult women: A two-country pragmatic randomized controlled trial

Simon M. Wilksch
Flinders University

Anne O'Shea
Flinders University

C. B. Taylor
Stanford University Medical Center

Denise Wilfley
Washington University School of Medicine in St. Louis

Corinna Jacobi
Technische Universität Dresden

See next page for additional authors

Authors

Simon M. Wilksch, Anne O'Shea, C. B. Taylor, Denise Wilfley, Corinna Jacobi, and Tracey D. Wade

Online prevention of disordered eating in at-risk young-adult women: a two-country pragmatic randomized controlled trial

Simon M. Wilksch[1], Anne O'Shea[1], C. Barr Taylor[2], Denise Wilfley[3,4], Corinna Jacobi[5] and Tracey D. Wade[1]

[1]School of Psychology, Flinders University, South Australia, Australia; [2]Department of Psychiatry, Stanford University Medical Center, Stanford, California, USA; [3]Department of Psychology, Washington University in St. Louis, St. Louis, Missouri, USA; [4]Department of Psychiatry, Washington University School of Medicine in St. Louis, St. Louis, Missouri, USA and [5]Institute of Clinical Psychology and Psychotherapy, Technische Universität Dresden, Dresden, Germany

Abstract

Background. Disordered eating (DE) is a widespread, serious problem. Efficacious prevention programs that can be delivered at-scale are needed.

Methods. A pragmatic randomized controlled trial of two online programs was conducted. Participants were young-adult women from Australia and New Zealand seeking to improve their body image. *Media Smart-Targeted* (*MS-T*) and *Student Bodies* (*SB*) were both 9-module interventions released weekly, whilst control participants received positive body image information. Primary [Eating Disorder Examination–Questionnaire (EDE-Q) Global], secondary (DE risk factors) and tertiary (DE) outcome measures were completed at baseline, post-program, 6- and 12-month follow-up.

Results. Baseline was completed by 608 women (*M* age = 20.71 years); 33 were excluded leaving 575 randomized to: *MS-T* (*N* = 191); *SB* (*N* = 190) or control (*N* = 194). Only 66% of those randomized to *MS-T* or *SB* accessed the intervention and were included in analyses with controls; 78% of this sample completed measures subsequent to baseline. Primary intent-to-treat (ITT) analyses revealed no differences between groups, while measure completer analyses found *MS-T* had significantly lower EDE-Q Global than controls at 12-month follow-up. Secondary ITT analyses found *MS-T* participants reported significantly higher quality of life–mental relative to both *SB* and controls (6-month follow-up), while *MS-T* and controls had lower clinical impairment relative to *SB* (post-program). Amongst measure completers, *MS-T* scored significantly lower than controls and *SB* on 5 variables. Of those with baseline DE, *MS-T* participants were significantly less likely than controls to have DE at 12-month follow-up.

Conclusions. Given both programs were not therapist-moderated, *MS-T* has potential to achieve reductions in DE risk at low implementation costs.

Introduction

Disordered eating (DE) refers to sub-threshold variants of threshold eating disorders (TED: e.g., anorexia nervosa, bulimia nervosa and binge eating disorder) and include behaviours such as fasting, binge eating, vomiting, laxative use, or maintaining body mass index (BMI) < 18.5, in addition to substantial concerns about food, body weight and shape. The pursuit of efficacious DE risk reduction is of critical importance for many reasons (Wilksch, 2014; Austin, 2016). First, DE can be a risk factor for the development of TED (Patton *et al.* 1999; Wertheim *et al.* 2001; Stice *et al.* 2008*a*). Second, the prevalent nature of DE, as highlighted by an Australian cohort study (*N* = 9688) where DE was observed in 23% of young-adult women (Wade *et al.* 2012). Third, evidence suggests those with DE experience similar impairment in functioning and quality of life as those with TED (Fairburn & Cooper, 2011; Fairweather-Schmidt & Wade, 2014). Fourth, DE has been prospectively linked to symptoms of depression, anxiety, and suicidality (Braun *et al.* 1994; Franko & Keel, 2006). Fifth, DE in mid-adolescence is prospectively associated with the development of overweight and obesity at 6-year follow-up, independent of baseline or parental BMI (Herpertz-Dahlmann *et al.* 2015). Sixth, DE can persist or increase from adolescence into adulthood (Kotler *et al.* 2001; Neumark-Sztainer *et al.* 2011). A recent survey of 50 000 Australian females and males aged 11–24 years (Mission Australia, 2010) found that body image ranked as the highest personal concern by 28.1% of 11–14 year-olds, 33.3% of 15–19 year-olds, and 40.3% of 20–24 year-olds. These studies suggest that body image and eating pathology are not limited to

adolescence; rather, they increase during early adulthood. Clearly, the burden and consequences of DE are immense and require efficacious risk reduction programs that can be easily disseminated at-scale.

Prevention programs for DE usually occur across the spectrum of universal (e.g. large groups of participants regardless of baseline level of DE risk), selective [e.g. participants selected based on a prospectively identified risk factor (e.g. females)], and indicated (i.e. participant selection based on exhibiting clear precursors to an ED such as elevated weight concerns). Three indicated risk reduction programs have had favourable outcomes across multiple randomized controlled trials (RCTs): *The Body Project* (Stice *et al.* 2000); *Healthy Weight* (Stice *et al.* 2001); and, *Student Bodies* (SB: Winzelberg *et al.* 2000). The first two are delivered face-to-face, while *SB* is delivered online with therapist moderation. For the current study where the goal was to evaluate programs in a 'real-world' context, it was thought that online programs would be more scalable at lower cost, thus *SB* was included. Since the commencement of this research, an online version of *The Body Project* has been investigated (Stice *et al.* 2012).

Online risk reduction programs are becoming more common across the DE prevention spectrum (e.g. see Melioli *et al.* 2016). Online delivery holds the appeal of sidestepping two important gaps in reducing the burden of mental illness (Patel *et al.* 2011; Kazdin *et al.* 2017): the research-practice gap (efficacious delivery of evidence-based programs in the real world); and, the treatment gap (discrepancy between those who could benefit from services and those who receive such services). Specifically, 'pure self-help' online interventions (i.e. no personalized online therapist moderator) do not require training of health providers to deliver – the intervention is designed by the specialist, made available online and then accessed directly by individuals at scale. As such, online interventions have no limitation to the number of participants who can participate, unlike face-to-face services that are bound by the availability of service providers, training of these providers, and, assumptions intervention fidelity is maintained.

SB has been evaluated in 11 RCTs, seven in the USA (Winzelberg *et al.* 1998; Celio *et al.* 2000; Winzelberg *et al.* 2000; Bruning Brown *et al.* 2004; Low *et al.* 2006; Taylor *et al.* 2006; Kass *et al.* 2014; Taylor *et al.* 2016) and four in Germany (Jacobi *et al.* 2005, 2007; Jacobi *et al.* 2012). These samples have included young-adult or late adolescent females from college and high-school settings. The most methodologically rigorous trial was with college-aged women with elevated weight concerns (*M* age = 20.8 years: Taylor *et al.* 2006), showing significant differences of moderate effect size (ES) at 1-year follow-up favouring the *SB* for weight and shape concern, global measures of disordered eating, and ED risk factors. Although no main effects were shown for prevention of ED onset, prevention effects were found for two subsets of users at 2-year follow-up: those who were overweight at baseline (i.e. *SB* = 0% *v.* controls = 10.8%); and, at one site, amongst those engaging in compensatory behaviours at baseline (i.e. *SB* = 14.4% *v.* controls = 30%). A comparison of guided *v.* self-help versions of *SB* (conducted concurrently to the current trial) found the guided group had significantly lower weight concerns and lower odds of remaining at high risk of an ED than the non-guided group at post-intervention (*M* age = 20.1 years: Kass *et al.* 2014). While a recent RCT for those at 'very high risk' of an eating disorder (Taylor *et al.* 2016) found amongst participants with high shape and weight concerns, ED onset was significantly lower in the *SB* group compared with controls (*SB* = 20% *v.* controls = 42%).

