Habitual Behavior as a Mediator Between Food-Related Behavioral Activation and Change in Symptoms of Depression in the MooDFOOD Trial

Matthew Owens1, Ed Watkins1, Mariska Bot2,3,4, Ingeborg A. Brouwer3,5, Miquel Roca6, Elisabeth Kohls7, Brenda W. J. H. Penninx2, Gerard van Grootheest2,4, Ulrich Hegerl8, Margalida Gili6, and Marjolein Visser5, on behalf of the MooDFOOD Prevention Trial Investigators

1Department of Psychology, University of Exeter; 2Department of Psychiatry, Amsterdam UMC, Amsterdam, The Netherlands; 3Amsterdam Public Health Research Institute, Vrije Universiteit Amsterdam; 4GGZ inGeest Specialized Mental Health Care, Amsterdam, The Netherlands; 5Department of Health Sciences, Faculty of Science, Vrije Universiteit Amsterdam; 6Institut Universitari d’Investigació en Ciències de la Salut (IUNICS/IDISBA, Rediapp), School of Medicine, University of Balearic Islands; 7Department of Psychiatry and Psychotherapy, Medical Faculty, University Leipzig; and 8Department of Psychiatry, Psychosomatics and Psychotherapy, Goethe-Universität Frankfurt

Abstract
In this study, we tested potential mediators that may explain change in depressive symptoms following exposure to a food-related behavioral activation intervention (F-BA). These included behavioral activation, avoidance and rumination, eating styles, body mass index, and dietary behavior at baseline and 3-month and 12-month follow-up. The trial used a community sample of 1,025 overweight adults with elevated depressive symptoms without current major depression. Participants were randomly assigned to one of four trial arms: either daily nutritional supplements (vs. placebo) alone or in combination with F-BA (vs. no F-BA) over 12 months. Although F-BA did not significantly reduce depressive symptoms (standardized regression coefficient $b = -0.223, SE = 0.129; p = 0.084$), significant mediators included emotional eating ($b = -0.028, SE = 0.014; p = 0.042$) and uncontrolled eating ($b = -0.039, SE = 0.016; p = 0.013$), suggesting that learning adaptive responses to emotional and food cues may underlie effects of F-BA on depressive symptoms.

Keywords
depression, prevention, behavioral activation, habits, eating styles

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Depression is highly prevalent (Kessler & Bromet, 2013; Wittchen et al., 2011) and currently the leading global health problem (World Health Organization, 2017) with significant negative outcomes including low educational attainment, relationship disruption, unstable employment, and an increased risk of mortality through somatic illness and suicide (Kessler, 2012). Economically, major depression can be devastating, with annual associated costs estimated to stand at $1 trillion (Chisholm et al., 2016), driven largely by loss of productivity (Evans-Lacko & Knapp, 2016). For these reasons, coupled with the realization that treatment alone is unlikely to ever be sufficient to reach all those in need, depression prevention interventions have become a global priority (Cuijpers et al., 2012; Ebert & Cuijpers, 2018).

Being overweight and obese has become a global pandemic (Abarca-Gómez et al., 2017). Being overweight is associated with a range of physical health...
conditions such as diabetes (Verma & Hussain, 2017) and may have a complex, bidirectional relationship with depression (Knüppel et al., 2019; Luppino et al., 2010) with evidence for shared etiologies and biological mechanisms between the two (Milaneschi et al., 2019).

One potential theoretical framework to understand the link between body weight and depression is nutritional psychology, which hypothesizes that positive changes in diet and food-related (psychological) behavior may prevent depression (Jacka et al., 2017; Sarris et al., 2015). For example, adhering to a Mediterranean diet (e.g., plenty of fruit and vegetables, fish, and olive oil) is associated with a lower risk of developing depression (Elstgeest et al., 2019; Lassale et al., 2019; Molendijk et al., 2018; Nicolaou et al., 2019). However, randomized trials are needed to establish whether diet and food-related behavior can causally influence depression. The MooDFOOD trial (Bot et al., 2019; Roca et al., 2016) is one such randomized controlled prevention trial enabling the investigation of the role of nutritional and psychological factors on depression. The MooDFOOD trial tested the effect of both daily nutritional supplementation (omega-3 fatty acids, selenium, folic acid, and vitamin D plus calcium) compared with placebo and a protocol-based psychological intervention, food-related behavioral activation (F-BA), compared with no F-BA. F-BA was specifically designed to change habitual behavior and included behavioral activation approaches including self-monitoring, functional analysis, and activity scheduling and applied these to improving mood by changing dietary habits and food-related behaviors (e.g., snacking, emotional eating), increasing positive behaviors, and emphasizing a Mediterranean-style diet. For an overview of the MooDFOOD findings in the broader context of this emerging area, see Owens, Watkins, Bot, Brouwer, Roca, Kohls, Penninx, van Grootheest, Cabot, et al. (2020).

The trial found no significant effects of intervention on the primary outcome (incidence of de novo major depression over a 12-month period; Bot et al., 2019). However, because of the unexpectedly low incidence of depression, the trial was underpowered to detect such effects. Secondary analyses showed that F-BA was effective at preventing depression for participants who adhered to the intervention, however. Further analysis on depressive symptoms demonstrated two main points. First, there was evidence that the effect of nutritional supplementation on depressive symptoms was significantly worse than placebo. Second, there was some evidence that the behavioral intervention was effective at reducing symptoms, particularly for people with higher symptom severity at baseline. Subsequent analyses demonstrated that the F-BA is able to improve diet in participants (Grasso et al., 2019), reduce the tendency to engage in emotional eating, reduce uncontrolled eating, and increase cognitive restrained eating but did not significantly reduce body mass index (BMI; Paans et al., 2020). Although we have found the MooDFOOD F-BA to be a feasible intervention to deliver, one that most participants found acceptable to do and would recommend to a friend, the mediating factors underlying any reduction in depressive symptoms remain untested, making it difficult to design effective interventions in the future (Owens, Watkins, Bot, Brouwer, Roca, Kohls, Penninx, van Grootheest, Cabot, et al., 2020).

In the present study, we formally tested potential mediators that may help further understand underlying mechanisms at work in psychological interventions such as the F-BA. It is important to investigate hypothesized intermediate processes by testing the role of putative mediators, even in the absence of an overall treatment effect, for both statistical and conceptual reasons (O’Rourke & MacKinnon, 2018). First, it has been shown that the mediated effect may be statistically significant even when the total effect is not (O’Rourke & MacKinnon, 2015). For example, a simulation study showed that the power to detect a mediated effect was more than 10 times greater than the power to detect a total effect of the same size (O’Rourke & MacKinnon, 2018). Second, mediation analyses provide an opportunity to test known and hypothesized mediators, to perform “manipulation checks” to test whether an intervention has changed the behavior/index that it was designed to (MacKinnon, 1994), and to inform the development of theory. Identifying mediators can help identify potential ways to enhance intervention effects by identifying key processes to target and is recommended in intervention and prevention research (Kraemer et al., 2002; O’Rourke & MacKinnon, 2018).

F-BA was designed within the behavioral activation (BA) framework (Richards et al., 2016; Veale, 2008; Watkins et al., 2011, 2016), which is an empirically supported efficacious treatment for depression that encourages individuals to increase their engagement with activities rather than avoid them (Ekers et al., 2014; Richards et al., 2016). In addition, there was strong emphasis on changing habitual behavior by identifying triggers, planning alternative responses, and repeating behaviors routinely to develop good habits.

Habits can be defined as learned dispositions to repeat past responses (Wood & Neal, 2007). They are initiated by “features of the context,” or environment, that have often occurred together when the habitual behavior is performed in the past. These features may be external (e.g., locations, behavior, or particular people) or internal (e.g., thoughts or feelings; Orbell & Verplanken, 2010). Habits can be either overt behaviors...
(e.g., emotional eating) or internal covert behaviors such as patterns of repetitive negative thought (e.g., Watkins & Nolen-Hoeksema, 2014). In this conceptualization of habit, for some individuals, an internal state (e.g., low mood) will trigger learned, dysfunctional responses. Central to F-BA was identifying and reducing maladaptive habits related to mood (e.g., avoidance) and related to food (e.g., emotional eating, snacking) and building positive habits, both mood related (better coping strategies) and food related (increased adherence to Mediterranean diet).

We hypothesized that the effects of F-BA on reducing depression would be mediated by behavioral change, especially change in behavioral habits. In this general framework, there are a number of competing models that each generate distinct a priori hypotheses as to which specific changes in behavior would be potential mediators of treatment effects, as described below. We hypothesized that these factors would be less active in the nutritional supplement approach and hence tested effects of pill with placebo as a comparison, with the hypothesis that there would be no effect of these mediators in this comparison.

A core element of F-BA was identifying and scheduling positive activities that are rewarding and beneficial to the individual (Kanter et al., 2007). The theoretical model behind BA proposes that increasing activation and approach and reducing avoidance increases the likelihood of receiving positive reinforcement from the environment, which reduces symptoms of depression. There is evidence that avoidance is a maladaptive coping response (Nasrin et al., 2017), which predicts chronic and acute stress and depression (Holahan et al., 2005). Avoidance includes strategies to actively avoid powerful negative emotions and thoughts, such as social withdrawal, procrastination, retreating to bed, and drinking alcohol or excessive eating, which may reduce distress in the short term but contribute to the maintenance of an emotional disorder in the long term (Clancy et al., 2016; Moulds et al., 2007). One form of avoidance is depressive rumination, defined as the tendency to repetitively analyze the causes, meanings, and consequences of problems and symptoms of depression (Watkins, 2008) in which an individual is preoccupied with negative mood and upsetting events. Thus, a BA model of how F-BA works produces the hypotheses that the effects of F-BA on change in depressive symptoms would be mediated by (a) an increase in behavioral activation and (b) reduction in avoidance including rumination.

