4-24-2020

Cardiovascular recovery from emotional stress: An operationalization of equanamity following mindfulness-based emotional stress

Emma McBride
Rowan University

CARDIOVASCULAR RECOVERY FROM EMOTIONAL STRESS: AN OPERATIONALIZATION OF EQUANIMITY FOLLOWING MINDFULNESS-BASED STRESS REDUCTION

by

Emma E. McBride

A Thesis

Submitted to the
Department of Psychology
College Science and Mathematics
In partial fulfillment of the requirement
For the degree of
Master of Arts in Clinical Psychology
at
Rowan University
April 10th, 2020

Thesis Advisor: Jeffrey Greeson, Ph.D

Dedication

I would like to dedicate this manuscript to all those seeking to transform suffering through awakening, so all of us.

Acknowledgment

I would like to express my appreciation to Professor Jeffrey Greeson for his guidance and help throughout this research. I will carry the skills and knowledge I have gained from our mentorship relationship into my future professional endeavors.

I would like to thank my husband for his unwavering love and support through this project. I would also like to thank my meditation teachers and community for their constant inspiration and perspective.

This project was supported by the National Center for Complementary and Integrative Health (NCCIH), grant #R00AT004945.

Abstract

Emma McBride
CARDIOVASCULAR RECOVERY FROM STRESS: AN OPERATIONALIZATION OF EQUANIMITY FOLLOWING MINDFULNESS-BASED STRESS REDUCTION
2019-2020
Jeffrey Greeson, Ph.D
Master of Arts in Clinical Psychology

Theoretical models of mindfulness suggest that meditation may improve health, in part, by regulating stress physiology, including faster recovery of heart rate (HR) and blood pressure (SBP/DBP) after emotional stress. Furthermore, improved cardiovascular recovery (CR) may be a marker of equanimity, defined as increased acceptance of and reduced reactivity to stress. No studies have tested this hypothesis, partly because methodology for assessing CR remains controversial. Using a novel operationalization of equanimity and several methods of measuring CR, this project investigated whether (1) equanimity is associated with improved CR, (2) Mindfulness-Based Stress Reduction (MBSR) is associated with improved CR, and (3) increased equanimity following MBSR partly explains improved CR. Using a pretest-posttest repeated measures design, 56 healthy adults completed MBSR bracketed by stress testing. HR, SBP and DBP recovery were calculated using simple change scores, residualized change scores, and percent recovery. GLMs showed (1) no association between equanimity and CR, (2) improved BP recovery following MBSR, but only when CR was measured using simple change scores, and (3) that equanimity explained a small amount of the variance in BP recovery following MBSR but was not a statistically significant predictor. Results have important implications for statistical conclusions validity in stress recovery research and ultimately contradict theoretical models predicting faster physiological recovery from emotional stress following mindfulness training.

Table of Contents

Table of Contents (Continued)

List of Figures

List of Tables

Chapter 1

Introduction

Mindfulness-based interventions (MBIs) have been the focus of research investigating the nature of contemplative practices and their potential as stress-reduction interventions. In the ongoing effort to better understand how MBIs promote well-being, certain outcomes and mechanisms of mindfulness have received more attention than others (for review see Keng et al., 2011). This paper will attempt to shed light on two infrequently addressed constructs in mindfulness research: equanimity and cardiovascular recovery from stress. In so doing, we hope to encourage further research which uses rigorous statistical methods to integrate a Buddhist framework for understanding mindfulness with a focus on clinically-relevant biological outcomes.

Equanimity

Mindfulness is frequently defined as the capacity to pay attention to the present moment with intention and an attitude of non-judgment (Bishop et al., 2004; Kabat-Zinn, 1994). However, definitions of the construct vary and, over the last decade, mindfulness has been increasingly defined in terms of its components. There is the well-known two component model described above, which includes (1) self-regulated attention and (2) an attitude of openness and acceptance (Bishop et al., 2004). In addition, some have proposed three-component (Shapiro, Carlson, Astin, & Freedman, 2006) and even five-component (Baer et al., 2008) models of mindfulness. Notably, each of these models includes a common construct: an attitude of nonjudgmental, open acceptance.

This attitude of receptive and inquisitive acceptance has much in common with the traditional Buddhist concept of equanimity, which is recently receiving increased

attention in mindfulness research. In 2015, Gaelle Desbordes and colleagues published a call to operationalize equanimity as a construct distinct from mindfulness and worthy of scientific study, both as a potential outcome of mindfulness training and as a mechanism of action underlying mindfulness-based interventions. The hypotheses in this paper linking equanimity to cardiovascular stress recovery draw heavily from the theory proposed in this article.

Drawing from a Buddhist framework, the authors understand equanimity as a dispositional tendency toward even-mindedness, wherein mental and external events are received openly, without the natural inclination to prolong the pleasant sensations and avoid the unpleasant. Of note, the capacity to respond to a situation with equanimity is dependent on mindfulness – we cannot respond with unbiased openness unless we are *aware* of what is happening in the present moment. Thus, equanimity may develop over the course of meditation practice only *after* the practitioner has become adequately aware of their own thoughts and behavior patterns. In addition, Desbordes and colleagues propose that equanimity significantly overlaps with constructs more familiar to Western psychological theory, such as acceptance, emotion regulation, non-judgment, non-reactivity, decentering, and metacognition.

In the context of unpleasant events, such as a laboratory stress test, Desbordes suggests that equanimity is experienced as an internal process of decentering from and accepting the experience, without ruminating on it or avoiding the accompanying sensations. Individuals high in equanimity would be expected to maintain an unbiased, receptive, curious awareness when faced with an emotionally challenging situation. Notably, this is not indifference or apathy, as the experience is still received in awareness

and allowed to exist fully, just as it is. Instead, a state of equanimity is one in which motivation is not affected by whether an experience is pleasant or unpleasant, but instead by one's values, long-term goals, and aspirations (Hadash, Segev, Tanay, Goldstein, & Bernstein, 2016). Therefore, instead of a buffered response to stress, the authors proposed that the primary signature of equanimity is temporal: a more rapid return to a baseline state of calm receptivity. It is worth noting that other theoretical models of mindfulness, namely Lindsay & Creswell's Monitor and Acceptance Theory (MAT), mirror the central role of equanimity and related constructs (i.e. acceptance) but suggest that acceptance will be associated with *decreased* physiological reactivity to stress (Lindsay & Creswell, 2019). Differences between these theoretical models of mindfulness will be addressed in more detail in the discussion section of this paper. Lastly, Desbordes and colleagues suggest that *physiological* markers of stress recovery may be more useful markers of increased equanimity, given previously reported difficulties with developing self-report psychometrics in meditation research (Grossman, 2008).

In this context, we would expect (1) increased equanimity (understood here as a *disposition* toward a certain style of emotional responding) to develop following a mindfulness-based intervention and (2) increased equanimity to be evidenced by a shorter time course for the physiological return to baseline following stress, *without* an accompanying decrease (or increase) in reactivity (see Figure 1). In the laboratory, this hypothesis can be tested using a number of physiological indices that are responsive to stress and indicative of autonomic function, such as heart rate and blood pressure. Indeed, research is beginning to show some evidence of faster autonomic recovery following stress in long-term meditators *and* that the relationship between meditation practice and

recovery time may be mediated by acceptance (Gamaiunova, Brandt, Bondolfi, & Kliegel, 2019), one of the key components of equanimity. This research will be discussed in more detail following a general review of stress recovery research in the following section.

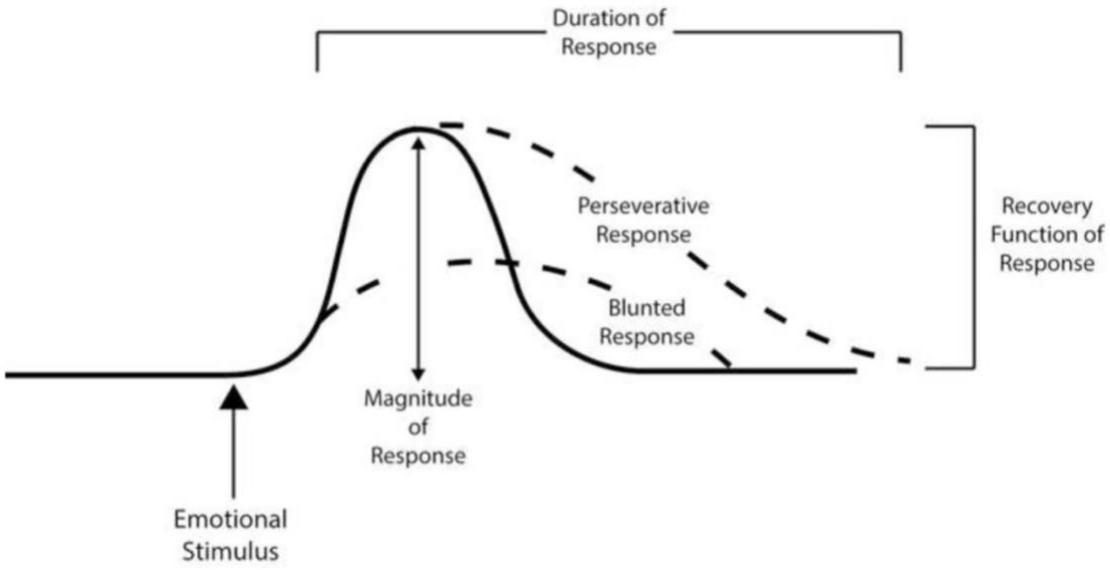

Figure 1. Generic representation of a typical physiological response to an emotional stimulus. The figure depicts different stress responses of different magnitudes and time courses. A more equanimous response is hypothesized as a rapid return to baseline and unchanged response magnitude (solid line). It is neither perseverative nor blunted (dotted lines). Reprinted from *Moving Beyond Mindfulness – Defining Equanimity as an Outcome Measure in Meditation and Contemplative Research*, by Desbordes et al., 2015, retrieved from https://link.springer.com/article/10.1007/s12671-013-0269-8.

Before proceeding with novel operationalizations of equanimity, it is important to assess the worth of pursuing research on an infrequently studied construct which already conceptually overlaps with several more well-researched constructs. A thoughtful reader may rightly ask why mindfulness researchers cannot simply continue to study mechanisms of mindfulness using familiar understandings of acceptance, decentering,

rumination, non-reactivity, and metacognition, rather than adding a similar, but novel, construct. The response is threefold:

Cultural competence. Modern Western instruction of meditation techniques is directly inspired by the Buddhist tradition. Despite this direct link between a specific sociocultural context and modes of contemplative practice being taught in the West, scientific understandings of mindfulness have not always aligned with Buddhist tradition (Anālayo, 2019; Dreyfus, 2011; Giles, 2019). There is significant semantic ambiguity around the term "mindfulness" (Van Dam et al., 2018) and some have argued this could be addressed by a more historically-rooted understanding of contemplative practice (Anālayo, 2019). Buddhist understandings of mindfulness and related concepts are by no means homogenous, but they do carry the accumulated knowledge of more than two thousand years of contemplative practice. If psychologists wish to better understand mindfulness, we would do well to leverage this sociocultural background not only in the way we teach contemplative practice, but in the constructs we use to study it. For example, identifying equanimity as a factor separate from mindfulness may actually help us build a more cohesive and complete understanding of related concepts (i.e. "distancing", "accepting", "non-judging") since these may be understood as lower-order factors of equanimity. Indeed, there is some indication that equanimity may be understood as a higher order factor reflecting the concept itself and two lower order factors: an attitude of acceptance and reduced reactivity to unpleasant hedonic tone (Hadash et al., 2016). This factor analysis, rooted in the related "Decoupling Model of Equanimity", was directly inspired by an integration of Buddhist thought and psychological science. In addition, an understanding of mindfulness that reflects the

Buddhist origins of meditation practice in the West is arguably *more* informative to clinicians seeking to teach and use contemplative techniques with clients (Desbordes et al., 2015).

Objective outcomes and falsifiable hypotheses. The present analysis was inspired by the theoretical justification for two specific, falsifiable hypotheses: (1) equanimity will be evidenced by a faster physiological return to a baseline state of calm receptivity following stress and (2) equanimity will develop gradually following sustained mindfulness practice (Desbordes et al., 2015). These are clear, testable, and directly informed by both Western psychological theory and Buddhist scholarship. There is little downside to investigating clinically and scientifically relevant hypotheses so cogently expressed.

Potential mechanism of mindfulness. Several potential mediators of the beneficial effects of mindfulness practice have been proposed, including most of the previously mentioned constructs related to equanimity (for review see Gu et al., 2015). There is at least some support for increased acceptance, metacognitive awareness, exposure, rumination, worry, emotional reactivity and behavioral control as potential mediators between MBIs and beneficial outcomes (Gu et al., 2015; Keng et al., 2011). Furthermore, Lindsay & Creswell's 2017 Monitor and Acceptance Theory (MAT) proposes that acceptance, defined as "a mental attitude of nonjudgment, openness and receptivity, and equanimity toward internal and external experiences", *must* be present for meditation practice to specifically improve stress-related outcomes like reactivity and recovery. Equanimity, therefore, can be thought of not only as a worthwhile outcome of contemplative practice in and of itself, but also as a historically and culturally relevant

construct with potential to partly explain the relationship between mindfulness practice and stress-related health outcomes. Although confusion about overlapping constructs remains (Creswell, Pacilio, Lindsay, & Brown, 2014; Van Dam et al., 2018), it is certainly beneficial to capitalize on recent interest in equanimity, build a more culturally competent understanding of mindfulness, and produce experimental research testing recent theoretical models of equanimity as a potential mechanism.

Cardiovascular Recovery from Stress

Just as equanimity has been an infrequently addressed concept in mindfulness research, psychophysiological research on stress responsivity has suffered from a lack of emphasis on recovery from a psychological stressor. This overreliance on models of stress reactivity rather than recovery is evident in acute stress responsivity research focusing on the hypothalamic-pituitary-adrenal (HPA) axis, the autonomic system, and the cardiovascular system. Due to the present study's focus on heart rate and blood pressure this review will concentrate on the cardiovascular system, but readers seeking a comprehensive review of systems are directed to Chida, Y. and Hamer, M. (2008).

The cardiovascular system is arguably the most frequently studied allostatic system and is typically investigated via stress-induced change in heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) (McEwen, 1998). Hyper-reactivity to laboratory stressors has been repeatedly linked with future cardiovascular risk status, including elevated blood pressure, clinical hypertension, left ventricular mass, atherosclerosis, and heart attack (for review see Chida & Steptoe, 2010). Chronic psychosocial factors such as hostility, aggression, and Type-A behavior have been repeatedly associated with *increased* cardiovascular reactivity (and with cardiovascular

disease in turn), whereas other negative emotional states such as anxiety, neuroticism, and negative affect have been associated with *reduced* cardiovascular reactivity (Y. Chida & Hamer, 2008).

Despite the clinically relevant information gleaned from a focus on cardiovascular reactivity, multiple researchers have proposed that it is equally and possibly more important to focus on cardiovascular recovery (Linden, Earle, Gerin, & Christenfeld, 1997; McEwen, 1998; Schuler & O'Brien, 1997; Stewart & France, 2001). When an organism responds to threat that stress response is twofold; we must activate the cardiovascular system to prepare for threat, then when the threat has passed we must be able to turn the response off (McEwen, 1998). When cardiovascular recovery is chronically inefficient allostatic load on the organism is increased over time, which in turn constitutes a risk factor for cardiovascular disease. Furthermore, there is some indication that prolonged cardiovascular recovery may be *more* strongly associated with end-organ damage than heightened cardiovascular reactivity (Pieper & Brosschot, 2005; Stewart & France, 2001; Trivedi, Sherwood, Strauman, & Blumenthal, 2008). In fact, inefficient cardiovascular recovery from stress has been linked with adverse health outcomes such as increased waist-hip ratio at 3-year follow-up (Steptoe & Wardle, 2005), hypertension status (Schuler & O'Brien, 1997; Stewart & France, 2001; Trivedi et al., 2008), and higher carotid atherosclerosis at 2-year follow-up (Puttonen et al., 2009). Like abnormal stress reactivity, prolonged stress recovery has also been associated with psychosocial factors. Specifically, general stress, anxiety, neuroticism, rumination, depressive symptoms, and negative affect have been linked with prolonged cardiovascular recovery from a laboratory-based stressor (Y. Chida & Hamer, 2008;

Gordon, Ditto, & D'Antono, 2012; Willmann, Langlet, Hainaut, & Bolmont, 2012). In the context of an anger-induction task, such as the anger recall task used in this study, rumination and dispositional hostility have been linked to prolonged cardiac recovery and post-task distraction is thought to improve cardiac recovery (Neumann, Waldstein, Sellers, Thayer, & Sorkin, 2004; Routledge, McFetridge-Durdle, Macdonald, Breau, & Campbell, 2015).

Overall, increased latency to cardiac recovery is likely predictive of future cardiovascular disease risk but remains somewhat neglected in stress responsivity research. Relatedly, psychosocial factors are potential mediators of the relationship between recovery from negative emotional states and cardiovascular disease risk, but research on these individual differences is still in its infancy. Lastly, interventions with the potential to improve cardiovascular recovery from stress, MBIs among them, may be promising ways to reduce cardiovascular disease risk, but investigation into these interventions using cardiac recovery as an outcome measure remains rare.

Calculating Latency to Cardiovascular Recovery

Despite the potential importance of cardiovascular recovery to models of stress-related disease, recovery has been infrequently studied, in part, because there is little consensus on the best practices for doing so. Apart from statistical methodology, addressed below, the stressor protocol itself must be designed to adequately capture the complete recovery curve of the system studied. For example, tasks which provoke anger, like the anger-induction used in the present study, typically induce a blood pressure response that persists for over 10 minutes, beyond the recovery period of many stress protocols (Linden et al., 1997). This is unfortunate because studying cardiovascular

recovery in the context of anger-provocation tasks is particularly useful given the substantial body of literature linking chronic anger and hostility to cardiovascular disease (Chida & Steptoe, 2009), but only if the protocol allows subjects' heart rate and blood pressure adequate time to return to near baseline levels (Linden et al., 1997).