To date *SB* has not been compared with another intervention. In the current study we compared it with *Media Smart-Targeted* (*MS-T*), an adapted online version of an 8-lesson school-based program (*MS*) that has been found to reduce ED risk factors relative to controls and active interventions in young-adolescent (Grades 7 and 8) girls and boys (Wilksch & Wade, 2009; Wilksch *et al.* 2015). *MS* is one of the most robust universal ED risk reduction programs (Levine & Smolak, 2016; Piran, 2015) and has been found to significantly lower shape and weight concerns in girls over a 2.5-year follow-up (Wilksch & Wade, 2009) and halve the risk of onset of clinical concerns about shape and weight at 12-month follow-up (Wilksch *et al.* 2015). The current study was the first evaluation of *MS-T*.

The World Health Organization estimates 22 as the peak age in the relative value of healthy lived years (Murray & Lopez, 1996) and thus DE risk reduction in young-adults is important to ensure this peak productivity is not compromised and to prevent impairment over future years (Wade *et al.* 2012). Thus the primary aim of this research was to evaluate the 'real world' effectiveness of an established online ED prevention program (*SB*) relative to a new online program (*MS-T*) and a control condition with young-adult women. Related to this, we sought to maximize the external validity of this research by: using a general community population as opposed to university and school-based settings typically investigated; and, allowing participants with higher baseline levels of eating pathology than in previous trials.

Methods

Participants

Participants were women aged 18–25 years from Australia and New Zealand who wished to improve their body image. Participants self-referred through advertisements posted around university campuses, social media online, and, health clinic waiting rooms. Exclusion criteria were minimal: suicide risk [e.g. have a suicide plan (Lecrubier *et al.* 1997)]; alcohol or substance abuse [e.g. the presence of withdrawal effects when reducing use (Lecrubier *et al.* 1997)]; or, self-reported BMI < 15.0. Women with premorbid DE were included in the study, to reflect the real-world likelihood of such women accessing online resources.

Conditions

SB has been described in detail (Winzelberg *et al.* 2000), as has *MS* (Wilksch & Wade, 2009). *SB* and *MS-T* are 9-module programs that follow the principles of effective prevention, such as interactive content and targeting prospectively identified ED risk factors (Stice *et al.* 2007). Consistent with previous *SB* trials (Taylor *et al.* 2016), both programs released modules weekly via a password-protected mobile internet-platform and both programs were a pure self-help format (no online therapist moderator). The *SB* program was delivered in its usual form on a mobile platform but without moderation. Participants were encouraged to view content (e.g. written, video), respond to questions, and complete accompanying assignments. Supplementary Table 1 provides a description of the programs. Both target weight concerns, a robust and proximal ED risk factor (Jacobi *et al.* in press), but do this in differing ways. *SB* focused on behavioural targets (e.g. eating, exercise) that are proximal to DE in risk factor

models while *MS-T* focused more on cognitive components (e.g. media internalization, ineffectiveness) that are more distal in such models (Stice, 2001; Fairburn *et al.* 2003).

The translation of *MS* to *MS-T* was guided by three rationale. First, the theoretical targets of *MS* (media internalization) are informed by the dual-pathway model, developed from risk factor research with young-adult women and thus relevant to this population (Stice, 2001). Second, *The Body Project* also targets this variable and has been found to be effective with this population (Stice *et al.* 2009). Third, given that results for universal samples are usually of a smaller effect than for university-aged, high-risk samples (Stice *et al.* 2007), it was expected that *MS-T* could produce at least comparable outcomes to *MS*.

MS-T was developed over an 18-month process of: writing and adapting each module to include age-appropriate content; pilot testing; ensuring accurate data collection; and, further revisions. For example where module 3 in *MS* provided a clip on standing up to peer pressure on teen drinking, in *MS-T* this was replaced by a post titled 'You look so different on Facebook' and then examining strategies to reduce pressures felt from social media usage. The new content included a focus on: emotion regulation, goal setting, and 'healthy eating', which included four tips informed by risk factor research: eat breakfast; eat regularly; drink water; and, dieting as unhelpful. *MS-T* modules were designed to be of comparable length and aesthetics to *SB*, to reduce confounds. There was one difference of note: *MS-T* participants received an automated weekly email to advise their module was available, whereas *SB* participants did not. The control condition involved one email containing 10 tips for improving body image as used in other targeted trials (e.g. Stice *et al.* 2011).

Procedure

Approval for this research was received from the Flinders University Social and Behavioural Research Ethics Committee. Potential participants were directed to the study website where they were provided with information, consent procedures, and baseline questionnaires. Upon completion and meeting inclusion criteria, participants were automatically randomized to one of the three groups. Randomization was stratified by age (18–21 years; 21.01 years- 25 years) and baseline scores on the Weight Concerns Scale (<47; >47: WCS: Killen *et al.* 1994). A WCS score of 47 or higher has been found to have a sensitivity of 79%, specificity of 67%, and positive predictive value of 13% for identifying adolescents who developed partial- or full-syndrome EDs (Jacobi *et al.* 2004).

Age was included as a proxy for duration of body image concerns (Rohde *et al.* in press). Whilst participants were accepted across the WCS score spectrum in line with the pragmatic RCT, we expected the majority of participants to score ⩾47. Participants allocated to an intervention were able to access their respective website immediately. All participants received an email reminder to complete post-program measures 10-weeks after baseline. This process was repeated at 6- and 12-month follow-up. If a participant did not commence the measures at any time point they received two further email reminders at weekly intervals. Participants who completed a minimum of three assessment points received a $50 gift voucher as reimbursement for their time. Intervention participation was not a requirement for this reimbursement.

Measures

Primary outcome

The Eating Disorder Examination Questionnaire (EDE-Q) Global was used to provide a continuous measure of eating pathology. The use of the EDE-Q was consistent with the pragmatic, 'pure self-help' nature of this trial where phone interview (i.e. EDE) could deter participation and would be unlikely in real-world implementation. The EDE-Q has only moderate diagnostic concordance against the EDE (Berg *et al.* 2012) and therefore diagnostic categories were not utilized.

Secondary outcomes

A range of risk factor and impairment measures in addition the EDE-Q were selected based upon the evidence supporting their construct validity (Sheehan et al. 1997; Espelage et al. 2003; Jacobi et al. 2004; Thompson et al. 2004; Henry & Crawford, 2005; Berg et al. 2011; Vannucci et al. 2012) and their frequent use in other prevention trials (e.g. Taylor *et al.* 2006; Wilksch & Wade, 2009). Higher scores indicated higher levels of risk for all but the quality of life mental scale, where a lower score indicated a poorer outcome (see Table 1).

Tertiary outcomes

Consistent with previous investigations (Bardone-Cone *et al.* 2010), DE was defined as having a global EDE-Q score ⩾1 SD of the community mean (i.e. 2.46: Mond *et al.* 2004), and the presence of one or more of the following in the previous 4-week period: objective bulimic episode; fasting; vomiting or laxatives to control weight; or BMI < 18.5. Thus DE was a dichotomous outcome and allowed exploration of both prevention (DE status at follow-up for those who did not have DE at baseline) and treatment effects (DE status at follow-up for those who had DE at baseline).

Statistical analyses

Participant flow, variables impacting intervention access and baseline differences

Demographic details were investigated (e.g. age, living location) using analysis of variance (ANOVA) and χ^2 analyses. Potential variables impacting on intervention access were examined univariately and then multivariately using linear regressions.

As data were only analyzed for those participants who accessed their intervention, consistent with previous investigations (e.g. Taylor *et al.* 2006), logistic regressions investigated baseline differences between participants who accessed their allocated intervention, *v.* those who did not. They were also used to compare rates of module completion across interventions. Baseline differences across the three groups were analyzed using ANOVAs for continuous measures and χ^2 for dichotomous outcomes, with an alpha level of 0.05.

Missing data and intervention completion rates

To determine whether data were missing at random, logistic regressions were used to assess whether 'measure completers' (those who completed baseline measures and at least one other assessment point, the minimum necessary given baseline was a covariate in analyses) differed from those who only completed baseline measures in the intervention conditions. Logistic regressions were also used to compare rates of module access and completion between *MS-T* and *SB*.

