

## Clinical Study

# Mindfulness Intervention for Stress Eating to Reduce Cortisol and Abdominal Fat among Overweight and Obese Women: An Exploratory Randomized Controlled Study

Jennifer Daubenmier,<sup>1</sup> Jean Kristeller,<sup>2</sup> Frederick M. Hecht,<sup>1</sup>  
Nicole Maninger,<sup>3</sup> Margaret Kuwata,<sup>1</sup> Kinnari Jhaveri,<sup>1</sup> Robert H. Lustig,<sup>4</sup>  
Margaret Kemeny,<sup>5</sup> Lori Karan,<sup>6</sup> and Elissa Epel<sup>5</sup>

<sup>1</sup> Osher Center for Integrative Medicine, Department of Medicine, University of California, San Francisco, CA 94115, USA

<sup>2</sup> Department of Psychology, Indiana State University, Terre Haute, IN 47809, USA

<sup>3</sup> California National Primate Research Center, University of California, Davis, CA 95616, USA

<sup>4</sup> Department of Pediatrics, University of California, San Francisco, CA 94143, USA

<sup>5</sup> Department of Psychiatry, University of California, San Francisco, CA 94143, USA

<sup>6</sup> Department of Medicine, University of California, San Francisco, CA 94143, USA

Correspondence should be addressed to Jennifer Daubenmier, Jennifer.daubenmier@ucsf.edu and Elissa Epel, eepel@lppi.ucsf.edu

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Psychological distress and elevated cortisol secretion promote abdominal fat, a feature of the Metabolic Syndrome. Effects of stress reduction interventions on abdominal fat are unknown. Forty-seven overweight/obese women (mean BMI = 31.2) were randomly assigned to a 4-month intervention or waitlist group to explore effects of a mindfulness program for stress eating. We assessed mindfulness, psychological distress, eating behavior, weight, cortisol awakening response (CAR), and abdominal fat (by dual-energy X-ray absorptiometry) pre- and posttreatment. Treatment participants improved in mindfulness, anxiety, and external-based eating compared to control participants. Groups did not differ on average CAR, weight, or abdominal fat over time. However, obese treatment participants showed significant reductions in CAR and maintained body weight, while obese control participants had stable CAR and gained weight. Improvements in mindfulness, chronic stress, and CAR were associated with reductions in abdominal fat. This proof of concept study suggests that mindfulness training shows promise for improving eating patterns and the CAR, which may reduce abdominal fat over time.

## 1. Introduction

Many of the adverse health effects of excess weight are associated with abdominal obesity independent of total weight. Visceral obesity, in particular, produces inflammatory molecules which promote insulin resistance and the Metabolic Syndrome [1]. Thus, abdominal adiposity is an important target for reducing risk of type 2 diabetes and cardiovascular disease (CVD) [2].

One modifiable risk factor that may contribute to abdominal adiposity is chronic psychological stress. Low socioeconomic status and job stress, two indicators of

chronic stress, are associated with greater abdominal adiposity in cross-sectional and prospective studies [3–5]. Stress can impact abdominal adiposity through repeated activation of the hypothalamic-pituitary-adrenal (HPA) axis, resulting in hypersecretion of cortisol. Cortisol binds to glucocorticoid receptors (GR) on fat cells activating lipoprotein lipase, an enzyme that converts circulating triglycerides into free fatty acids in adipocytes [6]. Increases in cortisol in combination with increased levels of insulin mobilize amino acids and fatty acids from peripheral to abdominal regions for immediate use by the liver for gluconeogenesis and ketones for energy use by the brain [7, 8]. A greater density of

GRs are found on visceral compared to peripheral fat cells partly explaining why fat stores are redistributed to intra-abdominal regions in the presence of elevated cortisol [9–11].

The link between elevated cortisol concentrations and increased abdominal fat was first observed in patients diagnosed with Cushing's syndrome who had adrenal tumors leading to hypercortisolemia [12]. Laboratory measures of increased HPA axis activity associated with abdominal adiposity include elevated cortisol secretion after lunch [13], elevated cortisol and ACTH levels after administration of corticotrophin-releasing hormone (CRH) [14], and elevated cortisol concentrations after challenges with CRH and arginine vasopressin [15, 16] and dexamethasone [17]. Healthy men and women who exhibit increased cortisol reactivity in response to laboratory stress tasks have greater abdominal adiposity [18–20], and among depressed, postmenopausal women, those with higher morning cortisol have greater levels of visceral fat as measured by computed tomography compared to those with lower cortisol levels [21] and healthy controls [22].

A naturalistic, noninvasive indicator of basal HPA activity, the cortisol awakening response (CAR), has been related to greater visceral adiposity as measured by waist to hip ratio in men [23–25] and magnetic resonance imaging in adolescent girls [26], although not all studies have shown a positive association [27]. Most people show a 50%–160% increase in cortisol concentrations in the first 30 minutes after awakening [28]. According to a recent meta-analysis, a heightened CAR is generally associated with greater job and life stress, and reduced responses tend to relate to positive psychological traits such as optimism and positive affect. However, a lower CAR is also related to fatigue and posttraumatic stress disorder, and norms have not been established to differentiate hypo- from hyper-CAR; thus, careful consideration of sample characteristics is needed when interpreting CAR [29]. Thoughts and emotions related to the upcoming day are theorized to accentuate the acute response because this rise is distinct from the circadian rise in the morning hours before awakening [30].

In addition to direct effects of chronic stress on abdominal obesity, psychological stress can also trigger consumption of high fat and sweet food, leading to overall weight gain [31–41]. Stress eating may also increase visceral adiposity independent of total weight gain. The combination of chronic stress and a high fat and sugar diet markedly increases visceral adipose tissue through stress-mediated upregulation of neuropeptide Y and its receptors in fat tissues of rodents [42]. Neuropeptide Y promotes fat angiogenesis and the proliferation and differentiation of new adipocytes. In humans, self-identified stress eaters tend to gain more abdominal fat during stressful periods compared to non self-identified stress eaters [43].

Psychological causes of stress eating or other types of emotional eating include poor awareness of internal physiological states and inability to differentiate between hunger cues and emotional arousal [44–47]. Some individuals are more susceptible to stress-induced eating than others and may adopt a self-regulation strategy for coping with aversive

states in which attention is shifted away from negative self-appraisal or affect and towards the immediate stimulus environment, such as food [48, 49]. Individuals who are identified as “emotional eaters” are more vulnerable to weight gain compared to nonemotional eaters, [43, 50] and they may regain more weight after successful weight loss through either diet and exercise [51] or bariatric surgery [52].

