EXPOSURE WRITING AS AN EXPOSURE BASED INTERVENTION

EXPRESSIVE WRITING AS AN EXPOSURE BASED THERAPY FOR DEPRESSION: AN INVESTIGATION OF EMOTION, COGNITION, AND PHYSIOLOGY

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LAY ABSTRACT

The current study examines the emotion, cognition, and physiology associated with an intervention, expressive writing (EW), which is commonly used as a treatment for anxiety and depression. Research has suggested that EW produces therapeutic outcome because it increases exposure to negative feelings. The current study tests the hypothesis that EW increases depressive emotion, cognition, and physiology. Results suggest that EW increases depressive emotion and cognition but does not alter some of the physiological parameters that have been associated with depression in prior research. An unpredicted exploratory result was that EW affected the relationship between heart rate and respiratory rate. Further investigation into the relationship between heart rate and respiratory rate during EW is warranted.

ABSTRACT

Although we have several therapeutic interventions for depression, we lack an understanding of the mechanisms that underlie these interventions. To gain a better understanding of the mental health conditions we treat, diagnoses we make, and interventions we use, mechanistic understandings are necessary. There is evidence that exposure to depressive emotion and cognitions can yield therapeutic outcome. The current study examines the physiology associated with an intervention, expressive writing (EW), which other research has shown to produce therapeutic outcomes because it increases exposure to negative feelings. The current study tests the hypothesis that EW increases depressive emotion, cognition, and physiology. Depression has been associated with decreased respiratory sinus arrhythmia (RSA) and increased heart rate (HR). RSA and HR were measured while participants did either EW or a control writing (CW) task. Because measures of RSA can be confounded by respiratory rate (RR), RR was also measured and statistically controlled for. Results revealed that EW does not alter RSA or HR. Interestingly, exploratory regression analyses between HR and RR during EW suggest that EW might trigger exposure to a depressive physiological state. Further investigation into the relationship between HR and RR during EW is warranted.

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TABLES AND FIGURES

LIST OF ABBREVIATIONS

AR = analytical rumination score
ARH = Analytical rumination hypothesis
ARQ = analytical rumination questionnaire
ARQ-M = analytical rumination questionnaire - modified
ASR = analyze-to-solve rumination
AUR = analyze-to-understand rumination
BMI = body mass index
CW = control writing
DMN = dorsal motor nucleus
DSM = Diagnostic and Statistical Manual of Mental Disorders
EBT = exposure based therapy
ECG = electrocardiogram
EW = expressive writing
GEE = generalized estimating equations
GLM = generalized linear model
HR = heart rate
HRV = heart rate variability
LIWC = Linguistic Inquiry Word Count
NA = nucleus ambiguus
NIMH = National Institute of Mental Health
PQ = problem questionnaire
PSNS = parasympathetic nervous system
RDoC = Research Domain Criteria
rmANCOVA = repeated measures analysis of covariance
RR = respiratory rate
RRS = ruminative response scale
RSA = respiratory sinus arrhythmia
VAMP = valence-arousal mood profile
W15 = first 15 minutes of writing task
W10 = last 10 minutes of writing task

1. 0 INTRODUCTION

Depression is the most common psychological condition for which therapeutic intervention is sought (Pincus et al., 1999). According to the World Health Organization (2016, April) depression significantly contributes to the overall global burden of disease and affects an estimated 350 million people worldwide. Estimates of lifetime prevalence rates of depression range from 16.6% to 41.4% (Kessler et al., 2005; Moffit et al., 2010). A diagnosis of major depressive disorder is characterized by a host of symptoms including: sad or depressed mood, anhedonia, changes in sleep, appetite, and energy, lowered ability to concentrate, abnormal amounts of guilt, and suicidal ideation (American Psychiatric Association, 2013). Furthermore, research has shown that depression is associated with a suite of changes in numerous body systems including the nervous, cardiovascular, endocrine, and immune system (Hasler, Drevets, Manji, & Charney, 2004; Andrews, Bharwani, Lee, Fox, & Thomson, 2015).