F-BA also targeted food-related behaviors, particularly, uncontrolled eating, defined as the tendency to overeat in an uncontrolled way, and emotional eating, defined as eating in response to negative emotions (van Strien et al., 2016). Both increased emotional eating and uncontrolled eating are associated with increased depression (e.g., Goldschmidt et al., 2014; Ouwens et al., 2009). Both involve frequent maladaptive and involuntary responses to particular contexts (the experience of internal emotional states for emotional eating, the experience of hunger or food and eating cues for uncontrolled eating), that is, are unhelpful mood- and food-related habits. F-BA aims to build positive eating habits and to shift patients from maladaptive (e.g., eating snacks) to adaptive coping responses (e.g., doing a positive activity) to negative mood and environmental cues. Thus, one hypothesis of how F-BA works is that it helps individuals to learn more adaptive responses to these cues and gain control over involuntary habits, as indexed by change in depressive symptoms being mediated by reductions in emotional eating and uncontrolled eating.

The final element in F-BA was an attempt to increase the uptake of a Mediterranean-style diet and reduce consumption of processed food and snacks. A habit change approach was used because of evidence of the importance of habit in eating behavior (Cleo et al., 2017; Verhoeven et al., 2012). Because a significant proportion of depressed individuals have a preference for sugary, high-fat foods and carbohydrate (Christensen, 2001; Elstgeest et al., 2019), whereas adherence to a Mediterranean diet is associated prospectively with a lower risk of depression, nutritional psychology models have proposed that increasing nutrients and decreasing fats and sugars in a whole-diet approach will lead to a reduction in depressive symptoms (Marx et al., 2019). Results from observational studies have suggested that lower levels of nutrients (e.g., omega-3, folic acid, vitamin D, selenium, and calcium) are associated with depression (Gao et al., 2012; Grosso et al., 2014; Milaneschi et al., 2019; Mlyniec et al., 2014; Rucklidge & Kaplan, 2013), and meta-analyses aggregating randomized controlled trials using specific nutrients as nutraceutical adjunctive or monotherapy in depressed patients (Firth et al., 2019; Sarris et al., 2016) have provided evidence for reduced depressive symptoms compared with placebo for several nutrients (e.g., omega-3 and vitamin D).

Because F-BA was able to improve diet quality in the MooDFOOD trial (Grasso et al., 2019), we can test this nutritional psychology model, which hypothesizes that the effects of F-BA on change in depressive symptoms would be mediated by a change in whole diet, as indexed by the extent to which people followed the MooDFOOD diet (based on a Mediterranean diet), reduced snacking, and shifted away from habitually eating sugar and fat. In addition, although weight change was not an explicit goal in the MooDFOOD trial, an alternative hypothesis tested here is that change
in BMI will mediate the effect of F-BA on depressive symptoms. This is plausible given that diets, such as that encouraged in the F-BA, can lead to healthy weight loss that may itself improve depressive symptoms. For example, several studies have shown positive effects of behavioral weight control on both depressed mood and weight (Brinkworth et al., 2009; Faulconbridge et al., 2018). To test these mediation hypotheses, we used depressive symptoms as the outcome in this analysis rather than diagnosis to increase the statistical power to detect the presence of mediation effects should they exist.

Method

Participants

This study used the data from the MooDFOOD randomized controlled prevention trial (Bot et al., 2019) cohort of overweight or obese adults (N = 1,025; mean age = 46.51 years, range = 18–75; mean Patient Health Questionnaire [PHQ-9] score = 7.42; mean BMI = 31.38) recruited from four sites in European countries (the Netherlands, Spain, Germany, and the United Kingdom).

Study design

The trial followed a 2 × 2 factorial design testing the effect of multinutrient supplements compared with placebo and/or food-related behavioral change on incidence of major depressive disorder (MDD) over a period of 1 year. Main eligibility criteria included being between 18 and 75 years old, being overweight or obese (BMI = 25–40 kg/m²), and having at least mild depressive symptoms as operationalized by a PHQ-9 score of 5 or more. Measures were collected at baseline (T0), 3 months (T3), 6 months (T6), and 12 months (T12; end of trial). The trial received approval from the local regional ethics committees and was conducted according to Helsinki good practice guidelines (World Medical Association, 2013).

Interventions

Food-related behavioral activation. The F-BA intervention was developed specifically for the MooDFOOD trial using the combined expertise within the consortium, including psychologists and nutritionists. The MooDFOOD F-BA consisted of a protocol-based manualized intervention using standard approaches of BA that are effective in depression treatment, including self-monitoring, functional analysis, and activity scheduling. This therapeutic approach has been employed in clinical trials that have proven to be effective with depressed patients (Richards et al., 2017; Watkins et al., 2011). When designing the F-BA intervention to change diet, it was recognized that patterns of eating are often habitual, and so models of habit change such as identifying and removing triggering cues to habits, learning and rehearsing new responses (Marteau et al., 2012; Wood & Neal, 2007), and using if–then plans (Gollwitzer & Sheeran, 2006) were also included. This was thought to be particularly important given that habit is one of the most powerful predictors of eating behavior (van’t Riet et al., 2011). Therefore, in addition to the original focus of BA on mood-related behaviors such as approach, avoidance, and rumination, the intervention in the MooDFOOD trial also included a focus on increasing positive food-related behaviors (e.g., eating a Mediterranean diet) and reducing potentially problematic food-related behaviors such as snacking and emotional eating (Owens et al., 2009).

F-BA was provided by trained psychologists familiar with BA (15 individual sessions, six group sessions, for a 1-year period), and a dietician was available to all study sites to provide advice. Initial training for psychologists was carried out over 4 days at one site (Exeter) in June 2015, and a refresher course was attended by all therapists at a midtrial consortium meeting in Reykjavik in May 2016. Intervention sessions were recorded (audio or video), and psychologists were supervised by a more experienced clinical psychologist who provided regular feedback on the intervention provided. In addition, online sessions led by the trial lead clinical psychologist (E. Watkins) were held to discuss the intervention and answer questions on the intervention protocol. Psychologist fidelity to the manualized F-BA was assessed by rating a random sample of therapy sessions against 14 key components at the end of the trial.

The MooDFOOD F-BA has been shown to be a feasible intervention to deliver in this population and one that is acceptable to participants (Owens, Watkins, Bot, Brouwer, Roca, Kohls, Penninx, van Grootheest, Cabout, et al., 2020). In the trial intervention, psychologists helped participants to set goals on mood- and food-related behaviors and introduced them to the idea of healthy eating. Goals were revisited and modified when necessary during subsequent sessions. During the intervention, participants kept a record of daily activities and habits with the aim of identifying triggers to habits and engagement in self-monitoring to improve food-related behaviors (e.g., regular meals per day, less snacking) and habitual eating patterns (e.g., decrease emotional eating). In addition, a participant manual was created providing more detail on the central concepts to help guide individuals through the intervention.

The intervention was delivered over a maximum of 21 sessions with up to 15 half-hour individual sessions,
provided in single or double (1-hr) meetings. Individual sessions were initially given weekly (Sessions 1–8) and then held every 2 weeks (Sessions 9–15). These were followed by up to six group sessions (maximum of 10 participants per group and lasting approximately 1 hr) occurring monthly initially (Sessions 16–18) and finally once every 2 months (Sessions 19–21). A range of potential strategies was employed, including food-related strategies and food group targets to aim for. These were introduced and explained to participants in the course of the first eight sessions and subsequently revisited and reviewed over the remaining sessions. An introduction to how eating behavior may be associated with mood improvement was provided in the third therapy session (2 weeks after the first F-BA session), which involved the provision of dietary guidelines based on a Mediterranean-style dietary pattern, referred to as the MooDFOOD dietary guidelines (Grasso et al., 2019). The F-BA was designed to start with individual sessions, leading on to group sessions; the former were focused on individualized review of food and mood habits and planning of new approaches and the latter were focused on consolidation and maintenance of what was learned.

Multinutrient supplements. Participants in the trial received either multinutrient supplements (1,412 mg of eicosapentaenoic and docosahexaenoic omega-3 fatty acids, ratio 3:1; 30 μg of selenium; 400 μg of folic acid; and 20 μg of vitamin D3 coupled with 100 mg of calcium) or placebos provided in two pills per day, to be taken daily for 1 year. Further details on the multinutrient supplements have been reported elsewhere (Bot et al., 2019; Roca et al., 2016).

Measures

Depressive symptoms. The PHQ-9 (Kroenke et al., 2001) is a well-validated nine-item self-report instrument that assesses current symptoms of depression over the preceding 2 weeks based criteria for major depression from the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013), which is used extensively in primary care and clinical trials (Gilbody et al., 2007). The internal consistency (α) in the present study ranged from .93 to .99.

Major depressive disorder. A previous history prevalence of MDD was measured using a brief standardized diagnostic interview (duration of 15–30min): the Major Depression module from the Mini International Neuropsychiatric Interview (Lecrubier et al., 1997; Sheehan et al., 1997). This measure was also used in the trial to assess current depression (within the preceding 6 months), which was one of the exclusion criteria (Bot et al., 2019).

Behavioral activation. Activation and avoidance/rumination were measured using the Behavioral Activation for Depression Scale (BADS). The BADS is a well-validated 25-item self-report instrument measuring the extent to which people become more activated over time, developed to assess change during BA therapy (Kanter et al., 2007, 2009). The BADS includes four factors of Activation, Avoidance/Rumination, Work/School impairment, and Social impairment as well as a total score. We used the Avoidance/Rumination and Activation subscales in the present analysis. The BADS has demonstrated good internal consistency and 1-week test–retest reliability in the general population (α = .87, r = 0.74, respectively; Kanter et al., 2007). The internal consistency in the present data was acceptable for the Avoidance/Rumination subscale (α = .83–.86) and the Activation subscale (αs = .80–.83). The BADS has previously shown to be sensitive as a mediator in previous BA depression trials (e.g., Richards et al., 2017).