Most behavioral weight loss interventions do not aim to reduce psychological stress as a primary goal, if at all, and stress may be one factor contributing to the modest success of long-term weight loss maintenance [51, 53]. Furthermore, most interventions focus on weight loss rather than on reduction of abdominal adiposity. Despite evidence linking stress to overeating and abdominal fat accumulation, to our knowledge, no published studies have examined whether behavioral interventions designed to improve stress, stress eating, and/or cortisol responses lead to reductions in abdominal adiposity. A mindfulness-based intervention may be effective in reducing stress and improving stress-related overeating as previous studies suggest that mindfulness training reduces psychological stress and enhances psychological well-being for a variety of health conditions, [54–58] may improve cortisol patterns, [59] may reduce binge eating and other eating disorder symptoms among patients with eating disorders, and may reduce weight among obese and nonobese adults [60–63]. We hypothesized that mindfulness training would enhance awareness of and responsiveness to bodily sensations and reduce psychological distress, emotional eating, and cortisol secretion, all of which, in turn, would reduce amount of abdominal adiposity, our main outcome. The current randomized waitlist-controlled pilot study explored the effects of a mindfulness-based intervention for stress eating on abdominal adiposity. We assessed changes in weight and the relative distribution of body fat as secondary outcomes. Given recent evidence that upper trunk as well as visceral fat is associated with increased insulin resistance [64] and leg fat is associated with lower metabolic risk [65, 66], we also examined the overall change in ratio of total trunk to leg fat as an index of relative body fat distribution. Finally, because stress eating is more common in women than in men [67], and women and men differ in fat distribution profiles, we targeted overweight and obese women who felt that stress influenced their eating behavior and weight for recruitment.

## 2. Materials and Methods

**2.1. Study Design.** The study was a randomized waitlist-controlled pilot study designed to explore the effects of a mindfulness intervention on abdominal adiposity among overweight and obese women. The study was approved by the Institutional Review Board of the University of California, San Francisco (UCSF), and all participants provided informed consent. The intervention was provided free of charge and participants were compensated for their time during assessment visits.

**2.2. Participants.** Female participants were recruited through media outlets and flyers posted in the San Francisco Bay Area. Recruitment was aimed at women who were stressed and wanted to control the effects of stress on their eating behavior. Potential participants attended an orientation session in which the intervention was described as a program to address relationships between stress, eating, and abdominal fat but was not specifically designed to facilitate weight loss. Participants were not blinded to study hypotheses.

Participants were eligible if their body mass index (BMI) was between 25–40 and they weighed less than 300 lbs (due to limitations of the densitometer) and they had no medical issues such as diabetes or medication use such as hormonal supplements that could affect weight loss, insulin resistance, or abdominal fat. Postmenopausal women were excluded because fat is redistributed to visceral depots after menopause and is primarily determined by alterations in estrogen levels [68]. Women were eligible if they had no history of a bilateral oophorectomy, total hysterectomy, or polycystic ovary syndrome; had no active endocrinologic disorder; were not pregnant, were less than one year postpartum, or breastfeeding; were not currently on an active diet plan; had no current self-reported eating disorder or alcohol or drug addiction; had a negative urine test for diabetes and opiate use; were not taking steroids or antipsychotic medications, though antidepressant medication use was permitted; had no prior experience with Mindfulness-Based Stress Reduction (MBSR) or current meditation or yoga practice; and were English literate.

**2.3. Randomization.** Participants were randomized to the treatment or control group in a 1:1 ratio and stratified on BMI category (overweight: BMI 25–29.99 versus obese: 30–39.99), age ( $\geq 40$  years), and current antidepressant medication use ( $n = 7$ ) because these factors are known to influence weight and may impact change in abdominal fat over time. Computer-generated random numbers were used by the site statistician at the UCSF General Clinical Research Center (GCRC) to assign group condition. After all participants had completed baseline assessments, this information was given to study staff who informed participants of their group condition.

**2.4. Intervention Groups.** A preliminary, novel intervention was developed drawing on components from Mindfulness-Based Stress Reduction (MBSR), [54] Mindfulness-Based Cognitive Therapy (MBCT), [56] and Mindfulness-Based Eating Awareness Training (MB-EAT) [69, 70]. Mindfulness is characterized by an open, nonjudgmental stance towards present-moment experience as a way to disidentify with and interrupt habitual patterns of thoughts, emotions, and behaviors to allow for more adaptive responses to occur. Mindfulness is cultivated through systematic training of a focused state of awareness through repeated attendance to bodily and other sensory experiences, thoughts, and emotions. MB-EAT promotes awareness of bodily experiences related to physical hunger, satiety, taste satisfaction, and emotional triggers for overeating. The program was

originally developed for binge eating disorder (BED), and in an uncontrolled pilot study and a randomized clinical trial, it was associated with reductions in binge-eating, depression, other indicators of regulation of food intake, as well as weight loss in proportion to amount of mindfulness practice [62, 70].

In the current study, the intervention program consisted of nine 2.5-hour classes and one 7-hour silent day of guided meditation practice after class 6. Classes were held on a weekly basis on the weekend. Participants were instructed in the body scan, mindful yoga stretches, sitting and loving kindness meditations as taught in MBSR, and the “3 minute breathing space” as taught in MBCT. Participants were also led through guided meditations as a way to introduce mindful eating practices of paying attention to physical sensations of hunger, stomach fullness, taste satisfaction, and food cravings; identification of emotional and eating triggers; self-acceptance; and inner wisdom as taught in MB-EAT [69]. Meditations on awareness of negative emotions in general and loving kindness and forgiveness towards others were included as supplemental meditations. Each session opened with a mindfulness practice (body scan, yoga, sitting meditation, loving kindness, or forgiveness) followed by a discussion of the practice and review of progress and challenges over the previous week, and then guided meditations and discussions were used to introduce new eating or emotional awareness practices. On the retreat day, participants entered into silence to practice the meditations they had been taught and had a potluck meal to practice mindful eating skills. Participants were encouraged to engage in daily home assignments that included up to 30 minutes per day of formal mindfulness practices 6 days per week and mindful practices before and during meals.

Participants randomly assigned to the waitlist group were offered the mindfulness program after completion of all posttreatment assessments. To provide guidelines for healthy eating and exercise during the intervention and to control the effects of such information on study outcomes, both groups participated in a 2-hour nutrition and exercise information session aimed at moderate weight loss midway through the intervention, in which mindfulness was not discussed.

**2.5. Measures.** If eligible by initial phone screen, participants completed two assessment visits. Study nurses, blind to participant condition, performed the anthropometric and body composition assessments and blood draws. Research assistants administered the computerized questionnaires and provided instructions for the home saliva sampling procedure, but were not blind to participant condition at posttreatment assessments.