Generally, there has been concern that the diagnostic criteria for depression lead to overdiagnosis and that the criteria should be made more stringent (Frances & Nardo, 2013; Spitzer & Wakefield, 1999). When research showed that the third edition of the Diagnostic and Statistical Manual (DSM) of Mental Disorders yielded surprisingly high estimates of disorder, it prompted changes in diagnostic criteria in the DSM-IV to increase stringency (Kessler et al., 1994; Spitzer & Wakefield, 1999). Specifically, the criteria for depression now included that the diagnosed must experience clinically significant impairment or distress along with meeting symptom criteria. The data of Kessler, Berglund, Demler, Jin, & Walters (2005), suggest that this change made little effect on the criteria's stringency. When the fifth edition of the DSM was being introduced, there was again controversy over the new diagnostic criteria. Specifically, the bereavement exclusion criterion for depression has been removed. Researchers have argued that the DSM-5 makes current criteria less stringent by now including what could be normal prolonged grief after bereavement (Theilman & Cacciatore, 2014; Wakefield, 2013).

The symptoms of depression are thought to reflect a state of disorder (i.e. biological dysfunction; American Psychiatric Association, 2013). This view of biological dysfunction, however, is currently under debate. The diagnostic criteria for depression may not accurately distinguish a normal adaptive psychological response to a stressor from a state of biological dysfunction. Wakefield (1992) argues that understanding disordered states requires, first, an evolutionary understanding of normal functioning. When we understand the evolved function of a trait, we can make claims about dysfunction. Little is known about the evolved function of depression. In response to the questionable validity of the diagnostic criteria used for mental health conditions, the National Institute of Mental Health (NIMH) launched the Research Domain Criteria (RDoC) project. The RDoC suggests discarding current assumptions about diagnostic categories and to, instead, focus on understanding the neuroscience and genetics of emotion and cognition across the full range of human behaviour, from normal to

abnormal. The RDoC research approach consists of two steps (Cuthbert & Insel, 2013), which are in alignment with Wakefield (1992). The first step is to "inventory the fundamental, primary behavioural functions that the brain has evolved to carry out, and to specify the neural systems that are primarily responsible for implementing these function" (Cuthbert & Insel, 2013, p.4). The second step "involves a consideration of psychopathology in terms of dysfunction of various kinds and degrees in particular systems, as studied from an integrative, multi-systems point of view" (Cuthbert & Insel, 2013, p.4).

Depression and anxiety often occur concurrently; the prevalence rate for anxiety disorders in individuals with a diagnosis of depression is 51.2% (Kessler et al., 1996). Depression and anxiety disorders may also share common etiological pathways (Barlow, 2002). Given that depression and anxiety disorders may share etiological pathways and have high co-morbidity rates, Moses & Barlow (2006) have hypothesized that interventions targeted for one disorder may be similarly effective in treating the other. It has been shown that treating a particular anxiety disorder significantly reduced symptoms of other anxiety and mood problems not targeted by the intervention (Borkovec, Abel, & Newman, 1995; Brown, Antony, & Barlow, 1995; for review, see Barlow, Allen, & Choate, 2004).

Exposure to and engaging negative emotions has been shown to be an effective treatment for anxiety disorders (Ougrin, 2011; Powers et al., 2010). Interestingly, there is evidence that engagement in and exposure to depressive emotions and cognitions yields therapeutic outcome as well (Hayes et al 2005; Hayes et al., 2007; Hunt, 1998). Exposure based therapies (EBTs) involve engaging negative emotions and emotionally difficult cognitions. Several mechanisms are thought to underlie the effectiveness of EBTs (Tyron, 2005). EBTs are thought to promote habituation, emotional processing, and a correction of erroneous cognitions. Habituation refers to a decrease in response after repeated presentations of a stimulus. Emotional processing refers to change in emotional response patterns associated with thoughts, feelings, and actions.