Table 1. Summary and description of primary and secondary outcome self-report measures

Outcomes	Description (Cronbach's alpha) and example item
Primary outcomes	
Global EDE-Q	Eating Disorder Examination – Questionnaire (Fairburn & Beglin, 1994), 22 items ($\alpha = 0.94$)
	e.g. *How dissatisfied have you been with your weight?*, 0 = 'Not at All' to 6 = 'Marked'
Secondary outcomes	
Weight concerns	The Weight Concerns Scale (Killen *et al.* 1994) 5 items ($\alpha = 0.72$)
	e.g. *Do you ever feel fat*, 1 = 'Never' to 5 = 'Always', multiplied to generate score /100
Depression	Depression, Anxiety, Stress Scale (Lovibond & Lovibond, 1995), 21 items (depression $\alpha = 0.93$; anxiety $\alpha = 0.85$; stress $\alpha = 0.87$)
	e.g. *I felt that I had nothing to look forward to*, 0 = Not at All to 3 = Very much or most of the time
Media internalization	Sociocultural Attitudes Towards Appearance Questionnaire-3 (Thompson *et al.* 2004), 9 items ($\alpha = 0.95$)
	e.g. *I compare my body to the bodies of TV and movie stars*, 1 = 'definitely disagree' to 5 = 'definitely agree'
Ineffectiveness	Eating Disorder Inventory (EDI: Garner *et al.* 1983) 10 items ($\alpha = 0.91$)
	e.g. *I feel inadequate*, 6 = 'Always' to 1 = 'Never'
Clinical impairment	Clinical Impairment Assessment Questionnaire (Bohn & Fairburn, 2008), 16 items ($\alpha = 0.96$)
	e.g. *To what extent has your exercise/eating habits/feelings about weight or shape made it difficult to concentrate?* 0 = 'Not at all' to 3 = 'A lot'
Quality of life – mental	Medical Outcome Studies Short Form Scales – Mental Component Scale (Ware *et al.* 1993) ,5 items ($\alpha = 0.85$)
	e.g. *Have you felt calm and peaceful*, 100 = 'strongly agree' to 0 = 'strongly disagree'
Risk – suicide, drug, alcohol	MINI International Neuropsychiatric Interview (Lecrubier *et al.* 1997) 15 items.
	e.g. *In the past month did you think about suicide?*, 'No' to 'Yes'

Due to an error in the US web company hosting *SB*, program usage data for $n = 50$ consecutive new *SB* participants over a 2.5-month period was not available. Thus there were no means for identifying if these users accessed *SB*. Logistic regression analyses found no significant baseline differences between these $n = 50$ cases *v.* the other $n = 140$ *SB* participants for any outcome variable [e.g. EDE-Q Global: OR = 1.14, 95% CI (0.90–1.46)]. These participants were omitted from all further analyses.

Primary and secondary outcomes

Linear mixed model (LMM) analyses, with baseline observations used as a covariate to ensure that any observed effects for that variable were due to changes at post-program and follow-up, were used to compare the impact of the conditions. A 3 (group: *MS-T*, *SB*, Control) × 3 (time: post-program, 6-month follow-up; 12-month follow-up) design was used, allowing for direct comparisons between the groups at post-program and follow-up using Bonferroni-adjusted post-hoc analyses. Cohen's *d* between group ES with 95% confidence intervals (CI) were calculated using means and standard errors from the LMM. Analyses were run in two ways. First, intent-to-treat (ITT) analyses included all participants who accessed their intervention at least once (*MS-T* = 122; *SB* = 98) and all controls ($n = 194$; Total $N = 414$), regardless of if the participant completed any further measures after baseline. These ITT analyses were conducted using multiple imputation (MI) in SPSS to estimate missing values using Bayesian analysis (Enders, 2010). The imputation step of the procedure used all the outcome variables at each assessment and the number of modules completed (the control group was scored as having completed no modules). MI incorporates post-randomization variables that are not part of the analysis model (intervention models completed) in the imputation step and so

enable an analysis that is valid under a more realistic missing at random assumption (Sterne *et al.* 2009). Five data sets were imputed. Second, using all available data for measure completers (*MS-T* = 82; *SB* = 70; controls $n = 169$; $N = 321$).

Tertiary outcomes

Both prevention (DE status for those who did not have DE at baseline, $N = 92$) and treatment effects (DE status for those who had DE at baseline, $N = 169$) were investigated for measure completers at 12-month follow-up. Odds ratios (OR) and 95% CI from logistic regressions were used to compare DE status at 12-month follow-up in the intervention groups relative to the control group. These analyses were also run adjusting for receiving any external professional treatment (e.g. psychologist, psychiatrist) at any of the four assessment points.

Results

Participant flow

Participant flow can be seen in Fig. 1. Recruitment occurred over a 12-month period between 3 November 2014 and 1 November 2015, with the final 12-month follow-up assessment completed on 29 December 2016. $N = 608$ women (*M* age = 20.71 years, *SD* = 2.26, range 18–25 years) completed baseline measures, with 33 participants excluded due to one or more of the following: suicide risk ($n = 8$); alcohol abuse ($n = 11$); substance abuse ($n = 20$); and, BMI < 15.0 ($n = 3$). Thus $N = 575$ were randomized to *MS-T*, *SB* or control.

The most common sources of recruitment were: flyers at universities and colleges ($n = 179$; 29.7%); social media ($n = 164$; 27.2%); and, media reports ($n = 114$; 18.9%). Participants lived
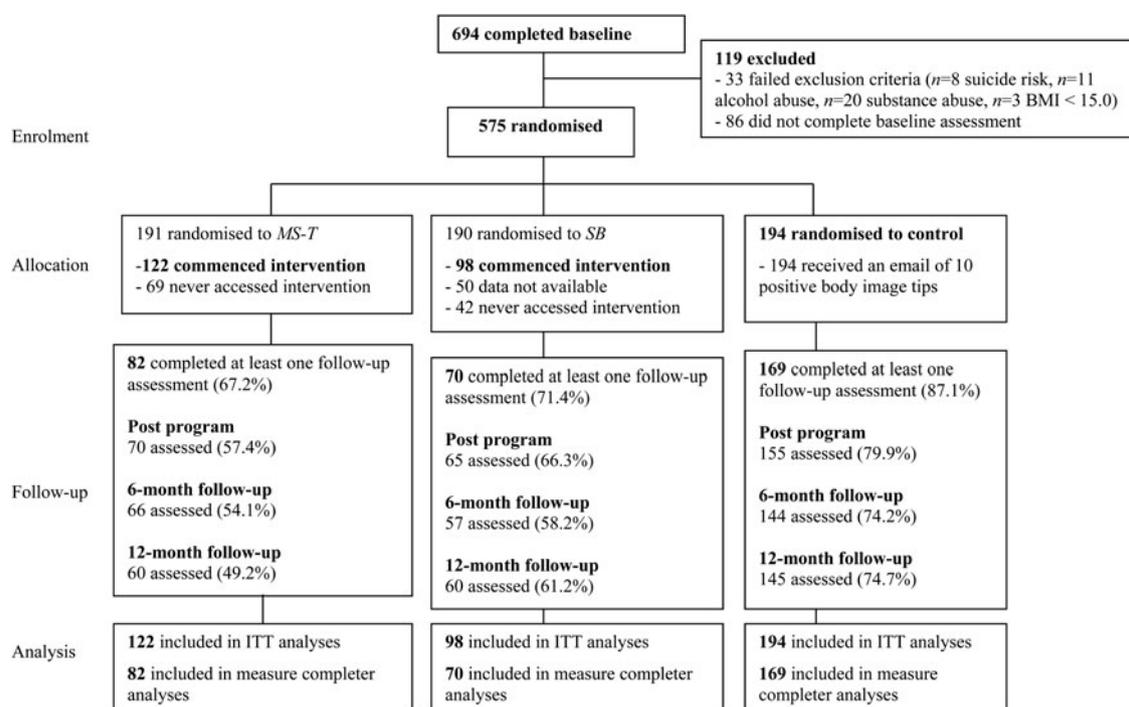
Fig. 1. Participant flow chart.

in each state and territory of Australia ($n = 460$) and NZ ($n = 144$). Capital cities ($n = 362$; 59.7%) were the most common location followed by regional towns ($n = 207$; 34.2%). Current undergraduate was the most common education level ($n = 229$; 37.9%), followed by completion of year 12 ($n = 169$; 27.9%) and completion of the undergraduate degree ($n = 169$; 18.8%). Ethnicity included: Caucasian – Australian ($n = 367$; 60.4%), followed by Caucasian – NZ ($n = 131$; 21.7%), and Asian ($n = 53$; 8.8%).