**2.5.1. Self-Report Measures.** Mindfulness was assessed using the Kentucky Inventory of Mindfulness Skills (KIMS) [71] questionnaire which measures four distinct, though somewhat correlated, mindfulness skills: Observing, which involves the ability to pay attention to internal and external sensory stimuli (e.g., body sensations, thoughts, sounds); Describing, which involves the ability to verbally express

one's experience; Acting with Awareness, which involves engaging in current activities with undivided attention; Accepting without Judgment, which assesses the ability to accept one's experience, particularly if it is unpleasant or unwanted, without judging it as good or bad or rushing to change it. Responses were rated on a 5-point scale ranging from 1 (never or very rarely true) to 5 (almost always or always true). The Body Responsiveness Scale assesses the importance of attending to bodily sensations to guide behavior and the degree of perceived integration between psychological and physical states (e.g., reverse coded item: "I suppress my bodily feelings and sensations") [72]. Responses were measured on a 7-point scale ranging from 1 (not at all true about me) to 7 (very true about me). Higher scores indicate greater body responsiveness.

The Wheaton Chronic Stress Inventory [73] measures the presence of chronic stressors in one's life related to work, relationships, financial difficulties, and general overload, which includes ratings of impact. Statements were rated according to a 5-point scale (0 = not at all true, 4 = extremely true) and averaged. The Perceived Stress Scale [74] evaluates one's perception of stressful events over the past month by using a 5-point scale (0 = never; 4 = very often). The State-Trait Anxiety Scale (trait form) [75] was used to assess general feelings of anxiety. Participants rated statements along a 4-point scale ranging from almost never = 1 to almost always = 4.

The Dutch Eating Behavior Questionnaire (DEBQ) [76] assesses three subscales of eating behaviors—dietary restraint, emotional eating, and external-based eating. The restrained eating subscale evaluates intentions and behaviors to restrict food intake due to concerns about weight. The emotional eating subscale measures overeating behaviors triggered by negative emotions, such as anger, boredom, anxiety, or fear. The external-based eating subscale assesses eating in response to food-related stimuli, such as the smell or taste of food, presence of others eating, or seeing food prepared. Responses were on a 5-point scale from 1 = never to 5 = very often.

**2.5.2. Treatment Adherence.** Weekly class attendance was recorded and participants completed logs of weekly minutes of formal home meditation practices and the number of meals they ate mindfully each week. Formal practices included the body scan meditation, sitting meditation focused on breath awareness, mindful yoga, loving kindness directed towards self and others, and self-forgiveness practice.

**2.5.3. Salivary Cortisol.** To measure the cortisol awakening response (CAR) and cortisol slope, participants collected saliva samples at home on 4 days, pre- and posttreatment. One day of CAR assessment has been shown to be highly influenced by situational factors and 2–6 days of assessment on work days are needed to achieve sufficient reliability as a trait measure [77]. Four days of sampling was chosen to maximize reliability without excessive participant burden. Samples were collected immediately upon awakening, 30

minutes after awakening and just prior to bedtime. CAR was available for 4 days, but cortisol slope was available for 3 days because participants took an opioid antagonist (naltrexone) that affects cortisol concentrations on the fourth day at 1 pm as part of a separate study. Each sample was collected by drooling into a straw in 2 mL SaliCaps tubes (IBL, Hamburg, Germany). Participants were instructed to collect the first sample while in bed and not to eat, drink, brush their teeth, or engage in vigorous activity between the first two morning samples or for 20 minutes prior to all other samples. Hormone analysis was performed at Dresden Lab Service, overseen by Dr. Clemens Kirschbaum, at the Dresden University of Technology (Germany) using a commercial chemiluminescence immunoassay (CLIA, IBL, Hamburg, Germany). Values greater than 100 were excluded because they are believed to be physiologically not plausible. The CAR was computed by subtracting the 30-minute postwaking cortisol value from the morning value. Cortisol slope was calculated by subtracting the bedtime cortisol value from the morning value. In all cases, values were averaged across days. All participants who completed the saliva sampling at both pre- and post-intervention timepoints had a minimum of two days of cortisol data available at each time point for analysis, except for one participant whose incomplete cortisol data were excluded.

**2.5.4. Serum Cortisol.** Fasting morning blood samples were obtained from an indwelling forearm venous catheter. Serum cortisol concentrations were estimated in duplicate using commercial radioimmunoassay kits (Coat-A-Count Cortisol kit, Siemens Medical Solutions Diagnostics, Los Angeles, Calif, USA). The intra- and inter-assay coefficients of variation were 4.02% and 5.99%, respectively.

**2.5.5. Anthropometric Variables.** A standard stadiometer (Perspective Enterprises, Portage, Mich, USA) was used to measure height to the nearest 1/8 inch. A digital scale (Wheelchair Scale 6002, Scale-Tronix, Carol Stream, Ill, USA) was used to measure weight to the nearest 0.1 kg. Waist circumference was assessed with a tape measure at the umbilicus. The mean of the closest 2 of 3 measures falling within a range of .5 cm was calculated.

**2.5.6. Body Fat.** Whole-body dual energy X-ray absorptiometry (DEXA) scans were performed to assess body fat distribution. The DEXA densitometry (GE Healthcare Lunar Prodigy, Madison, Wis, USA) was adjusted to the fan beam mode and EnCore software version 9.15 was used. The primary region of interest was fat tissue from a rectangular region in the abdominal area defined by the upper boundary of the second lumbar vertebra to the lower edge of the fourth lumbar vertebra. The vertical sides were defined as the continuation of the lateral sides of the rib cage. Previous research established that this region correlates with magnetic resonance imaging of visceral fat among obese women ( $r = .74$ ) [78] and was used as an estimate of visceral fat in the present study. As a secondary measure, ratio of trunk to leg fat mass ratio was assessed as an indicator of fat distribution.

The trunk was defined as the area below the chin and above the trochanter neck. The coefficient of variation in assessing fat mass from the UCSF CCRC densitometer is 4%.

### 3. Statistical Analyses

To test the primary hypothesis, both intention-to-treat and treatment efficacy analyses were performed. Independent-samples *t*-tests and chi-square analyses were used to compare groups at baseline. Primary analyses used independent samples *t*-tests to test between group differences in change scores. Assuming participants lost to followup did not change over time, missing data at postintervention were imputed using preintervention values. Treatment efficacy analyses were also performed by including treatment participants who attended at least 4/10 classes and excluding one control participant who received liposuction treatment. To explore whether the intervention had a differential impact among overweight versus obese participants on outcome variables, intention-to-treat ANOVAs with 2 between subject factors of group (treatment versus control) and obesity status [overweight (BMI < 30) versus obese (BMI ≥ 30)] on change scores were conducted. Variables with skewed distributions underwent natural log transformation. Cohen's *d* was calculated to assess effect size.

For secondary analyses, multiple linear regression models were performed across groups and within the treatment group among participants with complete data to predict changes in abdominal fat and fat distribution, controlling for baseline levels and change in weight. Predictors included changes in psychological, eating behavior, and cortisol variables. Interactions between group assignment and predictors were also tested.