Eating styles. The revised 18-item Three Factor Eating Questionnaire (TFEQ-R18; Karlsson et al., 2000) was used to assess emotional (e.g., “When I feel blue, I often overeat”; “When I feel lonely, I console myself by eating”; “When I feel anxious, I find myself eating”), uncontrolled (e.g., “Sometimes when I start eating, I just can’t seem to stop”; “When I smell a delicious food, I find it very difficult to keep from eating, even if I have just finished a meal”; “When I see a real delicacy, I often get so hungry that I have to eat right away”), and restrained eating (e.g., “I consciously hold back at meals in order not to weight gain”). There is good psychometric evidence for the presence of this three-factor structure in the TFEQ-R18, in which each factor has demonstrated good internal consistency (Angle et al., 2009). The measure has also been found to be applicable to the general population (de Lauzon et al., 2004). The TFEQ-R18 uses a 4-point Likert response format for each item. Higher scale scores in the respective scales are indicative of greater emotional eating, uncontrolled eating, and cognitive restraint, respectively. Raw scale scores were transformed to a scale from 0 to 100 as follows: (raw score − lowest possible raw score)/possible raw score range × 100 (de Lauzon et al., 2004).

Snacking. The food-behavior questionnaire is a bespoke instrument designed to measure a range of food-related behaviors. We used four items to measure snacking levels (“Eating between meals e.g., while doing other things, travelling or just taking a break can be called snacking. How often in the preceding 7 days do the following statements describe your activities related to snacking?”). The four new items (“While watching TV I snack,” “While reading I snack,” “While using the computer/playing video games I snack,” and “I snack without being aware...
that I was eating”) are rated by participants using a 5-point Likert-type scale with answers of never (0), rarely (1), sometimes (2), often (3), and very often (4). We summed the items for an overall estimate of snacking levels. This bespoke instrument has not yet been formally psychometrically tested: The α values in the present study for snacking ranged from .69 to .71.

**Mediterranean diet score.** Participants reported their usual food intake during the previous 12 months by completing an online self-administered food frequency questionnaire (FFQ) at T0, T6, and T12 (end of trial). The GA2LEN FFQ was used because it has been shown to be an appropriate tool to estimate dietary intake across Europe regardless of cultural and linguistic differences (Garcia-Larsen et al., 2011). The FFQ included 210 food items that were categorized into 18 food groups: (a) vegetables, (b) fruit, (c) fish, (d) meat, (e) egg/soy, (f) pulses/legumes, (g) nuts, (h) potatoes, (i) whole grains, (j) refined grains, (k) low-fat dairy products, (l) high-fat dairy products, (m) olive oil, (n) other fats/oils, (o) sweets/extras, (p) soft drinks, (q) alcoholic beverages, and (r) water/coffee/tea. A Mediterranean diet score was calculated following the procedure outlined elsewhere (Grasso et al., 2019).

**Food habits.** We measured eating diets high in sugar and fat as a habit using six items from the Self-Reported Habit Index (Verplanken & Orbell, 2003), including four items identified as representing automaticity in the Self-Reported Behavioural Automaticity Index (e.g., “I do without thinking”) and two further items that assessed the frequency of behavior (e.g., “I do frequently”), with respect to eating a diet high in sugar and fats. The stem used was “Eating a diet high in sugar and fats (e.g., sweets, crisps, biscuits, chocolates, ice cream, fizzy drinks, cake, fast food) is something….” Each item is rated on a 7-point Likert scale that ranges from strongly disagree to strongly agree, giving scores from 6 to 42. The α values in the present study ranged from .94 to .95.

**Psychologist F-BA fidelity ratings.** F-BA sessions were rated by experienced supervising clinical psychologists against 14 key components (e.g., “Provide education about eating patterns, healthy diet” and “Link behaviors to triggers and warning signs – i.e., cues to habits”) using a Likert-type response scale ranging from 0 to 6 (poor to excellent). Mean fidelity scores were calculated over all 14 items. The a priori threshold for good fidelity was set at 4.

**Statistical approach**

As recommended for tests of mediation, our analyses examined the change in a putative mediator before change in outcome to address the issue of temporal precedence and reduce the likelihood of backward causation (Kraemer et al., 2002). The statistical models presented here tested whether any change in depressive symptoms (PHQ-9) from T3 to T12, adjusting for baseline levels, was explained by change in the putative mediators from baseline to T3 (but for Mediterranean diet score, change from baseline to T6, adjusting for baseline). The analysis of multiple measurements of depressive symptoms has the benefit of increased statistical power and, crucially, allows for a test of potential prospective change in the mediator that precedes symptom change.

We used the Mplus program (Version 8.1; Muthén & Muthén, 2017). Indirect effects (mediation) models were conducted using the statistically powerful product of coefficients approach (MacKinnon et al., 2002). We requested 10,000 random bootstrapped samples with replacement (Shrout & Bolger, 2002) calculating biascorrected 95% confidence intervals (CI) to account for the skew in the bootstrapped distribution. We used effect coding (−1, +1) to estimate indirect effects of trial arm (F-BA vs. no F-BA) on depressive symptoms at 12 months via the potential mediators. As a comparison analysis, we assessed indirect effects in the nutritional supplement arm compared with placebo in the same way. Both interventions were included in the same model. All models were adjusted for site, sex, and a history of clinical depression. Full information maximum likelihood estimation was used to account for missing data, which is a robust, unbiased, and efficient technique outperforming traditional approaches to missing data (Enders & Bandalos, 2001). We report unstandardized regression coefficients and ratio of these to the standard error, which is an approximately normally distributed variable (analogous to a z score). We also report the indirect and total effects and calculate the proportion mediated (PM) as a mediation effect size. The PM is a measure of the relative magnitude of mediation and is stable in samples of 500 (MacKinnon, 1994). This measure is the ratio of the total effect to indirect effect and provides an estimate of the importance of the potential mediator (Vanderweele, 2013). The proportion represents how much of the total effect operates via the mediator (e.g., 10%). For significant indirect effects, we tested whether these differed significantly between F-BA and supplement groups using Wald χ² tests.

**Results**

Participant characteristics are shown in Table 1. Incomplete trial data on the outcome (depressive symptoms) was defined as having missing data for the PHQ-9 at
both T6 (trial midpoint) and T12 (trial end point). Of those participants randomly assigned to one of the MooDFOOD trial arms (1,025), 233 (22.73%) were classified as having incomplete outcome data. Differences in baseline characteristics between this group and the remaining sample without this level of incomplete data (792, 77.27%) are reported in Table 2. Participants with incomplete data were younger, were more likely to be non-White, and had higher baseline BMIs on average. In addition, participants with complete data were overrepresented by the trial site in Germany, and there were more participants with incomplete data at the Spanish trial site.

**Psychologist fidelity to the F-BA intervention**

A total of 102 F-BA sessions were rated by experienced supervising clinical psychologists for levels of psychologist fidelity to the F-BA protocol. The majority of sessions (89, 87.25% vs. 13, 12.75%) met the a priori specified criteria for fidelity (average score of 4 overall). The mean fidelity rating was 4.41 (SD = 0.43; range = 3.50–5.85).

**Effect of F-BA on depressive symptoms**

There was no significant main effect of F-BA compared with no F-BA on change in depressive symptoms at T12 after adjusting for history of MDD, site, and sex (standardized regression coefficient $|b| = 0.223$, $SE = 0.129$, $b/SE = -1.727$, $p = .084$).

**Effect of F-BA on putative mediators**

There were six statistically significant effects of F-BA on the putative mediators. Relative to no F-BA, F-BA reduced emotional eating ($b = -1.767$, $SE = 0.709$, $b/SE = -2.493$, $p = .013$), reduced uncontrolled eating ($b = -1.842$, $SE = 0.532$, $b/SE = -3.460$, $p = .001$), and increased cognitive restraint ($b = 2.520$, $SE = 0.575$, $b/SE = 4.380$, $p < .001$). F-BA also reduced snacking ($b = -0.364$, $SE = 0.086$, $b/SE = -4.241$, $p < .000$) and habitual diet of sugar and fat ($b = -0.851$, $SE = 0.287$, $b/SE = -2.964$, $p = .003$) and increased the Mediterranean diet score ($b = 0.923$, $SE = 0.187$, $b/SE = 4.940$, $p < .001$). See Tables 3 and 4 for full results.

**Association of putative mediators with change in depression**

Change in five of the putative mediators were significantly associated with subsequent change in depressed symptoms: BADS activation, BADS avoidance and rumination, emotional eating, uncontrolled eating, and cognitive restraint (see Table 4).

**Test of mediation**

There were significant indirect mediation effects on depressive symptoms via emotional eating ($b = -0.028$, $SE = 0.014$, $b/SE = -2.030$, $p = .042$; bias-corrected 95% CI = [-0.063, -0.007]) and uncontrolled eating ($b = -0.039$, $SE = 0.016$, $b/SE = -2.479$, $p = .013$; bias-corrected 95% CI = [-0.080, -0.002]).
CI = [-0.077, -0.014]). The indirect effect for emotional eating and uncontrolled eating, respectively, accounted for 20% and 32% of the total effect of F-BA on depressive symptoms. There were no significant indirect effects for the other putative mediators (see Tables 3 and 4).