Expressive writing (EW) interventions have been used to help alleviate symptoms of depression and anxiety (Koopman et al., 2005; Gortner, Rude, & Pennebaker, 2006; Sloan, Marx, Epstein, & Lexington, 2007; Baikie, Liesbeth, & Wilhelm, 2012; Krpan et al., 2013). Furthermore, there is a literature showing that EW has physical and psychological benefits for several populations including non-clinical ones (Pennebaker & Francis, 1996; Richards, Beall, Seagal, & Pennebaker, 2000; Koopman et al., 2005; Gortner Rude & Pennebaker, 2006). EW involves writing about one's deepest thoughts and feelings about a troubling experience or personal problem (Pennebaker & Beall, 1986). However, the underlying mechanism through which EW achieves therapeutic effect is unclear. Some research has suggested that EW achieves therapeutic effect through exposure-based mechanisms (Hunt, 1998; Hayes et al., 2005; Hayes et al., 2007).

In the current study, I explore whether EW is an EBT. Above, I reviewed concerns that the current diagnostic criteria may erroneously pathologize normal adaptive emotional responses to stressors. Given this information, it seems plausible that EW may achieve its therapeutic effect by promoting exposure to a normal, adaptive depressive process. If EW is an EBT, EW should increase exposure to depressive emotion, cognition, and physiology. Research done by Maslej, Rheaume, & Andrews (in prep) shows that EW increases exposure to depressive emotion and cognition. In the current study, I use a design similar to Maslej et al. (in prep) in an attempt to replicate her findings and extend them with a physiological investigation. In particular, I examined the cardiovascular physiology that has been associated with depression.

Maslej et al. (in prep) had participants consider a personal problem they were currently facing and then do an EW task that involved writing about their deepest thoughts and feelings about the selected personal problem. To have a comparison group, the researchers also had another group of participants do a control writing (CW) task that involved writing objectively about one's schedule over the last seven days. All participants wrote for duration of 25 minutes. Participants were an undergraduate sample. Below, I discuss the findings of Maslej et al. (in prep) in the context of previous literature.

1. 1 Does EW increase exposure to depressive emotion?

Previous literature has had mixed results when investigating whether EW induces negative emotion. Previous research has shown that a session of EW induces negative emotion (Pennebaker & Beall, 1986; Pennebaker & Susman, 1988), however, some studies have not replicated this (Mackenzie, Wiprzycka, Hasher, & Goldstein, 2007; Smyth, Hockemeyer & Tulloch, 2008). One explanation for the failure to replicate may include the timing of mood measurement. All studies measure emotional state before and after a writing session. After a writing session is done, the emotional effects of tasks may dissipate. Thus, some studies may fail to replicate because negative emotions felt during the writing task may have dissipated after the writing task is done.

In an attempt to capture emotional effects prior to dissipation, Maslej et al. (in prep) suggested that an emotional measurement part way through the writing task might be a more valid measure of emotional states triggered by EW. In two studies, Maslej et al. (in prep) measured emotion after 15 minutes of writing during a 25 minute writing session and found that sadness had increased during EW compared to CW.

1. 2 Does EW increase exposure to depressive cognition?

Depressive rumination has been studied extensively using the *Ruminative Response Scale* (RRS; Treynor, Gonzalez, & Nolen-Hoeksema, 2003). The RRS initially conceptualized rumination as unproductive processing (Nolen-Hoeksema, 1987; Morrow & Nolen-Hoeksema, 1990; Papageorgiou & Wells, 2003). However, factor analysis of RRS items revealed two distinct types of rumination (Treynor et al., 2003). These two styles were termed the *reflective pondering factor* and the *brooding factor*. Reflective

pondering is thought to represent rumination about coping and problem solving and is thought to be an adaptive form of rumination (Joormann, Dkane, & Gotlib, 2006; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Brooding is thought to represent rumination about critical thoughts about oneself, others, or fate, and it is thought to be a maladaptive form of rumination (Treynor et al., 2003; Nolen-Hoeksema et al., 2008).