### 4. Results

**4.1. Participant Characteristics.** Of 322 potential participants who were screened for eligibility from November 2006 to March 2007, 53 met eligibility criteria and chose to enroll (see Figure 1). The most common reasons for ineligibility were BMI outside of range and postmenopausal status. Of the 53 eligible participants, 47 went on to the randomization stage, with 24 randomized to the treatment and 23 to the control group. The overall sample was 62% White, 15% Hispanic/Latino, 15% Asian/Pacific Islander, and 9% other. Groups did not differ in overall ethnic composition, with 63% of the treatment and 61% of the control group identifying as White ( $P = .91$ ).

The sample reported significantly greater levels of perceived stress compared to a representative sample of US women in 2006, as assessed by total scores on the Perceived Stress Scale ( $19.0 \pm 5.9$  versus  $16.1 \pm 7.7$ ;  $t(46) = -166.0$ ;  $P < .001$ ) [79]. The sample also reported a high level of emotional eating, as evidenced by significantly higher scores on the DEBQ emotion eating subscale compared to a representative sample of overweight (BMI > 25) Dutch citizens ( $3.42 \pm 0.8$  versus  $2.61 \pm 0.9$ ;  $t(45) = 7.2$ ,  $P < .001$ ) [80]. These differences were to be expected given

that recruitment targeted women who were stress eaters. As shown in Table 1, no significant differences between treatment and control groups were observed at baseline, except that treatment participants reported lower scores on the mindfulness "Observing" subscale compared to control participants.

**4.2. Lost to Followup and Treatment Adherence.** Four treatment participants did not receive the minimum treatment dose. Five treatment and two control participants were lost to followup for the primary analysis (see Figure 1). One control participant received liposuction and was included in the intention-to-treat analyses but was excluded from treatment efficacy and secondary analyses involving any biological outcomes.

Class attendance was 68% among all participants and 79% among those who received the minimum dose. To include adherence data from all participants, mean weekly minutes of meditation practice were based on a minimum of 4 weeks of adherence logs. Participants who attended at least one class reported practicing meditation an average of  $98 \pm 79$  minutes and eating  $5.9 \pm 4.4$  meals mindfully per week. The "as treated" participants reported a mean of  $108 \pm 75$  minutes of meditation practice and  $6.5 \pm 4.2$  mindful meals per week.

#### 4.3. Treatment Effects

**4.3.1. Psychological Variables.** Results of the intention-to-treat and treatment efficacy analyses are summarized and instances in which results vary are noted (see Table 2). The treatment group reported significantly greater increases on 3 of the 4 mindfulness subscales and on the Body Responsiveness Scale compared to the control group (in the treatment efficacy analysis). Effect sizes were medium to large, except for the Describing subscale of the KIMS which did not differ between groups.

Means were in the predicted directions for chronic and perceived stress with chronic stress remaining constant in the intervention group and going up in the control group, and perceived stress going down in the intervention group and remaining constant in the control group. The effect size was small for chronic stress and medium for perceived stress, although not statistically significant given the sample size. The treatment group significantly decreased in trait anxiety compared to the control group in the treatment efficacy analysis with a moderate effect size (the effect was marginally significant in intention-to-treat analysis).

The treatment group showed a slight increase in restrained eating and the control group showed a minor decrease; however, the effect size was small and nonsignificant. Both groups decreased in emotional and external-based eating, but the treatment group reported significantly greater decreases in external eating compared to the control group, while the treatment effect on emotional eating was marginally significant. The effect size was moderate for emotional eating and moderate to large for external eating.

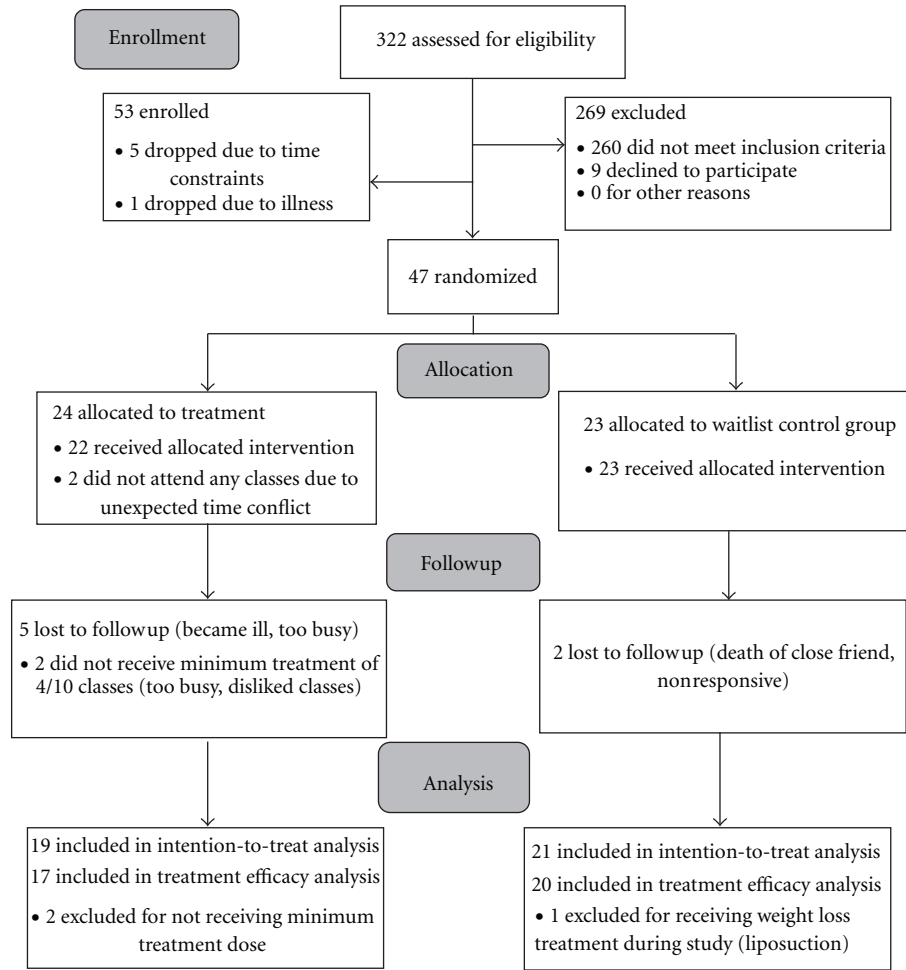


FIGURE 1: Flow diagram with abdominal fat as primary analysis.