Comparison analysis: effect of nutritional supplements compared with placebo

There was one significant effect of supplements on the BADS behavioral activation measure ($b = -0.739, SE = 0.254, b/SE = -2.912, p = .004$), indicating that participants receiving the active supplements were less behaviorally active than participants taking placebo. There was also a significant indirect mediation effect for supplements through BA ($b = 0.040, SE = 0.019, b/SE = 2.129, p = .033; bias-corrected 95% CI = [0.012, 0.088]) showing that decrease in BA in the supplements group was associated with a subsequent increase in depressive symptoms (see Table 4). For all significant indirect effects of F-BA and/or supplements, we subsequently tested whether these differed between F-BA and supplement groups. The significant indirect effects of F-BA for emotional eating (Wald $\chi^2 = 4.55, p = .03$) and uncontrolled eating (Wald $\chi^2 = 6.59, p = .01$) were significantly different to those found for supplements. The indirect effect of supplements observed for BA did not differ from the indirect effect in the F-BA group (Wald $\chi^2 = 2.55, p = .11$).

Although we were not powered to test effects of moderated mediation in the present study, exploratory supplementary analysis showed no evidence that the significant mediation effects were moderated by gender, a history of clinical depression, or the baseline depressive
Table 3. Means and Standard Deviations of Symptoms and Potential Mediators Over Time by Intervention Group

| Variable and time point | No F-BA Placebo | Supplements | F-BA Placebo | Supplements |
|-------------------------|-----------------|-------------|--------------|-------------|
| PHQ                     |                 |             |              |             |
| T0                      | 7.34 (4.14)     | 7.93 (4.39) | 7.30 (4.41)  | 7.12 (4.04) |
| T3                      | 5.33 (4.02)     | 6.42 (3.99) | 5.14 (4.11)  | 5.32 (3.29) |
| T12                     | 4.46 (3.81)     | 5.21 (4.24) | 3.70 (3.32)  | 4.56 (3.95) |
| BADS                    |                 |             |              |             |
| T0                      | 19.15 (7.64)    | 19.70 (8.14) | 18.89 (8.07) | 19.92 (8.17) |
| T3                      | 21.18 (8.51)    | 20.70 (8.17) | 22.84 (8.88) | 21.14 (8.05) |
| T12                     | 22.99 (7.66)    | 21.26 (8.53) | 23.59 (8.79) | 22.59 (8.21) |
| RUM                     |                 |             |              |             |
| T0                      | 32.10 (10.11)   | 32.86 (9.54) | 33.05 (9.40) | 33.58 (9.01) |
| T3                      | 35.10 (9.45)    | 34.90 (9.56) | 35.19 (9.42) | 35.33 (8.47) |
| T12                     | 36.60 (9.23)    | 36.33 (9.25) | 37.05 (8.52) | 37.24 (8.56) |
| FBQ                     |                 |             |              |             |
| T0                      | 5.07 (3.20)     | 5.37 (3.55)  | 5.41 (3.40)  | 5.06 (3.45)  |
| T3                      | 4.52 (3.16)     | 4.88 (3.37)  | 3.88 (2.75)  | 4.04 (2.91)  |
| T12                     | 3.97 (2.72)     | 4.32 (3.09)  | 3.38 (2.60)  | 3.82 (3.00)  |
| SRBAI                   |                 |             |              |             |
| T0                      | 24.33 (10.28)   | 23.60 (9.67) | 22.94 (10.13) | 23.34 (10.19) |
| T3                      | 21.43 (10.46)   | 21.67 (9.78) | 19.39 (9.93) | 19.56 (9.13) |
| T12                     | 20.63 (9.61)    | 19.71 (9.12) | 16.86 (9.03) | 18.69 (8.93) |
| MFDS                    |                 |             |              |             |
| T0                      | 51.68 (7.56)    | 51.63 (6.78) | 51.91 (6.93) | 51.42 (6.85) |
| T6                      | 52.36 (7.77)    | 52.54 (6.97) | 54.88 (6.48) | 54.04 (6.34) |
| T12                     | 52.46 (7.10)    | 52.99 (6.51) | 55.56 (6.58) | 54.94 (6.39) |
| TFEQ EE                 |                 |             |              |             |
| T0                      | 54.61 (31.29)   | 58.93 (29.92) | 56.74 (30.11) | 56.28 (30.26) |
| T3                      | 44.44 (31.48)   | 51.59 (30.66) | 43.65 (31.01) | 44.39 (27.94) |
| T12                     | 40.55 (30.83)   | 46.45 (30.16) | 34.54 (27.79) | 40.54 (27.56) |
| TFEQ UE                 |                 |             |              |             |
| T0                      | 49.72 (23.42)   | 52.83 (22.80) | 48.99 (24.07) | 48.22 (23.24) |
| T3                      | 42.39 (22.61)   | 48.13 (22.89) | 39.32 (22.31) | 40.52 (21.14) |
| T12                     | 40.44 (23.76)   | 42.69 (21.58) | 32.43 (19.07) | 38.64 (21.00) |
| TFEQ CR                 |                 |             |              |             |
| T0                      | 41.21 (19.77)   | 43.42 (19.86) | 43.15 (19.61) | 41.53 (19.60) |
| T3                      | 44.20 (21.06)   | 45.92 (20.95) | 51.17 (19.83) | 49.97 (19.36) |
| T12                     | 46.33 (20.17)   | 44.92 (20.17) | 52.91 (18.73) | 51.34 (17.73) |
| BMI                     |                 |             |              |             |
| T0                      | 31.37 (4.0)     | 31.29 (4.02) | 31.16 (3.91) | 31.70 (3.90) |
| T3                      | 31.37 (3.98)    | 31.10 (4.22) | 31.15 (4.06) | 31.54 (4.03) |
| T12                     | 31.31 (4.09)    | 31.03 (4.38) | 30.59 (3.86) | 31.21 (4.23) |

Note: Values are means with standard deviations in parentheses. F-BA = food-related behavioral activation intervention; PHQ = Patient Health Questionnaire (Kroenke et al., 2001); BADS = Behavioural Activation for Depression Scale (Kanter et al., 2007, 2009); RUM = Avoidance and Rumination Scale of BADS; FBQ = food-behavior questionnaire; SRBAI = Self-Reported Behavioural Automaticity Index (Gardner et al., 2012); MFDS = MooDFOOD diet score (Grasso et al., 2019); TFEQ = Three Factor Eating Questionnaire (Karlsson et al., 2000); EE = emotional eating; UE = uncontrolled eating; CR = cognitive restraint; T0 = baseline; T3 = 3-month follow-up; T6 = 6-month follow-up; T12 = 12-month follow-up.
Discussion

We found that F-BA in a sample of overweight or obese adults reduced potentially unhealthy eating (i.e., the habitual tendency to consume foods high in sugar and fat and snacking), increased the Mediterranean diet score, decreased habitual emotional eating and uncontrolled eating, and increased cognitive restraint. These results indicate that F-BA was successful at producing the intended behavioral change: It was designed to use BA approaches such as self-monitoring, functional analysis, and activity scheduling to change food-related behaviors and mood-food-related behaviors such as emotional eating. Moreover, several of the observed outcomes used indices that match closely to conceptualizations of habits, including automaticity (habitual tendency to consume foods high in sugar and fat) and a repeated response to the same cue (emotional eating, uncontrolled eating), indicating that, as intended, F-BA was successful at reducing potentially maladaptive habits. F-BA did not significantly reduce avoidance and rumination or increase levels of BA and did not have a significant effect on depressive symptom reduction.

Consistent with prior research (e.g., Nolen-Hoeksema et al., 2008) and theoretical models, reductions in avoidance and rumination and increases in BA were associated with decreases in symptoms of depression. Likewise, changes in eating styles were associated with decreases in symptoms of depression.

Critically, of the potential mediating factors, only emotional eating and uncontrolled eating were significant mediators of the effects of F-BA on change in depressive symptoms. That is, we found that the prevention F-BA intervention significantly reduced both emotional and uncontrolled eating, which in turn were associated with a subsequent reduction in depressive symptoms. The greater the extent to which emotional eating and uncontrolled eating were reduced by F-BA, the greater was the symptomatic improvement. Although F-BA significantly reduced snacking and habitually consuming foods high in sugar and fat and increased the Mediterranean diet score, changes in these factors were not significantly related to change in depressive symptoms in this sample of overweight individuals. Thus, of the alternative behavioral change models of how F-BA might work, the pattern of findings is not consistent with the BA model or with a nutritional psychology model but most consistent with the hypothesis that F-BA works by helping individuals to learn more adaptive responses to mood and food cues and gain control over involuntary habits.

Indeed, the emotional eating measure is an index of a habitual response to negative mood (i.e., eating) that is potentially physically unhealthy and unlikely to be helpful at improving mood in the long run. Likewise, the uncontrolled eating measure provides an index of a habitual response to food and eating cues that may be maladaptive. Their observed role as mediators therefore is consistent with the hypothesis that the mechanism by which F-BA works is by individuals weakening a problematic automatic response to a cue. Because emotional eating was our only measure to assess adoption of an unhelpful response to negative emotions, we cannot distinguish whether this mediator effect reflects specifically a reduction in eating to low mood or whether it is a proxy for a more general shift away from maladaptive coping in response to low mood. After all, a key focus of F-BA is for participants to deliberately plan to do an alternative action to improve mood, such as talking to a friend or going for a walk, rather than to eat when in a low mood. Thus, although uncontrolled eating is a food-related behavior and emotional eating is a mood-food-related behavior, these measures may act as a proxy for learning to break and replace unhelpful habits more generally.