The analytical rumination hypothesis (ARH) suggests that depression is triggered by complex problems and evolved to promote analytical rumination (Andrews & Thomson, 2009; Andrews, Bharwani, Lee, Fox, & Thomson, 2015). Analytical rumination is based on the cognitive construct of analysis. Analysis involves breaking down a complex problem into manageable components to be analyzed in turn. The ARH suggests that engaging in and promoting depressive cognitions may yield therapeutic effect. The ARH suggests that analytical rumination promotes problem solving and problem solving reduces depressive symptoms (Andrews & Thomson, 2009). Indeed, Bartoskova et al., (in prep) analyzed data for four subclinical samples and one clinical sample and found that depression promotes a two-stage analytical rumination process that helps understand a personal problem and generate solutions to resolve the problem. The first of these processes has been termed analyze-to-understand rumination (AUR) while the second process has been termed analyze-to-solve rumination (ASR).

If EW is an EBT, then it should trigger depressive cognition. Maslej et al. (in prep) measured analytical rumination during EW using a modified version of the analytical rumination questionnaire (ARQ-M; Barbic, Durisko, & Andrews, 2014). Maslej et al. utilized the ARQ-M over the RRS for several reasons. First, the psychometric properties of the RRS are poor compared to the ARQ-M. Bartoskova et al. (in prep) have shown that the factors of the RRS vary across sex, culture, and diagnostic status; AUR and ASR, on the other hand, are invariant across these categories. Second, the RRS originally conceptualized rumination as an unproductive thinking process (Nolen-Hoeksema, 1987; Morrow & Nolen-Hoeksema, 1990; Papageorgiou & Wells, 2003). Given the concerns about pathologizing normal adaptive processes reviewed above, Maslej et al. (in prep) utilized a rumination scale based on an adaptive hypothesis for rumination. Maslej et al. (in prep) found that EW increases analytical rumination. These results suggest that EW induces exposure to normal adaptive depressive cognitive processes.

1.3 Extending the findings of Maslej et al. (in prep)

In my thesis, I try to extend the findings of Maslej et al (in prep) by studying the physiology associated with EW. In particular, I examined two cardiovascular variables that have been associated with depression. These variables are respiratory sinus arrhythmia (RSA) and heart rate. Specifically, there is evidence that the parasympathetic activation of the heart may be altered in depression (Lehofer et al., 1999; Rottenberg, 2007; Kemp et al., 2014). Below, I provide a background to understand these variables and the relevant literature on depression. After that, I present the current investigation.

1.4 Parasympathetic regulation of cardiac activity

The parasympathetic nervous system (PSNS) consists of several cranial nerves including the vagus nerve (Cranial nerve X). There are two vagal motor systems that descend from different brainstem source nuclei that innervate the heart. One efferent pathway descends from the dorsal motor nucleus (DMN), while the other descends from the nucleus ambiguus (NA). The efferents from the NA innervate the sinoatrial (SA) node of the heart, which is where the heart beat originates. The vagal efferents from the NA are influenced by a common cardiopulmonary oscillator circuit in the brainstem. Consequently, the vagal efferents from the NA have a phasic respiratory rhythm (Porges, 1995, 1997, 2007). There is phasic waxing and waning of vagal inhibition on the heart, with respiration.

As a result of the vagal innervation of the SA node, during inhalation heart rate accelerates and during exhalation heart rate decelerates. This variability in heart rate related respiration is called respiratory sinus arrhythmia (RSA). RSA can be measured by examining the frequency band of heart rate variability (HRV) that coincides with the human respiratory rate (0.15-0.40 Hz). RSA has been used as a non-invasive index of cardiac vagal tone (Berntson, Cacioppo, & Grossman, 2007).

During rest, vagal efferents from the NA provide tonic inhibition to pacemaker cells in the SA node (Porges, 1995, 1997, 2007). When there is an increase in metabolic demand, tonic inhibition is rapidly withdrawn (within milliseconds) to cause a rapid increase in heart rate (Berger, Saul, Cohen, 1989). When vagal tone decreases, RSA decreases. After metabolic demands decrease to resting levels, vagal tone increases and, consequently, RSA increases and heart rate decrease. Thus rapid changes in vagal tone allow for an individual to rapidly change between metabolic states and adapt to changing environmental circumstances.