**4.3.2. Cortisol, Abdominal Fat, Fat Distribution, and Weight.** Treatment participants showed a nonsignificant trend for greater reductions in CAR over time compared to the control group (moderate effect size). Neither group showed substantial changes in the cortisol slope or morning serum cortisol concentrations. Groups did not differ substantially over time on amount of abdominal fat, fat distribution (the ratio of trunk to leg fat), or overall weight.

**4.3.3. Subgroup Analyses by Obesity Status.** Exploratory intention-to-treat analyses revealed significant interactions between treatment group and obesity status for the CAR ( $F(1,37) = 4.3$ ,  $P = .046$ ; see Figure 2) and weight ( $F(1,37) = 4.1$ ,  $P = .049$ ). Inspection of the CAR means indicated significant reductions among obese participants in the treatment group ( $-9.4 \pm 11.0$  nmol/L,  $P = .03$ ) but not in the control group ( $0.2 \pm 9.7$  nmol/L,  $P = .96$ ; independent samples  $t$ -test comparing groups:  $t(16) = -1.9$ ,  $P = .07$ ), while the mean CARs of overweight participants in the treatment group ( $1.5 \pm 4.8$  nmol/L,  $P = .33$ ) and control group ( $-0.3 \pm 8.7$  nmol/L,  $P = .92$ ) did not differ over time ( $t(14) = 0.6$ ,  $P = .54$ ). Secondly, among obese participants, those assigned to the treatment group maintained weight

( $-0.4 \pm 3.5$  kg,  $P = .70$ ) while those in the control group gained weight ( $1.7 \pm 1.5$  kg,  $P = .01$ ; independent samples  $t$ -test comparing groups:  $t(18) = -1.6$ ,  $P = .12$ ). Mean weight did not change among overweight participants in the treatment group ( $0.4 \pm 1.8$  kg,  $P = .53$ ) or control group ( $-0.2 \pm 1.8$  kg,  $P = .71$ ; independent samples  $t$ -test comparing groups:  $t(22) = 0.7$ ,  $P = .47$ ). No other interactions between treatment group and obesity status were significant.

**4.4. Predictors of Changes in Abdominal Fat.** Results of multiple linear regressions predicting change in abdominal adiposity are shown in Table 3. Increases in the KIMS subscale, Acting with Awareness, were marginally related to decreases in abdominal adiposity across groups. A significant interaction between changes in body responsiveness and group condition was observed such that increases in body responsiveness were significantly related to greater decreases in abdominal fat among treatment but not control group participants. A significant interaction between changes in chronic stress and group condition was also observed, indicating that among treatment group participants, greater decreases in chronic stress were related to greater decreases

TABLE 1: Baseline characteristics of treatment and control participants.

| Variable                             | Treatment ( <i>n</i> = 24) <sup>a</sup> |       | Control ( <i>n</i> = 23) <sup>a</sup> |       | <i>P</i> value |
|--------------------------------------|-----------------------------------------|-------|---------------------------------------|-------|----------------|
|                                      | Mean                                    | SD    | Mean                                  | SD    |                |
| Age                                  | 40.42                                   | 8.0   | 41.39                                 | 6.7   | .65            |
| Weight (kg)                          | 84.40                                   | 14.2  | 85.17                                 | 14.7  | .86            |
| Body mass index                      | 31.40                                   | 4.7   | 30.77                                 | 4.8   | .65            |
| Waist circumference (cm)             | 104.14                                  | 10.9  | 103.22                                | 11.6  | .78            |
| Mindfulness-Act with Awareness       | 2.65                                    | 0.4   | 2.79                                  | 0.4   | .24            |
| Mindfulness-Observe                  | 3.01                                    | 0.4   | 3.52                                  | 0.5   | .001           |
| Mindfulness-Describe                 | 3.53                                    | 0.7   | 3.26                                  | 0.8   | .21            |
| Mindfulness-Nonjudging               | 3.13                                    | 0.9   | 3.05                                  | 0.8   | .73            |
| Body Responsiveness                  | 3.65                                    | 0.9   | 4.11                                  | 0.9   | .09            |
| Wheaton Chronic Stress Inventory     | 1.96                                    | 0.5   | 1.95                                  | 0.5   | .87            |
| Perceived stress                     | 1.96                                    | 0.5   | 1.86                                  | 0.7   | .59            |
| Anxiety                              | 2.25                                    | 0.4   | 2.15                                  | 0.5   | .43            |
| Restrained eating                    | 2.79                                    | 0.6   | 2.80                                  | 0.5   | .96            |
| Emotional eating                     | 3.42                                    | 0.7   | 3.42                                  | 0.8   | .99            |
| External-based eating                | 3.57                                    | 0.5   | 3.50                                  | 0.5   | .64            |
| Cortisol awakening response (nmol/L) | 6.72                                    | 8.1   | 7.26                                  | 7.9   | .83            |
| Cortisol slope (nmol/L)              | 15.67                                   | 5.9   | 13.52                                 | 5.2   | .22            |
| Serum morning cortisol (ln)          | 2.20                                    | 0.4   | 2.38                                  | 0.4   | .12            |
| Abdominal fat, L2-L4 region (g)      | 2238.81                                 | 675.0 | 2002.78                               | 652.2 | .23            |
| Trunk/leg fat mass ratio             | 1.68                                    | 0.5   | 1.51                                  | 0.3   | .15            |

<sup>a</sup> Variables with missing values in the treatment group included the cortisol awakening response (*n* = 3) and cortisol slope (*n* = 3), and in the control group, the mindfulness and eating variables (*n* = 1), cortisol awakening response (*n* = 2), and cortisol slope (*n* = 2).

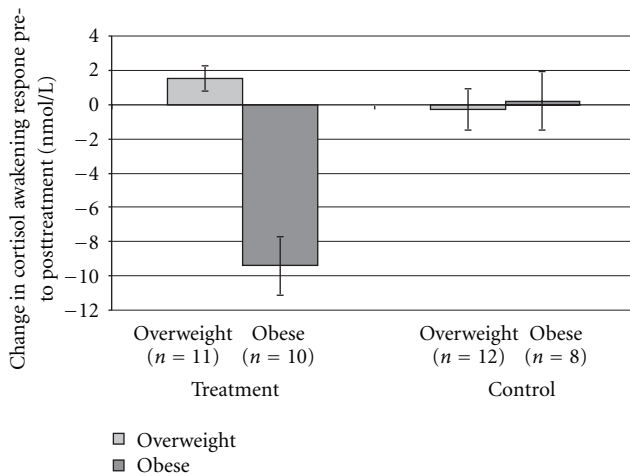


FIGURE 2: Mean weight change and standard errors by group condition among overweight versus obese participants.

in abdominal fat but not among control group participants. Decreases in CAR and increases in the cortisol slope tended to be related to decreases in abdominal fat, although these effects were not statistically significant when groups were combined. When examined separately, reductions in CAR were significantly related to reductions in abdominal fat