Assuming the stress responsivity protocol captures the full recovery curve following an emotional stressor, the question of how to analyze that curve becomes paramount. Analyses of cardiovascular reactivity to stress are typically done using simple change scores in which the subject's average baseline level is subtracted from their average stress level. Similarly, researchers studying cardiovascular recovery from stress have traditionally calculated recovery change scores by subtracting baseline levels *or* stress levels from recovery levels (Neumann et al., 2004). However, multiple stress researchers have explored the disadvantages of change scores, especially in the context of recovery (Christenfeld, Glynn, & Gerin, 2000; Linden et al., 1997; Llabre, Spitzer, Siegel, Saab, & Schneiderman, 2004). In short, the reliability of a change score depends on the reliability of each of its components, in this case baseline, the stressor, and recovery, as well as on the correlation between those components and the resulting change score. For example, if baseline blood pressure (BP) is positively correlated with simple recovery change scores the change score is then more reflective of between-subjects differences in baseline BP than of recovery proper. This situation, in which participants who start lower often recover "better", is quite common in stress recovery research (Linden et al., 1997). A multigroup design also complicates this methodology: if groups are different from one another at baseline and/or if the correlation between baseline and recovery varies by group, change scores will be confounded by group

differences at *baseline* and thus less reflective of group differences in recovery proper. Overall, in a multigroup design, the utility of change scores depends on (1) baseline values being uncorrelated with change during stress and recovery, (2) this being the case for each group (i.e. pre- and post-intervention, in a repeated measures design), and (3) lack of significant baseline difference between each group. In a recovery design this situation is quite difficult to come by (Linden et al., 1997). Several researchers (Hutchinson & Ruiz, 2011; Stewart, Janicki, & Kamarck, 2006) have addressed these assumptions by using residualized change scores, which effectively adjust for the impact of baseline and reactivity on recovery for each participant (Linden et al., 1997). Percent recovery ([stress-recovery]/[stress-baseline]*100) is another option which controls for the fact that degree of reactivity is highly likely to influence both change from baseline and change from stress levels (Linden et al., 1997).

Of note, the strategies mentioned above (simple change scores, residualized change scores, and percent recovery) do not solve every methodological issue in calculating a recovery outcome variable. For example, in all these methods researchers typically average across multiple measures at baseline and during the stressor in order to increase the reliability of the outcome variable. Unfortunately, this strategy is less useful for any type of recovery change score because there is no task-based way to decide where a recovery period "ends" (Christenfeld et al., 2000). The slope of the recovery curve is also lost in any method wherein a researcher averages multiple measures to describe a single focus area. More sophisticated curve-fitting techniques are increasingly being used to solve these problems (Christenfeld et al., 2000; Llabre et al., 2004). These methods use all available data points and can describe the recovery curve using a mathematical

equation with multiple parameters. The estimates derived are also independent of baseline/stress levels and remain reliable even if a subject does not return to a prestress level during the recovery period (Linden et al., 1997).

Although this methodology is likely superior to both residualized change scores and percent recovery, multilevel or latent growth curve modelling can be complex and difficult to execute correctly. In addition, contemporary stress recovery research still remains relatively reliant on change scores. In most cases, researchers calculate the difference between the recovery period and the baseline period, many using simple change scores (Gordon et al., 2012; Keogh & Creaven, 2017; Routledge et al., 2015; Steffen & Larson, 2015; Trivedi et al., 2008) and others calculating residualized change scores (Hutchinson & Ruiz, 2011; Stewart et al., 2006). This continued use of more simple data analysis methods is partly due to the reliance of curve-fitting techniques on statistical software that may be novel to many researchers, and partly due to the frequent assessments of BP or HR required across the reactivity-recovery curve, which are not always available. While acknowledging the likely superiority of curve-fitting techniques, the present analysis will focus on more accessible methodology because of (1) the introductory nature of this thesis project and (2) the continued use of simple change scores and need for direct comparison between these and more reliable equivalents.

Mindfulness, Equanimity, and Cardiovascular Recovery

Like stress responsivity research, research investigating mindfulness and psychological stress has overwhelmingly focused on the potential of mindfulness-based interventions (MBIs) to attenuate physiologic *reactivity*, with somewhat inconsistent results. Several studies have examined MBIs and physiologic reactivity to a laboratory

stressor, with some reporting buffered reactivity (Arch et al., 2014; Brown, Weinstein, & Creswell, 2012; Hoge et al., 2013; Nyklicek, Mommersteeg, Van Beugen, Ramakers, & Van Boxtel, 2013; Rosenkranz et al., 2016; Skinner et al., 2008; Steffen & Larson, 2015) but others reporting no association between MBIs and stress-related physiologic reactivity (Creswell et al., 2014; Gex-Fabry et al., 2012; Nyklicek et al., 2013; Pace et al., 2009). There is even some indication that meditation training may be associated with *increased* physiologic (salivary cortisol) reactivity to stress (Creswell et al., 2014). As discussed above, although increased reactivity to psychological stress has been associated with negative health outcomes (Cohen et al., 2002; Matthews, Woodall, & Allen, 1993), it is important to note that prolonged recovery represents a separate and, arguably, equally important mechanism underlying stress-related disease (Epel, McEwen, & Ickovics, 1998; Mezzacappa, Kelsey, Katkin, & Sloan, 2001).

Within mindfulness research, a focus on stress recovery is still nascent. There is some indication that recovery from emotional tasks may be accelerated when there is an opportunity to cope with the source of the distress (Neumann et al., 2004; Pieper & Brosschot, 2005; Routledge et al., 2015), with mindfulness representing one such opportunity. Mindfulness may also provide a means of coping with perseverative cognition such as worry, rumination and negative emotional states, all of which have been shown to prolong cardiovascular recovery from emotional induction stressors (Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; Key, Campbell, Bacon, & Gerin, 2008; Pieper & Brosschot, 2005; Brosschot et al., 2006). In this vein, Gamaiunova and colleagues (2019) recently published an intriguing study demonstrating that long-term meditators had faster cortisol recovery but unchanged heart rate (HR) and heart rate

variability (HRV) recovery to the Trier Social Stress Test (TSST). Notably, groups did not differ in stress reactivity on any physiological variable. The researchers calculated simple change scores for recovery but controlled for baseline and stress levels in an ANCOVA. This method essentially adjusts for baseline and reactivity based on the regression slope of the whole group, rather than at the level of the individual, as in residualized change scores. Although this is certainly superior to simple change scores, this may be less reliable than residualized change scores in a multigroup design if the regression slopes for all groups are not parallel (Linden et al., 1997). Methodology aside, although this study did not find group differences in *cardiovascular* recovery, they do note that the inclusion of a non-homogenous group of meditators may be masking the effects of meditation training on cardiovascular functioning. The group also conducted a mediation analysis supporting the role of acceptance as a mediator of the relationship between long-term meditation practice and improved cortisol recovery from stress. This finding is particularly notable in the context of the present analysis due to the crossover between acceptance and equanimity.

Studies on mindfulness and cardiovascular recovery have also been conducted with meditation naive participants. Grant et al., 2013 found *increased* latency to blood pressure (BP) recovery following a physical stressor (cold pressor task) in a population of meditation naïve college students who experienced a brief mindfulness induction. Reactivity was again unchanged, as was HR recovery. These unexpected results are understood by the researchers in light of the effect of meditation on physical discomfort in beginning practitioners: practicing mindful breathing for the first time may be (1) stressful in and of itself and (2) may draw participant's attention toward physical

sensations they were otherwise not attending to, thereby increasing and/or prolonging physiologic arousal. Similarly, Steffen et al. (2015) found no effect of a brief mindfulness induction on cardiovascular recovery in a meditation-naïve population. These studies both highlight the importance of using a homogenous group of individuals *with* some significant meditation experience in order to more rigorously assess mindfulness and physiologic recovery. In addition, and in light of the findings from Gamaiunova et al. (2019), perhaps a brief and specific mindfulness induction focusing on acceptance and equanimity rather than mindful awareness would be a more effective way to quickly improve recovery in meditation-naïve populations.

Lastly, Crosswell et al. (2017) recently examined the effect of a 6-week mindfulness-based intervention on cardiovascular recovery in younger female breast cancer survivors. This study is comparable to the present analysis and is therefore reviewed in some detail here. Using personal negative emotion induction task, the authors found that women in the intervention group experienced *more* sadness and anger than controls, as well as a more efficient diastolic blood pressure (DBP) recovery from the stressor. HR recovery was not different between groups nor was reactivity on any cardiovascular variable. Whereas the three studies cited above used either simple change scores (Grant et al., 2013; Steffen & Larson, 2015) or ANCOVA controlling for baseline and stress (Gamaiunova et al., 2019), in this study recovery was analyzed using multilevel mixed-effects modeling. This method is used to examine data with multiple time points nested within individuals and thereby allows for the inclusion of all available data points. In so doing, this technique allows for a regression equation at the level of the individual and the parameters in that equation can be used to test for individual

differences in patterns of recovery over time (Hoffman & Rovine, 2007). They also examined the recovery trajectory using three different change periods: (1) the overall recovery pattern (change from during stressor to the last 6 minutes of recovery), (2) the beginning of the recovery period (change from during the stressor to the first 3 minutes of recovery), and (3) the end of the recovery period (change from immediately after the stressor to the end of the recovery period). Results were specific to initial BP recovery from stress, indicating that methodology which enables researchers to examine multiple phases of the recovery curve may more effectively capture group differences. The authors propose that mindfulness training may help individuals develop new emotion regulation strategies in which they learn to "non-judgmentally observe and accept, rather than react to, their thoughts and feelings" (Crosswell et al., 2017, pg. 79). Although not directly referenced in the article, this interpretation is highly similar to the understanding of equanimity put forward by Desbordes et al. (2015). The authors suggest that future research continue to interrogate the relevance of improved cardiovascular recovery as an outcome of mindfulness-based interventions and begin to assess potential mediators of the effect of mindfulness training on recovery.

We propose that the above review of the literature suggests an opportunity to combine two under-studied constructs in mindfulness and stress physiology research: equanimity and cardiovascular recovery from stress. The purpose of the current study is to use a pretest-posttest repeated measures design to examine whether a Mindfulness-Based Stress Reduction intervention improves cardiovascular recovery from induced negative affect. In addition, we are specifically interested in the role of self-reported equanimity since this construct, whether trait-like or learned, may be one factor which

protects mindful individuals from the negative or perseverative cognitive patterns that prolong cardiovascular recovery from stress (Farb, Anderson, & Segal, 2012).

Four hypotheses were tested in this study, listed below. All hypotheses were examined using three methods of analyzing recovery: simple change scores, residualized change scores, and percent recovery, with the hope that comparing these methods will contribute to solidifying best practices in stress recovery research.

1. High self-reported equanimity will be associated with more efficient heart rate (HR), systolic blood pressure (SBP) and diastolic blood pressure (DBP) recovery from an anger recall stressor, both before and after MBSR.
2. Self-reported equanimity will *not* be associated with altered cardiovascular (HR, SBP, DBP) reactivity to the stressor, either before or after MBSR.
3. Completion of MBSR will be associated with more efficient cardiovascular (HR, SBP, DBP) recovery from the anger recall stressor.
4. Self-reported equanimity will party explain the relationship between Time (pre-/post-MBSR) and cardiovascular (HR, SBP, DBP) recovery.

Chapter 2

Methods

Study Design

Data are from a study of 64 medically healthy adults who participated in a larger open trial of an 8-week Mindfulness-Based Stress Reduction (MBSR) program investigating biological signatures of mindfulness training. This study used a pretest-posttest repeated measures design in which subjects served as their own controls. Each participant completed questionnaires assessing self-reported equanimity as well as an in-person stress-testing protocol before and after the intervention. The MBSR course as well as pre- and post-MBSR laboratory sessions were held at the Duke Clinical Research Unit at Duke University in Durham, North Carolina.

Participants

Participants were eligible for the study if they were between the ages of 18 and 65, medically healthy, and able to attend one of the MBSR courses offered. Fifty-six participants completed the post-MBSR session, and therefore pre-post analyses were performed on the remaining sample (n = 64 enrolled, 56 completed, age 22-64, 67% Female, 84% White).

Intervention

Mindfulness training was delivered via Mindfulness-Based Stress Reduction (MBSR). MBSR is a standardized, secular, 8-week program that provides intensive training in mindfulness meditation. The program consists of eight 2.5-hour classes held weekly and one full-day silent meditation “retreat”. Participants are also asked to complete 45 minutes of daily meditation practice independently. The course teaches

participants the core principles and practices of mindfulness: non-judgmental present-focused attention, emotion regulation via awareness and non-reactivity, compassion and kindness, and behavioral self-regulation. Instructors encourage participants to purposefully experience and observe the thoughts, emotions, and sensations which arise without judging them. Participants are also taught more advanced meditation practices in later sessions, including choiceless awareness (meta-cognitive awareness), loving-kindness (compassion and kindness toward self and others), and mindful interpersonal communication (speaking authentically and listening deeply without reacting). Reviews and meta-analyses have shown significant positive effects of MBSR training on the mind-body system, as well as increases in self-reported mindfulness following course completion (Grossman, Niemann, Schmidt, & Walach, 2004; Ludwig & Kabat-Zinn, 2008).

Procedure for Pre- and Post-MBSR Assessment

Two laboratory sessions, one pre-MBSR and one post-MBSR, bracketed the 8-week MBSR course. After providing written informed consent at the first laboratory session, participants completed questionnaires assessing self-reported equanimity. Participants were then fitted with an automated vital signs monitor and a manually activated blood pressure monitor (Avobus GE Dinamap ProCare 400), seated in a comfortable recliner, and instructed to rest for 30-minutes to acclimatize to the testing environment and provide accurate baseline measurements. Heart rate (HR) and blood pressure (BP) were recorded every minute through a 10-minute baseline period. Participants then completed a 5-minute stressful laboratory task [Anger Recall Task; (Suarez, Saab, Llabre, Kuhn, & Zimmerman, 2004)] in which they were instructed to

think and speak about a situation that made them angry at the time and continues to make them angry. To ensure the chosen topic was capable of inducing a sufficient emotional response, research assistants asked participants to rate their stress level during the experience from 1 to 10. If they reported a score less than 7 research assistants probed for a different, more stressful event. Participants were then asked to spend one minute visualizing the event and four minutes speaking about the feelings, thoughts, and sensations they felt during the event. If participants had trouble describing the event research assistants prompted participants with follow-up questions (e.g. "What was the most stressful part of that event?", "How did your body feel at the time?", "What were you thinking when that happened?"). HR and BP were recorded every minute during the task. Following the anger recall task, HR and BP were recorded every minute for the first 15 minutes of a 30-minute recovery period, after which measurements were taken every 2 minutes. During this period participants were instructed to sit quietly. Of note, at the post-MBSR lab visits, half of participants were assigned to practice 15 minutes of meditation during the first half of the recovery period. Since post-stress meditation practice is not directly related to the present examination of equanimity, we first analyzed whether in-lab meditation practice resulted in accelerated recovery. Results showed no difference between those who meditated and those who did not, which informed our decision to pool recovery data across groups. Stress testing procedures were identical pre- and post-MBSR, with the exemption of informed consent at the initial session and debrief at the concluding session. For the anger recall task, participants supplied two different life incidents, each of which were rated for anger (1-10) and stress (1-10), and then randomly assigned across pre- and post-intervention lab visits.

Self-Report Measures

Several researchers have proposed self-report scales that, to some degree, assess the construct of equanimity as described here by assessing resilience to stress, acceptance, and/or emotion regulation in difficult circumstances (Kraus & Sears, 2009; Lundman, Strandberg, Eisemann, Gustafson, & Brulin, 2007; Mack et al., 2008). However, these scales do not share the theoretical framework proposed above and vary widely in their conceptualization of equanimity. To examine equanimity in the absence of validated self-report measures of the construct we drew on a recent factor analysis showing that equanimity may entail one higher order factor reflecting the concept itself and two lower order factors representing its two manifestations: (1) an attitude of acceptance toward experiences regardless of whether they are pleasant or unpleasant and (2) reduced reactivity to unpleasant experiences (Hadash et al., 2016). In light of this research, this study will combine three facets of the Five Facet Mindfulness Questionnaire to yield a measure of trait equanimity: observing, non-judging and non-reactivity. The items included in the non-judging facet (ex: "I criticize myself for having irrational or inappropriate emotions") closely parallel the construct of acceptance proposed by Hadash et al. (2016), as do those included in the non-reactivity facet. In addition, the observing facet is included to account for the baseline level of awareness needed to support the development of equanimity (Desbordes et al., 2015) and a "Monitor + Acceptance" understanding of mechanisms of mindfulness training (Lindsay & Creswell, 2017). To support the validity of this measure of equanimity we will also examine whether scores on this measure (FFMQ Observing + FFMQ Non-Judging +

FFMQ Non-Reactivity) are significantly different before and after MBSR, since the intervention is likely to reliably elicit an increase in equanimity (Desbordes et al., 2015).

Statistical Analyses

All hypotheses were addressed by first assessing psychometrics, univariate distributions, graphics, and assumptions. Results are interpreted using parameter estimates and effect sizes in addition to p-values. In general, statistical analyses were conducted with an emphasis on (1) plotting raw data, (2) using sensitivity analyses when appropriate (D. A. Fife, in press). Manipulation checks and analyses for hypotheses I-IV were planned a priori and are roughly confirmatory. Multiple imputation was used for hypotheses III and IV to account for attrition at Time 2 (Enders, 2017). Sensitivity analysis refers here to the use of robust regression was used when assumptions of normality or homoskedasticity were violated (Erceg-Hurn & Mirosevich, 2008). The robust and general linear models were then compared to see whether the robust model suggests a difference in interpretation. R code for sensitivity analyses can be found in Appendix B. Results of robust or imputed models are reported only when relevant for ease of interpretation, but interested readers are directed to Appendix B and the applicable dataset is available upon request. Post-hoc exploratory analyses are noted explicitly and were performed where appropriate using graphics and measures of effect size (Fife & Rodgers, 2019).

Cardiovascular recovery was examined using simple change scores, residualized change scores, and percent recovery. Reactivity (hypothesis II) was assessed using only residualized change scores.

Baseline, anger recall, and recovery levels were calculated as the mean of the readings obtained during each period. The mean of the baseline period was calculated as the mean of the last 5 minutes of baseline to account for participants who were still acclimating during the first 5 minutes. The mean of the recovery period was calculated as the mean of the first 10 minutes of recovery. This decision was made following review of typical recovery period analyses of cardiovascular variables following an emotion induction (Crosswell et al., 2017; Gerin et al., 2006; Linden et al., 1997). It is meant to capture the full curve for most participants but to avoid capturing rising HR/BP later in the recovery period, which occurs for a minority of participants.