Variables impacting on intervention access

Five variables were significantly associated with likelihood of accessing an intervention: age [OR = 1.14, 95% CI (1.02–1.27)]; attitude towards program participation [OR = 0.53, 95% CI (0.35–0.82)]; EDE-Q – Global [OR = 1.21, 95% CI (1.02–1.44)]; clinical impairment [OR = 1.03, 95% CI (1.01–1.05)]; and quality of life – mental [OR = 0.99, 95% CI (0.98–1.00)]. Attitude towards program participation was the only predictor to remain significant [OR = 0.56, 95% CI (0.37–0.86)] in a simultaneous multiple regression, where participants were significantly more likely to access the intervention if they identified themselves as 'committed to trying the program'.

Baseline measures

Table 2 presents baseline scores by group for those who accessed their intervention (MS-T = 122; SB = 98) and controls ($n = 194$). No significant differences were found.

Missing data

No significant differences were found between intervention participants who completed more than one assessment point ($n = 150$: MS-T = 82; SB = 68) and those who completed baseline only

($n = 70$: MS-T = 40; SB = 30). EDE-Q Global [OR = 1.05, 95% CI (0.85–1.29)], risk factors [e.g. internalization: OR = .87, 95% CI (0.64–1.17)], BMI [OR = 1.25, 95% CI (0.78–1.99)], and, DE [OR = .81, 95% CI (0.50–1.33)], were all non-significant. There was no significant difference in rates of post-program and follow-up measure completion between MS-T and SB [OR = .90, 95% CI (0.51–1.60)]. Module completion was significantly positively associated with assessment completion at post-program and follow-up [OR = 2.02, 95% CI (1.44–2.84)], indicating the inclusion of module completion as a variable in the determination of MI.

Intervention completion

Table 2 presents completion rates of each module for SB ($n = 140$) and MS-T ($n = 191$). A total of $n = 111$ (33.5%: SB = 42 + MS-T = 69 = 111/331) of participants did not open their allocated intervention. There were no significant differences in rates of first accessing the interventions or completion of module 1. From module 2 through 9, a significantly higher proportion of participants completed MS-T than SB. Of the participants who completed module 1 of their programs (SB = 63; MS-T = 63), $n = 2$ (3.2%) and $n = 26$ (41.2%) completed the full SB and MS-T programs, respectively.

Primary outcome

ITT analyses found no significant differences in EDE-Q Global outcomes across the three groups (see Table 3). Measure completer analyses found MS-T participants had significantly lower EDE-Q Global scores than controls at 12-month follow-up. Main effects for group favouring MS-T were found for comparisons with both the control and SB groups. ES were small.

Table 2. Rates of access and completion of Student Bodies and Media Smart-Targeted and baseline scores across conditions

	SB (n = 140)	MS-T (n = 191)	Odds ratio (95% CI)	
Opened module 1	98 (70%)	122 (63.9%)	0.76 (0.48–1.21)	
Completed module 1	63 (45%)	63 (32.9%)	0.60 (0.38–0.94)	
Completed module 2	17 (12.1%)	54 (28.3%)	**2.85 (1.57–5.18)**	
Completed module 3	8 (5.7%)	40 (20.9%)	**4.37 (1.98–9.67)**	
Completed module 4	7 (5.0%)	39 (20.4%)	**4.88 (2.11–11.26)**	
Completed module 5	4 (4.3%)	31 (16.2%)	**6.59 (2.27–19.13)**	
Completed module 6	4 (2.9%)	34 (17.8%)	**7.36 (2.55–21.28)**	
Completed module 7	4 (2.9%)	26 (13.6%)	**5.36 (1.83–15.73)**	
Completed module 8	4 (2.9%)	28 (14.7%)	**5.84 (2.00–17.06)**	
Completed module 9	2 (1.4%)	26 (13.6%)	**10.87 (2.54–46.63)**	
	SB (n = 98)	MS-T (n = 122)	Controls (n = 194)	Odds Ratio (95% Confidence Interval)/F value
ED treatment in last 12 months	11 (11.2%)	18 (14.8%)	32 (16.7%)	χ (2) = 1.52, p = 0.467
Psychiatric medication	17 (17.7%)	25 (20.5%)	30 (15.5%)	χ (2) = 1.27, p = 0.530
Suicidal thoughts	28 (28.9%)	35 (28.7%)	49 (25.7%)	χ (2) = 0.50, p = 0.779
Non suicidal self-harm	18 (18.6%)	18 (14.8%)	30 (15.7%)	χ (2) = 0.62, p = 0.734
Age (years)	20.89 (2.29)	21.05 (2.20)	20.65 (2.29)	$F_{(2,410)}$ = 1.23, p = 0.293
BMI	24.11 (7.52)	24.57 (7.17)	24.20 (7.00)	$F_{(2,411)}$ = 0.15, p = 0.864
Global EDE-Q (0–6)	3.55 (1.26)	3.35 (1.42)	3.20 (1.40)	$F_{(2,407)}$ = 1.87, p = 0.156
Weight concerns (0–100)	68.42 (20.93)	66.00 (21.40)	64.05 (22.24)	$F_{(2,404)}$ = 1.31, p = 0.270
Weight concerns ⩾47	96 (78.7%)	77 (80.2%)	149 (78.8%)	χ (2) = 0.92, p = 0.955
DASS – Total (0–3)	1.15 (0.64)	1.07 (0.61)	1.04 (0.67)	$F_{(2,410)}$ = 1.06, p = 0.348
Media internalization (1–5)	3.65 (1.02)	3.55 (0.95)	3.53 (1.02)	$F_{(24\,007)}$ = .46, p = 0.633
Ineffectiveness (1–6)	3.71 (1.05)	3.57 (0.98)	3.41 (1.00)	$F_{(2,409)}$ = 3.02, p = 0.050
Quality of life – mental (0–100)	49.44 (20.17)	51.30 (18.87)	52.29 (19.58)	$F_{(2,410)}$ = 2.27, p = 0.105
Clinical impairment (0–48)	23.10 (12.38)	22.03 (12.89)	20.62 (12.95)	$F_{(2,408)}$ = 1.30, p = 0.275
DE	72 (73.4%)	79 (64.8%)	121 (62.7%)	χ (2) = 5.09, p = 0.079

Note: ED, eating disorder; BMI, body mass index; EDE-Q, eating disorder examination – questionnaire; DASS, depression, anxiety, stress scale; DE, disordered eating case. Bonferroni-adjusted p value for multiple testing in rates of module completion (0.05/9) to p = 0.06. Frequencies (and percentages) are reported for dichotomous variables while means (and standard deviations) are provided for continuous measures. Bolded values indicate significant odds ratios (p < 0.05).

Secondary outcomes

ITT analyses revealed significant effects of small ES for two variables. Specifically, SB participants had significantly higher levels of clinical impairment at post-program than both MS-T and controls. MS-T participants had significantly higher quality of life-mental at 6-month follow-up than both controls and SB (see Table 3). Amongst measure completers, MS-T participants reported significantly lower scores than both controls and SB on 5 variables with ES ranging from small to medium in magnitude: depression (6- and 12-month follow-up); internalization (post-program); ineffectiveness (12-month follow-up); clinical impairment (12-month follow-up); and quality of life mental (6-month follow-up). MS-T participants also scored significantly higher than SB at each assessment point for the quality of life – mental.

Tertiary outcomes

Prevention effects

Of measure completer participants with 12-month follow-up data, a total of n = 15 met DE criteria who did not meet DE at baseline

(n = 92). Table 4 provides the frequency and percentage of participants from each group that developed DE at 12-month follow-up. It shows MS-T and SB participants had an 85% and 69% lower likelihood than controls of becoming a new DE case at 12-month follow-up respectively. Amongst those who did not seek external treatment at any point in the study, MS-T and SB participants were 78% and 55% less likely than controls respectively to become a new DE case at 12-month follow-up. No results relating to prevention effects were significant.