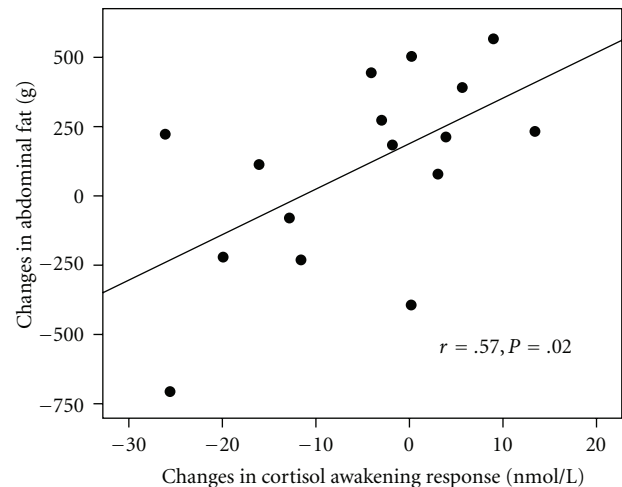


FIGURE 3: Scatter plot of correlation between changes in cortisol awakening response and changes in abdominal fat among treatment group participants.

among treatment but not control group participants (see Figure 3).

**4.5. Predictors of Changes in Fat Distribution.** Across groups, increases in the KIMS subscales, Acting with Awareness and Describing, were related to decreases in trunk/leg fat ratio

TABLE 2: Change from baseline for intention-to-treat and treatment efficacy analyses.

| Outcome                               | N<br>(t,c) | Intention to treat |                   |                                 | ES <sup>b</sup> | N<br>(t,c) | Treatment efficacy |                   |                                   | P    | ES   |
|---------------------------------------|------------|--------------------|-------------------|---------------------------------|-----------------|------------|--------------------|-------------------|-----------------------------------|------|------|
|                                       |            | Treat M<br>(SD)    | Control<br>M (SD) | Mean diff.<br>T - C<br>(95% CI) |                 |            | Treat M<br>(SD)    | Control<br>M (SD) | Mean diff.<br>(T - C)<br>(95% CI) |      |      |
| Mindfulness-Aware                     | 24<br>22   | 0.18<br>(0.5)      | -0.06<br>(0.3)    | 0.24<br>(0.0-0.5)               | .56             | 19<br>21   | 0.25<br>(0.5)      | -0.07<br>(0.3)    | 0.31<br>(0.0-0.6)                 | .02  | .72  |
| Mindfulness-Observe                   | 24<br>22   | 0.17<br>(0.5)      | -0.08<br>(0.3)    | 0.24<br>(0.0-0.5)               | .58             | 19<br>21   | 0.26<br>(0.5)      | -0.08<br>(0.3)    | 0.34<br>(0.1-0.6)                 | .01  | .85  |
| Mindfulness-Describe                  | 24<br>22   | 0.14<br>(0.5)      | 0.19<br>(0.5)     | -0.06<br>(-0.4-0.3)             | -.12            | 19<br>21   | 0.17<br>(0.6)      | 0.20<br>(0.5)     | 0.0<br>(-0.4-0.3)                 | .86  | -.05 |
| Mindfulness-Accept                    | 24<br>22   | 0.47<br>(0.7)      | .06<br>(0.5)      | 0.41<br>(0.1-0.8)               | .66             | 19<br>21   | 0.60<br>(0.7)      | 0.07<br>(0.5)     | 0.54<br>(0.2-0.9)                 | .007 | .83  |
| Body responsiveness                   | 24<br>22   | 0.27<br>(0.8)      | -0.26<br>(1.0)    | 0.53<br>(0.0-1.1)               | .56             | 19<br>21   | 0.36<br>(0.9)      | -0.27<br>(1.1)    | 0.63<br>(0.0-1.3)                 | .047 | .63  |
| Chronic stress                        | 24<br>23   | 0.03<br>(0.5)      | 0.15<br>(0.5)     | -0.12<br>(-0.4-0.2)             | -.26            | 19<br>21   | 0.02<br>(0.5)      | 0.16<br>(0.5)     | -0.14<br>(-0.4-0.2)               | .37  | -.30 |
| Perceived stress                      | 24<br>23   | -0.20<br>(0.5)     | 0.03<br>(0.6)     | -0.23<br>(-0.6-0.1)             | -.41            | 19<br>21   | -0.25<br>(0.5)     | 0.03<br>(0.6)     | -0.29<br>(-0.7-0.9)               | .14  | -.51 |
| Anxiety                               | 24<br>23   | -0.17<br>(0.4)     | 0.00<br>(0.3)     | -0.17<br>(-0.4-0.0)             | -.49            | 19<br>21   | -0.23<br>(0.4)     | 0.00<br>(0.4)     | -0.23<br>(-0.5-0.1)               | .045 | -.64 |
| Restrained eating                     | 24<br>22   | 0.10<br>(0.5)      | -0.03<br>(0.5)    | 0.13<br>(-0.2-0.4)              | .28             | 19<br>21   | 0.06<br>(0.5)      | -0.03<br>(0.5)    | 0.09<br>(-0.2-0.4)                | .56  | .19  |
| Emotional eating                      | 24<br>22   | -0.42<br>(0.4)     | -0.21<br>(0.4)    | -0.22<br>(-0.5-0.3)             | -.52            | 19<br>21   | -0.47<br>(0.5)     | -0.22<br>(0.4)    | -0.25<br>(-0.5-0.0)               | .08  | -.57 |
| External eating                       | 24<br>22   | -0.33<br>(0.4)     | -0.09<br>(0.4)    | -0.26<br>(-0.5-0.0)             | -.62            | 19<br>21   | -0.41<br>(0.4)     | -0.09<br>(0.4)    | -0.31<br>(-0.6-0.0)               | .02  | -.70 |
| Cort awake resp.<br>(nmol/L)/(nmol/L) | 21<br>21   | -3.7<br>(9.9)      | -0.1<br>(8.4)     | -3.6<br>(-9.3-2.1)              | -.39            | 15<br>17   | -5.49<br>(11.1)    | -0.10<br>(9.4)    | -5.39<br>(-12.8-2.0)              | .15  | -.53 |
| Cortisol slope (nmol/L)               | 21<br>21   | 1.2<br>(12.3)      | 0.9<br>(6.0)      | 0.25<br>(-5.8-6.3)              | .03             | 15<br>17   | 2.42<br>(14.3)     | 0.28<br>(5.8)     | 2.1<br>(-5.6-9.8)                 | .58  | .20  |
| Serum morn cortisol (ln)              | 24<br>23   | 0.00<br>(0.4)      | -0.02<br>(0.4)    | 0.02<br>(-0.2-0.3)              | .05             | 18<br>18   | .00<br>(0.5)       | -0.05<br>(0.5)    | 0.05<br>(-0.3-0.4)                | .78  | .10  |
| Weight (kg)                           | 24<br>23   | -0.03<br>(2.7)     | 0.38<br>(1.9)     | -0.40<br>(-1.8-1.0)             | -.17            | 18<br>20   | -0.06<br>(3.1)     | 0.58<br>(1.9)     | -0.64<br>(-2.3-1.0)               | .45  | -.26 |
| Abdominal fat (g)                     | 24<br>23   | 68.2<br>(318.9)    | 50.0<br>(305.4)   | 18.2<br>(-165.4-201.6)          | .06             | 17<br>20   | 54.4<br>(358.2)    | 41.6<br>(322.3)   | 12.8<br>(-214.3-239.9)            | .91  | .04  |
| Trunk/Leg fat ratio                   | 24<br>23   | 0.02<br>(0.1)      | 0.02<br>(0.1)     | .00<br>(-1-0.07)                | .01             | 17<br>20   | 0.03<br>(0.1)      | 0.02<br>(0.1)     | 0.01<br>(-0.1-0.1)                | .79  | .10  |