Simple change scores were calculated by subtracting mean baseline HR/BP from mean recovery HR/BP. A score ≤ 0 indicates complete recovery, a higher score indicates a less complete return to pre-stress HR/BP. Residualized change scores for reactivity were calculated by regressing the mean anger recall level on the mean baseline level to control for the potential influence of baseline on anger recall level. Similarly, the mean recovery level was regressed on the mean baseline level *and* the mean anger recall level to calculate residualized change scores for recovery. Like simple change scores, a residualized change score ≤ 0 indicates complete or more efficient cardiovascular recovery. Percent recovery was calculated using the following formula:

$$\%Recovery = \frac{(mStress - mRecovery)}{mStress - mBaseline} \times 100$$

In contrast to change scores, percent recovery scores ≥ 100 indicate complete or more efficient cardiovascular recovery.

For hypotheses I-III, general linear models (GLMs) were used to predict recovery or reactivity from dispositional equanimity or Time (pre-MBSR vs. post-MBSR). Time

was dummy coded as (0,1). Separate regression analyses were conducted for each cardiovascular measure (HR, SBP, DBP). Assumptions of normality, homoskedasticity, and linearity were checked prior to each analysis. Robust models were checked for agreement with GLMs in cases where residuals for the outcome variable were not normally distributed or where there were notable outliers. Multiple imputation was used to account for attrition at Time 2. Each nested model comparison controlled for traditional predictors of HR and BP: Body Mass Index (BMI), Age, and Gender. For each analysis, a reduced model was constructed predicting the variable of interest from control variables. A corresponding full model was then built adding the predictor of interest. The full and reduced models were compared, and results were interpreted using p-values, semi-partial R_2, Akaike Information Criterion (AIC), Bayesian Information Criterion (BIC), and Bayes factors (BF). Support for the full model was inferred given (1) a smaller AIC and BIC relative to the reduced model, (2) a larger BF relative to the reduced model *and* BF>10, and (3) a statistically significant ($p<.05$) ΔR_2. Standardized β was used as a measure of effect size for dispositional equanimity and Time (pre- vs. post-MBSR) effects, respectively. Planned model comparisons in regression notation are provided in Appendix A.

For hypothesis IV, GLMs were constructed only for outcome variables which were significantly associated with Time (pre- vs. post-MBSR) in hypothesis III. To test whether dispositional equanimity party explained this relationship, reduced models predicting cardiovascular recovery from control variables and Time were compared with GLMs including dispositional equanimity (see Appendix A). Evidence for partial

mediation was assessed by comparing estimates (standardized β, p-value, ΔR^2) for Time before and after adding equanimity to the model.

Post-hoc exploratory analyses were conducted to explore varying results across simple change scores, residualized change scores, and percent recovery. All analyses were conducted using R Studio and SPSS. Access to R code for all main analyses can be found in Appendix B and the datasets used in this analysis are available upon request.

Chapter 3

Results

Sample Characteristics

64 participants ages 22-64 were enrolled in the study and completed pre-MBSR self-report measures and laboratory stress testing. 56 participants completed the post-MBSR measures and stress testing, yielding a follow-up rate of 88% at the primary endpoint. Participants were majority female and Caucasian. Demographic characteristics are described in detail in Table 1. Chi-square and independent samples t-tests showed no significant differences between those who completed MBSR and those who dropped-out.

Among the 64 participants who were enrolled in MBSR, the average total number of minutes of formal mindfulness practice during the 8-week program was 2,205 (range, 475-4820 minutes).

Table 1

Demographic Characteristics of Study Participants

Characteristic	Enrolled (n=64)	Completed (n=56)
Age: Mean (range)	39.7 (22-64)	39.7 (23-64)
Sex, % Female	67.2	69.6
Race, % White	84.4	83.9
Ethnicity, % Non-Hispanic	90.6	89.3
Married, %	54.7	57.1
Income >$100K, %	46.9	46.4
Employed, % full-time	82.8	82.1
Education, % with graduate degree	50.0	50.0
Religious Affiliation, %		
Christian	37.5	39.3
Buddhist	1.6	1.8
Other	17.2	16.1
None	43.8	42.9
Prior Meditation Experience, % Yes	15.6	17.0
BMI: Mean (range)	23.2 (19-29)	23.2 (19-29)

Note. There were no significant differences on demographic variables between those who completed the study and those who did not.

Stress Manipulation

The Anger Recall Task successfully elicited significant increases in heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) both before MBSR (HR: $t = -10.885$, $p<.001$, $d = .93$; SBP: $t = -17.076$, $p<.001$, $d = 1.27$; DBP: $t = -19.686$, $p<.001$, $d = 1.28$) and after MBSR (HR: $t = -8.753$, $p<.001$, $d = .86$; SBP: $t = -13.598$, $p<.001$, $d = 1.22$; DBP: $t = -11.562$, $p<.001$, $d = 1.34$). Significant decreases over the recovery period were also observed both before MBSR (HR: $t = 12.260$, $p<.001$, $d = 1.03$; SBP: $t = 12.197$, $p<.001$, $d = .87$; DBP: $t = 14.847$, $p<.001$, $d = 1.11$) and after MBSR (HR: $t = 11.211$, $p<.001$, $d = .97$; SBP: $t = 11.112$, $p<.001$, $d = .93$; DBP: $t = 13.354$, $p<.001$, $d = 1.32$) (See Figure 2a, 2b, and 2c). Table 2 presents unadjusted mean

values for HR, SBP, and DBP in each focus area (baseline, stress, and recovery). Higher values are indicative of increased cardiovascular activation

Table 2

Mean Heart Rate and Blood Pressure by Focus Area

	Unadjusted Means (SD)					
	Baseline		Stress		Recovery	
	Pre-MBSR	Post-MBSR	Pre-MBSR	Post-MBSR	Pre-MBSR	Post-MBSR
HR	61.5 (9.1)	61.3 (8.2)	70.8 (11.0)	69.9 (11.5)	60.6 (8.6)	60.3 (8.1)
SBP	137.1 (13.0)	133.3 (12.6)	155.1 (15.2)	149.3 (13.7)	143.1 (12.5)	137.1 (12.8)
DBP	72.8 (8.2)	71.9 (6.1)	83.5 (8.6)	81.4 (7.9)	74.8 (7.1)	71.7 (6.6)

Note. HR = heart rate; SBP = systolic blood pressure; DBP = diastolic blood pressure; SD = standard deviation

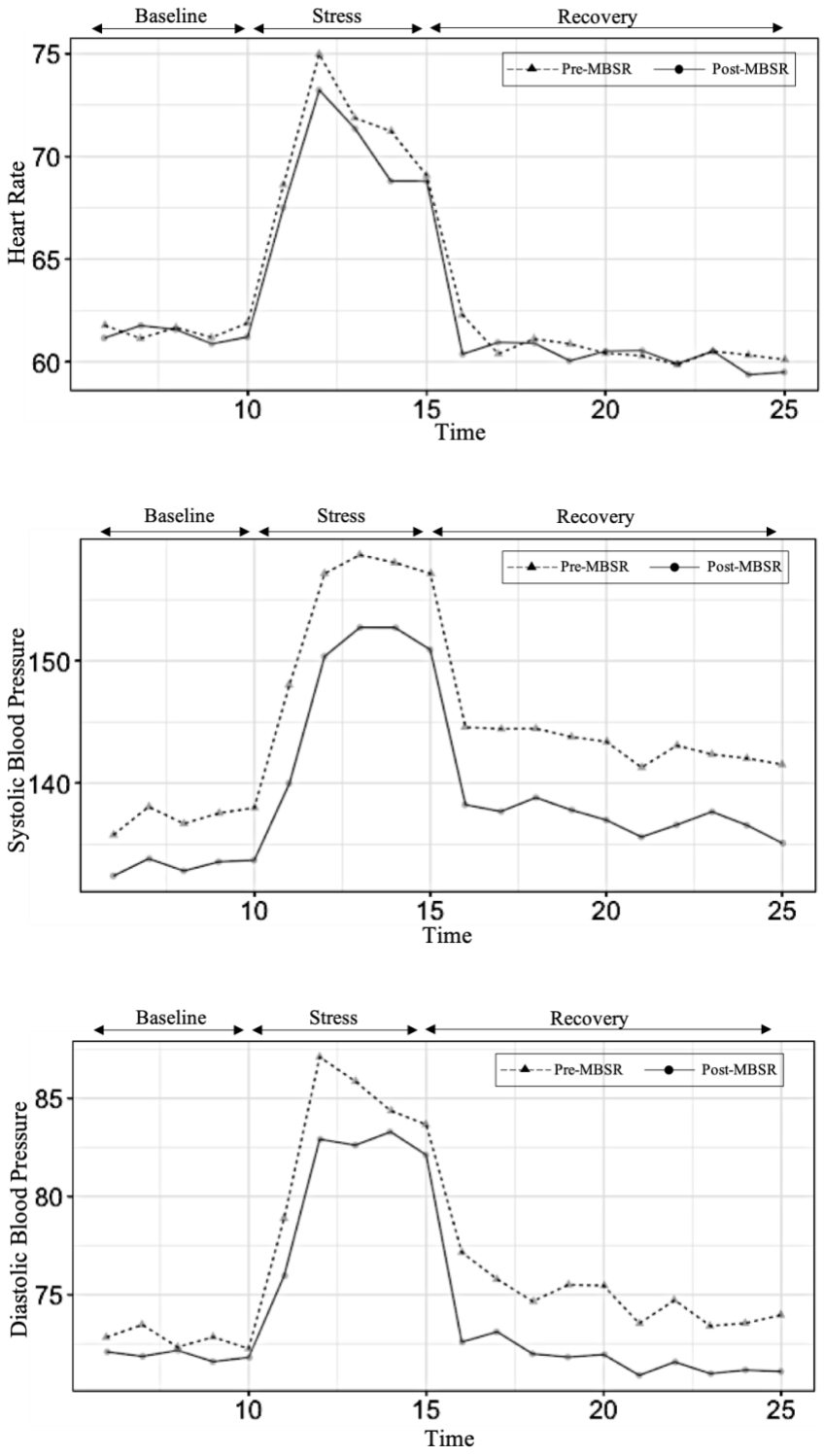

Figure 2. HR, SBP, and DBP increased and decreased as expected before and after the anger recall task.

Equanimity Pre- vs. Post-MBSR

Alpha coefficients for all facets of self-reported equanimity were in the good to excellent range (.83 to .93). As expected, self-reported equanimity (FFMQ Observing + FFMQ Non-Reactivity + FFMQ Non-Judgment) was significantly higher post-MBSR (M = 86.54, SD = 12.8) than pre-MBSR (M = 72.4, SD = 14.4) ($t = -5.609$, $p<.001$, $d = 1.03$). Higher scores are indicative of greater self-reported equanimity (See Figure 3).

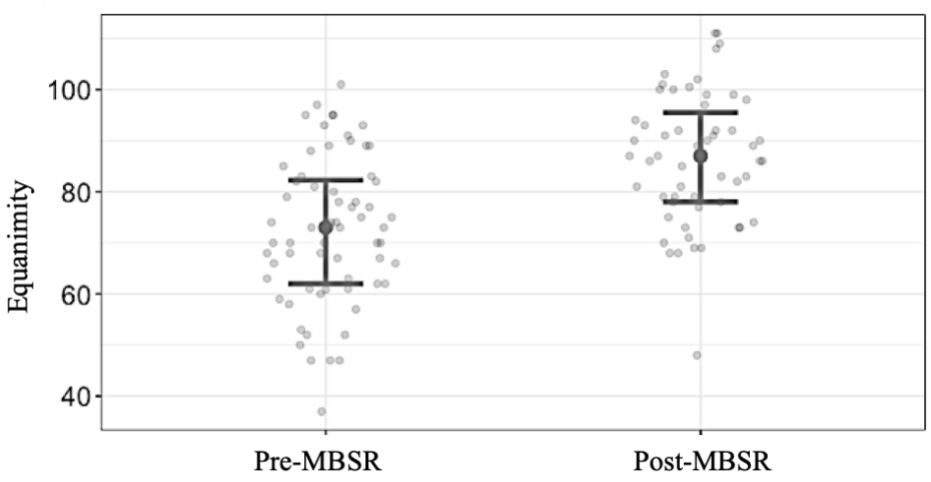

Figure 3. Equanimity increased after MBSR. Whiskers of plot represent the upper and lower quartiles.

Hypothesis I: Self-Reported Equanimity and Cardiovascular Recovery

Means and standard deviations for each measure of cardiovascular recovery are summarized in Table 3. Contrary to our hypothesis, results of planned model comparisons predicting cardiovascular recovery indicated that adding equanimity to the model did not improve fit for any outcome variable, either before or after MBSR. Results were equivalent regardless of which outcome variable (simple change scores,

residualized change scores, or percent recovery) was used. Results are summarized in Tables 4 and 5.

Table 3

Mean Recovery Score for Simple Change Scores, Residualized Change Scores, and Percent Recovery

	Unadjusted Means (SD)					
	HR		SBP		DBP	
	Pre-MBSR	Post-MBSR	Pre-MBSR	Post-MBSR	Pre-MBSR	Post-MBSR
Simple Change Scores	-.83 (3.2)	-1.1 (2.9)	5.95 (5.1)	3.84 (5.7)	2.01 (3.9)	-.19 (3.8)
Residualized Change Scores	0.00 (2.9)	0.00 (2.4)	0.00 (4.5)	0.00 (5.2)	0.00 (3.2)	0.00 (3.3)
Percent Recovery	127.94 (190.4)	137.14 (172.0)	71.07 (40.7)	108.39 (205.8)	85.44 (46.5)	115.45 (166.1)

Note. Means of residualized change scores are zero by default. Scores represent the proportion of the recovery score that is *unpredictable* from baseline and stress. Each individual score is calculated relative to the regression line ($y - \hat{y}$) but the *mean* difference between observed and predicted scores is always zero because the regression line has been fit to minimize error.

Table 4

Association between Self-Reported Equanimity and Cardiovascular Recovery Pre-MBSR

DV	Model	AIC	BIC	BF	R_2	P
Simple Change Scores	DV = HR Recovery					
	Full	342.7	355.6	.13	.01	.903
	Reduced	340.7	351.5	7.94	.009	
	DV = SBP Recovery					
	Full	399.4	412.4	.13	.04	.916
	Reduced	397.5	408.2	7.95	.04	
	DV = DBP Recovery					
	Full	366.8	379.8	.13	.012	.990
	Reduced	364.8	375.6	8.00	.012	
Residualized Change Scores	DV = HR Recovery					
	Full	329.5	342.4	.14	.019	.677
	Reduced	327.7	338.4	7.28	.016	
	DV = SBP Recovery					
	Full	382.6	395.5	.14	.025	.652
	Reduced	380.8	391.6	7.16	.022	
	DV = DBP Recovery					
	Full	341.1	354.0	.13	.024	.966
	Reduced	339.1	349.9	8.00	.024	
Percent Recovery	DV = HR Recovery[a]					
	Full	861.0	873.0	.22	.053	.318
	Reduced	860.1	870.9	4.64	.037	
	DV = SBP Recovery					
	Full	666.1	679.0	.14	.013	.689
	Reduced	664.3	675.0	7.33	.010	
	DV = DBP Recovery					
	Full	682.8	695.8	.15	.017	.559
	Reduced	681.2	692.0	6.64	.011	

Model Predictors	
Full	Age, Sex, BMI, Self-reported Equanimity
Reduced	Age, Sex, BMI

Note. There was no associated between pre-MBSR equanimity and CR.

[a] Sensitivity analyses accounting for outliers were conducted for this dependent variable. The full GLM was compared with a robust regression model using the same predictors. Model comparison showed no meaningful difference between the robust and general linear model.

Table 5

Association between Self-Reported Equanimity and Cardiovascular Recovery Post-MBSR

DV	Model	AIC	BIC	BF	R_2	P
Simple Change Scores	DV = HR Recovery					
	Full	278.3	290.4	.14	.072	.971
	Reduced	276.3	286.4	7.41	.072	
	DV = SBP Recovery[a]					
	Full	357.3	369.3	.26	.051	.273
	Reduced	356.6	366.7	3.80	.027	
	DV = DBP Recovery[a]					
	Full	311.7	323.8	.19	.027	.432
	Reduced	310.4	320.4	5.26	.015	
Residualized Change Scores	DV = HR Recovery[a]					
	Full	261.2	273.3	.14	.038	.738
	Reduced	259.4	269.4	6.97	.035	
	DV = SBP Recovery					
	Full	346.1	358.1	.33	.040	.203
	Reduced	345.9	355.9	3.01	.008	
	DV = DBP Recovery[a]					
	Full	294.0	306.0	.21	.044	.375
	Reduced	292.9	302.9	4.79	.029	
Percent Recovery	DV = HR Recovery[a]					
	Full	718.6	730.5	.24	.046	.309
	Reduced	717.7	727.7	4.13	.025	
	DV = SBP Recovery[a]					
	Full	753.3	765.3	.14	.014	.932
	Reduced	751.3	761.3	7.39	.014	
	DV = DBP Recovery[a]					
	Full	728.4	740.4	.14	.034	.991
	Reduced	726.4	736.4	7.42	.034	

Model Predictors	
Full	Age, Sex, BMI, Self-reported Equanimity
Reduced	Age, Sex, BMI

Note. There was no association between post-MBSR equanimity and CR.

[a] Sensitivity analyses accounting for outliers and non-linearity were conducted for these dependent variables. The full GLMs were compared with robust regression models using the same predictors. Model comparison showed no meaningful difference between the robust and general linear models for all DVs.

Hypothesis II: Self-Reported Equanimity and Cardiovascular Reactivity

As hypothesized, results of most planned model comparisons predicting cardiovascular reactivity indicated that adding equanimity did not improve fit for any outcome variable, either before or after MBSR (see Table 6). There was a trend toward improvement when equanimity was added to the model predicting SBP reactivity pre-MBSR ($\beta = .23$, $\Delta R^2 = .046$, $p = .088$). AIC also favored the full model. In this model, contrary to expectations, increased equanimity was associated with increased SBP reactivity (see Figure 4).