RSA is both an individual difference variable (i.e. resting baseline RSA varies between individuals) and a response variable (i.e. RSA changes in response to a task). As an individual difference variable, it has been used to index and predict both physical and mental health status (Kemp & Quintana, 2013; Beauchaine & Thayer, 2015). As a response variable, it has been used to index emotion regulation, cognitive effort, working memory load, and attention (Reynard et al., 2011; Duschek et al., 2009; Hansen, Johnsen, Thayer, 2003).

1. 5 Cardiorespiratory physiology of depression

Carney et al. (1988) first reported increased resting heart rate (HR) and reduced resting heart rate variability (HRV) in depressed individuals. Some studies have replicated this finding (Lehofer et al., 1999; Rottenberg, 2007; Kemp et al., 2014) but others have not (Moser et al., 1988; Lehofer et al., 1997; LeMoult, Yoon, Joormann, 2016). Some researchers have suggested that depression may be associated with vagal dysfunction (Kemp et al., 2014; Sgoifo et al., 2015).

It is unclear how respiratory physiology changes with depression. In depression induction experiments, depressed emotion has been associated with either no change in respiratory rate (RR) or decreases in RR (Rehwoldt, 1911; Averill, 1969). In studies involving depressed patients, depression has been associated with increased RR (Damas-Mora, Grant, Kenyon, Patel, & Jenner 1976; Damas-Mora, Souster, & Jenner, 1982).

The ARH suggests that depression promotes analytical rumination by reallocating energetic resources towards cognition and by promoting distraction resistance (Andrews & Thomson, 2009; Andrews, Bharwani, Lee, Fox, & Thomson, 2015). The ARH suggests that symptoms of depression are a consequence of adaptive trade-offs and promotion of a body state conducive to analytical rumination. For example, the ARH suggests that there may be an up-regulation of energetic resources for cognition related to the solving the triggering complex problem and a down-regulation of resources to things like growth, reproduction, and social activity. The ARH suggests that the physiology of depression is coordinated to promote analytical rumination.

Analytical rumination may be a highly demanding metabolic process that needs to be supported by aerobic glycolysis (Andrews, Bharwani, Lee, Fox, & Thomson, 2015). Glycolysis is a glycolytic pathway that can rapidly metabolize glucose without the use of oxygen (Ferrier, 2013). It's plausible that the high metabolic demands of analytical rumination may alter cardiovascular and respiratory physiology. For instance, the increased HR and decreased RSA seen in depression may support the energetic demands of analytical rumination.

1.6 The current investigation

In the current investigation, I examine the hypothesis that EW is an EBT. Maslej et al. have shown that both sadness and analytical rumination increase with EW. EW triggers depressive emotions and cognition. I replicated these findings and further test the hypothesis that EW is an EBT by examining if EW triggers exposure to depressive physiology. In this study, I use an experimental design similar to Maslej et al. (in prep), with the addition of measuring cardiac and respiratory activity. I predicted that, if EW triggers exposure to depressive physiology, then EW would decrease RSA and increase HR. Because measures of RSA can be confounded by respiratory rate (RR), I also measured RR and statistically controlled for its influence on RSA (Grossman, Karemaker, Wieling, 1991; Grossman & Taylor, 2007; Ritz & Dahme, 2006). Results revealed that EW does not alter RSA or HR. However, exploratory correlational analyses suggested that EW might alter the relationship between HR and RR.

2.0 METHODS AND MATERIALS

2.1 Participants

All participants were recruited through an online posting for Introductory Psychology course students at McMaster University. Participants provided informed consent to participate in the experiment and were compensated for two hours of their time

with course credit. The experiment consisted of participants being assigned to one of two writing task groups: EW group and CW group. Participants were naïve to the presence of two participant groups and to the purpose of the study.

Data was collected from 75 participants. Two participants were excluded because of incomplete electrocardiogram (ECG) data. Three participants were excluded because their baseline ECG measurements included high levels of artefact. Two participants were excluded because of incomplete respiratory data. One participant was excluded because their respiratory data contained high levels of artefact. Collectively, this brought the sample size to 66 participants. Of these participants, only 3 males and 12 males made up the EW group and CW group, respectively. Because there is insufficient data to test for effects of gender, within and between conditions, all males were excluded from analysis. The participants in the sample of the analyzed data are 25 females in the EW group and 26 in the CW group.