<sup>a</sup> P = P value of independent t-tests comparing mean changes over time between groups; <sup>b</sup> ES = Cohen's d effect size.

TABLE 3: Estimated effects of changes in predictors on changes in abdominal fat adjusted for baseline abdominal fat and weight change.

| Predictor                       | Across groups                   |              |                   |                | Treatment                   |                    |              |      | Control |                    |               |      |     |
|---------------------------------|---------------------------------|--------------|-------------------|----------------|-----------------------------|--------------------|--------------|------|---------|--------------------|---------------|------|-----|
|                                 | Est effect <sup>a</sup><br>(SE) | 95% CI       | St C <sup>b</sup> | P <sup>c</sup> | Gx <sup>pd</sup><br>P value | Est Effect<br>(SE) | 95% CI       | St C | P       | Est effect<br>(SE) | 95% CI        | St C | P   |
| Attendance                      | —                               | —            | —                 | —              | —                           | −40.7<br>(30.3)    | −105.4–24.0  | −.30 | .20     | —                  | —             | —    | —   |
| Mindful meals (#)               | —                               | —            | —                 | —              | —                           | −17.0<br>(20.6)    | −60.9–26.8   | −.21 | .42     | —                  | —             | —    | —   |
| Meditation (min)                | —                               | —            | —                 | —              | —                           | 1.2<br>(1.1)       | −1.2–3.6     | .25  | .30     | —                  | —             | —    | —   |
| Mindfulness-Act w Aware         | −217.3<br>(115.9)               | −454.8–20.2  | −.29              | .07            | .97                         | −274.5<br>(151.0)  | −598.3–49.3  | −.40 | .09     | −247.5<br>(223.6)  | −724.0–229.0  | −.25 | .29 |
| Mindfulness-Observe             | −63.0<br>(123.3)                | −313.5–187.5 | −.08              | .61            | .54                         | −78.4<br>(155.9)   | −410.7–253.9 | −.12 | .62     | −275.7<br>(296.1)  | −906.9–355.49 | −.23 | .37 |
| Mindfulness-Describe            | −73.9<br>(114.0)                | −305.6–157.8 | −.10              | .52            | .56                         | −0.4<br>(199.6)    | −425.8–425.0 | .00  | .99     | −172.3<br>(154.3)  | −501.2–156.5  | −.27 | .28 |
| Mindfulness-Accept              | −5.0<br>(79.0)                  | −165.6–155.6 | −.01              | .95            | .73                         | −74.9<br>(123.2)   | −337.6–187.8 | −.15 | .55     | −27.79<br>(149.7)  | −346.8–291.2  | −.05 | .86 |
| Body responsiveness             | −43.7<br>(51.3)                 | −147.9–60.4  | −.13              | .40            | .04                         | −218.8<br>(84.3)   | −398.7–9.0   | −.50 | .02     | 15.9<br>(67.4)     | −127.7–159.4  | .06  | .82 |
| Chronic stress                  | 15.8<br>(115.4)                 | −218.7–250.3 | .02               | .89            | .01                         | 338.2<br>(154.1)   | 9.7–666.7    | .47  | .04     | −291.8<br>(159.4)  | −631.6–48.1   | −.44 | .09 |
| Perceived stress                | 60.8<br>(95.4)                  | −133.0–254.6 | .11               | .53            | .32                         | 210.6<br>(164.9)   | −141.8–561.1 | .30  | .22     | 32.4<br>(127.1)    | −238.5–303.3  | .06  | .80 |
| Anxiety                         | −81.7<br>(140.3)                | −366.8–203.5 | −.09              | .56            | .70                         | 4.9<br>(207.9)     | −438.3–448.1 | .01  | .98     | −96.5<br>(230.2)   | −587.2–394.2  | −.10 | .68 |
| Restrained eating               | −27.0<br>(106.3)                | −243.0–189.0 | −.04              | .80            | .37                         | −146.9<br>(188.0)  | −547.6–253.8 | −.19 | .45     | 81.1<br>(159.5)    | −258.8–421.0  | .13  | .62 |
| Emotional eating                | 75.4<br>(124.3)                 | −177.2–328.0 | .10               | .55            | .67                         | 103.1<br>(172.7)   | −265.1–471.3 | .14  | .56     | 217.5<br>(226.0)   | −264.1–699.1  | .22  | .35 |
| External eating                 | 150.0<br>(119.4)                | −92.7–392.7  | .20               | .22            | .49                         | 306.3<br>(181.8)   | −81.2–693.7  | .36  | .11     | 160.3<br>(183.6)   | −231.0–551.5  | .21  | .40 |
| Cortisol awake rsp.<br>(nmol/L) | 13.9<br>(19.3)                  | −25.7–53.6   | .21               | .43            | .83                         | 26.3<br>(11.1)     | 2.1–50.4     | .85  | .04     | 10.4<br>(9.1)      | −9.4–30.1     | .29  | .28 |
| Cortisol slope (nmol/L)         | −7.9<br>(5.3)                   | −18.6–2.8    | −.25              | .14            | .87                         | −10.7<br>(6.5)     | −24.9–3.6    | −.44 | .13     | −6.8<br>(14.6)     | −38.3–24.7    | −.12 | .65 |
| Serum morn. cortisol (ln)       | 114.8<br>(117.1)                | −123.5–353.0 | .15               | .33            | .63                         | 59.4<br>(176.5)    | −316.8–435.6 | .08  | .74     | 195.9<br>(172.1)   | −173.3–565.1  | .27  | .27 |

<sup>a</sup> Est effect = unstandardized regression coefficient; <sup>b</sup> St C = standardized regression coefficient; <sup>c</sup> P = significance level of regression coefficient; <sup>d</sup> GxP P value = significance level of the group × predictor interaction term.