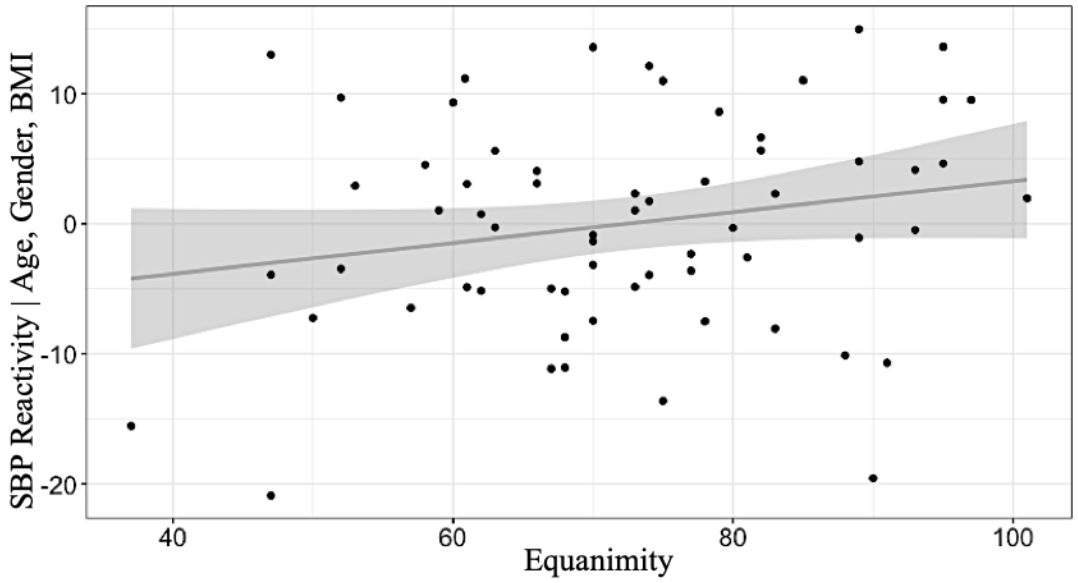

Figure 4. Higher equanimity was associated with higher SBP reactivity pre-MBSR ($\beta = .23$, $\Delta R^2 = .046$, $p = .088$).

Table 6

Association Between Self-Reported Equanimity and Cardiovascular Reactivity

DV	Model	AIC	BIC	BF	R_2	P
			DV = HR Reactivity[a]			
	Full	435.1	448.0	.13	.056	.772
	Reduced	433.1	443.9	7.64	.055	
			DV = SBP Reactivity			
Pre-MBSR	*Full*	458.8	471.8	.62	.097	.088
	Reduced	460.0	470.8	1.63	.051	
			DV = DBP Reactivity			
	Full	373.8	386.7	.22	.080	.316
	Reduced	372.9	383.7	4.61	.064	
			DV = HR Reactivity[a]			
	Full	381.3	393.4	.14	.096	.729
	Reduced	379.5	389.5	6.94	.094	
			DV = SBP Reactivity			
Post-MBSR	*Full*	401.7	413.7	.14	.058	.942
	Reduced	399.7	409.7	7.39	.058	
			DV = DBP Reactivity			
	Full	364.6	376.6	.17	.022	.534
	Reduced	363.0	373.0	5.98	.015	

Model Predictors	
Full	Age, Sex, BMI, Self-reported Equanimity
Reduced	Age, Sex, BMI

Note. There was no association between equanimity and cardiovascular reactivity, exempting a trend wherein equanimity was positively associated with SBP reactivity pre-MBSR. Reactivity was calculated using residualized change scores exclusively.

[a] Sensitivity analyses accounting for outliers, skewness, and heteroskedasticity were conducted for these dependent variables. The full GLMs were compared with robust regression models using the same predictors. Model comparison showed no meaningful difference between the robust and general linear models for all DVs.

Hypothesis III: MBSR and Cardiovascular Recovery

As hypothesized, MBSR was associated with more efficient SBP ($\beta = -.201$, $\Delta R_2 = .039$, $p < .05$) and DBP ($\beta = -.289$, $\Delta R_2 = .076$, $p < .05$) recovery (see Figures 5a and 5b). However, the effect of MBSR on SBP and DBP recovery was not present when

recovery was measured using residualized change scores or percent recovery. There was no effect of MBSR on HR recovery. Results are summarized in Tables 7 and 8.

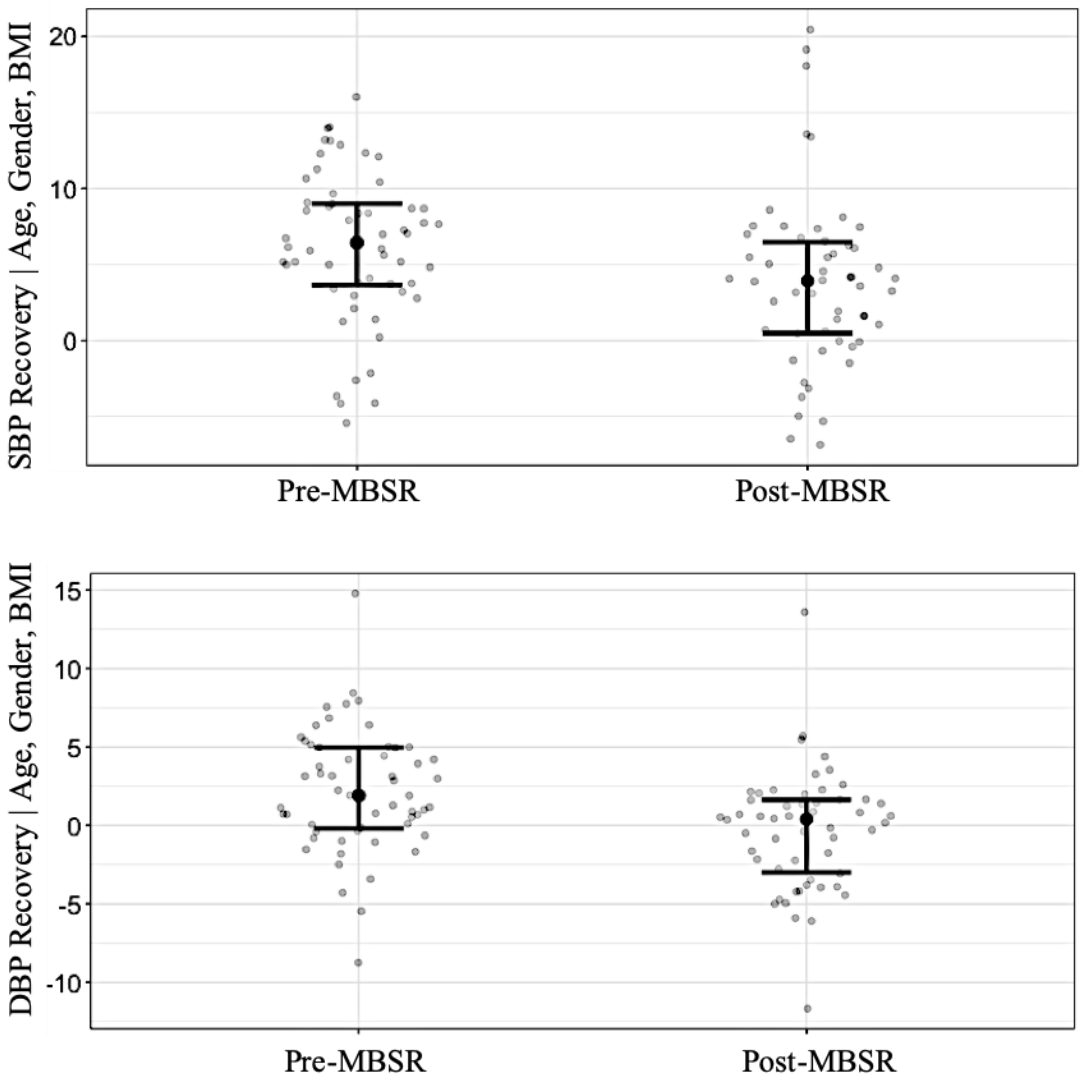

Figure 5. BP recovery improved after MBSR when measured via simple change score.

Table 7

Association between MBSR and Cardiovascular Recovery

	Model	AIC	BIC	BF	R_2	P
Simple Change Scores	DV = HR Recovery					
	Full	619.3	636.0	.10	.006	.684
	Reduced	617.4	631.4	10.05	.005	
	DV = SBP Recovery					
	Full	752.8	769.5	1.05	.065	.031*
	Reduced	755.7	769.6	.96	.026	
	DV = DBP Recovery					
	Full	674.9	691.6	10.51	.080	.003*
	Reduced	682.4	696.3	.10	.004	
Residualized Change Scores	DV = HR Recovery					
	Full	587.2	604.0	.09	.011	.991
	Reduced	585.2	599.2	10.95	.011	
	DV = SBP Recovery					
	Full	726.5	743.2	.09	.004	.986
	Reduced	724.5	738.5	11.0	.004	
	DV = DBP Recovery					
	Full	633.5	650.3	.09	.008	.978
	Reduced	631.5	645.5	10.95	.008	
Percent Recovery	DV = HR Recovery[a]					
	Full	1585.4	1602.1	.10	.009	.784
	Reduced	1583.5	1597.4	10.49	.009	
	DV = SBP Recovery[a]					
	Full	1542.2	1558.9	.25	.021	.167
	Reduced	1542.2	1556.1	4.02	.004	
	DV = DBP Recovery[a]					
	Full	1494.6	1511.3	.24	.030	.177
	Reduced	1494.5	1508.5	4.21	.014	
	Model Predictors					
	Full	Age, Sex, BMI, Time (pre-MBSR, post-MBSR)				
	Reduced	Age, Sex, BMI				

Note. MBSR was associated with improved SBP and DBP recovery when these were measured using simple change scores. Multiple imputation was conducted for these models to account for missing data post-MBSR. Estimates were comparable for both models.

[a] Sensitivity analyses accounting for outliers, skewness, and heteroskedasticity were conducted for these dependent variables. The full GLMs were compared with robust regression models using the same predictors. Model comparison showed no meaningful difference between the robust and general linear models for all DVs.

Table 8

Partial Explanatory Role of Equanimity in the Association between Cardiovascular Recovery and MBSR

	Cohen's d	Standardized β	Semi-partial R_2	P
	DV = SBP Recovery (Simple Change)			
Reduced	-.40	-.201	.039	.031*
Full	-.34	-.169	.022	.10
	DV = DBP Recovery (Simple Change)			
Reduced	-.56	-.289	.076	.003*
Full	-.54	-.261	.053	.011*
	Model Predictors			
Reduced	Age, Sex, BMI, Time (pre-MBSR, post-MBSR)			
Full	Age, Sex, BMI, Time (pre-MBSR, post-MBSR), equanimity			

Note. Parameter estimates for Time before and after adding equanimity to GLMs predicting SBP and DBP recovery (simple change scores). Equanimity was not a significant predictor in either model.

Hypothesis IV: Equanimity as an Explanatory Process

Since models predicting SBP and DBP recovery via simple change score were the only models showing the expected pattern in hypothesis III, equanimity was tested as a potential explanatory variable for only these models. Equanimity was added as a fifth predictor (see Appendix A) and the degree of change in parameters for Time (pre- vs. post-MBSR) was interpreted as suggestive or not suggestive of partial mediation. Including equanimity in the model predicting SBP recovery reduced the absolute size of the coefficient by 16% and removed its statistical significance ($\beta = -.169$, $\Delta R_2 = .017$, $p = .10$). However, equanimity itself was not a significant predictor of SBP recovery. This means that the difference in SBP recovery between pre-MBSR and post-MBSR became smaller by 16% after the addition of equanimity (see Figure 6a). Including equanimity in the model predicting DBP recovery reduced the size of the regression coefficient by 10%

and attenuated the accompanying p-value, but the effect of time (pre vs. post MBSR) remained statistically significant (see Figure 6b). Results are summarized in Table 8.

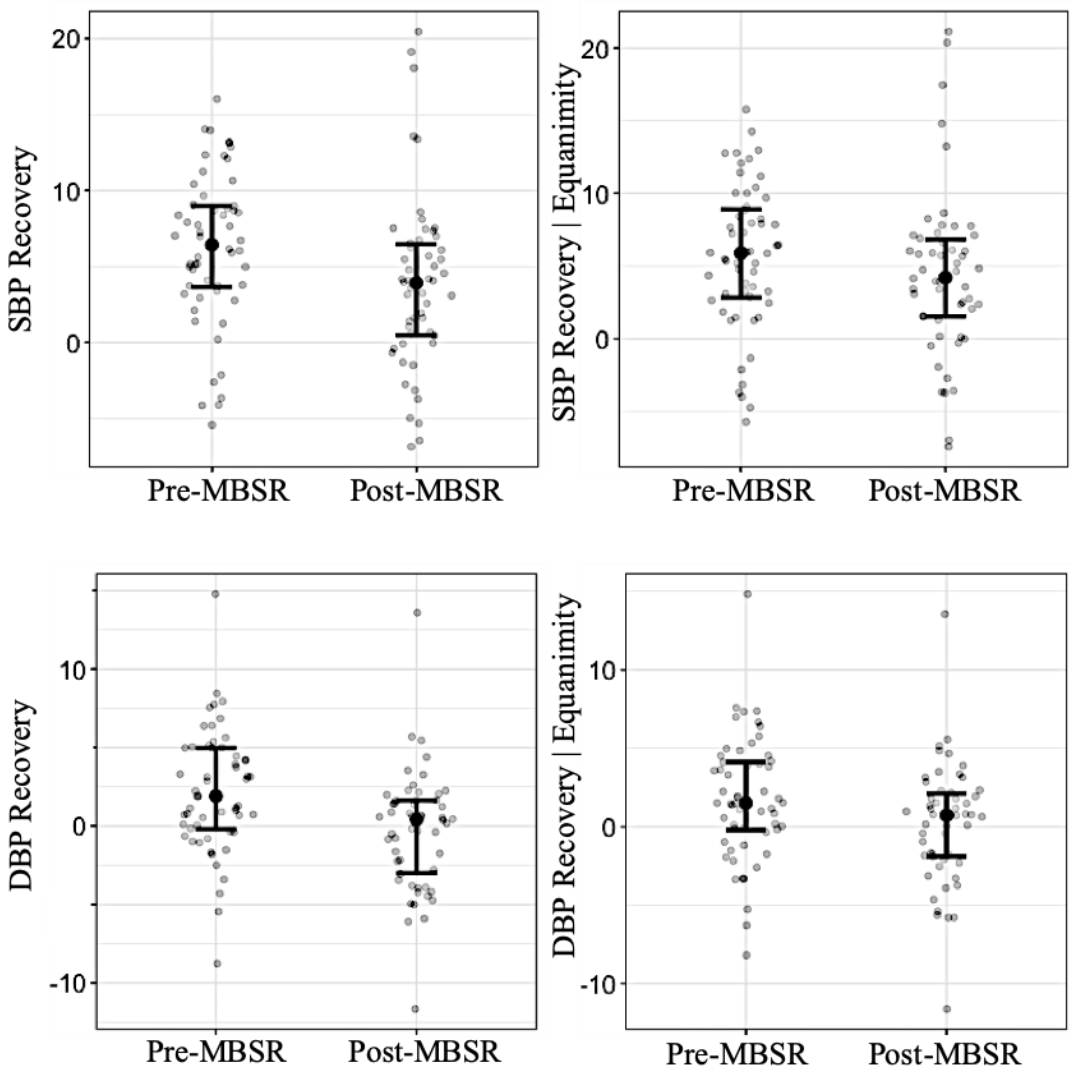

Figure 6. Added variable plots depicting better BP recovery after MBSR (left), followed by the same relationship with the effect of equanimity covaried out (right). Equanimity explained a small portion of the difference in SBP recovery post-MBSR, but not DBP recovery.

Post-Hoc Exploratory Analysis

Simple change scores are theoretically prone to one type of measurement error due to correlation between this score and baseline averages. Specifically, baseline averages are often positively correlated with simple change scores for recovery (those who start lower recover "better"). The result of this situation, quite common in stress recovery research, is that simple change scores are more reflective of between-subjects differences in baseline averages than of recovery proper. In addition, in a multigroup design the reliability of simple change scores also depends on group equivalence (i.e. pre- vs. post-MBSR) at baseline *and* on the degree of correlation between baseline and later change being consistent across Time. If groups are different from one another at baseline group differences in recovery score may simply be a function of this disparity. Residualized change scores and percent recovery scores, which mathematically control for baseline and stress levels, should negate the effect of these confounds, if present.

Post-hoc analyses were conducted to confirm these explanations for the differences between operationalizations of recovery for BP in hypothesis III. As expected, baseline SBP ($r = -.21$, $p<.05$) and baseline DBP ($r = -.34$, $p<.05$) were correlated with simple change scores for recovery, but not in the expected direction (those who started lower recovered "worse"). In addition, baseline SBP was an average of 3.87 points lower post-MBSR than pre-MBSR ($d = -.30$, $p = .10$). Baseline DBP did not differ between timepoints ($d = -.12$, $p = .52$). Moderation models showed that the relationship between baseline and recovery score did not significantly vary by timepoint for either SBP ($\beta = -.029$, $p = .71$) or DBP ($\beta = -.125$, $p = .20$). In general, some, but not

all, of the expected patterns which make simple change scores difficult to interpret in a recovery design were present in this dataset.

Chapter 4

Discussion

The present study investigated associations between self-reported equanimity, mindfulness training, and cardiovascular recovery (CR) from stress. Four hypotheses were addressed: (1) that high self-reported equanimity is associated with more efficient CR, (2) that self-reported equanimity is *not* associated with cardiovascular *reactivity*, (3) that completing MBSR is associated with more efficient CR, and (4) that self-reported equanimity partly explains the relationship between MBSR and CR.

Our findings indicate that self-reported equanimity (FFMQ Observing + FFMQ Non-reactivity + FFMQ Non-Judging) was *not* associated with CR, as hypothesized. Equanimity was also not associated with cardiovascular *reactivity*, exempting one trend wherein high equanimity was associated with high SBP reactivity *before* MBSR. Mindfulness training (MBSR) was not associated with better HR recovery, but was associated with more efficient SBP and DBP recovery. However, this effect only held when recovery was measured via simple change scores but not with residualized change or percent change. Post-hoc analyses showed that this discrepancy may be due to a significant correlation between baseline BP and simple change scores and lower baseline SBP post-MBSR, both of which reduce the reliability of simple change scores (Linden et al., 1997). This calls the statistical conclusions validity of these models into question. Equanimity explained small portions of the association between MBSR and SBP/DBP recovery, but these results are difficult to interpret given that (1) the validity of the original model is in question and (2) the lack of clear agreement between parameters (β, p-values, Cohen's d, ΔR_2).

Discussion of these results can be divided into two themes: the operationalization of equanimity and measurement validity in stress recovery research. For clarity, these will be addressed in two separate sections.