2.2 Description of questionnaires and tasks

Emotional state of the participant was measured pre-, during, and post-task. Physiology was measured before, during, and after the task. Furthermore, questionnaires were administered before and after the task. Questionnaires and tasks are described in detail below. See Figure 1. for flowchart of protocol.

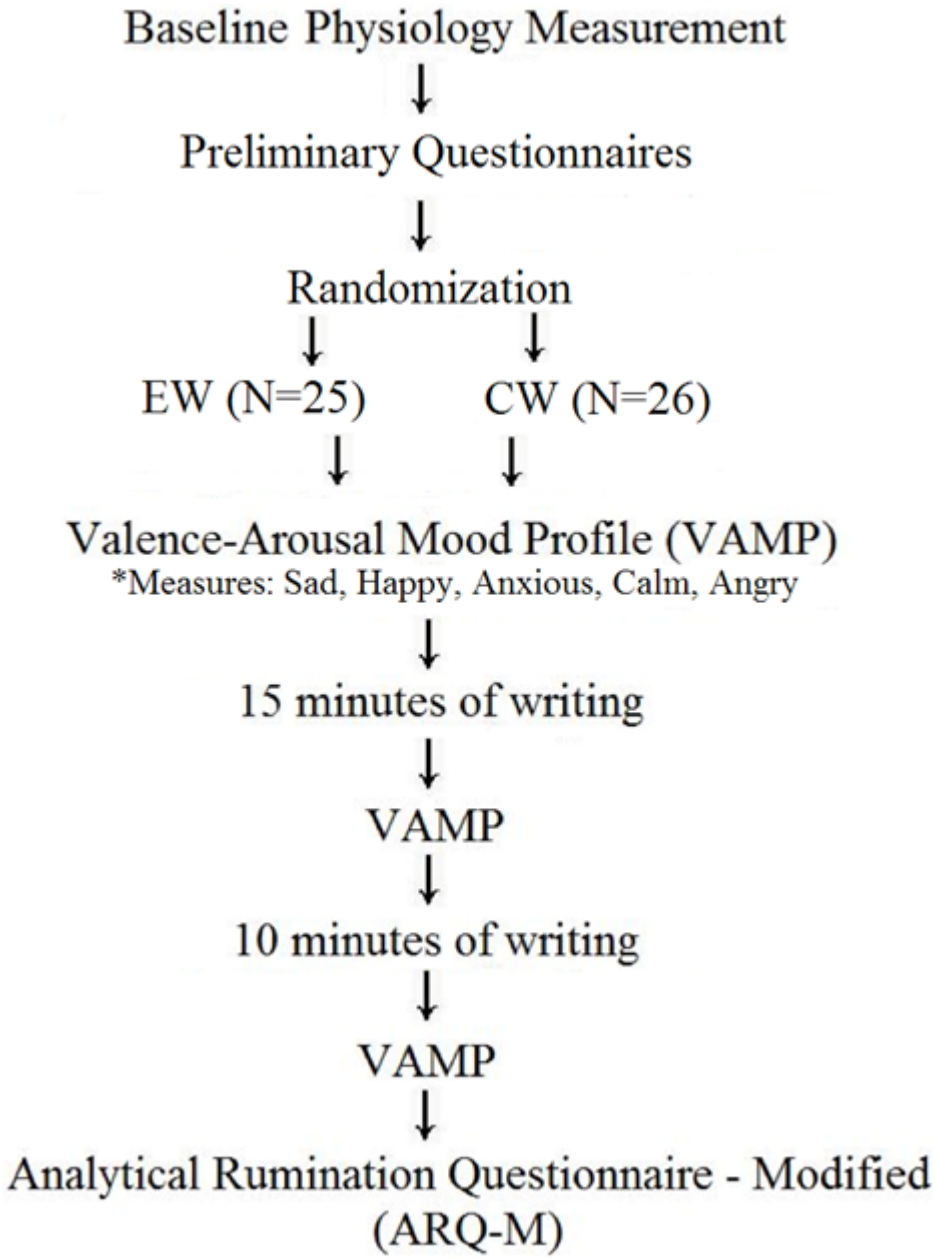

Figure 1. Flowchart of study protocol.