Furthermore, because reducing snacking or increasing Mediterranean diet or reducing habitual eating of foods high in sugar and fat were not found to be mediators of the effects of F-BA on depression, this suggests that changing diet itself is not sufficient to improve depressive symptoms. Given that the type of foods eaten during uncontrolled and emotional eating and snacking often overlap with snacking and eating increased sugar and fat, this in turn suggests that it was not the direct eating or dietary effects of reducing uncontrolled eating and emotional eating that underpins their role as mediators.

Instead, an alternative explanation is that reductions in emotional eating and uncontrolled eating reflect increases in control over involuntary behaviors and an increased sense of personal agency, which is a plausible psychological mechanism by which these changes could reduce depressive symptoms. We speculate that the underlying mechanism for F-BA is not simply changing food-related habits but rather more generally learning to gain control over unhelpful habits that are associated with negative mood. This hypothesis was not directly tested in the present study because we did not include measures of other unhelpful and helpful coping strategies in response to low mood (e.g., rumination to low mood, increasing activation to low mood): This hypothesis predicts that these would also be found to be mediators of F-BA on depression.

Note that in addition to becoming a habitual response, emotional eating is likely to be an attempt
## Table 4. Potential Mediators of Effects Between Intervention and Depressive Symptoms

| Potential mediator                                                                 | Effect on mediator | Association of mediator with outcome | F-BA | SUP | Effect on mediator | Association of mediator with outcome | F-BA | SUP | Effect on mediator | Association of mediator with outcome | F-BA | SUP | Effect on mediator | Association of mediator with outcome | F-BA | SUP | Effect on mediator | Association of mediator with outcome | F-BA | SUP | Effect on mediator | Association of mediator with outcome | F-BA | SUP | Effect on mediator | Association of mediator with outcome |
|------------------------------------------------------------------------------------|--------------------|--------------------------------------|------|-----|--------------------|--------------------------------------|------|-----|--------------------|--------------------------------------|------|-----|--------------------|--------------------------------------|------|-----|--------------------|--------------------------------------|------|-----|--------------------|--------------------------------------|------|-----|--------------------|--------------------------------------|------|-----|--------------------|--------------------------------------|------|-----|--------------------|--------------------------------------|
| **BADS activation**                                                                | 0.423              | 0.258                                | .101 | −0.739          | 0.254     | .004                | −0.056 | 0.015 | <.001              | −0.023 | 0.016               | .156            | −0.147 | 0.121              | .222          | −.16                          |
| **RUM avoidance and rumination**                                                    | −0.262             | 0.257                                | .307 | −0.272          | 0.259     | .295                | −0.075 | 0.017 | <.001              | 0.020            | 0.020               | .324            | −0.139 | 0.120              | .247          | −.14                          |
| **TFEQ**                                                                           | −1.767             | 0.709                                | .013 | 0.905           | 0.711     | .203                | 0.016  | 0.004 | <.001              | −0.028 | 0.014               | .042            | −0.140 | 0.121              | .245          | .20                           |
| **Uncontrolled eating**                                                             | −1.842             | 0.532                                | .001 | 0.888           | 0.555     | .097                | 0.021  | 0.006 | <.001              | −0.039 | 0.016               | .013            | −0.122 | 0.120              | .309          | .32                           |
| **Cognitive restraint**                                                             | 2.520              | 0.575                                | <.001 | 0.085          | 0.581     | .883                | −0.015 | 0.007 | .041              | −0.037 | 0.020               | .068            | −0.143 | 0.121              | .236          | .26                           |
| **FBQ snacking**                                                                   | −0.364             | 0.086                                | <.001 | 0.140           | 0.086     | .104                | 0.065  | 0.040 | .102              | −0.024 | 0.015               | .126            | −0.139 | 0.121              | .248          | .17                           |
| **SRB AI habits**                                                                  | −0.851             | 0.287                                | .003 | 0.185           | 0.289     | .129                | −0.007 | 0.013 | .596              | 0.006  | 0.011               | .618            | −0.139 | 0.120              | .247          | −.4                           |
| **MFDS diet**                                                                      | 0.923              | 0.187                                | <.001 | −0.087          | 0.188     | .641                | −0.013 | 0.017 | .457              | −0.012 | 0.016               | .454            | −0.185 | 0.114              | .105          | .6                            |
| **BMI**                                                                           | −0.008             | 0.035                                | 820   | −0.022          | 0.035     | .530                | −0.002 | 0.031 | .936              | 0.000  | 0.000               | .940            | −0.138 | 0.122              | .256          | 0                            |

Note: The outcome is final follow-up (T12) Patient Health Questionnaire-9 score, adjusted for baseline levels and 3-month follow-up (T3). Boldface type indicates statistically significant p values (<.05). b = standardized regression coefficient. F-BA = food-related behavioral activation arm; SUP = nutritional supplementation arm; BADS = Behavioural Activation for Depression Scale (Kanter et al., 2007, 2009); RUM = Avoidance and Rumination Scale of BADS; TFEQ = Three Factor Eating Questionnaire (Karlsson et al., 2000); FBQ = food-behavior questionnaire; SRBAI = Self-Reported Behavioural Automaticity Index (Gardner et al., 2012); MFDS = MooDFOOD Diet Score (Grasso et al., 2019); BMI = body mass index; PM = proportion mediated.

*The indirect effect as a proportion of the total effect. All models were adjusted for site, sex, and a history of clinical depression.*
by the individual at affect regulation (Fuller-Tyszkiewicz et al., 2014; Haedt-Matt & Keel, 2011), that is, eating to reduce the negative effects of low mood through comfort or distraction. Although this approach may be successful in the short term, these behaviors may ultimately become maladaptive. Therefore, future work should also explore the interrelationship between affect regulation and habit formation.

Mediation analyses are recommended for intervention trials to identify potential ways to enhance intervention effects in the future (Kraemer et al., 2002; O’Rourke & MacKinnon, 2018). The current results suggest that enhancing the effect of F-BA on emotional eating and uncontrolled eating could strengthen the overall intervention effect: If the intervention was adapted to more effectively change emotional and uncontrolled eating, then this could increase its efficacy and result in a significant overall effect of F-BA on depression. More specifically, our proposed hypothesis suggests that F-BA would be more efficacious by concentrating more on changing unhelpful habits by identifying their triggers and repeatedly practicing alternative behaviors to these triggers while removing the activity scheduling and psychoeducation intended to increase a Mediterranean diet. If this hypothesis is correct, then such adaptations to F-BA would be predicted to improve outcomes if tested in future trials.

The pattern of results suggests that F-BA did not significantly reduce avoidance and rumination or increase BA, in contrast to other studies of BA (e.g., Richards et al., 2017; Topper et al., 2017; Watkins et al., 2011). There are differences in trial designs, populations, and interventions that might account for why the MooDFOOD trial did not replicate these effects. First, this was a prevention study, using participants with mildly elevated symptoms of depression and elevated BMI, rather than a treatment study in acute major depression (Richards et al., 2016; Watkins et al., 2011) or a prevention study explicitly targeting individuals with high levels of rumination (Topper et al., 2017). Thus, levels of avoidance and activation in the MooDFOOD sample may be closer to the population mean than in clinical populations, affording less scope for change. Second, the increased focus on food-related behaviors in the MooDFOOD BA intervention (which is not present in other BA interventions) may have made this aspect more salient. Conversely, focus on increasing activation and reducing avoidance may have been relatively reduced compared with other BA interventions. Third, because the MooDFOOD sample was selected to be overweight and obese, the study patients may have been more engaged in and motivated by food and weight-related goals than by mood-related goals.

We used the nutritional supplementation compared with pill placebo trial arms to test the specificity of any mediational effects to F-BA. As expected, there was no positive effects of supplementation relative to placebo on putative moderators. There was, however, evidence of significantly reduced BA in the supplement group, which was, in turn, related to increased depressive symptoms. This effect is consistent with the BA hypothesis and with recent nutraceutical (cf. pharmaceutical) research in the context of treating depression, in which the nutritional supplementation performed significantly worse than placebo (Sarris et al., 2019), which has led to a call for caution in recommending supplements for depression (Berk & Jacka, 2019).

The results did not support an alternative hypothesis that change in depressive symptoms would be mediated by change in BMI. However, this is likely explained by the fact that the MooDFOOD trial was not explicitly designed to facilitate weight loss in participants. Previous research has shown that weight loss through controlled dieting can be accompanied by improved mood (Brinkworth et al., 2009; Faulconbridge et al., 2018), but this was not a focus of the MooDFOOD trial. Furthermore, the relationship between mood and weight appears to a complex one. For example, both weight gain and weight loss, compared with stable weight, are associated with psychological distress over the long term (Knüppel et al., 2019). Future research should aim to incorporate long-term follow-up periods to further test these hypotheses.

**Strengths and limitations**

The present study has a number of strengths. The data were drawn from a multicountry European prevention trial for depression, which included a large sample size and a range of standardized measurements and benefited from randomization to treatment allocation. There were also a number of limitations. First, although the trial 12-month follow-up rate was reasonable (76%; Bot et al., 2019), it is possible that attrition bias influenced the findings, specifically, participants who dropped out of the trial and were not contactable before final follow-up differed from the overall sample on certain characteristics. Second, one needs to be cautious about generalizing from the MooDFOOD study to other populations because it was a selective sample of overweight individuals. In addition, there was limited ethnic diversity, and so caution is required in generalizing the findings to non-White participants. Third, baseline symptoms of depression were lower, and a smaller proportion of the sample reported a history of depression compared with other preventive studies (e.g., van Zoonen et al., 2014), which may have affected the ability to detect a significant effect on symptoms. It remains unresolved whether reductions in symptoms of depression would be statistically and clinically significant in a sample of people with greater symptoms.
The results confirmed the presence of the F-BA effect of improving diet as measured by the FFQ (Grasso et al., 2019), although no mediation effects for change in diet were found on depressive symptoms in the present study. Nevertheless, it is important to interpret these findings with caution because it has been established that FFQs can be prone to bias (Kipnis et al., 2002). Future research could use web-based or smartphone applications to reduce bias and participant burden (Naska et al., 2017) and explore more novel technologies such as apps using mobile phone cameras (Kong & Tan, 2012).