Operationalizing Equanimity

The present study was inspired by an idea of equanimity as a construct distinct from mindfulness and worthy of independent study, but which currently lacks an established operationalization or measurement methodology (Desbordes et al., 2015; Hadash et al., 2016). This analysis was planned in response to the related suggestion that equanimity, understood as a learned disposition toward a certain style of emotional responding, may be operationalized as a faster resolution of the physiologic stress response. Because of well-known difficulties with self-report measures of mindfulness and related constructs (Grossman, 2008; Grossman, 2011; Van Dam et al., 2018), we assessed this hypothesis using both a novel self-report measure of equanimity and an operationalization of *learned* equanimity: completing an 8-week MBSR course centered on developing a daily mindfulness meditation practice. In both cases, our hypothesis – that equanimity would be linked with more efficient cardiovascular recovery – was not well supported.

There are several ways of interpreting these results, all of which point to a need for further research. The first and most straightforward is that the hypothesis is incorrect and equanimity is in fact not associated with more efficient cardiovascular recovery from stress. This is certainly possible, especially given that the integration of Buddhist and Western psychological thought on this construct is in its infancy. In addition, it is important to "mind the hype" and note that mindfulness training and/or the cultivation of

equanimity may not be associated with the kind of clinically relevant "benefits" researchers often hope to see (Van Dam et al., 2018). That said, the idea that cultivating "an attitude capable of embracing pleasure or pain without reflexively reacting to them" (Olendzki, 2006, p. 258) would be associated with a unique physiological response to stress is certainly not worth abandoning, especially given the growing body of research linking meditation training and altered stress responsivity (Crosswell et al., 2017; Gamaiunova et al., 2019; Lindsay, Young, Smyth, Brown, & Creswell, 2018; Rosenkranz et al., 2016).

Alternatively, our population was medically healthy and we may not expect to see physiological changes in stress responsivity in a population of healthy adults who are not significantly stressed. Creswell and Lindsay's 2014 stress-buffering account of mindfulness suggests that disease-related health effects of meditation practice, such as improved cardiovascular recovery from stress, are most likely to be seen in high-stress populations. Thus, it remains possible that equanimity and/or mindfulness training *would* have been associated with improved recovery in a higher-stress sample. Our population, in contrast, may have already been recovering relatively efficiently and therefore had little remove to improve, even as equanimity increased.

Another possible explanation for our findings is that the self-report operationalization of equanimity we employed lacked construct validity. We chose to use the Observing, Non-Judgment, and Non-Reactivity facets of the Five Facet Mindfulness Questionnaire following a review of Hadash & colleague's (2016) "Decoupling Model of Equanimity" and Lindsay & Creswell's (2017) Monitor and Acceptance Theory (MAT) of mindfulness. The measure is meant to reflect (1) that equanimity is manifested via

acceptance of (FFMQ Non-Judging) and reduced **reactivity** to (FFMQ Non-Reactivity) unpleasant events (Hadash et al., 2016) and (2) that equanimity, like acceptance, is dependent on a basic level of **awareness** (FFMQ Observing) cultivated through sustained mindfulness practice (Lindsay & Creswell, 2017). However, this theory may not have been adequately reflected in the scales we used. For example, the Non-Judging scale of the FFMQ refers specifically to "non-judging *of inner experience*" (emphasis added) and is assessed using items like "some of my emotions are bad and I shouldn't feel them" (Baer et al., 2008). While it certainly extends to internal experiences, the attitude of acceptance relevant to equanimity refers more to an attitude of balanced openness to *all* experience, wherein pleasant experiences are not grasped at and unpleasant experiences are not pushed away (Olendzki, 2006). Relatedly, the "attitude of acceptance" identified as a lower-order factor of equanimity by Hadash and colleagues was measured using the Distress Tolerance Scale (DTS) and the White Bear Suppression Inventory (WBSI). These measures tap distress tolerance and experiential avoidance, respectively, both of which do not map directly onto Non-Judging as assessed in the FFMQ. The Non-Reactivity subscale maps quite well onto the proposed definition of equanimity and onto those used by Hadash and colleagues (Anxiety Sensitivity Index-3, the Leiden Index of Depression Sensitivity-Revised), but again refers more to responses to *internal* stressors than to a more comprehensive range of experience. Lastly, although the Observe subscale has been shown to reliably differentiate meditators from non-meditators (Baer et al., 2008) assessments of awareness are also particularly prone to response bias and demand characteristics, especially in a sample of novice meditators (Grossman, 2011).

Upon reflection, we believe it may have been more appropriate to understand completing MBSR as cultivating a basic level of mindful awareness, to limit operationalizing equanimity to Non-Judging and Non-Reactivity, and to test hypothesis I following mindfulness training *exclusively*. After all, the present understanding of equanimity indicates that we might not expect large variation in this trait in a meditation naïve sample, which in turn may make it difficult to detect physiological markers of high equanimity. Future research may also make use of self-report measures which more explicitly integrate Buddhist understandings of acceptance and non-reactivity. The Non-Attachment Scale (Sahdra, Shaver, & Brown, 2010) and the Self-Other Four Immeasurables Scale (Kraus & Sears, 2009) may be useful alternatives. Of note, these measures include items assessing the respondent's attitude toward pleasant experiences (sukha), which is equally important in a Buddhist understanding of equanimity (Olendzki, 2006).

Construct validity aside, it is also possible that equanimity simply did not develop sufficiently following an 8-week mindfulness training program. Although scores on our self-report measure of equanimity did increase as expected, this study did not include an active control group and thus we cannot be sure that increases in this measure were not a function of expectancies and demand characteristics. Although the increase in equanimity was large (d=1.03), it is worth noting that pre-post increases in self-report measures of mindfulness have also been reported following active control conditions and the validity of self-report questionnaires following MBIs is still in question (Visted et al., 2015). In addition, Buddhist scholarship implies that equanimity is best understood as a "way of being" that is the end result of sustained mindfulness training (Thrangu Rinpoche, 2002).

It may not make sense to expect this capacity to develop following two months of meditation practice. In fact, Hadash and colleagues (2016) found no improvement in "an attitude of acceptance", one of the two lower-order factors of equanimity, following a 4-week mindfulness-based intervention. This is consistent with Monitor and Acceptance Theory (MAT), which posits that acceptance (broadly defined to include equanimity and related constructs) may develop more slowly than the capacity to monitor attention (Lindsay & Creswell, 2017).

MAT also posits that improvements in biological stress reactivity and recovery are facilitated by improvements in acceptance, and indeed there is now some evidence to support this theory (Gamaiunova et al., 2019; Lindsay et al., 2018). However, this hypothesis was not strongly supported in our sample. Equanimity explained only a small portion of the association between mindfulness training and blood pressure recovery and was not independently associated with improved recovery post-MBSR. In addition, although we did see improvements in blood pressure recovery following MBSR, these were only evident when recovery was measured using simple change scores. This being the case, it is difficult to draw conclusions about the role of equanimity when the statistical model itself is suspect. This is addressed in detail in the following section.

This study also focused specifically on the cardiovascular system, to the exclusion of other autonomic systems sensitive to emotional stress. Heart rate reactivity and recovery were unchanged in all hypotheses, which is consistent with previous mindfulness research showing no effect of mindfulness training on this physiological response (Crosswell et al., 2017; Gamaiunova et al., 2019; Grant et al., 2013). In the case of heart rate recovery, this may be the result of normative quick heart rate recovery from

stress, which is difficult to capture in a stress response protocol (Linden et al., 1997). Continuous beat-by-beat measurement of heart rate, a focus on heart-rate-variability (HRV), and multilevel modelling techniques may help clarify whether effects of mindfulness training on cardiovascular recovery are specific to blood pressure. In addition, other indices of autonomic function such as stress hormones (i.e., cortisol), and immune markers (i.e., cytokines) are also worthy of study. These biological systems have been linked to emotion regulation in the context of a laboratory stressor (Steptoe, Hamer, & Chida, 2007) and are frequently studied in the context of mindfulness-based interventions (Morgan, Irwin, Chung, & Wang, 2014; O'Leary, O'Neill, & Dockray, 2016). It is possible that the effect of equanimity on stress recovery would be more apparent using other indices of autonomic function. That said, the clinical relevance of cardiovascular recovery from stress is well established (Schuler & O'Brien, 1997), and future mindfulness research should continue to investigate the cardiovascular system as a potential means of addressing stress-related chronic illness.

Regarding reactivity, we found that, as hypothesized, equanimity was *not* associated with changes in cardiovascular reactivity. This hypothesis reflects the traditional distinction between equanimity and indifference. Buddhist literature clearly warns against this "near-enemy" of equanimity: an attitude of apathy toward experience that can be understood as a pernicious form of aversion (Salzberg, 1995). Extending this theory to the physiological stress response, we would *not* expect individuals high in equanimity to show a buffered response to emotional arousal. In fact, we may even expect *increased* reactivity (Crosswell et al., 2017). We did find a trend toward *increased* SBP reactivity in participants high in equanimity, but this was only the case *before*

MBSR and is therefore inconsistent with the mindfulness training study cited above. It is also important to note that this understanding of the relationship between equanimity and stress reactivity is not ubiquitous. MAT, which heavily informed this analysis, posits that acceptance is a critical component for *reducing* affective and biological reactivity following mindfulness training. The same research team recently published the first experimental evidence showing that mindfulness training which specifically includes acceptance *reduced* SBP reactivity, whereas training which built only attention monitoring did not (Lindsay et al., 2018). Cardiovascular recovery was not assessed. An analysis of reactivity before and after MBSR was beyond the scope of this paper, but future research should continue to clarify the theoretical foundations of stress reactivity research with meditators. Furthermore, this and other studies typically understand equanimity among novice meditators to reflect lower levels of the same construct present in long-term meditators when they may in fact be distinct phenomena (Hadash et al., 2016). For example, it is possible that in beginning meditators equanimity/acceptance is manifested as an increased ability to decenter from and not react to emotions (buffered reactivity, unchanged recovery) whereas equanimity/acceptance in long-term meditators may be manifested as an attitude of openness, compassion, and willingness to feel one's emotions without grasping or aversion (unchanged/increased reactivity, improved recovery). Future studies comparing populations of novice and experienced meditators may provide a useful means of clarifying the role of equanimity in stress reactivity.

Overall, this study provides the first direct examination of the association between equanimity, mindfulness training, and cardiovascular recovery from stress. There is theoretical justification to expect that meditation training may improve cardiovascular

recovery from emotional stress and for this effect to be, in part, a function of increased equanimity and related constructs (acceptance, non-reactivity). However, our results did not strongly support this hypothesis. Our conclusions are limited by (1) lack of a matched control group, (2) a population of novice meditators, (3) lack of agreed upon measurement methodology for equanimity, and (4) concerns about measurement validity for cardiovascular recovery. This analysis was designed specifically to address the latter limitation and is addressed in detail in the following section.

Measurement Validity in Stress Recovery Research

This study provides evidence that the conclusions drawn from recovery models using simple change scores may differ from models using residualized change or percent recovery. Models predicting SBP and DBP recovery from Time (pre-MBSR vs. post-MBSR) and covariates were significant only when simple change scores were used as the outcome variable, but *not* when residualized change scores or percent recovery scores were used. Our results demonstrate that methods of calculating recovery may be subject to measurement error when they do not adjust for (1) correlation between baseline and subsequent change, (2) group differences in baseline, and (3) group differences in the correlation between baseline and subsequent change. Post-hoc analyses indicated that our dataset was subject to confounds (1) and (2), which are common in stress recovery research (Linden et al., 1997). Because of this, we cannot conclude that our results supported the hypothesis that mindfulness training and equanimity are associated with improved cardiovascular recovery from emotional stress. In fact, it is likely that significance in the simple change score models was a function of lower baseline blood pressure post-MBSR and a significant correlation between baseline blood pressure and

subsequent change (i.e. those who "recovered better" also started higher). Figures 7a and 7b provide a visualization of the former confound in which post-MBSR SBP and DBP have been adjusted to account for the average between-group difference during baseline and stress. Visually, when both groups start at the same place it is easier to see that SBP and DBP recovery do not appear more efficient post-MBSR. Residualized change scores and percent recovery scores adjust for this confound, and consequently the "effect" disappeared.

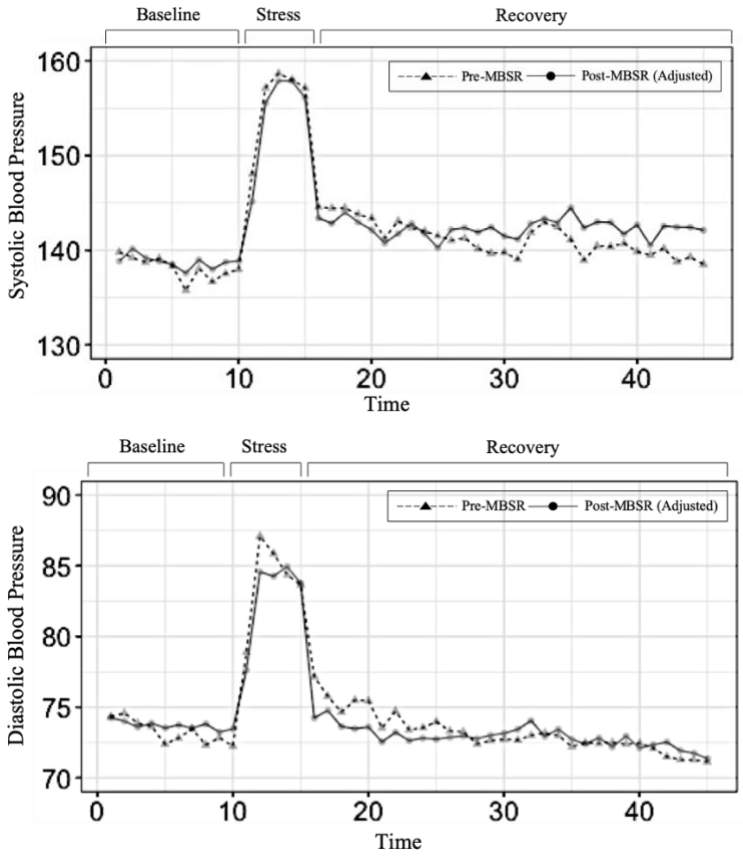

Figure 7. The average difference between-group difference between baseline and stress was added to post-MBSR SBP and DBP. Visual differences in recovery are now less apparent.

It is worth noting that the conclusions drawn in this paper, about mindfulness training and equanimity specifically, would have been very different had we not directly addressed measurement validity in stress recovery research. In fact, the answers to two of our research questions – does mindfulness training improve cardiovascular recovery and does equanimity partly explain that improvement? – were contingent on the methodology used to operationalize recovery. As previously mentioned, multiple researchers have explored the disadvantages of change scores in the context of stress recovery (Christenfeld et al., 2000; Linden et al., 1997; Llabre et al., 2004). In response, many researchers have abandoned the use of change scores for more sophisticated curve-fitting techniques (discussed below), but nonetheless it remains possible to find many papers using simple change scores in isolation (Gordon et al., 2012; Keogh & Creaven, 2017; Routledge et al., 2015; Steffen & Larson, 2015; Trivedi et al., 2008). It is important to note that sometimes researchers using simple change scores will include baseline and stress levels as covariates (i.e. ANCOVA), which attempts to control for baseline and stress in the same way residualized change scores and percent recovery scores do. However, this strategy assumes that residual variability is due to the task, not to individual differences in baseline levels, and thereby bases adjustments on the regression slope of the whole group rather than on the impact of baseline/stress on recovery for each individual. To satisfy this assumption in a multigroup design, the researcher must show that regression slopes for all groups are parallel (i.e. there is no group difference in the correlation between baseline and subsequent change) (Linden et al., 1997). This consideration was infrequently addressed in our review of the stress recovery literature. Overall, it is our hope that this analysis demonstrates the importance of measurement

validity in stress recovery research *and* that it is relatively easy to use alternate means of measuring recovery if curve-fitting techniques are not accessible.

On the subject of curve-fitting techniques, it is important to highlight that residualized change scores and percent recovery scores are not ideal means of measuring recovery; they are merely better than simple change scores. Because they are calculated by collapsing several measurements into a single score, just as simple change scores do, they are also highly variable. This makes Type II errors more possible in the context of Null Hypothesis Significance Testing (NHST), which was partially accounted for in this analysis by the use of Bayesian estimates and model comparison. However, collapsing many measurements into a single score also forfeits much of the detail collected over the testing period. For example, no method used in this paper enables the researcher to examine the slope of the recovery curve, which arguably is the one parameter which most directly addresses whether recovery is more efficient. As discussed in the Introduction section of this paper, curve fitting techniques use all available data points, are not confounded by baseline and stress levels, and describe the recovery curve using multiple parameters (i.e. slope, asymptote). This methodology is increasingly being used in general stress recovery research (Christenfeld et al., 2000; Llabre et al., 2004) as well as in mindfulness research specifically (Crosswell et al., 2017; Lindsay et al., 2018). Due to the introductory nature of this project curve-fitting techniques were not used, but it remains possible that these methods would reveal differences in cardiovascular recovery that were not identifiable using the present methods.

Strengths and Limitations

The present study provides a useful reminder of the importance of presenting null findings, especially given increased focus on methodological rigor. Scientific study of mindfulness meditation has been particularly vulnerable to exaggerated positive claims and lack of attention to null findings (Van Dam et al., 2018). Although our hypotheses were not strongly supported, this study was strengthened by research questions rooted in recent theory, clinically relevant biological outcome measures, and an explicit focus on construct, measurement and statistical conclusions validity. At the same time, this study was also limited by other methodological considerations which commonly plague mindfulness research, namely our lack of an active control group, population of novice meditators, and use of a novel self-report measure of equanimity which may lack construct validity.

Chapter 5

Conclusion

Our findings suggest that, contrary to expectations, neither self-reported equanimity nor mindfulness training are linked with improved cardiovascular recovery from emotional stress in a healthy adult sample. In addition, we used multiple means of operationalizing cardiovascular recovery and found that mindfulness training was associated with more efficient blood pressure recovery when simple change scores were used but not when residualized change scores and percent recovery scores were used. Post-hoc analyses indicated that this discrepancy was likely the result of confounds which complicate the interpretation of simple change scores, rather than genuine differences in recovery post-MBSR. This is the first analysis to address a possible biological basis for equanimity and is unique in its integration of a clinically relevant biological outcome measure with this traditional Buddhist construct. This being the case, future research should continue to address equanimity as an outcome and potential mechanism of mindfulness training, both from a physiological *and* self-report perspective. More broadly, research linking mindfulness and related constructs with stress reactivity or recovery will also benefit from a focus on measurement and statistical conclusions validity. Although our hypotheses were not strongly supported in a healthy sample, our hope is that this paper encourages further research in clinical populations and in non-clinical populations with higher levels of stress, using thoughtful statistical methods to integrate Buddhist theory with relevant biological outcomes. Robust theoretical foundations, statistical rigor, a historically-rooted understanding of contemplative practice, and a strong inclination to "mind the hype" will, we hope, help our field better

understand the pathways through which mindfulness meditation training may enhance health and well-being.