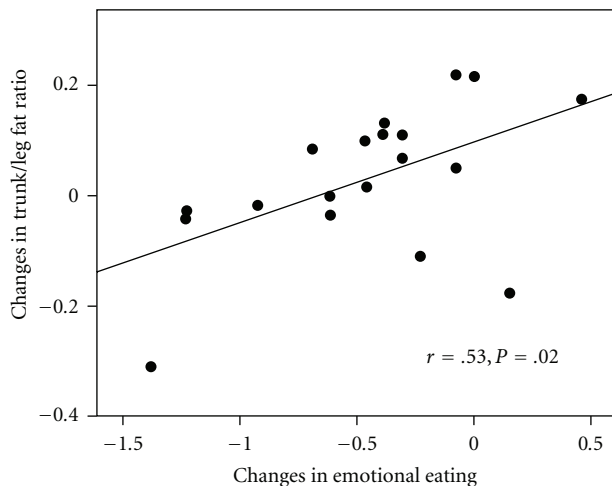


FIGURE 4: Scatter plot of correlation between changes in emotional eating and changes in trunk to leg fat ratio among treatment group participants.

(Acting with Awareness:  $b = -0.08(.04)$ ; 95% CI =  $-0.17-0.00$ ;  $P = .05$ ; Describing:  $b = -0.09(.04)$ , 95% CI =  $-0.17- -0.01$ ;  $P = .03$ ). Within the treatment group, decreases in emotional eating predicted decreases in the trunk/leg fat ratio ( $b = 0.13(.06)$ ; 95% CI =  $0.01-0.3$ ;  $P = .03$ ; see Figure 4). Across groups, decreases in morning serum cortisol levels were significantly related to decreases in trunk/leg fat ratio ( $b = 0.08(.04)$ ; 95% CI =  $0.01-0.16$ ;  $P = .03$ ).

## 5. Discussion

To our knowledge, this is the first study to explore effects of a novel mindful eating and stress reduction program on abdominal adiposity and fat distribution. The intervention was successful in increasing mindfulness and responsiveness to bodily sensations, reducing anxiety and eating in response to external food cues, and tended to reduce eating in response to emotions. The CAR showed a greater reduction among treatment participants compared to the control group, and the effect size was moderate, although non-significant. However, a significant reduction was observed among the subgroup of obese participants in the treatment group suggesting promising results for larger studies, particularly among obese adults. Despite these encouraging outcomes, the treatment did not reduce abdominal adiposity located between lumbar vertebrae 2–4, a region highly associated with amount of visceral fat, nor influence distribution of trunk to leg fat. However, we did observe the expected dose response relationships: intervention participants who reported the greatest improvements in mindfulness, responsiveness to bodily sensations, and chronic stress had the largest reductions in abdominal fat, supporting the theory that improvements in these psychological processes targeted by the intervention may lead to changes in abdominal adiposity. Furthermore, reductions in CAR were related to

reductions in abdominal fat among treatment group participants. Previous research established that CAR is associated with increased abdominal adiposity cross-sectionally; however, to our knowledge, this is the first study to demonstrate that longitudinal reductions in CAR are associated with corresponding reductions in abdominal adiposity. These results suggest that successful efforts to reduce CAR may reduce visceral adiposity over time.

We also observed the predicted dose response relationships between several changes in mediators with changes in relative fat distribution. Increases in mindfulness, decreases in serum morning cortisol levels, and, among treatment participants, reductions in emotional eating were associated with decreases in central to peripheral fat distribution as measured by the trunk/leg fat ratio. These findings are congruent with those of rat studies demonstrating a link between stress eating and fat distribution [9]. Specifically, chronic stress and elevated glucocorticoids induce a shift in preference of food intake in rats from chow to fat and sugar (“comfort foods”), which, in combination with elevated insulin, reorganize energy stores from peripheral to central regions. In turn, abdominal fat depots are highly correlated with reductions in HPA reactivity to acute stressors, suggesting the presence of a metabolic negative feedback signal. These animal studies suggest that ingestion of “comfort foods” may provide a short-term relief of stress in humans, albeit at the expense of increased abdominal adiposity. Mindfulness training may improve the ability to cope effectively with stressful experiences and reduce the reliance on “comfort foods” to manage stress or other negative emotions promoting more favorable body fat distribution over time.

The intervention was not designed to induce total weight loss, as guidelines for reducing caloric intake or increasing exercise were not an active part of the program. However, secondary analyses revealed that the intervention stabilized weight among those who were obese, as obese control group participants gained a mean of 1.7 kilograms during the same time period. Furthermore, a greater frequency of eating meals mindfully was marginally related to weight loss ( $r = -.41$ ,  $P = .08$ ). These results indicate that mindfulness practices by themselves may not reliably induce decreased caloric intake in this population of women but may prevent periodic increases in overeating and eventual weight gain. Minimally, these techniques may support weight maintenance efforts, and actual weight loss might occur for those participants who eat a high proportion of meals mindfully. Unfortunately, we were not able to examine longer-term changes in the current study. It is possible that these group differences in weight maintenance might have increased, or disappeared, during a longer term followup.

## 6. Limitations

Important limitations include the exploratory nature of the study with a large number of analyses, small sample size, and moderate percentage of participants that was lost to followup. Many of the associations between improvements

in psychological variables and cortisol levels and abdominal fat were observed only among intervention participants and were not found across groups or within the control group. This tendency may be due to greater changes and variability in the predictor variables as a result of the intervention. It also should be noted that participants were unblinded to the hypotheses of the study about stress and abdominal fat, which could affect behavior in both groups. In addition, the study relied on an indirect measure of visceral adiposity; future research could examine actual changes in visceral adiposity with imaging. Finally, participants were relatively healthy, premenopausal women who reported high levels of stress and emotional eating, and thus it is not clear if the results would generalize to other types of women, men, or individuals with type 2 diabetes or the Metabolic Syndrome.

In summary, this exploratory study shows promise for mindfulness training benefiting obese women at risk for the Metabolic Syndrome by improving patterns of overeating and decreasing the cortisol awakening response, which may contribute to reduced abdominal fat over time. Although the intervention was not effective in reducing abdominal adiposity or improving fat distribution across all participants, improvements were observed among those who increased in mindfulness and decreased in chronic stress, emotional eating, and CAR. We also observed a prevention of weight gain in the obese subgroup of participants. Future research could examine the effects of introducing mindfulness techniques after initial weight loss on long-term weight maintenance in an obese population, or whether these techniques facilitate initial weight loss attempts in combination with nutrition and exercise guidelines designed for weight loss. Integrating this program with active weight loss strategies may lead to targeted decreases in abdominal fat.

## Conflict of Interests

The authors declare no conflict of interests.

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