Treatment effects

Of measure completer participants with 12-month follow-up data, a total of n = 52 no longer met DE criteria who did meet DE at baseline (n = 169). Table 4 shows MS-T participants who met criteria for eating pathology at baseline had a significantly lower likelihood than controls of still meeting criteria at 12-month follow-up, including when adjusting for accessing ED treatment. Amongst those who did access external ED treatment, MS-T participants were seven times less likely than controls to meet DE criteria at 12-month follow-up. The comparison between SB and

Table 3. Effect sizes (Cohen's d) for between-groups pairwise comparisons for primary and secondary outcomes

Outcomes	ITT sample (n = 414)				Measure Completer sample (n = 284)			
	Post-program d (95% CI)	6-month follow-Up d (95% CI)	12-month follow-Up d (95% CI)	Overall group comparison d (95% CI)	Post-program d (95% CI)	6-month follow-up d (95% CI)	12-month follow-up d (95% CI)	Overall group comparison d (95% CI)
Primary Outcome								
EDE-Q Global								
MS-T v. Con	−0.07 (−0.30 to 0.16)	−0.10 (−0.32 to 0.13)	−0.09 (−0.31 to 0.14)	−0.13 (−0.36 to 0.09)	−0.22 (−0.50 to 0.07)	−0.20 (−0.49 to 0.08)	**−0.33 (−0.63 to −0.03)**	**−0.31 (−0.59 to −0.02)**
SB v. Con	0.08 (−0.16 to 0.32)	0.09 (−0.15 to 0.34)	0.11 (−0.13 to 0.35)	0.14 (0.10 to 0.38)	0.03 (−.27 to 0.32)	0.10 (−0.21 to 0.41)	0.02 (−0.28 to 0.32)	0.06 (−0.24 to 0.35)
MS-T v. SB	−0.14 (−0.41 to 0.12)	−0.19 (−0.46 to 0.08)	−0.16 (−0.43 to .11)	−0.24 (−0.51 to −0.03)	−0.25 (−0.59 to 0.10)	−0.31 (−0.66 to 0.05)	−0.35 (−71 to 0.01)	**−0.37 (−0.71 to 0.02)**
Secondary outcomes								
WCS								
MS-T v. Con	−0.16 (−0.39 to 0.07)	.01 (−0.21 to 0.24)	0.03 (−0.20 to 0.25)	−0.04 (−0.27 to 0.18)	−0.14 (−0.43 to 0.14)	.08 (−0.21 to 0.38)	−0.14 (−0.44 to 0.16)	−0.08 (−0.36 to 0.20)
SB v. Con	−0.14 (−0.38 to 0.10)	−0.01 (−0.25 to 0.23)	0.00 (−0.24 to 0.24)	−0.07 (−0.31 to 0.18)	−0.13 (−0.43 to 0.16)	−0.02 (−0.32 to 0.29)	−0.03 (−0.33 to 0.26)	−0.07 (−0.37 to 0.22)
MS-T v. SB	−0.01 (−0.28 to 0.26)	0.02 (−0.25 to 0.28)	0.03 (−0.24 to 0.29)	0.02 (−0.25 to 0.28)	−0.01 (−0.35 to 0.34)	0.10 (−0.26 to 0.46)	−0.11 (−0.46 to 0.25)	−0.01(−0.35 to 0.34)
DASS Total								
MS-T v. Con	0.10 (−0.12 to 0.33)	−0.05 (−0.30 to 0.17)	−0.01 (−0.23 to 0.22)	0.02 (−0.21 to 0.25)	0.04 (−0.24 to 0.33)	**−0.33 (−0.62 to 0.03)**	**−0.32 (−0.62 to −0.02)**	−0.25 (−0.54 to 0.03)
SB v. Con	0.15 (−0.09 to 0.39)	0.07 (−0.18 to 0.31)	0.19 (−0.05 to 0.43)	0.18 (−0.06 to 0.43)	0.15 (−0.15 to 0.44)	0.11 (−0.20 to 0.42)	0.22 (−0.08 to 0.52)	0.21 (−0.09 to 0.54)
MS-T v. SB	−0.05 (−0.31 to 0.22)	−0.11 (−0.38 to 0.15)	−0.18 (−0.45 to 0.09)	−0.17 (−0.43 to 0.10)	−0.14 (−0.48 to 0.21)	**−0.45 (−0.81 to −0.09)**	**−0.56 (−0.93 to 0.20)**	**−0.47 (−0.81 to −0.12)**
SATAQ-3								
MS-T v. Con	−0.17 (−0.40 to 0.05)	−0.04 (−0.27 to 0.18)	−0.01 (−0.24 to 0.21)	−0.11 (−0.34 to 0.12)	**−0.33 (−0.61 to −0.04)**	−0.07 (−0.37 to 0.22)	−0.15 (−0.45 to 0.15)	−0.22 (−0.51 to 0.56)
SB v. Con	−0.05 (−0.30 to 0.19)	−0.03 (−0.28 to 0.21)	−0.15 (−0.39 to 0.09)	−0.11 (−0.36 to 0.13)	0.03 (−0.26 to 0.33)	0.07 (−0.23 to 0.39)	−0.12 (−0.48 to 0.24)	0.00 (−0.29 to 0.30)
MS-T v. SB	−0.11 (−0.38 to 0.16)	−0.02 (−0.28 to 0.25)	0.09 (−0.17 to 0.36)	−0.01 (−0.28 to 0.25)	**−0.36 (−0.71 to −0.02)**	−0.15 (−0.51 to 0.20)	−0.05 (−0.41 to −0.31)	−0.23 (−0.57 to 0.12)
Ineffectiveness								
MS-T v. Con	−0.03 (−0.26 to 0.20)	−0.05 (−0.27 to 0.18)	−0.08 (−0.30 to 0.15)	−0.03 (−0.25 to 0.20)	−0.05 (−0.33 to 0.24)	−0.19 (−0.48 to 0.11)	**−0.32 (−0.62 to −0.02)**	−0.22, (−0.51 to 0.06)
SB v. Con	0.03 (−0.21 to 0.28)	0.04 (−0.20 to 0.28)	0.08 (−0.16 to 0.32)	0.05 (−0.19 to 0.29)	0.23 (−0.06 to 0.53)	0.13 (−0.18 to 0.43)	0.15 (−0.15 to 0.45)	0.16 (−0.14 to 0.45)
MS-T v. SB	−0.07 (−0.33 to 0.20)	−0.10 (−0.36 to 0.17)	−0.14 (−0.40 to 0.13)	−0.07 (−0.34 to 0.19)	−0.30 (−0.64 to 0.04)	−0.32 (−0.68 to 0.04)	**−0.51 (−0.88 to −0.15)**	**−0.39 (−0.73 to −0.04)**
Clinical impairment								
MS-T v. Con	−0.04 (−0.27 to 0.18)	−0.02 (−0.25 to 0.21)	−0.08 (−0.31 to 0.14)	−0.06 (−0.29 to 0.16)	−0.06 (−0.34 to 0.22)	−0.12 (−0.42 to 0.17)	**−0.39 (−0.69 to −0.08)**	−0.23 (−0.51 to 0.06)
SB v. Con	**0.29 (0.05–0.53)**	0.00 (−0.24 to 0.24)	0.10 (−0.14 to 0.35)	0.16 (−0.09 to 0.40)	**0.36 (0.06–0.65)**	0.16 (−0.15 to 0.47)	0.21 (−0.09 to 0.51)	0.28 (−0.01 to 0.58)
MS-T v. SB	**−0.36 (−0.63 to −0.10)**	−0.01 (−0.28 to 0.25)	−0.18 (−0.45 to 0.08)	−0.18 (−0.42 to 0.07)	**−0.42 (−0.77 to 0.07)**	−0.29 (−0.65 to 0.07)	**−0.61 (−0.97 to −0.24)**	**−0.52 (−0.87 to −0.17)**
QOL-M								
MS-T v. Con	0.15 (−0.07 to 0.38)	**0.24 (0.02–0.47)**	−0.01 (−0.24 to 0.21)	0.18 (−0.05 to 0.40)	0.28 (−0.01 to 0.56)	**0.44 (0.14–0.73)**	0.28 (−0.02 to 0.58)	**0.41 (0.12–0.69)**
SB v. Con	−0.14 (−0.37 to 0.09)	−0.12 (−0.37 to 0.12)	−0.16 (−0.40 to 0.09)	−0.20 (−0.44 to 0.05)	−0.18 (−0.47 to 0.12)	−0.14 (−0.45 to 0.17)	−0.16 (−0.46 to 0.14)	−0.19 (−0.49 to 0.10)
MS-T v. SB	0.24 (−0.02 to 0.51)	**0.34 (0.08 to 0.61)**	0.14 (−0.13 to 0.40)	**0.31 (0.05–0.58)**	**0.46 (0.11–81)**	**0.59 (0.22–0.95)**	**0.46 (0.10–0.82)**	**0.61 (0.26–0.96)**

(Continued)

Table 3. (*Continued.*)