Conclusions

Finally, although we have outlined a potential habit-based theoretical perspective that may help to guide future intervention strategies to improve mood and prevent depression, we acknowledge that there is likely to be a complex interaction between factors in explaining behavior that leads to increases in depressive symptoms and depressive states. This is likely to be further complicated by heterogeneity in depression eating behavior.

In conclusion, an analysis of potential mediators of F-BA in the MooDFOOD randomized controlled prevention trial found that changes in emotional eating and uncontrolled eating mediated the effects of F-BA on reducing symptoms of depression. We interpreted this as indicating that F-BA works by helping individuals to learn more adaptive responses to emotional and food cues and to gain control over these involuntary habits. This suggests that further targeting change in these habitual responses may improve interventions for depression in this sample.

Transparency

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Author Contributions

M. Owens and E. Watkins were the lead authors on the manuscript, and M. Owens analyzed and interpreted the data with E. Watkins. M. Visser and I. A. Brouwer obtained funding for the MooDFOOD project, designed the MooDFOOD prevention trial, and coordinated the MooDFOOD project. B. W. J. H. Pennix, M. Bot, and E. Watkins contributed to the design of the MooDFOOD prevention trial. E. Watkins led the development and training of the MooDFOOD Food-Related Behavioural Change Intervention. E. Kohls and U. Hegerl coordinated the recruitment, interventions, and follow-ups at the trial center in Germany, University Leipzig. B. W. J. H. Pennix and M. Bot coordinated the recruitment, interventions, and follow-ups at the trial center in the Netherlands, VU University Medical Center Amsterdam. E. Watkins and M. Owens coordinated the recruitment, interventions, and follow-ups at the trial center in the United Kingdom, University of Exeter. M. Roca and M. Gili coordinated the recruitment, interventions, and follow-ups at the trial center in Spain, University of Balearic Islands. G. van Grootheest set up the logistics for the trial’s data collection. All of the authors contributed to the writing of the manuscript and approved the final manuscript for submission.

Declaration of Conflicting Interests

The author(s) declared that there were no conflicts of interest with respect to the authorship or the publication of this article.

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ORCID iDs

Matthew Owens https://orcid.org/0000-0001-7407-6540
Ed Watkins https://orcid.org/0000-0002-2432-5577

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For a complete list of the MooDFOOD Prevention Trial Investigators, see https://www.moodfood-vu.eu.

Supplemental Material

Additional supporting information can be found at http://journals.sagepub.com/doi/suppl/10.1177/2167702620979785

References

Abarca-Gómez, L., Abdeen, Z. A., Hamid, Z. A., Abu-Rmeileh, N. M., Acosta-Cazares, B., Acuin, C., Adams, R. J., Aekplakorn, W., Afşana, K., Aguilar-Salinas, C. A., Agyemang, C., Ahmadvand, A., Ahrens, W., Ajlouni, K., Akhtaeva, N., Al-Hazzaa, H. M., Al-Othman, A. R., Al-Raddadi, R., Al Bu Haiman, F., . . . Ezzati, M. (2017). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: A pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. Lancet, 390(10113), 2627–2642. https://doi.org/10.1016/S0140-6736(17)32129-3

American Psychiatric Association. (1994). Diagnostic and statistical manual of mental disorders (4th ed.).

Angle, S., Engblom, J., Eriksson, T., Kautiainen, S., Saha, M. T., Lindfors, P., Lehtinen, M., & Rimpela, A. (2009). Three factor eating questionnaire-R18 as a measure of cognitive restraint, uncontrolled eating and emotional eating in a sample of young Finnish females. International Journal of Behavioral Nutrition and Physical Activity, 6, Article 41. https://doi.org/10.1186/1479-5868-6-41
Elstgeest, L. E. M., Visser, M., Penninx, B. W. J. H., Colpo, M., Bandinelli, S., & Brouwer, I. A. (2019). Bidirectional associations between food groups and depressive symptoms: Longitudinal findings from the Invecchiare in Chianti (InCHIANTI) study. *British Journal of Nutrition*, 121(4), 439–450. https://doi.org/10.1017/S0007114518003205

Enders, C. K., & Bandolos, D. L. (2001). The relative performance of full information maximum likelihood estimation for missing data in structural equation models. *Structural Equation Modeling*, 8(3), 430–457. https://doi.org/10.1207/S15328007sem0803_5

Evans-Lacko, S., & Knapp, M. (2016). Global patterns of workplace productivity for people with depression: Absenteeism and presenteeism costs across eight diverse countries. *Social Psychiatry and Psychiatric Epidemiology*, 51(11), 1525–1537. https://doi.org/10.1007/s00127-016-1278-4

Faulconbridge, L. F., Driscoll, C. F. B., Hopkins, C. M., Bailer Benforado, B., Bishop-Gilyard, C., Carvajal, R., Berkowitz, R. I., DeRubeis, R., & Wadden, T. A. (2018). Combined treatment for obesity and depression: A pilot study. *Obesity*, 26(7), 1144–1152. https://doi.org/10.1002/oby.22209

Firth, J., Teasdale, S. B., Allott, K., Siskind, D., Marx, W., Cotter, J., Veronese, N., Schuch, F., Smith, L., Solmi, M., Carvalho, A. F., Vancampfort, D., Berk, M., Stubbs, B., & Sarris, J. (2019). The efficacy and safety of nutrient supplements in the treatment of mental disorders: A meta-review of meta-analyses of randomized controlled trials. *World Psychiatry*, 18(3), 308–324. https://doi.org/10.1002/wps.20672

Fuller-Tyszkiewicz, M., Richardson, B., Skouteris, H., Austin, D., Castle, D., Busija, L., Klein, B., Holmes, M., & Broadbent, J. (2014). Optimizing prediction of binge eating episodes: A comparison approach to test alternative conceptualizations of the affect regulation model. *Journal of Eating Disorders*, 2(1), 1–8. https://doi.org/10.1186/1743-0339-2-1

Gao, S., Jin, Y., Unverzagt, F. W., Liang, C., Hall, K. S., Cao, J., Ma, F., Murrell, J. R., Cheng, Y., Li, P., Bian, J., & Hendrie, H. C. (2012). Selenium level and depressive symptoms in a rural elderly Chinese cohort. *BMC Psychiatry*, 12, Article 72. https://doi.org/10.1186/1471-244X-12-72

Garcia-Larsen, V., Luczynska, M., Kowalski, M. L., Voutilainen, H., Ahlstrom, M., Haahetla, T., Tossala, E., Bockelbrink, A., Lee, H. H., Vassiliopoulou, E., Papadopoulos, N. G., Ramallo, R., Moreira, A., Delgado, L., Castel-Branco, M. G., Calder, P. C., Childs, C. E., Bakolis, I., Hooper, R., & Burney, P. G., for the working group of GA2LEN-WP 1.2 ‘‘Epidemiological and Clinical Studies.’’ (2011). Use of a common Food Frequency Questionnaire (FFQ) to assess dietary patterns and their relation to allergy and asthma in Europe: Pilot study of the GA2LEN FFQ. *European Journal of Clinical Nutrition*, 65(6), 750–756. https://doi.org/10.1038/ejcn.2011.15

Gardner, B., Abraham, C., Lally, P., & de Brujin, G.-J. (2012). Towards parsimony in habit measurement: Testing the convergent and predictive validity of an automaticity subscale of the Self-Report Habit Index. *International Journal of Behavioral Nutrition and Physical Activity*, 9, Article 102. https://doi.org/10.1186/1479-5868-9-102

Gilbody, S., Richards, D., & Barkham, M. (2007). Diagnosing depression in primary care using self-completed instruments: UK validation of PHQ–9 and CORE–OM. *British Journal of General Practice*, 57(541), 650–652.
Goldschmidt, A. B., Crosby, R. D., Engel, S. G., Crow, S. J., Cao, L., Peterson, C. B., & Durkin, N. (2014). Affect and eating behavior in obese adults with and without elevated depression symptoms. *The International Journal of Eating Disorders, 47*(3), 281–286. https://doi.org/10.1002/eat.22188

Gollwitzer, P. M., & Sheeran, P. (2006). Implementation intentions and goal achievement: A meta-analysis of effects and processes. In M. P. Zanna (Ed.), *Advances in experimental social psychology* (Vol. 38, pp. 69–119). Academic Press. https://doi.org/10.1016/S0065-2600(06)80021-1

Grasso, A. C., Othof, M. R., van Dooren, C., Roca, M., Gili, M., Visser, M., Cabout, M., Bot, M., Penninx, B. W. J. H., van Grootheest, G., Kohls, E., Hegerl, U., Owens, M., Watkins, E., Brouwer, I. A., Penninx, B., Paans, N., Thesing, C., Gibson-Smith, D., . . . Verkerk, B. (2019). Effect of food-related behavioral activation therapy on food intake and the environmental impact of the diet: Results from the MooDFOOD prevention trial. *European Journal of Nutrition, 59*, 2579–2591. https://doi.org/10.1007/s00394-019-02106-1

Grosso, G., Galvano, F., Marventano, S., Malaguarnera, M., Bucolo, C., Drago, F., & Caraci, F. (2014). Omega-3 fatty acids and depression: Scientific evidence and biological mechanisms. *Oxidative Medicine and Cellular Longevity, 2014*, Article 313570. https://doi.org/10.1155/2014/313570

Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the affect regulation model of binge eating: A meta-analysis of studies using ecological momentary assessment. *Psychological Bulletin, 137*(4), 660–681. https://doi.org/10.1037/a0023660

Holahan, C. J., Moos, R. H., Holahan, C. K., Brennan, P. L., & Schutte, K. K. (2005). Stress generation, avoidance coping, and depressive symptoms: A 10-year model. *Journal of Consulting and Clinical Psychology, 73*(4), 658–666. https://doi.org/10.1037/0022-006X.73.4.658

Jacka, F. N., O’Neil, A., Opie, R., Isiopoulos, C., Cotton, S., Mohiebbi, M., Castle, D., Dash, S., Mihalopoulos, C., Chatterton, M., Lou Brazionis, L., Dean, O. M., Hodge, A. M., & Berk, M. (2017). A randomised controlled trial of dietary improvement for adults with major depression (the “SMILES” trial). *BMC Medicine, 15*(1), Article 23. https://doi.org/10.1186/s12916-017-0791-y

Kanter, J. W., Mulick, P. S., Busch, A. M., Berlin, K. S., & Martell, C. R. (2007). The Behavioral Activation for Depression Scale (BADS): Psychometric properties and factor structure. *Journal of Psychopathology and Behavioral Assessment, 29*(3), 191–202. https://doi.org/10.1007/s10862-006-9038-5

Kanter, J. W., Rusch, L. C., Busch, A. M., & Sedivy, S. K. (2009). Validation of the Behavioral Activation for Depression Scale (BADS) in a community sample with elevated depressive symptoms. *Journal of Psychopathology and Behavioral Assessment, 31*(1), 36–42. https://doi.org/10.1007/s10862-008-9088-y

Karlsso, J., Persson, L. O., Sjőström, L., Sullivan, M., Sjostrom, L., & Sullivan, M. (2000). Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. *International Journal Obesity Related Metabolic Disorders, 24*(12), 1715–1725. https://doi.org/10.1038/sj.ijo.0801442

Kessler, R. C. (2012). The costs of depression. *Psychiatric Clinics of North America, 35*(1), 1–14. https://doi.org/10.1016/j.psc.2011.11.005

Kessler, R. C., & Bremet, E. J. (2013). The epidemiology of depression across cultures. *Annual Review of Public Health, 34*(1), 119–138. https://doi.org/10.1146/annurev-publichealth-031912-114009

Kipnis, V., Midthune, D., Friedman, L., Bingham, S., Day, N. E., Riboli, E., Ferrari, P., & Carroll, R. J. (2002). Bias in dietary-report instruments and its implications for nutritional epidemiology. *Public Health Nutrition, 5*(6a), 915–923. https://doi.org/10.1079/phn2002383

Knüppel, A., Shipley, M. J., Llewellyn, C. H., & Brunner, E. J. (2019). Weight change increases the odds of psychological distress in middle age: Bidirectional analyses from the Whitehall II Study. *Psychological Medicine, 49*(15), 2505–2514. https://doi.org/10.1017/s0033291718003379

Kong, F., & Tan, J. (2012). DietCam: Automatic dietary assessment with mobile camera phones. *Pervasive and Mobile Computing, 8*(1), 147–163. https://doi.org/10.1016/j.pmcj.2011.07.003

Kraemer, H. C., Wilson, G. T., Fairburn, C. G., & Agras, W. S. (2002). Mediators and moderators of treatment effects in randomized clinical trials. *Archives of General Psychiatry, 59*(10), 877–883. https://doi.org/10.1001/archpsyc.59.10.877

Kroenke, K., Spitzer, R. L., & Williams, J. B. W. (2001). The PHQ-9. *Journal of General Internal Medicine, 16*(9), 606–613. https://doi.org/10.1046/j.1525-1497.2001.016009606.x

Lassele, C., Batty, G. D., Baghdadi, A., Jacka, F., Sánchez-Villegas, A., Kivimäki, M., & Akbaraly, T. (2019). Healthy dietary indices and risk of depressive outcomes: A systematic review and meta-analysis of observational studies. *Molecular Psychiatry, 24*(7), 965–986. https://doi.org/10.1038/s41388-018-0257-8

Lecrubier, Y., Sheehan, D. V., Weiller, E., Amorim, P., Bonora, I., Sheehan, K. H., Janavs, J., & Dunbar, G. C. (1997). The Mini International Neuropsychiatric Interview (MINI). A short diagnostic structured interview: Reliability and validity according to the CIDI. *European Psychiatry, 12*(5), 224–231. https://doi.org/10.1016/S0924-9389(97)83296-8

Luppino, F. S., De Wit, L. M., Bouvy, P. F., Stijnen, T., Cuijpers, P., Penninx, B. W. J. H., & Zitman, F. G. (2010). Overweight, obesity, and depression: A systematic review and meta-analysis of longitudinal studies. *Archives of General Psychiatry, 67*(3), 220–229. https://doi.org/10.1001/archgenpsychiatry.2010.2

MacKinnon, D. P. (1994). Analysis of mediating variables in prevention and intervention research. *NIDA Research Monograph Series, 139*, 127–153. https://archives.drugabuse.gov/sites/default/files/monograph139.pdf

MacKinnon, D. P., Lockwood, C. M., Hoffman, J. M., West, S. G., & Sheets, V. (2002). A comparison of methods to test mediation and other intervening variable effects. *Psychological Methods, 7*(1), 83–104. https://doi.org/10.1037/1082-989X.7.1.83

Marteau, T. M., Hollands, G. J., & Fletcher, P. C. (2012). Changing human behavior to prevent disease: The importance of...
targeting automatic processes. Science, 337(6101), 1492–1495. https://doi.org/10.1126/science.1226918
Marx, W., Hoare, E., & Jacka, F. (2019). Diet and depression: From epidemiology to novel therapeutics. In J. Quevedo, A. Carvalho, & C. Zarate (Eds.), Neurobiology of depression (pp. 285–292). Elsevier. https://doi.org/10.1016/b978-0-12-813533-0-00025-1
Milaneschi, Y., Peyrot, W. J., Nivard, M. G., Mbarek, H., Boomsma, D. I., & Penninx, B. W. J. H. (2019). A role for vitamin D and omega-3 fatty acids in major depression? An exploration using genomics. Translational Psychiatry, 9(1), Article 219. https://doi.org/10.1038/s41398-019-0554-y
Mlyniec, K., Davies, C. L., De Agüero Sánchez, I. G., Pytka, K., Budziszewska, B., & Nowak, G. (2014). Essential elements in depression and anxiety. Part i. Pharmacological Reports, 66(4), 534–544. https://doi.org/10.1016/j.pharep.2014.03.001
Molendijk, M., Molero, P., Ortuño Sánchez-Pedreño, F., Van der Does, W., & Angel Martínez-González, M. (2018). Diet quality and depression risk: A systematic review and dose-response meta-analysis of prospective studies. Journal of Affective Disorders, 226, 346–354. https://doi.org/10.1016/j.jad.2017.09.022
Moulds, M. L., Kandris, E., Starr, S., & Wong, A. C. M. (2007). The relationship between rumination, avoidance and depression in a non-clinical sample. Behaviour Research and Therapy, 45(2), 251–261. https://doi.org/10.1016/j.brat.2006.03.003
Muthén, L. K., & Muthén, B. O. (2017). Mplus user's guide (8th ed.). Los Angeles, CA: Author.
Naska, A., Lagiou, A., & Lagiou, P. (2017). Dietary assessment methods in epidemiological research: Current state of the art and future prospects. PLoS ONE, 6, Article 926. https://doi.org/10.12688/f1000research.10703.1
Nasrini, R., Rimes, K., Reinecke, A., Rinck, M., & Barrohofer, T. (2017). Effects of brief behavioural activation on approach and avoidance tendencies in acute depression: Preliminary findings. Behavioural and Cognitive Psychotherapy, 45(1), 58–72. https://doi.org/10.1017/s1352465816000394
Nicolaou, M., Colpo, M., & Vermeulen, E. (2019). Association of a priori dietary patterns with depressive symptoms: A harmonized meta-analysis of observational studies. Psychological Medicine, 50(11), 1872–1883. https://doi.org/10.1017/S0033291719001958
Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking rumination. Perspectives on Psychological Science, 3(5), 400–424. https://doi.org/10.1111/j.1745-6924.2008.00088.x
Orbell, S., & Verplanken, B. (2010). The automatic component of habit in health behavior: Habit as cue-contingent automaticity. Health Psychology, 29(4), 374–383. https://doi.org/10.1037/a0019506
O’Rourke, H. P., & MacKinnon, D. P. (2015). When the test of mediation is more powerful than the test of the total effect. Behavior Research Methods, 47(2), 424–442. https://doi.org/10.3758/s13428-014-0481-z
O’Rourke, H. P., & MacKinnon, D. P. (2018). Reasons for testing mediation in the absence of an intervention effect: A research imperative in prevention and intervention research. Journal of Studies on Alcohol and Drugs, 79(2), 171–181. https://doi.org/10.15288/jsad.2018.79.171
Ouwens, M. A., van Strien, T., & van Leeuwe, J. F. J. (2009). Possible pathways between depression, emotional and external eating. A structural equation model. Appetite, 53(2), 245–248. https://doi.org/10.1016/j.appet.2009.06.001
Ouwens, M., Watkins, E., Bot, M., Brouwer, I. A., Roca, M., Kohls, E., Penninx, B. W. J. H., van Grootheest, G., Cabout, M., Hegerl, U., Gili, M., & Visser, M. (2020). Acceptability and feasibility of two interventions in the MooDFOOD trial: A food-related depression prevention randomised controlled trial in overweight adults with subsyndromal symptoms of depression. BMJ Open, 10(9). https://doi.org/10.1136/bmjopen-2019-034025
Ouwens, M., Watkins, E., Bot, M., Brouwer, I. A., Roca, M., Kohls, E., Penninx, B. W. J. H., van Grootheest, G., Hegerl, U., Gili, M., & Visser, M. (2020). Nutrition and depression: Summary of findings from the EU-funded MooDFOOD depression prevention randomised controlled trial and a critical review of the literature. Nutrition Bulletin, 45(4), 403–414. https://doi.org/10.1111/nbu.12447
Paans, N. P. G., Bot, M., Brouwer, I. A., Visser, M., Gili, M., Roca, M., Hegerl, U., Kohls, E., Owens, M., Watkins, E., & Penninx, B. W. J. H. (2020). Effects of food-related behavioral activation therapy on eating styles, diet quality and body weight change: Results from the MooDFOOD Randomized Clinical Trial [Manuscript submitted for publication].
Richards, D. A., Ekers, D., McMillan, D., Taylor, R. S., Byford, S., Warren, F. C., Barrett, B., Farrand, P. A., Gilbody, S., Kuyken, W., O’Mahen, H., Watkins, E. R., Wright, K. A., Hollon, S. D., Reed, N., Rhodes, S., Fletcher, E., & Finning, K. (2016). Cost and outcome of behavioural activation versus cognitive behavioural therapy for depression (COBRA): A randomised, controlled, non-inferiority trial. Lancet, 388(10047), 871–880. https://doi.org/10.1016/S0140-6736(16)31140-0
Richards, D. A., Rhodes, S., Ekers, D., McMillan, D., Taylor, R. S., Byford, S., Barrett, B., Finning, K., Ganguli, P., Warren, F., Farrand, P., Gilbody, S., Kuyken, W., O’Mahen, H., Watkins, E., Wright, K., Reed, N., Fletcher, E., Hollon, S. D., & Woodhouse, R. (2017). Cost and outcome of behavioural activation (COBRA): A randomised controlled trial of behavioural activation versus cognitive-behavioural therapy for depression. Health Technology Assessment, 21(46), i–365. https://doi.org/10.3310/hta21460
Roca, M., Kohls, E., Gili, M., Watkins, E., Owens, M., Hegerl, U., van Grootheest, G., Bot, M., Cabout, M., Brouwer, I. A., Visser, M., & Penninx, B. W. (2016). Prevention of depression through nutritional strategies in high-risk persons: Rationale and design of the MooDFOOD prevention trial. BMC Psychiatry, 16(1), Article 192. https://doi.org/10.1186/s12888-016-0900-z
Rucklidge, J. J., & Kaplan, B. J. (2013). Broad-spectrum micronutrient formulas for the treatment of psychiatric symptoms: A systematic review. Expert Review of Neurotherapeutics, 13(1), 49–73. https://doi.org/10.1586/ern.12.145
Sarris, J., Byrne, G. J., Stough, C., Bousman, C., Mischoulon, D., Murphy, J., Macdonald, P., Adams, L., Nazareth, S., Oliver, G., Cribb, L., Savage, K., Menon, R., Chamoli, S., Berk, M., & Ng, C. (2019). Nutraceuticals for major depressive disorder—more is not merrier: An 8-week double-blind, randomised, controlled trial. *Journal of Affective Disorders*, 245, 1007–1015. https://doi.org/10.1016/j.jad.2018.11.092