References

Anālayo, B. (2019). Adding historical depth to definitions of mindfulness. *Current Opinion in Psychology, 28*, 11-14. doi:https://doi.org/10.1016/j.copsyc.2018.09.013

Arch, J. J., Brown, K. W., Dean, D. J., Landy, L. N., Brown, K. D., & Laudenslager, M. L. (2014). Self-compassion training modulates alpha-amylase, heart rate variability, and subjective responses to social evaluative threat in women. *Psychoneuroendocrinology, 42*, 49-58. doi:https://doi.org/10.1016/j.psyneuen.2013.12.018

Baer, R. A., Smith, G. T., Lykins, E., Button, D., Krietemeyer, J., Sauer, S., . . . Williams, J. M. G. (2008). Construct Validity of the Five Facet Mindfulness Questionnaire in Meditating and Nonmeditating Samples. *Assessment, 15*(3), 329-342. doi:10.1177/1073191107313003

Bishop, S. R., Lau, M., Shapiro, S., Carlson, L., Anderson, N. D., Carmody, J., . . . Devins, G. (2004). Mindfulness: A Proposed Operational Definition. *Clinical Psychology: Science and Practice, 11*(3), 230-241. doi:10.1093/clipsy.bph077

Brown, K. W., Weinstein, N., & Creswell, J. D. (2012). Trait mindfulness modulates neuroendocrine and affective responses to social evaluative threat. *Psychoneuroendocrinology, 37*(12), 2037-2041. doi:10.1016/j.psyneuen.2012.04.003

Chida, Y., & Hamer, M. (2008). Chronic psychosocial factors and acute physiological responses to laboratory-induced stress in healthy populations: a quantitative review of 30 years of investigations. *Psychol Bull, 134*(6), 829-885. doi:10.1037/a0013342

Chida, Y., & Steptoe, A. (2009). The Association of Anger and Hostility With Future Coronary Heart Disease. *Journal of the American College of Cardiology, 53*(11), 936. doi:10.1016/j.jacc.2008.11.044

Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: a meta-analysis of prospective evidence. *Hypertension, 55*(4), 1026-1032. doi:10.1161/hypertensionaha.109.146621

Christenfeld, N., Glynn, L. M., & Gerin, W. (2000). On the reliable assessment of cardiovascular recovery: An application of curve-fitting techniques. *Psychophysiology, 37*(4), 543-550. doi:10.1017/S0048577200982246

Cohen, S., Hamrick, N., Rodriguez, M. S., Feldman, P. J., Rabin, B. S., & Manuck, S. B. (2002). Reactivity and Vulnerability to Stress-Associated Risk for Upper Respiratory Illness. *Psychosomatic Medicine, 64*(2), 302-310. Retrieved from

https://journals.lww.com/psychosomaticmedicine/Fulltext/2002/03000/Reactivity_and_Vulnerability_to_Stress_Associated.14.aspx

Creswell, J. D., Pacilio, L. E., Lindsay, E. K., & Brown, K. W. (2014). Brief mindfulness meditation training alters psychological and neuroendocrine responses to social evaluative stress. *Psychoneuroendocrinology, 44*, 1-12. doi:10.1016/j.psyneuen.2014.02.007

Crosswell, A. D., Moreno, P. I., Raposa, E. B., Motivala, S. J., Stanton, A. L., Ganz, P. A., & Bower, J. E. (2017). Effects of mindfulness training on emotional and physiologic recovery from induced negative affect. *Psychoneuroendocrinology, 86*, 78-86. doi:10.1016/j.psyneuen.2017.08.003

Desbordes, G., Gard, T., Hoge, E. A., Hölzel, B. K., Kerr, C., Lazar, S. W., . . . Vago, D. R. (2015). Moving Beyond Mindfulness: Defining Equanimity as an Outcome Measure in Meditation and Contemplative Research. *Mindfulness, 6*(2), 356-372. doi:10.1007/s12671-013-0269-8

Dreyfus, G. (2011). Is mindfulness present-centred and non-judgmental? A discussion of the cognitive dimensions of mindfulness. *Contemporary Buddhism, 12*(1), 41-54. doi:10.1080/14639947.2011.564815

Enders, C. K. (2017). Multiple imputation as a flexible tool for missing data handling in clinical research. *Behav Res Ther, 98*, 4-18. doi:10.1016/j.brat.2016.11.008

Epel, E. S., McEwen, B. S., & Ickovics, J. R. (1998). Embodying Psychological Thriving: Physical Thriving in Response to Stress. *Journal of Social Issues, 54*(2), 301-322. doi:10.1111/j.1540-4560.1998.tb01220.x

Erceg-Hurn, D. M., & Mirosevich, V. M. (2008). Modern robust statistical methods: an easy way to maximize the accuracy and power of your research. *Am Psychol, 63*(7), 591-601. doi:10.1037/0003-066x.63.7.591

Farb, N. A. S., Anderson, A. K., & Segal, Z. V. (2012). The mindful brain and emotion regulation in mood disorders. *Canadian journal of psychiatry. Revue canadienne de psychiatrie, 57*(2), 70-77. doi:10.1177/070674371205700203

Fife, D., & Rodgers, J. L. (2019). Exonerating EDA, Expanding CDA: A Pragmatic Solution to the Replication Crisis. doi:10.31234/osf.io/5vfq6

Fife, D. A. (in press). The Eight Steps of Data Analysis: A Graphical Framework to Promote Sound Statistical Analysis. *Perspectives on Psychological Science*. doi:https://doi.org/10.31234/OSF.IO/R8G7C

Gamaiunova, L., Brandt, P. Y., Bondolfi, G., & Kliegel, M. (2019). Exploration of psychological mechanisms of the reduced stress response in long-term meditation practitioners. *Psychoneuroendocrinology, 104*, 143-151. doi:10.1016/j.psyneuen.2019.02.026

Gerin, W., Davidson, K. W., Christenfeld, N. J., Goyal, T., & Schwartz, J. E. (2006). The role of angry rumination and distraction in blood pressure recovery from emotional arousal. *Psychosom Med, 68*(1), 64-72. doi:10.1097/01.psy.0000195747.12404.aa

Gex-Fabry, M., Jermann, F., Kosel, M., Rossier, M. F., Van der Linden, M., Bertschy, G., . . . Aubry, J.-M. (2012). Salivary cortisol profiles in patients remitted from recurrent depression: One-year follow-up of a mindfulness-based cognitive therapy trial. *Journal of Psychiatric Research, 46*(1), 80-86. doi:https://doi.org/10.1016/j.jpsychires.2011.09.011

Giles, J. (2019). Relevance of the no-self theory in contemporary mindfulness. *Current Opinion in Psychology, 28*, 298-301. doi:https://doi.org/10.1016/j.copsyc.2019.03.016

Gordon, J. L., Ditto, B., & D'Antono, B. (2012). Cognitive depressive symptoms associated with delayed heart rate recovery following interpersonal stress in healthy men and women. *Psychophysiology, 49*(8), 1082-1089. doi:10.1111/j.1469-8986.2012.01397.x

Grant, C., Hobkirk, A., Persons, E., Hwang, V., & Danoff-Burg, S. (2013). Cardiovascular Reactivity to and Recovery from Stressful Tasks Following a Mindfulness Analog in College Students with a Family History of Hypertension. *Journal of Alternative & Complementary Medicine, 19*(4), 341-346. doi:10.1089/acm.2011.0880

Grossman, P. (2008). On measuring mindfulness in psychosomatic and psychological research. Journal of Psychosomatic Research, 64, 405-408. *Journal of Psychosomatic Research, 64*, 405-408. doi:10.1016/j.jpsychores.2008.02.001

Grossman, P. (2011). Defining mindfulness by how poorly I think I pay attention during everyday awareness and other intractable problems for psychology's (re)invention of mindfulness: comment on Brown et al. (2011). *Psychol Assess, 23*(4), 1034-1040; discussion 1041-1036. doi:10.1037/a0022713

Grossman, P., Niemann, L., Schmidt, S., & Walach, H. (2004). Mindfulness-based stress reduction and health benefits: A meta-analysis. *Journal of Psychosomatic Research, 57*(1), 35-43. doi:https://doi.org/10.1016/S0022-3999(03)00573-7

Gu, J., Strauss, C., Bond, R., & Cavanagh, K. (2015). How do mindfulness-based cognitive therapy and mindfulness-based stress reduction improve mental health and wellbeing? A systematic review and meta-analysis of mediation studies. *Clinical Psychology Review, 37*, 1-12. doi:https://doi.org/10.1016/j.cpr.2015.01.006

Hadash, Y., Segev, N., Tanay, G., Goldstein, P., & Bernstein, A. (2016). The Decoupling Model of Equanimity: Theory, Measurement, and Test in a Mindfulness Intervention. *Mindfulness, 7*(5), 1214-1226. doi:10.1007/s12671-016-0564-2

Hoffman, L., & Rovine, M. J. (2007). Multilevel models for the experimental psychologist: Foundations and illustrative examples. *Behavior Research Methods, 39*(1), 101-117. doi:10.3758/BF03192848

Hoge, E. A., Bui, E., Marques, L., Metcalf, C. A., Morris, L. K., Robinaugh, D. J., . . . Simon, N. M. (2013). Randomized controlled trial of mindfulness meditation for generalized anxiety disorder: effects on anxiety and stress reactivity. *J Clin Psychiatry, 74*(8), 786-792. doi:10.4088/JCP.12m08083

Hutchinson, J. G., & Ruiz, J. M. (2011). Neuroticism and Cardiovascular Response in Women: Evidence of Effects on Blood Pressure Recovery. *Journal of Personality, 79*(2), 277-302. doi:10.1111/j.1467-6494.2010.00679.x

Kabat-Zinn, J. (1994). *Wherever you go, there you are : mindfulness meditation in everyday life*. New York: Hyperion.

Keng, S. L., Smoski, M. J., & Robins, C. J. (2011). Effects of mindfulness on psychological health: a review of empirical studies. *Clin Psychol Rev, 31*(6), 1041-1056. doi:10.1016/j.cpr.2011.04.006

Keogh, K., & Creaven, A.-M. (2017). Evaluating the Impact of a Brief Artistic Intervention on Cardiovascular Recovery From Acute Stress. *Art Therapy: Journal of the American Art Therapy Association, 34*(4), 167-175. doi:10.1080/07421656.2017.1386038

Key, B. L., Campbell, T. S., Bacon, S. L., & Gerin, W. (2008). The influence of trait and state rumination on cardiovascular recovery from a negative emotional stressor. *Journal of Behavioral Medicine, 31*(3), 237-248. doi:10.1007/s10865-008-9152-9

Kraus, S., & Sears, S. (2009). Measuring the immeasurables: Development and initial validation of the Self-Other Four Immeasurables (SOFI) Scale based on Buddhist teachings on loving kindness, compassion, joy, and equanimity. *Social Indicators Research, 92*(1), 169-181. doi:10.1007/s11205-008-9300-1

Linden, W., Earle, T. L., Gerin, W., & Christenfeld, N. (1997). Physiological stress reactivity and recovery: conceptual siblings separated at birth? *J Psychosom Res, 42*(2), 117-135.

Lindsay, E. K., & Creswell, J. D. (2017). Mechanisms of mindfulness training: Monitor and Acceptance Theory (MAT). *Clinical Psychology Review, 51*, 48-59. doi:https://doi.org/10.1016/j.cpr.2016.10.011

Lindsay, E. K., & Creswell, J. D. (2019). Mindfulness, acceptance, and emotion regulation: perspectives from Monitor and Acceptance Theory (MAT). *Current Opinion in Psychology, 28*, 120-125. doi:https://doi.org/10.1016/j.copsyc.2018.12.004

Lindsay, E. K., Young, S., Smyth, J. M., Brown, K. W., & Creswell, J. D. (2018). Acceptance lowers stress reactivity: Dismantling mindfulness training in a randomized controlled trial. *Psychoneuroendocrinology, 87*, 63-73. doi:https://doi.org/10.1016/j.psyneuen.2017.09.015

Llabre, M. M., Spitzer, S., Siegel, S., Saab, P. G., & Schneiderman, N. (2004). Applying latent growth curve modeling to the investigation of individual differences in cardiovascular recovery from stress. *Psychosom Med, 66*(1), 29-41.

Ludwig, D. S., & Kabat-Zinn, J. (2008). Mindfulness in medicine. *Jama, 300*(11), 1350-1352. doi:10.1001/jama.300.11.1350

Lundman, B., Strandberg, G., Eisemann, M., Gustafson, Y., & Brulin, C. (2007). Psychometric properties of the Swedish version of the Resilience Scale. *Scand J Caring Sci, 21*(2), 229-237. doi:10.1111/j.1471-6712.2007.00461.x

Mack, J. W., Nilsson, M., Balboni, T., Friedlander, R. J., Block, S. D., Trice, E., & Prigerson, H. G. (2008). Peace, Equanimity, and Acceptance in the cancer experience (PEACE). *Cancer, 112*(11), 2509-2517. doi:10.1002/cncr.23476

Matthews, K. A., Woodall, K. L., & Allen, M. T. (1993). Cardiovascular reactivity to stress predicts future blood pressure status. *Hypertension, 22*(4), 479-485. doi:10.1161/01.hyp.22.4.479

McEwen, B. S. (1998). Stress, adaptation, and disease. Allostasis and allostatic load. *Ann N Y Acad Sci, 840*, 33-44. doi:10.1111/j.1749-6632.1998.tb09546.x

Mezzacappa, E. S., Kelsey, R. M., Katkin, E. S., & Sloan, R. P. (2001). Vagal Rebound and Recovery From Psychological Stress. *Psychosomatic Medicine, 63*(4), 650-657. Retrieved from https://journals.lww.com/psychosomaticmedicine/Fulltext/2001/07000/Vagal_Rebound_and_Recovery_From_Psychological.18.aspx

Morgan, N., Irwin, M. R., Chung, M., & Wang, C. (2014). The Effects of Mind-Body Therapies on the Immune System: Meta-Analysis. *PLoS One, 9*(7), e100903. doi:10.1371/journal.pone.0100903

Neumann, S. A., Waldstein, S. R., Sellers, J. J., III, Thayer, J. F., & Sorkin, J. D. (2004). Hostility and Distraction Have Differential Influences on Cardiovascular Recovery From Anger Recall in Women. *Health Psychology, 23*(6), 631-640. doi:10.1037/0278-6133.23.6.631

Nyklicek, I., Mommersteeg, P. M., Van Beugen, S., Ramakers, C., & Van Boxtel, G. J. (2013). Mindfulness-based stress reduction and physiological activity during acute stress: a randomized controlled trial. *Health Psychol, 32*(10), 1110-1113. doi:10.1037/a0032200

O'Leary, K., O'Neill, S., & Dockray, S. (2016). A systematic review of the effects of mindfulness interventions on cortisol. *Journal of Health Psychology, 21*(9), 2108-2121. doi:10.1177/1359105315569095

Olendzki, A. (2006). The transformative impact of non-self. In D. K. Nauriyal, M. S. Drummond, & Y. B. Lal (Eds.), *Buddhist thought and applied psychological research: Transcending the boundaries* (pp. pp. 250-261). Abingdon, UK: Routledge.

Pace, T. W. W., Negi, L. T., Adame, D. D., Cole, S. P., Sivilli, T. I., Brown, T. D., . . . Raison, C. L. (2009). Effect of compassion meditation on neuroendocrine, innate immune and behavioral responses to psychosocial stress. *Psychoneuroendocrinology, 34*(1), 87-98. doi:https://doi.org/10.1016/j.psyneuen.2008.08.011

Pieper, S., & Brosschot, J. F. (2005). Prolonged stress-related cardiovascular activation: Is there any? *Annals of Behavioral Medicine, 30*(2), 91-103. doi:10.1207/s15324796abm3002_1

Puttonen, S., Kivimaki, M., Elovainio, M., Pulkki-Raback, L., Hintsanen, M., Vahtera, J., . . . Keltikangas-Jarvinen, L. (2009). Shift work in young adults and carotid artery intima-media thickness: The Cardiovascular Risk in Young Finns study. *Atherosclerosis, 205*(2), 608-613. doi:10.1016/j.atherosclerosis.2009.01.016

Rinpoche, K. T. (2002). *Essential Practice*. Ithica, NY: Snow Lion Publications.

Rosenkranz, M. A., Lutz, A., Perlman, D. M., Bachhuber, D. R. W., Schuyler, B. S., MacCoon, D. G., & Davidson, R. J. (2016). Reduced stress and inflammatory responsiveness in experienced meditators compared to a matched healthy control group. *Psychoneuroendocrinology, 68*, 117-125. doi:https://doi.org/10.1016/j.psyneuen.2016.02.013

Routledge, F. S., McFetridge-Durdle, J. A., Macdonald, M., Breau, L., & Campbell, T. (2015). The effect of exercise and distraction on blood pressure recovery following an anger-provoking stressor in normotensive young adults. *Journal of Psychophysiology, 29*(2), 45-54. doi:10.1027/0269-8803/a000133

Sahdra, B., Shaver, P., & Brown, K. (2010). A Scale to Measure Nonattachment: A Buddhist Complement to Western Research on Attachment and Adaptive Functioning. *Journal of personality assessment, 92*, 116-127. doi:10.1080/00223890903425960

Salzberg, S. (1995). *Loving-Kindness: The Revolutionary Art of Happiness*. Boston, MA: Shambhala Publications.

Schuler, J. L., & O'Brien, W. H. (1997). Cardiovascular recovery from stress and hypertension risk factors: a meta-analytic review. *Psychophysiology, 34*(6), 649-659.