Outcomes	ITT sample (n = 414)				Measure Completer sample (n = 284)			
	Post-program d (95% CI)	6-month follow-Up d (95% CI)	12-month follow-up d (95% CI)	Overall group comparison d (95% CI)	Post-program d (95% CI)	6-month follow-up d (95% CI)	12-month follow-up d (95% CI)	Overall group comparison d (95% CI)
BMI								
MS-T v. Con	0.01 (−0.22 to 0.23)	0.12 (−0.11 to 0.34)	−0.05 (−0.28 to 0.18)	0.02 (−0.21 to 0.25)	0.10 (−0.19 to 0.38)	0.18 (−0.11 to 0.48)	−0.02 (−0.32 to 0.28)	0.09 (−0.19 to 0.38)
SB v. Con	0.06 (−0.18 to 0.31)	0.00 (−0.24 to 0.24)	0.18 (−0.06 to 0.35)	0.11 (−0.14 to 0.35)	0.13 (−0.16 to 0.43)	−0.11 (−0.42 to 0.20)	0.26 (−0.04 to 0.56)	0.13 (−0.17 to 0.42)
MS-T v. SB	−0.06 (−0.32 to 0.21)	0.11 (−0.15 to 0.38)	−0.20 (−0.47 to 0.07)	−0.08 (−0.35 to 0.18)	−0.04 (−0.38 to 0.31)	0.30 (−0.06 to 0.66)	−0.29 (−0.65 to 0.07)	−0.03 (−0.38 to 0.31)

Note: MS, *Media Smart*; SB, *Student Bodies*; Con=, control; CI=, confidence intervals; EDE-Q, eating disorder examination- questionnaire; WCS, weight concerns scale; DASS, depression, anxiety, stress scales; SATAQ-3, sociocultural attitudes towards appearance questionnaire-3; QOL-M, quality of life – mental; BMI, body mass index; ITT, intent-to-treat sample using multiple imputation for missing data; Measure completer sample, participants who completed baseline and at least one other assessment point. For continuous outcomes, a negative *d* indicates group 1 scoring at lower risk than group 2 for all measures apart from QOL-M, where a positive *d* indicates group 1 had a higher QOL-M. Between-group comparisons that are significantly different are bolded (*p* < 0.05). *12-month follow-up frequencies for new disordered cases at 12-month follow-up that did not meet diagnosis at baseline. ^12-month follow-up frequencies for cases who did meet disordered eating diagnosis at baseline but who did not meet diagnosis at 12-month follow-up. Between-group comparisons that are significantly different are bolded (*p* < 0.05).

controls was not significant. Supplementary Table 2 provides breakdowns of specific DE behaviours.

Discussion

The aim of this pragmatic RCT was to assess the real-world effectiveness of two online DE prevention programs. This study differed to earlier trials in that: participants were sought from the broader community; programs were pure 'self-help' rather than moderated by a personalized online therapist; and, less exclusion criteria for eating pathology were used.

What type of participants enrolled and accessed their allocated intervention?

Participants were experiencing higher levels of baseline eating pathology than any previous *SB* trial (EDE-Q Global *M* = 3.37), including those 'at very high risk' of an eating disorder (*M* = 2.31: Taylor *et al.* 2016), and clinical impairment scores (*M* = 21.61: Bohn & Fairburn, 2008) were in the clinical range (⩾16). This mimics how these interventions might be used in community settings, where website access would not be limited and where those with greater pathology might be more likely to access interventions.

While higher pathology and clinical impairment were associated with greater likelihood of accessing the intervention, the only unique predictor was identification as being 'committed to trying the program'. One-third of participants allocated to *SB* (30.0%) or *MS-T* (36.1%) never opened the website, consistent with the median engagement level of 38% found in a review of online engagement (Waller & Gilbody, 2009). There could be value in providing brief motivation enhancement strategies at the completion of baseline measures to increase the likelihood of accessing a program. Alternatively, motivation could be an inclusion criterion, recommending guided or face-to-face options to those who are less motivated.

Rates of intervention completion were low. Of those who opened their allocated intervention, 51.6% (63/122) and 64.3% (63/98) completed module 1 of *MS-T* and *SB*, respectively, with attrition for *SB* significantly greater than for *MS-T* and the full *MS-T* program completed at a 10-fold higher rate than *SB*. Modules were released weekly, in line with earlier *SB* trials, as it was hoped that this would give participants time to complete homework and consolidate learning. However, this might have been a deterrent to some and future trials could investigate whether allowing open access to the full intervention is associated with greater completion. Also, *MS-T* participants received an automated weekly email alert that their next module was available, whereas *SB* participants did not, suggesting that reminders result in higher completion rates. The early and high drop-off rates for *SB* were inconsistent with previous *SB* trials. In the most comparable study, Kass *et al.* (2014) reported that approximately half the participants in an unmoderated group completed at least half the *SB* program, much higher usage than observed in the current trial. The Kass *et al.* trial included journal log prompts and guided discussions, both of which may reinforce participation.

MS-T was designed to be aesthetically similar to *SB* with comparable module length and learning activities, thus it is unlikely that the differing completion rates were due to program appearance factors. One explanation relates to the difference in the way weight concerns were targeted: *SB* initially focuses on a variety of cognitive issues related to body image with a focus on

Table 4. Tertiary outcomes, presence of disordered eating at 12-month follow-up (significant results are bolded)

Analysis	MS-T N (%)	SB N (%)	Control N (%)	OR (95% CI) MS-T v. control	OR (95% CI) SB v. control
Prevention effects[a]					
1. Not adjusting for treatment	1/23 (4.3)	1/12 (8.3)	13/57 (22.8)	0.15 (0.02–1.25)	0.31 (0.04–2.61)
2. Adjusting for treatment				0.23 (0.03–1.94)	0.46 (0.05–4.03)
No treatment	1/23 (4.3)	1/12 (8.3)	8/48 (16.7)	0.22 (0.03–1.93)	0.45 (0.05–4.03)
Yes treatment	–	–	5/9 (55.6)	–	–
Treatment effects[b]					
1.Not adjusting for treatment	16/37 (43.2)	15/45 (33.3)	21/87 (24.1)	**0.42 (0.19–0.94)**	0.64 (0.29–1.40)
2. Adjusting for treatment				**0.41 (0.18–0.94)**	0.63 (0.29–1.40)
No treatment	11/28 (39.3)	13/34 (38.2)	18/67 (26.9)	0.57 (0.22–1.44)	0.59 (0.25–1.43)
Yes treatment	5/9 (55.6)	2/11 (18.2)	3/20 (15.0)	**0.14 (0.02–0.85)**	0.79 (0.11–5.66)

Note: [a]Proportion per condition of new cases of disordered eating at 12-month follow-up: only participants who did not have DE at baseline.
[b]Proportion per condition of no longer being a disordered eating case: only participants who did have DE at baseline. Bolded values indicate significant odds ratios ($p < 0.05$)

self-directed behavioural change in later modules; while *MS –T* focuses on critiquing media messages and developing strategies to resist these and related pressures. Whilst completion rates were low making it difficult to draw conclusions about the program targets, it could be that *MS-T* participants found their content of personal relevance, which might explain higher completion rates.

What were the outcomes for those who accessed their allocated intervention?

ITT analyses revealed minimal differences in outcomes across the groups and no differences at 12-month follow-up. No significant effect was found for the primary outcome (EDE-Q Global) and only two effects for secondary outcome variables with low ES - *MS-T* participants reported higher quality of life–mental relative to both controls and *SB* (6-month follow-up), and *MS-T* and controls had lower clinical impairment relative to *SB* (post-program). These minimal differences are likely explained by low rates of intervention completion.

SB did not perform as well as previous findings (e.g. Jacobi *et al.* 2012; Taylor *et al.* 2016), though findings are difficult to interpret given very low completion rates. The most likely reason for this was the absence of a personalized online moderator, which has previously been recommended as an important for internet interventions to work (Andersson *et al.* 2009). This is consistent with the recent comparison of guided *v.* self-help versions of *SB* where the guided group had superior outcomes (Kass *et al.* 2014). Second, *SB* was implemented and hosted by a different web team in the USA than earlier trials and did not support some basic features of the program, such as notifications and interactivity or feedback. At the time of this trial, the team was working on a number of larger *SB* trials where it is possible that program delivery in the current trial was sub-optimal.