Sarris, J., Logan, A. C., Akbaraly, T. N., Amminger, G. P., Balanzá-Martínez, V., Freeman, M. P., Hibbeln, J., Matsuoka, Y., Mischoulon, D., Mizoue, T., Nanri, A., Nishi, D., Ramsey, D., Rucklidge, J. J., Sanchez-Villegas, A., Scholey, A., Su, K. P., & Jacka, F. N. (2015). Nutritional medicine as mainstream in psychiatry. *Lancet Psychiatry*, 2(3), 271–274. https://doi.org/10.1016/S2215-0366(14)00051-0

Sarris, J., Murphy, J., Mischoulon, D., Papakostas, G. I., Fava, M., Berk, M., & Ng, C. H. (2016). Adjunctive nutraceuticals for depression: A systematic review and meta-analyses. *American Journal of Psychiatry*, 173(6), 575–587. https://doi.org/10.1176/appi.ajp.2016.15091228

Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Janavs, J., Weiller, E., Keskiner, A., Schinka, J., Knapp, E., Sheehan, K. H., Janavs, J., Sanchez-Villegas, A., Scholey, A., Su, K. P., & Jacka, F. N. (2015). Nutritional medicine as mainstream in psychiatry. *Lancet Psychiatry*, 2(3), 271–274. https://doi.org/10.1016/S2215-0366(14)00051-0

Shrout, P. E., & Bolger, N. (2002). Mediation in experimen
tal and nonexperimental studies: New procedures and recommendations. *Psychological Methods*, 7(4), 422–445.

Topper, M., Emmelkamp, P. M. G., Watkins, E., & Ehring, T. (2017). Prevention of anxiety disorders and depression by targeting excessive worry and rumination in adolescents and young adults: A randomized controlled trial. *Behaviour Research and Therapy*, 90, 123–126. https://doi.org/10.1016/j.brat.2016.12.015

Vanderweele, T. J. (2013). Policy-relevant proportions for direct effects. *Epidemiology*, 24(1), 175–176. https://doi.org/10.1097/EDE.0b013e318278a140

van Strien, T., Donker, M. H., & Ouwens, M. A. (2016). Is desire to eat in response to positive emotions an “obese” eating style: Is KummerSpeck for some people a misnomer? *Appetite*, 100, 225–235. https://doi.org/10.1016/j.appet.2016.02.035

van’t Riet, J., Sijtsma, S. J., Dagevos, H., & de Bruijn, G. J. (2011). The importance of habits in eating behaviour. An overview and recommendations for future research. *Appetite*, 57(3), 585–596. https://doi.org/10.1016/j.appet.2011.07.010

van Zoonen, K., Buntrock, C., Ebert, D. D., Smit, F., Reynolds, C. F., Beekman, A. T., & Cuijpers, P. (2014). Preventing the onset of major depressive disorder: A meta-analytic review of psychological interventions. *International Journal of Epidemiology*, 43(2), 318–329. https://doi.org/10.1093/ije/dyt175

Veale, D. (2008). Behavioural activation for depression. *Advances in Psychiatric Treatment*, 14(1), 29–36. https://doi.org/10.1192/apt.bp.107.004051

Verhoeven, A. A. C., Adriaanse, M. A., Evers, C., & de Ridder, D. T. D. (2012). The power of habits: Unhealthy snacking behaviour is primarily predicted by habit strength. *British Journal of Health Psychology*, 17(4), 758–770. https://doi.org/10.1111/j.2044-8287.2012.02070.x

Verma, S., & Hussain, M. E. (2017). Obesity and diabetes: An update. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*, 11(1), 73–79. https://doi.org/10.1016/j.dsx.2016.06.017

Verplanken, B., & Orbell, S. (2003). Reflections on past behavior: A self-report index of habit strength. *Journal of Applied Social Psychology*, 33(6), 1313–1330. https://doi.org/10.1177/002190290303600407

Watkins, E. R. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin*, 134(2), 163–206. https://doi.org/10.1037/0033-2909.134.2.163

Watkins, E. R., Mullan, E., Wingrove, J., Rimes, K., Steiner, H., Bathurst, N., Eastman, R., & Scott, J. (2011). Ruminations-focused cognitive-behavioural therapy for residual depression: Phase II randomised controlled trial. *British Journal of Psychiatry*, 199(4), 317–322. https://doi.org/10.1192/bjp.bp.110.090282

Watkins, E. R., Newbold, A., Tester-Jones, M., Javaid, M., Cadman, J., Collins, L. M., Graham, J., & Mostazir, M. (2016). Implementing multifactorial psychotherapy research in online virtual environments (IMPROVE-2): Study protocol for a phase III trial of the MOST randomized component selection method for internet cognitive-behavioural therapy for depression. *BMC Psychiatry*, 16(1), Article 345. https://doi.org/10.1186/s12888-016-1054-8

Watkins, E. R., & Nolen-Hoeksema, S. (2014). A habit-goal framework of depressive rumination. *Journal of Abnormal Psychology*, 123(1), 24–34. https://doi.org/10.1037/a0035540

Wittchen, H. U., Jacobi, F., Rehm, J., Gustavsson, A., Svensson, M., Jonsson, B., Olesen, J., Allgulander, C., Alonso, J., Faravelli, C., Fratiglioni, L., Jemmun, P., Lieb, R., Maercker, A., van Os, J., Preisig, M., Salvador-Carulla, L., Simon, R., & Steinhausen, H.-C. (2011). The size and burden of mental disorders and other disorders of the brain in Europe 2010. *European Neuropsychopharmacology*, 21(9), 655–679. https://doi.org/10.1016/j.euroence.2011.07.018

Wood, W., & Neal, D. T. (2007). A new look at habits and the habit-goal interface. *Psychological Review*, 114(4), 843–863. https://doi.org/10.1037/0033-295X.114.4.843

World Health Organization. (2017). *Depression and other common mental disorders: Global health estimates*. http://apps.who.int/iris/bitstream/handle/10665/254610/WHO-MSD-MER-2017.2-eng.pdf;jsessionid=FF5A7B1D7D63B6405DEA1C5E6732B9?sequence=1

World Medical Association. (2013). World Medical Association Declaration of Helsinki: Ethical principles for medical research involving human subjects World Medical Association Declaration of Helsinki Special Communication. *JAMA*, 310(20), 2191–2194. https://doi.org/10.1001/jama.2013.281053