Shapiro, S. L., Carlson, L. E., Astin, J. A., & Freedman, B. (2006). Mechanisms of mindfulness. *J Clin Psychol, 62*(3), 373-386. doi:10.1002/jclp.20237

Skinner, T. C., Anstey, C., Baird, S., Foreman, M., Kelly, A., & Magee, C. (2008). Mindfulness and stress reactivity: a preliminary investigation. *Spirituality and Health International, 9*(4), 241-248. doi:doi:10.1002/shi.356

Steffen, P. R., & Larson, M. J. (2015). A Brief Mindfulness Exercise Reduces Cardiovascular Reactivity During a Laboratory Stressor Paradigm. *Mindfulness, 6*(4), 803-811. doi:10.1007/s12671-014-0320-4

Steptoe, A., Hamer, M., & Chida, Y. (2007). The effects of acute psychological stress on circulating inflammatory factors in humans: a review and meta-analysis. *Brain, behavior, and immunity, 21*(7), 901-912. doi:10.1016/j.bbi.2007.03.011

Steptoe, A., & Wardle, J. (2005). Cardiovascular stress responsivity, body mass and abdominal adiposity [Press release]

Stewart, J. C., & France, C. R. (2001). Cardiovascular recovery from stress predicts longitudinal changes in blood pressure. *Biol Psychol, 58*(2), 105-120.

Stewart, J. C., Janicki, D. L., & Kamarck, T. W. (2006). Cardiovascular reactivity to and recovery from psychological challenge as predictors of 3-year change in blood pressure. *Health Psychology, 25*(1), 111-118. doi:10.1037/0278-6133.25.1.111

Suarez, E. C., Saab, P. G., Llabre, M. M., Kuhn, C. M., & Zimmerman, E. (2004). Ethnicity, gender, and age effects on adrenoceptors and physiological responses to emotional stress. *Psychophysiology, 41*(3), 450-460. doi:10.1111/j.1469-8986.00161.x

Trivedi, R., Sherwood, A., Strauman, T. J., & Blumenthal, J. A. (2008). Laboratory-based blood pressure recovery is a predictor of ambulatory blood pressure. *Biological Psychology, 77*(3), 317-323. doi:https://doi.org/10.1016/j.biopsycho.2007.11.004

Van Dam, N. T., van Vugt, M. K., Vago, D. R., Schmalzl, L., Saron, C. D., Olendzki, A., . . . Meyer, D. E. (2018). Mind the Hype: A Critical Evaluation and Prescriptive Agenda for Research on Mindfulness and Meditation. *Perspect Psychol Sci, 13*(1), 36-61. doi:10.1177/1745691617709589

Visted, E., Vøllestad, J., Nielsen, M. B., & Nielsen, G. H. (2015). The Impact of Group-Based Mindfulness Training on Self-Reported Mindfulness: a Systematic Review and Meta-analysis. *Mindfulness, 6*(3), 501-522. doi:10.1007/s12671-014-0283-5

Willmann, M., Langlet, C., Hainaut, J.-P., & Bolmont, B. (2012). The time course of autonomic parameters and muscle tension during recovery following a moderate cognitive stressor: Dependency on trait anxiety level. *International Journal of Psychophysiology, 84*(1), 51-58. doi:https://doi.org/10.1016/j.ijpsycho.2012.01.009

Appendix A

Statistical Analyses in Regression Notation

Hypothesis 1

Reduced model:
Recovery = $b_0 + b_1(\text{Age}) + b_2(\text{Sex}) + b_3(\text{BMI})$
Full model:
Recovery = $b_0 + b_1(\text{Age}) + b_2(\text{Sex}) + b_3(\text{BMI}) + b_4(\text{Equanimity})$

Hypothesis 2

Reduced model:
Reactivity = $b_0 + b_1(\text{Age}) + b_2(\text{Sex}) + b_3(\text{BMI})$
Full model:
Reactivity = $b_0 + b_1(\text{Age}) + b_2(\text{Sex}) + b_3(\text{BMI}) + b_4(\text{Equanimity})$

Hypothesis 3

Reduced model:
Recovery = $b_0 + b_1(\text{Age}) + b_2(\text{Sex}) + b_3(\text{BMI})$
Full model:
Recovery = $b_0 + b_1(\text{Age}) + b_2(\text{Sex}) + b_3(\text{BMI}) + b_4(\text{Time})$

Hypothesis 4

Reduced model:
Recovery = $b_0 + b_1(\text{Age}) + b_2(\text{Sex}) + b_3(\text{BMI}) + b_4(\text{Time})$
Full model:
Recovery = $b_0 + b_1(\text{Age}) + b_2(\text{Sex}) + b_3(\text{BMI}) + b_4(\text{Time}) + b_5(\text{Equanimity})$

Appendix B

R Code for Statistical Analyses

Datasets are available upon request

Visualizations and analyses make use of the following packages: fifer, flexplot, ggplot2, cowplot

Hypothesis I

```
d$Gender = factor(d$Gender)

#Simple Change Scores
added.plot(HRrec_sct1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(SBPrec_sct1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(DBPrec_sct1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(HRrec_sct2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
added.plot(SBPrec_sct2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
added.plot(DBPrec_sct2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
 #model comparison
   #HR T1
mod.schr.hyp1.t1.full = lm(HRrec_sct1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.schr.hyp1.t1.full)
mod.schr.hyp1.t1.reduced = lm(HRrec_sct1~Age+Gender+BMI, data = d)
visualize(mod.schr.hyp1.t1.reduced)
compare.fits(HRrec_sct1~equa_t1|Age+Gender+BMI, data = d, mod.schr.hyp1.t1.full,
        mod.schr.hyp1.t1.reduced)
model.comparison(mod.schr.hyp1.t1.full, mod.schr.hyp1.t1.reduced)
   #SBP T1
mod.scsbp.hyp1.t1.full = lm(SBPrec_sct1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.scsbp.hyp1.t1.full)
mod.scsbp.hyp1.t1.reduced = lm(SBPrec_sct1~Age+Gender+BMI, data = d)
visualize(mod.scsbp.hyp1.t1.reduced)
compare.fits(SBPrec_sct1~equa_t1|Age+Gender+BMI, data = d, mod.scsbp.hyp1.t1.full,
        mod.scsbp.hyp1.t1.reduced)
model.comparison(mod.scsbp.hyp1.t1.full, mod.scsbp.hyp1.t1.reduced)
   #DBP T1
mod.scdbp.hyp1.t1.full = lm(DBPrec_sct1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.scdbp.hyp1.t1.full)
mod.scdbp.hyp1.t1.reduced = lm(DBPrec_sct1~Age+Gender+BMI, data = d)
visualize(mod.scdbp.hyp1.t1.reduced)
compare.fits(DBPrec_sct1~equa_t1|Age+Gender+BMI, data = d, mod.scdbp.hyp1.t1.full,
        mod.scdbp.hyp1.t1.reduced)
model.comparison(mod.scdbp.hyp1.t1.full, mod.scdbp.hyp1.t1.reduced)
   #HR T2
```

```
mod.schr.hyp1.t2.full = lm(HRrec_sct2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.schr.hyp1.t2.full)
mod.schr.hyp1.t2.reduced = lm(HRrec_sct2~Age+Gender+BMI, data = d)
visualize(mod.schr.hyp1.t2.reduced)
compare.fits(HRrec_sct2~equa_t2|Age+Gender+BMI, data = d, mod.schr.hyp1.t2.full,
        mod.schr.hyp1.t2.reduced)
model.comparison(mod.schr.hyp1.t2.full, mod.schr.hyp1.t2.reduced)
   #SBP T2
mod.scsbp.hyp1.t2.full = lm(SBPrec_sct2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.scsbp.hyp1.t2.full)
mod.scsbp.hyp1.t2.reduced = lm(SBPrec_sct2~Age+Gender+BMI, data = d)
visualize(mod.scsbp.hyp1.t2.reduced)
compare.fits(SBPrec_sct2~equa_t2|Age+Gender+BMI, data = d, mod.scsbp.hyp1.t2.full,
        mod.scsbp.hyp1.t2.reduced)
model.comparison(mod.scsbp.hyp1.t2.full, mod.scsbp.hyp1.t2.reduced) #reduced
   #robust model due to positive skew in outcome variable
mod.scsbp.hyp1.t2.robust = rlm(SBPrec_sct2~Age+Gender+BMI+equa_t2, data = d)
compare.fits(SBPrec_sct2~equa_t2|Age+Gender+BMI, data = d, mod.scsbp.hyp1.t2.full,
        mod.scsbp.hyp1.t2.robust)
model.comparison(mod.scsbp.hyp1.t2.full, mod.scsbp.hyp1.t2.robust)
   #DBP T2
mod.scdbp.hyp1.t2.full = lm(DBPrec_sct2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.scdbp.hyp1.t2.full)
mod.scdbp.hyp1.t2.reduced = lm(DBPrec_sct2~Age+Gender+BMI, data = d)
visualize(mod.scdbp.hyp1.t2.reduced)
compare.fits(DBPrec_sct2~equa_t2|Age+Gender+BMI, data = d, mod.scdbp.hyp1.t2.full,
        mod.scdbp.hyp1.t2.reduced)
model.comparison(mod.scdbp.hyp1.t2.full, mod.scdbp.hyp1.t2.reduced)
   #robust model due to positive skew in outcome variable
mod.scdbp.hyp1.t2.robust = rlm(DBPrec_sct2~Age+Gender+BMI+equa_t2, data = d)
compare.fits(DBPrec_sct2~equa_t2|Age+Gender+BMI, data = d, mod.scdbp.hyp1.t2.full,
        mod.scdbp.hyp1.t2.robust)
model.comparison(mod.scdbp.hyp1.t2.full, mod.scdbp.hyp1.t2.robust)

#Residualized Change
added.plot(HRrec_rest1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(SBPrec_rest1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(DBPrec_rest1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(HRrec_rest2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
added.plot(SBPrec_rest2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
added.plot(DBPrec_rest2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
 #model comparison
   #HR T1
mod.reshr.hyp1.t1.full = lm(HRrec_rest1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.reshr.hyp1.t1.full)
mod.reshr.hyp1.t1.reduced = lm(HRrec_rest1~Age+Gender+BMI, data = d)
```

```
visualize(mod.reshr.hyp1.t1.reduced)
compare.fits(HRrec_rest1~equa_t1|Age+Gender+BMI, data = d, mod.reshr.hyp1.t1.full,
        mod.reshr.hyp1.t1.reduced)
model.comparison(mod.reshr.hyp1.t1.full, mod.reshr.hyp1.t1.reduced)
   #SBP T1
mod.ressbp.hyp1.t1.full = lm(SBPrec_rest1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.ressbp.hyp1.t1.full)
mod.ressbp.hyp1.t1.reduced = lm(SBPrec_rest1~Age+Gender+BMI, data = d)
visualize(mod.reshr.hyp1.t1.reduced)
compare.fits(SBPrec_rest1~equa_t1|Age+Gender+BMI, data = d,
mod.ressbp.hyp1.t1.full, mod.ressbp.hyp1.t1.reduced)
model.comparison(mod.ressbp.hyp1.t1.full, mod.ressbp.hyp1.t1.reduced)
   #DBP T1
mod.resdbp.hyp1.t1.full = lm(DBPrec_rest1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.resdbp.hyp1.t1.full)
mod.resdbp.hyp1.t1.reduced = lm(DBPrec_rest1~Age+Gender+BMI, data = d)
visualize(mod.resdbp.hyp1.t1.reduced)
compare.fits(DBPrec_rest1~equa_t1|Age+Gender+BMI, data = d,
mod.resdbp.hyp1.t1.full, mod.resdbp.hyp1.t1.reduced)
model.comparison(mod.resdbp.hyp1.t1.full, mod.resdbp.hyp1.t1.reduced)
   #HR T2
mod.reshr.hyp1.t2.full = lm(HRrec_rest2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.reshr.hyp1.t2.full)
mod.reshr.hyp1.t2.reduced = lm(HRrec_rest2~Age+Gender+BMI, data = d)
visualize(mod.reshr.hyp1.t2.reduced)
compare.fits(HRrec_rest2~equa_t2|Age+Gender+BMI, data = d, mod.reshr.hyp1.t2.full,
        mod.reshr.hyp1.t2.reduced)
model.comparison(mod.reshr.hyp1.t2.full, mod.reshr.hyp1.t2.reduced)
   #robust model due to outlier and non-linearity
mod.reshr.hyp1.t2.robust = rlm(HRrec_rest2~Age+Gender+BMI+equa_t2, data = d)
compare.fits(HRrec_rest2~equa_t2|Age+Gender+BMI, data = d, mod.reshr.hyp1.t2.full,
        mod.reshr.hyp1.t2.robust)
model.comparison(mod.reshr.hyp1.t2.full, mod.reshr.hyp1.t2.robust)
   #SBP T2
mod.ressbp.hyp1.t2.full = lm(SBPrec_rest2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.ressbp.hyp1.t2.full)
mod.ressbp.hyp1.t2.reduced = lm(SBPrec_rest2~Age+Gender+BMI, data = d)
visualize(mod.ressbp.hyp1.t2.reduced)
compare.fits(SBPrec_rest2~equa_t2|Age+Gender+BMI, data = d,
mod.ressbp.hyp1.t2.full, mod.ressbp.hyp1.t2.reduced)
model.comparison(mod.ressbp.hyp1.t2.full, mod.ressbp.hyp1.t2.reduced)
   #DBP T2
mod.resdbp.hyp1.t2.full = lm(DBPrec_rest2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.resdbp.hyp1.t2.full)
mod.resdbp.hyp1.t2.reduced = lm(DBPrec_rest2~Age+Gender+BMI, data = d)
visualize(mod.resdbp.hyp1.t2.reduced)
```

```
compare.fits(DBPrec_rest2~equa_t2|Age+Gender+BMI, data = d,
mod.resdbp.hyp1.t2.full, mod.resdbp.hyp1.t2.reduced)
model.comparison(mod.resdbp.hyp1.t2.full, mod.resdbp.hyp1.t2.reduced)
   #robust model comparison due to outlier in outcome variable
mod.resdbp.hyp1.t2.robust = rlm(DBPrec_rest2~Age+Gender+BMI+equa_t2, data = d)
compare.fits(DBPrec_rest2~equa_t2|Age+Gender+BMI, data = d,
mod.resdbp.hyp1.t2.full, mod.resdbp.hyp1.t2.robust)
model.comparison(mod.scdbp.hyp1.t2.full, mod.scdbp.hyp1.t2.robust)

#Percent recovery
added.plot(HRrec_perct1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(SBPrec_perct1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(DBPrec_perct1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(HRrec_perct2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
added.plot(SBPrec_perct2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
added.plot(DBPrec_perct2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
 #model comparison
   #HR T1
mod.perchr.hyp1.t1.full = lm(HRrec_perct1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.perchr.hyp1.t1.full)
mod.perchr.hyp1.t1.reduced = lm(HRrec_perct1~Age+Gender+BMI, data = d)
visualize(mod.perchr.hyp1.t1.reduced)
compare.fits(HRrec_perct1~equa_t1|Age+Gender+BMI, data = d,
mod.perchr.hyp1.t1.full, mod.perchr.hyp1.t1.reduced)
model.comparison(mod.perchr.hyp1.t1.full, mod.perchr.hyp1.t1.reduced)
   #robust model due to outliers
mod.perchr.hyp1.t1.full.robust = rlm(HRrec_perct1~Age+Gender+BMI+equa_t1, data =
d)
compare.fits(HRrec_perct1~equa_t1|Age+Gender+BMI, data = d,
mod.perchr.hyp1.t1.full, mod.perchr.hyp1.t1.full.robust)
model.comparison(mod.perchr.hyp1.t1.full, mod.perchr.hyp1.t1.full.robust)
   #SBP T1
mod.percsbp.hyp1.t1.full = lm(SBPrec_perct1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.percsbp.hyp1.t1.full)
mod.percsbp.hyp1.t1.reduced = lm(SBPrec_perct1~Age+Gender+BMI, data = d)
visualize(mod.percsbp.hyp1.t1.reduced)
compare.fits(SBPrec_perct1~equa_t1|Age+Gender+BMI, data = d,
mod.percsbp.hyp1.t1.full, mod.percsbp.hyp1.t1.reduced)
model.comparison(mod.percsbp.hyp1.t1.full, mod.percsbp.hyp1.t1.reduced)
   #DBP T1
mod.percdbp.hyp1.t1.full = lm(DBPrec_perct1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.percdbp.hyp1.t1.full)
mod.percdbp.hyp1.t1.reduced = lm(DBPrec_perct1~Age+Gender+BMI, data = d)
visualize(mod.percdbp.hyp1.t1.reduced)
compare.fits(DBPrec_perct1~equa_t1|Age+Gender+BMI, data = d,
mod.percdbp.hyp1.t1.full, mod.percdbp.hyp1.t1.reduced)
```

```
model.comparison(mod.percdbp.hyp1.t1.full, mod.percdbp.hyp1.t1.reduced)
   #HR T2
mod.perchr.hyp1.t2.full = lm(HRrec_perct2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.perchr.hyp1.t2.full
mod.perchr.hyp1.t2.reduced = lm(HRrec_perct2~Age+Gender+BMI, data = d)
visualize(mod.perchr.hyp1.t2.reduced)
compare.fits(HRrec_perct2~equa_t2|Age+Gender+BMI, data = d,
mod.perchr.hyp1.t2.full, mod.perchr.hyp1.t2.reduced)
model.comparison(mod.perchr.hyp1.t2.full, mod.perchr.hyp1.t2.reduced)
   #robust due to non-normality
mod.perchr.hyp1.t2.full.robust = rlm(HRrec_perct2~Age+Gender+BMI+equa_t2, data =
d)
compare.fits(HRrec_perct2~equa_t2|Age+Gender+BMI, data = d,
mod.perchr.hyp1.t2.full, mod.perchr.hyp1.t2.full.robust)
model.comparison(mod.perchr.hyp1.t2.full, mod.perchr.hyp1.t2.full.robust)
   #SBP T2
mod.percsbp.hyp1.t2.full = lm(SBPrec_perct2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.percsbp.hyp1.t2.full)
mod.percsbp.hyp1.t2.reduced = lm(SBPrec_perct2~Age+Gender+BMI, data = d)
visualize(mod.percsbp.hyp1.t2.reduced)
compare.fits(SBPrec_perct2~equa_t2|Age+Gender+BMI, data = d,
mod.percsbp.hyp1.t2.full, mod.percsbp.hyp1.t2.reduced)
model.comparison(mod.percsbp.hyp1.t2.full, mod.percsbp.hyp1.t2.reduced)
   #robust model due to outlier
mod.percsbp.hyp1.t2.full.robust = rlm(SBPrec_perct2~Age+Gender+BMI+equa_t2, data
= d)
compare.fits(SBPrec_perct2~equa_t2|Age+Gender+BMI, data = d,
mod.percsbp.hyp1.t2.full, mod.percsbp.hyp1.t2.full.robust)
model.comparison(mod.percsbp.hyp1.t2.full, mod.percsbp.hyp1.t2.full.robust)
   #DBP T2
mod.percdbp.hyp1.t2.full = lm(DBPrec_perct2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.percdbp.hyp1.t2.full)
mod.percdbp.hyp1.t2.reduced = lm(DBPrec_perct2~Age+Gender+BMI, data = d)
visualize(mod.perchr.hyp1.t2.reduced)
compare.fits(DBPrec_perct2~equa_t2|Age+Gender+BMI, data = d,
mod.percdbp.hyp1.t2.full, mod.percdbp.hyp1.t2.reduced)
model.comparison(mod.percdbp.hyp1.t2.full, mod.percdbp.hyp1.t2.reduced)
   #robust due to outliers
mod.percdbp.hyp1.t2.full.robust = rlm(DBPrec_perct2~Age+Gender+BMI+equa_t2, data
= d)
compare.fits(DBPrec_perct2~equa_t2|Age+Gender+BMI, data = d,
mod.percdbp.hyp1.t2.full, mod.percdbp.hyp1.t2.full.robust)
model.comparison(mod.percdbp.hyp1.t2.full, mod.percdbp.hyp1.t2.full.robust)
```