Of those who did access their intervention, *MS-T* achieved some promising results. For the measure completer sample, *MS-T* participants reported significant benefits relative to both controls and *SB* at varying time points for EDE-Q Global, depression, ineffectiveness, clinical impairment, media internalization, and quality of life – mental. Amongst measure completers at

12-month follow-up, *MS-T* participants reported significantly lower Global EDE-Q scores than controls and lower levels than both controls and *SB* for depression (also at 6-month follow-up); ineffectiveness; and, clinical impairment. Given measure completion was twice as likely by those who completed more modules than those who did not, these findings suggest participants needed to participate in *MS-T* to experience benefit.

Whilst not significant, *MS-T* participants who did not meet DE criteria at baseline were 85% less likely than controls to meet criteria at 12-month follow-up. The rates of reduction in onset of eating pathology were comparable with that found for face-to-face delivery of *The Body Project* (60%) and *Healthy Weight* (61%) and in earlier trials of *SB* (88% for those with elevated BMI at baseline: Taylor *et al.* 2006), though these respective findings were over a 2–3 year follow-up (Stice *et al.* 2008b) using diagnostic criteria. Of those who met DE criteria at baseline, those in the *MS-T* group were significantly less likely (59%) than controls to still meet diagnosis at 12-month follow-up, including controlling for accessing external ED treatment. To the best of our knowledge, this was the first time a program has achieved a treatment effect of this type. Most previous studies have excluded participants with elevated levels of baseline eating pathology. *MS-T* is well-suited to a clinical population given that the program is not face-to-face and it does not include open discussion groups where other participants could be exposed to the unhelpful content. It was of interest that amongst those who did access external ED treatment, *MS-T* participants were 85% less likely than controls to have DE at 12-month follow-up. This suggests that *MS-T* might augment treatment response and warrants further investigation.

Limitations and strengths

Four limitations were present in this study, the first two related to the pragmatic RCT approach. First, measure completion was self-report rather than an interview which has been used in other prevention trials (e.g. Taylor *et al.* 2006). Second, rates of measure completion were lower than in other targeted prevention trials where this was likely exacerbated by online self-report assessment and findings for the ITT analyses should be interpreted cautiously. Third, the lost program usage data for *n* = 50 new *SB*

participants was beyond our control and this was managed in a conservative fashion (i.e. confirming no differences between these and the remaining *SB* participants and omitting from analyses). Finally, the follow-up period was shorter than some previous prevention trials (Wilksch & Wade, 2009)

There were also strengths, including the: evaluation of multiple programs by two research groups; a large, community-based sample recruited in just 12-months; minimal exclusion criteria allowing for investigation of outcomes by those with pre-existing eating pathology; and, the inclusion of indicators of clinical impairment. Whilst the fully automated online screening and program allocation procedures allowing for real-world application with minimal cost were also a strength, strategies for increasing user engagement and reducing dropout need to be explored. Of the two programs, *SB* is a far more established program that has shown consistent benefit in previous trials and is likely better suited to guided delivery using a personalized online therapist. The results indicate that *MS-T* shows promise as a 'pure self-help' program where further investigations are indicated.

References

Andersson G, Carlbring P, Berger T, Almlöv J and Cuijpers P (2009) What makes internet therapy work? *Cognitive Behaviour Therapy* **38**, 55–60.

Austin SB (2016). Accelerating progress in eating disorders prevention: a call for policy translation research and training. *Eating Disorders*, **24**, 6–19.

Bardone-Cone AM, Harney MB, Maldonado CR, Lawson MA, Robinson DP, Smith R *et al.* (2010) Defining recovery from an eating disorder: conceptualization, validation, and examination of psychosocial functioning and psychiatric comorbidity. *Behaviour Research and Therapy* **48**, 194–202.

Berg KC, Peterson CB, Frazier P and Crow SJ (2011) Convergence of scores on the interview and questionnaire versions of the eating disorder examination: a meta-analytic review. *Psychological Assessment* **23**, 714–724.

Berg KC, Stiles-Shields EC, Swanson SA, Peterson CB, Lebow J and Le Grange D (2012) Diagnostic concordance of the interview and questionnaire versions of the eating disorder examination. *International Journal of Eating Disorders* **45**, 850–855.

Bohn K and Fairburn CG (2008) The clinical impairment assessment questionnaire (CIA). In Fairburn CG (ed.). *Cognitive Behavioral Therapy for Eating Disorders*. New York: Guilford Press, pp. 315–317.

Braun D, Sunday S and Halmi K (1994) Psychiatric comorbidity in patients with eating disorders. *Psychological Medicine* **24**, 859–867.

Bruning Brown J, Winzelberg AJ, Abascal LB and Taylor CB (2004). An evaluation of an internet-delivered eating disorder prevention program for adolescents and their parents. *Journal of Adolescent Health* **35**, 290–296.

Celio AA, Winzelberg AJ, Wilfley DE, Eppstein-Herald D, Springer EA, Dev P *et al.* (2000) Reducing risk factors for eating disorders: comparison of an internet- and a classroom-delivered psychoeducational program. *Journal of Consulting and Clinical Psychology* **68**, 650–657.

Enders CK (2010). *Applied Missing Data Analysis*. New York: Guilford Press.

Espelage DL, Mazzeo SE, Aggen SH, Quittner AL, Sherman R and Thompson R (2003) Examining the construct validity of the Eating Disorder Inventory. *Psychological Assessment* **15**, 71–80.

Fairburn CG and Beglin SJ (1994) Assessment of eating disorders: interview or self-report questionnaire? *International Journal of Eating Disorders* **16**, 363–370.

Fairburn CG and Cooper Z (2011) Eating disorders, DSM-5 and clinical reality. *The British Journal of Psychiatry* **198**, 8–10.

Fairburn CG, Cooper Z and Shafran R (2003) Cognitive behaviour therapy for eating disorders: a 'transdiagnostic' theory and treatment. *Behaviour Research and Therapy* **41**, 509–528.

Fairweather-Schmidt AK and Wade TD (2014) DSM-5 eating disorders and other specified eating and feeding disorders: is there a meaningful differentiation? *International Journal of Eating Disorders* **47**, 524–533.

Franko DL and Keel PK (2006) Suicidality in eating disorders: occurrence, correlates, and clinical implications. *Clinical Psychology Review* **26**, 769–782.

Garner DM, Olmstead MP and Polivy J (1983) Development and validation of a multidimensional Eating Disorder Inventory for anorexia nervosa and bulimia nervosa. *International Journal of Eating Disorders* **2**, 15–34.

Henry JD and Crawford JR (2005) The short-form version of the Depression Anxiety Stress Scales (DASS-21): construct validity and normative data in a large non-clinical sample. *The British Journal of Clinical Psychology* **44**, 227–239.

Herpertz-Dahlmann B., Dempfle A., Konrad K., Klasen F. and Ravens-Sieberer U. BELLA Study Group (2015). Eating disorder symptoms do not just disappear: the implications of adolescent eating-disordered behaviour for body weight and mental health in young adulthood. *European Child & Adolescent Psychiatry* **24**, 675–684.

Jacobi C, Abascal L and Taylor CB (2004) Screening for eating disorders and high-risk behavior: caution. *International Journal of Eating Disorders* **36**, 280–295.

Jacobi C, Hütter K and Fittig E (in press). Psychosocial risk factors for eating disorders. In Agras S (ed.). *The Oxford Handbook of Eating Disorders*. New York: Oxford University Press.

Jacobi C, Morris L, Beckers C, Bronisch-Holtze J, Winter J, Winzelberg AJ *et al.* (2005) Reduction of risk factors for disordered eating behavior: adaptation and initial results of an internet-supported prevention program. *Zeitschrift fur Gesundheitspsychologie* **13**, 92–101.

Jacobi C, Morris L, Beckers C, Bronisch-Holtze J, Winter J, Winzelberg AJ *et al.* (2007) Maintenance of internet-based prevention: a randomized controlled trial. *International Journal of Eating Disorders* **40**, 114–119.

Jacobi C, Völker U, Trockel MT and Taylor CB (2012) Effects of an internet-based intervention for subthreshold eating disorders: a randomized controlled trial. *Behaviour Research and Therapy* **50**, 93–99.

Kass AE, Trockel M, Safer DL, Sinton MM, Cunning D, Rizk MT *et al.* (2014) Internet-based preventive intervention for reducing eating disorder risk: a randomized controlled trial comparing guided with unguided self-help. *Behaviour Research and Therapy* **63**, 90–98.

Kazdin AE, Fitzsimmons-Craft EE and Wilfley DE (2017) Addressing critical gaps in the treatment of eating disorders. *International Journal of Eating Disorders* (in press).

Killen JD, Taylor C, Hayward C, Wilson DM, Haydel KF, Hammer LD, et al. (1994) Pursuit of thinness and onset of eating disorder symptoms in a community sample of adolescent girls: a three-year prospective analysis. *International Journal of Eating Disorders* **16**, 227–238.

Kotler LA, Cohen P, Davies M, Pine DS and Walsh B (2001) Longitudinal relationships between childhood, adolescent, and adult eating disorders. *Journal of the American Academy of Child & Adolescent Psychiatry* **40**, 1434–1440.

Lecrubier Y, Sheehan DV, Weiller E, Amorim P, Bonora I, Harnett Sheehan K *et al.* (1997) The Mini International Neuropsychiatric Interview (MINI). A short diagnostic structured interview: reliability and validity according to the CIDI. *European Psychiatry* **12**, 224–231.

Levine MP and Smolak L (2016) The role of protective factors in the prevention of negative body image and disordered eating. *Eating Disorders*, **24**, 39–46.

Lovibond P and Lovibond S (1995) The structure of negative emotional states: comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour Research and Therapy* **33**, 335–343.

Low KG, Charanasomboon S, Lesser J, Reinhalter K, Martin R, Jones H et al. (2006) Effectiveness of a computer-based interactive eating disorders prevention program at long-term follow-Up. *Eating Disorders: The Journal of Treatment & Prevention* 14, 17–30.

Melioli T, Bauer S, Franko DL, Moessner M, Ozer F, Chabrol H et al. (2016) Reducing eating disorder symptoms and risk factors using the internet: a meta-analytic review. *International Journal of Eating Disorders* 49, 19–31.

Mission Australia (2010). *National Survey of Young Australians.* Sydney: Mission Australia.

Mond JM, Hay PJ, Rodgers B, Owen C and Beumont PJV (2004) Validity of the Eating Disorder Examination Questionnaire (EDE-Q) in screening for eating disorders in community samples. *Behaviour Research and Therapy* 42, 551–567.

Murray CJL and Lopez AD (eds.) (1996). *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability From Diseases, Injuries and Risk Factors in 1990 to 2020.* Cambridge, MA: Harvard University Press.

Neumark-Sztainer D, Wall M, Larson NI, Eisenberg ME and Loth K (2011) Dieting and disordered eating behaviors from adolescence to young adulthood: findings from a 10-year longitudinal study. . *Journal of the American Dietetic Association* 111, 1004–1011.

Patel V, Chowdhary N, Rahman A and Verdeli H (2011) Improving access to psychological treatments: lessons from developing countries. *Behaviour Research and Therapy* 49, 523–528.

Patton GC, Selzer R, Coffey C, Carlin JB and Wolfe R (1999) Onset of adolescent eating disorders: population based cohort study over 3 years. *British Medical Journal* 318, 765–768.

Piran N (2015) New possibilities in the prevention of eating disorders: the introduction of positive body image measures. *Body Image* 14, 146–157.

Rohde P, Stice E, Shaw H, Gau JM and Ohls OC (in press) Age effects in eating disorder baseline risk factors and prevention intervention effects. *International Journal of Eating Disorders.*

Sheehan DV, Lecrubier Y, Harnett Sheehan K, Janavs J, Weiller E, Keskiner A et al. (1997) The validity of the Mini International Neuropsychiatric Interview (MINI) according to the SCID-P and its reliability. *European Psychiatry* 12, 232–241.

Sterne JA, White IR, Carlin JB, Spratt M, Royston P, Kenward MG et al. (2009) Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. *BMJ: British Medical Journal* 338, b2393.

Stice E (2001) A prospective test of the dual-pathway model of bulimic pathology: mediating effects of dieting and negative affect. *Journal of Abnormal Psychology* 110, 124–135.

Stice E, Chase A, Stormer S and Appel A (2001) A randomized trial of a dissonance-based eating disorder prevention program. *International Journal of Eating Disorders* 29, 247–262.

Stice E, Davis K, Miller NP and Marti C (2008a). Fasting increases risk for onset of binge eating and bulimic pathology: a 5-year prospective study. *Journal of Abnormal Psychology* 117, 941–946.

Stice E, Marti C, Spoor S, Presnell K and Shaw H (2008b) Dissonance and healthy weight eating disorder prevention programs: long-term effects from a randomized efficacy trial. *Journal of Consulting and Clinical Psychology* 76, 329–340.

Stice E, Mazotti L, Weibel D and Agras WS (2000) Dissonance prevention program decreases thin-ideal internalization, body dissatisfaction, dieting, negative affect, and bulimic symptoms: a preliminary experiment. *International Journal of Eating Disorders* 27, 206–217.

Stice E, Rohde P, Durant S and Shaw H (2012) A preliminary trial of a prototype internet dissonance-based eating disorder prevention program for young women With body image concerns. *Journal of Consulting and Clinical Psychology*, 80, 907–916.

Stice E, Rohde P, Gau J and Shaw H (2009) An effectiveness trial of a dissonance-based eating disorder prevention program for high-risk adolescent girls. *Journal of Consulting & Clinical Psychology* 77, 825–834.

Stice E, Rohde P, Shaw H and Gau J (2011) An effectiveness trial of a selected dissonance-based eating disorder prevention program for female high school students: long-term effects. *Journal of Consulting and Clinical Psychology* 79, 500–508.

Stice E, Shaw H and Marti CN (2007) A meta-analytic review of eating disorder prevention programs: encouraging findings. *Annual Review of Clinical Psychology* 3, 207–231.

Taylor C, Bryson S, Luce KH, Cunning D, Doyle AC, Abascal LB et al. (2006) Prevention of eating disorders in At-Risk College-Age women. *Archives of General Psychiatry* 63, 881–888.

Taylor CB, Kass AE, Trockel M, Cunning D, Weisman H, Bailey J et al. (2016) Reducing eating disorder onset in a very high risk sample with significant comorbid depression: a randomized controlled trial. *Journal of Consulting and Clinical Psychology* 84, 402.

Thompson J, van den Berg P, Roehrig M, Guarda AS and Heinberg LJ (2004) The sociocultural attitudes towards appearance scale-3: development and validation. *International Journal of Eating Disorders* 35, 293–304.

Vannucci A, Kass AE, Sinton MM, Aspen V, Weisman H, Bailey JO et al. (2012) An examination of the clinical impairment assessment among women at high risk for eating disorder onset. *Behaviour Research and Therapy* 50, 407–414.

Wade TD, Wilksch SM and Lee C (2012) A longitudinal investigation of the impact of disordered eating on young women's quality of life. *Health Psychology* 31, 352–359.

Waller R and Gilbody S (2009) Barriers to the uptake of computerized cognitive behavioural therapy: a systematic review of the quantitative and qualitative evidence. *Psychological Medicine* 39, 705–712.

Ware JE, Snow KK, Kosinski M and Gandek B (1993). *SF-36 Health Survey: Manual and Interpretation Guide.* The Health Institute, New England Medical Center.

Wertheim EH, Koerner J and Paxton SJ (2001) Longitudinal predictors of restrictive eating and bulimic tendencies in three different age groups of adolescent girls. *Journal of Youth and Adolescence* 30, 69–81.

Wilksch SM (2014) Where did universal eating disorder prevention go? *Eating Disorders: The Journal of Treatment & Prevention* 22, 184–192.

Wilksch SM, Paxton SJ, Byrne SM, Austin SB, McLean SA, Thompson KM et al. (2015) Prevention across the spectrum: a randomized controlled trial of three programs to reduce risk factors for both eating disorders and obesity. *Psychological Medicine* 45, 1811–1823.

Wilksch SM and Wade TD (2009) Reduction of shape and weight concern in young adolescents: a 30-month controlled evaluation of a media literacy program. *Journal of the American Academy of Child and Adolescent Psychiatry* 48, 652–661.

Winzelberg AJ, Eppstein D, Eldredge KL, Wilfley D, Dasmahapatra R, Dev P et al. (2000) Effectiveness of an internet-based program for reducing risk factors for eating disorders. *Journal of Consulting and Clinical Psychology* 68, 346–350.

Winzelberg AJ, Taylor CB, Sharpe T, Eldredge KL, Dev P and Constantinou PS (1998) Evaluation of a computer-mediated eating disorder intervention program. *International Journal of Eating Disorders* 24, 339–349.