Hypothesis II

```
d$Gender = factor(d$Gender)

added.plot(HRrea_rest1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(SBPrea_rest1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(DBPrea_rest1~Age+Gender+BMI+equa_t1, data = d, method = "lm")
added.plot(HRrea_rest2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
added.plot(SBPrea_rest2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
added.plot(DBPrea_rest2~Age+Gender+BMI+equa_t2, data = d, method = "lm")
 #model comparison
   #HR T1
mod.reahr.hyp2.t1.full = lm(HRrea_rest1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.reahr.hyp2.t1.full)
mod.reahr.hyp2.t1.reduced = lm(HRrea_rest1~Age+Gender+BMI, data = d)
visualize(mod.reahr.hyp2.t1.reduced)
compare.fits(HRrea_rest1~equa_t1|Age+Gender+BMI, data = d, mod.reahr.hyp2.t1.full,
        mod.reahr.hyp2.t1.reduced)
model.comparison(mod.reahr.hyp2.t1.full, mod.reahr.hyp2.t1.reduced)
   #robust due to skew
mod.reahr.hyp2.t1.full.robust = rlm(HRrea_rest1~Age+Gender+BMI+equa_t1, data = d)
compare.fits(HRrea_rest1~equa_t1|Age+Gender+BMI, data = d, mod.reahr.hyp2.t1.full,
        mod.reahr.hyp2.t1.full.robust)
model.comparison(mod.reahr.hyp2.t1.full, mod.reahr.hyp2.t1.full.robust)
   #SBP T1
mod.reasbp.hyp2.t1.full = lm(SBPrea_rest1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.reasbp.hyp2.t1.full)
mod.reasbp.hyp2.t1.reduced = lm(SBPrea_rest1~Age+Gender+BMI, data = d)
visualize(mod.reasbp.hyp2.t1.reduced)
compare.fits(SBPrea_rest1~equa_t1|Age+Gender+BMI, data = d,
mod.reasbp.hyp2.t1.full, mod.reasbp.hyp2.t1.reduced)
model.comparison(mod.reasbp.hyp2.t1.full, mod.reasbp.hyp2.t1.reduced)
estimates(mod.reasbp.hyp2.t1.full)
summary(mod.reasbp.hyp2.t1.full)
   #DBP T1
mod.readbp.hyp2.t1.full = lm(DBPrea_rest1~Age+Gender+BMI+equa_t1, data = d)
visualize(mod.readbp.hyp2.t1.full)
mod.readbp.hyp2.t1.reduced = lm(DBPrea_rest1~Age+Gender+BMI, data = d)
visualize(mod.readbp.hyp2.t1.reduced)
compare.fits(DBPrea_rest1~equa_t1|Age+Gender+BMI, data = d,
mod.readbp.hyp2.t1.full, mod.readbp.hyp2.t1.reduced)
model.comparison(mod.readbp.hyp2.t1.full, mod.readbp.hyp2.t1.reduced) reduced
   #HR T2
mod.reahr.hyp2.t2.full = lm(HRrea_rest2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.reahr.hyp2.t2.full)
mod.reahr.hyp2.t2.reduced = lm(HRrea_rest2~Age+Gender+BMI, data = d)
```

```
visualize(mod.reahr.hyp2.t2.reduced)
compare.fits(HRrea_rest2~equa_t2|Age+Gender+BMI, data = d, mod.reahr.hyp2.t2.full,
        mod.reahr.hyp2.t2.reduced)
model.comparison(mod.reahr.hyp2.t2.full, mod.reahr.hyp2.t2.reduced)
   #robust due to skew
mod.reahr.hyp2.t2.full.robust = rlm(HRrea_rest2~Age+Gender+BMI+equa_t2, data = d)
compare.fits(HRrea_rest2~equa_t2|Age+Gender+BMI, data = d, mod.reahr.hyp2.t2.full,
        mod.reahr.hyp2.t2.full.robust)
model.comparison(mod.reahr.hyp2.t2.full, mod.reahr.hyp2.t2.full.robust)
   #SBP T2
mod.reasbp.hyp2.t2.full = lm(SBPrea_rest2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.reasbp.hyp2.t2.full)
mod.reasbp.hyp2.t2.reduced = lm(SBPrea_rest2~Age+Gender+BMI, data = d)
visualize(mod.reasbp.hyp2.t2.reduced)
compare.fits(SBPrea_rest2~equa_t2|Age+Gender+BMI, data = d,
mod.reasbp.hyp2.t2.full, mod.reasbp.hyp2.t2.reduced)
model.comparison(mod.reasbp.hyp2.t2.full, mod.reasbp.hyp2.t2.reduced)
   #DBP T2
mod.readbp.hyp2.t2.full = lm(DBPrea_rest2~Age+Gender+BMI+equa_t2, data = d)
visualize(mod.readbp.hyp2.t2.full)
mod.readbp.hyp2.t2.reduced = lm(DBPrea_rest2~Age+Gender+BMI, data = d)
visualize(mod.readbp.hyp2.t2.reduced)
compare.fits(DBPrea_rest2~equa_t2|Age+Gender+BMI, data = d,
mod.readbp.hyp2.t2.full, mod.readbp.hyp2.t2.reduced)
model.comparison(mod.readbp.hyp2.t2.full, mod.readbp.hyp2.t2.reduced)
```

Hypothesis III

```
q  = q %>% filter(Intervention != " ")
q$Gender =  factor(q$Gender)
q$Intervention = factor(q$Intervention)

#Simple change scores
added.plot(HRrec_sc~Age+Gender+BMI+Intervention, data = q, method = "lm")
added.plot(SBPrec_sc~Age+Gender+BMI+Intervention, data = q, method = "lm")
added.plot(DBPrec_sc~Age+Gender+BMI+Intervention, data = q, method = "lm")
 #Model comparison
   #HR
mod.schr.hyp3.full = lm(HRrec_sc~Age+Gender+BMI+Intervention, data = q)
visualize(mod.schr.hyp3.full)
mod.schr.hyp3.reduced = lm(HRrec_sc~Age+Gender+BMI, data = q)
visualize(mod.schr.hyp3.reduced)
compare.fits(HRrec_sc~Intervention|Age+Gender, data = q, mod.schr.hyp3.full,
        mod.schr.hyp3.reduced)
model.comparison(mod.schr.hyp3.full, mod.schr.hyp3.reduced)
   #SBP
```

```
mod.scsbp.hyp3.full = lm(SBPrec_sc~Age+Gender+BMI+Intervention, data = q)
visualize(mod.scsbp.hyp3.full, method = "lm")
mod.scsbp.hyp3.reduced = lm(SBPrec_sc~Age+Gender+BMI, data = q)
compare.fits(SBPrec_sc~Intervention|Age+Gender+BMI, data = q, mod.scsbp.hyp3.full,
        mod.scsbp.hyp3.reduced)
model.comparison(mod.scsbp.hyp3.full, mod.scsbp.hyp3.reduced)
estimates(mod.scsbp.hyp3.full, mc = FALSE)
summary(mod.scsbp.hyp3.full)
   #multiple imputation for missing data
imputed.estimates = impute.me(mod.scsbp.hyp3.full, data = q, silent = F, return.mod = F)
imputed.estimates
summary(mod.scsbp.hyp3.full)
model.comparison(mod.scsbp.hyp3.full, imputed.estimates)
imputed.estimates = impute.me(mod.scsbp.hyp3.full, data = q, silent = F, return.mod = T)
compare.fits(SBPrec_sc~Intervention|Age+Gender+BMI, data = q, mod.scsbp.hyp3.full,
imputed.estimates)
   #robust check
mod.scsbp.hyp3.full.robust = rlm(SBPrec_sc~Age+Gender+BMI+Intervention, data = q)
mod.scsbp.hyp3.full = lm(SBPrec_sc~Age+Gender+BMI+Intervention, data = q)
compare.fits(SBPrec_sc~Intervention, data=q, mod.scsbp.hyp3.full.robust,
mod.scsbp.hyp3.full)
model.comparison(mod.scsbp.hyp3.full.robust, mod.scsbp.hyp3.full)
   #DBP
mod.scdbp.hyp3.full = lm(DBPrec_sc~Age+Gender+BMI+Intervention, data = q)
visualize(mod.scdbp.hyp3.full)
mod.scdbp.hyp3.reduced = lm(DBPrec_sc~Age+Gender+BMI, data = q)
visualize(mod.scdbp.hyp3.reduced)
compare.fits(DBPrec_sc~Intervention|Age+Gender+BMI, data = q, mod.scdbp.hyp3.full,
        mod.scdbp.hyp3.reduced)
model.comparison(mod.scdbp.hyp3.full, mod.scdbp.hyp3.reduced)
summary(mod.scdbp.hyp3.full)
   #multiple imputation for missing data
imputed.estimates = impute.me(mod.scdbp.hyp3.full, data = q, silent = F, return.mod =
F)
imputed.estimates
summary(mod.scdbp.hyp3.full) #the same
model.comparison(mod.scdbp.hyp3.full, imputed.estimates)
imputed.estimates = impute.me(mod.scdbp.hyp3.full, data = q, silent = F, return.mod =
T)
compare.fits(DBPrec_sc~Intervention|Age+Gender+BMI, data = q, mod.scdbp.hyp3.full,
imputed.estimates)
   #robust check
mod.scdbp.hyp3.full.robust = rlm(DBPrec_sc~Age+Gender+BMI+Intervention, data = q)
mod.scdbp.hyp3.full = lm(DBPrec_sc~Age+Gender+BMI+Intervention, data = q)
compare.fits(DBPrec_sc~Intervention, data=q, mod.scdbp.hyp3.full.robust,
mod.scdbp.hyp3.full)
```

```
model.comparison(mod.scdbp.hyp3.full.robust, mod.scdbp.hyp3.full)

#Residualized change scores
added.plot(HRrec_res~Age+Gender+BMI+Intervention, data = q, method = "lm")
added.plot(SBPrec_res~Age+Gender+BMI+Intervention, data = q, method = "lm")
added.plot(DBPrec_res~Age+Gender+BMI+Intervention, data = q, method = "lm")
 #Model comparison
   #HR
mod.reshr.hyp3.full = lm(HRrec_res~Age+Gender+BMI+Intervention, data = q)
visualize(mod.reshr.hyp3.full)
mod.reshr.hyp3.reduced = lm(HRrec_res~Age+Gender+BMI, data = q)
visualize(mod.reshr.hyp3.reduced)
compare.fits(HRrec_res~Intervention|Age+Gender+BMI, data = q, mod.reshr.hyp3.full,
          mod.reshr.hyp3.reduced)
model.comparison(mod.reshr.hyp3.full, mod.reshr.hyp3.reduced)
   #SBP
mod.ressbp.hyp3.full = lm(SBPrec_res~Age+Gender+BMI+Intervention, data = q)
visualize(mod.ressbp.hyp3.full)
mod.ressbp.hyp3.reduced = lm(SBPrec_res~Age+Gender+BMI, data = q)
visualize(mod.ressbp.hyp3.reduced)
compare.fits(SBPrec_res~Intervention|Age+Gender+BMI, data = q,
mod.ressbp.hyp3.full, mod.ressbp.hyp3.reduced)
model.comparison(mod.ressbp.hyp3.full, mod.ressbp.hyp3.reduced)
summary(mod.ressbp.hyp3.full)
   #DBP
mod.resdbp.hyp3.full = lm(DBPrec_res~Age+Gender+BMI+Intervention, data = q)
visualize(mod.resdbp.hyp3.full)
mod.resdbp.hyp3.reduced = lm(DBPrec_res~Age+Gender+BMI, data = q)
visualize(mod.resdbp.hyp3.reduced)
compare.fits(DBPrec_res~Intervention|Age+Gender+BMI, data = q,
mod.resdbp.hyp3.full, mod.resdbp.hyp3.reduced)
model.comparison(mod.resdbp.hyp3.full, mod.resdbp.hyp3.reduced)

#Percent recovery
added.plot(HRrec_perc~Age+Gender+BMI+Intervention, data = q, method = "lm")
added.plot(SBPrec_perc~Age+Gender+BMI+Intervention, data = q, method = "lm")
added.plot(DBPrec_perc~Age+Gender+BMI+Intervention, data = q, method = "lm")
 #Model comparison
   #HR
mod.perchr.hyp3.full = lm(HRrec_perc~Age+Gender+BMI+Intervention, data = q)
visualize(mod.perchr.hyp3.full)
mod.perchr.hyp3.reduced = lm(HRrec_perc~Age+Gender+BMI, data = q)
visualize(mod.perchr.hyp3.reduced)
compare.fits(HRrec_perc~Intervention|Age+Gender+BMI, data = q,
mod.perchr.hyp3.full, mod.perchr.hyp3.reduced)
model.comparison(mod.perchr.hyp3.full, mod.perchr.hyp3.reduced)
```

```
   #robust model due to  outliers
mod.perchr.hyp3.full.robust = rlm(HRrec_perc~Age+Gender+BMI+Intervention, data =
q)
mod.perchr.hyp3.full = lm(HRrec_perc~Age+Gender+BMI+Intervention, data = q)
compare.fits(HRrec_perc~Intervention, data=q, mod.perchr.hyp3.full.robust,
mod.perchr.hyp3.full)
model.comparison(mod.perchr.hyp3.full.robust, mod.perchr.hyp3.full))
   #SBP
mod.percsbp.hyp3.full = lm(SBPrec_perc~Age+Gender+BMI+Intervention, data = q)
visualize(mod.percsbp.hyp3.full)
mod.percsbp.hyp3.reduced = lm(SBPrec_perc~Age+Gender+BMI, data = q)
visualize(mod.percsbp.hyp3.reduced)
compare.fits(SBPrec_perc~Intervention|Age+Gender+BMI, data = q,
mod.percsbp.hyp3.full, mod.percsbp.hyp3.reduced)
model.comparison(mod.percsbp.hyp3.full, mod.percsbp.hyp3.reduced)
   #robust due to outliers
mod.percsbp.hyp3.full.robust = rlm(SBPrec_perc~Age+Gender+BMI+Intervention, data
= q)
mod.percsbp.hyp3.full = lm(SBPrec_perc~Age+Gender+BMI+Intervention, data = q)
compare.fits(SBPrec_perc~Intervention, data=q, mod.percsbp.hyp3.full.robust,
mod.percsbp.hyp3.full)
model.comparison(mod.scsbp.hyp3.full.robust, mod.scsbp.hyp3.full)
   #DBP
mod.percdbp.hyp3.full = lm(DBPrec_perc~Age+Gender+BMI+Intervention, data = q)
visualize(mod.percdbp.hyp3.full)
mod.percdbp.hyp3.reduced = lm(DBPrec_perc~Age+Gender+BMI, data = q)
visualize(mod.percdbp.hyp3.reduced)
compare.fits(DBPrec_perc~Intervention|Age+Gender+BMI, data = q,
mod.percdbp.hyp3.full, mod.percdbp.hyp3.reduced)
model.comparison(mod.percdbp.hyp3.full, mod.percdbp.hyp3.reduced)
   #robust due to outliers
mod.percdbp.hyp3.full.robust = rlm(DBPrec_perc~Age+Gender+BMI+Intervention, data
= q)
mod.percdbp.hyp3.full = lm(DBPrec_perc~Age+Gender+BMI+Intervention, data = q)
compare.fits(DBPrec_perc~Intervention, data=q, mod.percdbp.hyp3.full.robust,
mod.percdbp.hyp3.full)
model.comparison(mod.percdbp.hyp3.full.robust, mod.percdbp.hyp3.full)
```

Hypothesis IV

```
q$Gender =  factor(q$Gender)
require(cowplot)

  #SBPSC~Intervention
a = added.plot(SBPrec_sc~Age+Gender+BMI+Intervention, data = q, method = "lm")
```

```
b = added.plot(SBPrec_sc~Age+Gender+BMI+equa+Intervention, data = q, method =
"lm")
plot_grid(a,b)
mod.scsbp.hyp4.reduced = lm(SBPrec_sc~Age+Gender+BMI+Intervention, data = q)
mod.scsbp.hyp4.full = lm(SBPrec_sc~Age+Gender+BMI+Intervention+equa, data = q)
  mod.1 = lm(SBPrec_sc~Intervention, data = q)
  mod.2 = lm(SBPrec_sc~Intervention+equa, data = q)
  compare.fits(SBPrec_sc~Intervention|equa, data = q, mod.1, mod.2)
summary(mod.scsbp.hyp4.reduced)
estimates(mod.scsbp.hyp4.reduced, mc = FALSE)
summary(mod.scsbp.hyp4.full)
estimates(mod.scsbp.hyp4.full, mc = FALSE)

  #DBPSC~Intervention
a2 = added.plot(DBPrec_sc~Age+Gender+BMI+Intervention, data = q, method = "lm")
b2 = added.plot(DBPrec_sc~Age+Gender+BMI+equa+Intervention, data = q, method =
"lm")
plot_grid(a2,b2)
mod.scdbp.hyp4.reduced = lm(DBPrec_sc~Age+Gender+BMI+Intervention, data = q)
mod.scdbp.hyp4.full = lm(DBPrec_sc~Age+Gender+BMI+Intervention+equa, data = q)
summary(mod.scdbp.hyp4.reduced)
estimates(mod.scdbp.hyp4.reduced, mc = FALSE)
summary(mod.scdbp.hyp4.full)
estimates(mod.scdbp.hyp4.full, mc = FALSE)
```