2.2.1 Questionnaires

Pre-task questionnaires included a background information survey, patient health questionnaire-9 (PHQ-9), problem questionnaire (PQ), and the Valence-Arousal Mood Profile (VAMP). To obtain measures of change in emotional state during the writing task, the VAMP was administered 15 minutes into the writing task. Post-task questionnaires included the VAMP and the ARQ-Modified (ARQ-M).

2.2.1.1 Background information survey

The background information survey consisted of questions about age, gender, ethnicity, socioeconomic status, height and weight to calculate body-mass index (kg/m^2), whether the participant had any caffeine or alcohol within the last 4 hours, whether the participant had smoked or had a large meal within the last 2 hours, how frequently they exercised (from 0 times a week to 3 or more times a week), and whether they had any recent or upcoming stressors in their day.

2.2.1.2 Patient Health Questionnaire-9 (PHQ-9)

The PHQ-9 is a 9-item questionnaire measuring the frequency of depressive symptoms over the last 2 weeks (Kroenke, Spitzer, & Williams, 2001). It consists of items scoring the DSM-IV criteria for MDD. The items are scored based on frequency of occurrence from 0 (not at all) to 3 (nearly every day). To obtain a PHQ-9 score, the results of all items were summed for each participant.

2.2.1.3 Problem Questionnaire (PQ)

The PQ consisted of two questions. The first question asked participants to indicate if they were experiencing personal problems from a given list of life areas. Example items include: romantic, interpersonal, financial, and health related personal problems. The second question asked participants to identify only one of the problems that they feel is the most serious problem. If the participant was in the expressive writing group, they were asked to recall and write about this problem.

2.2.1.4 Valence-Arousal Mood Profile (VAMP)

The VAMP consists of 20 adjectives that describe the participant's current emotional states of sadness, happiness, anxiousness, calmness, and anger. Each emotional state has 3-5 adjectives. Each adjective is rated on a 4-point likert scale ranging from a score of 1 (extremely inaccurate as a self-description) to 4 (extremely accurate as a self-description). Respective adjective scores were averaged to obtain a VAMP score for each emotional state.

2.2.1.5 Analytical Rumination Questionnaire - Modified (ARQ-M)

The ARQ-M is a modified version of the ARQ (Barbic, Durisko, & Andrews, 2014) that asks participants to indicate the frequency of analytical rumination during the

writing task instead of over the last 2 weeks. The ARQ-M is a 20-item questionnaire and consists of participants scoring items measuring the frequency of rumination from 1 (never) to 5 (all of the time). An analytical rumination score (AR) was constructed by summing all 20 items of the ARQ. Two additional rumination scores were constructed from the ARQ. The ARQ-M contains items that probe two different kinds of rumination (Bartoskova et al., in prep). The first is AUR. The AUR is constructed from the sum of 3 items of the ARQ that are related to rumination regarding understanding a personal problem. Example items include: "I tried to understand why I had these problems" and "I tried to figure out what I had done wrong." The second is ASR. The ASR is constructed from the sum of 3 items of the ARQ that are related to rumination regarding solving a personal problem. Example items include: "I tried to figure out how I could stick to my goals" and "I tried to figure out how to make the best out of a bad situation."

2.2.2 Writing tasks

Writing task instructions were adapted and modified from previous research (Pennebaker, 1997). The EW task asked participants to write about their deepest thoughts and feelings about a personal problem that they were facing. This personal problem was identified by the participant in the PQ. The following instructions were given to the participant.

> For the next 25 minutes, we would like you to write your very deepest thoughts and feelings about the personal problem you considered in the previous questionnaire. In your writing, we want you to really let go and explore your emotions and thoughts regarding this problem. All of your writing will be completely confidential.
>
> Do not worry about spelling or grammar, but try to make your text as legible as possible. After writing for 15 minutes, you will be asked to pause and complete a brief questionnaire. After completing the questionnaire, you will continue writing for another 10 minutes.

The CW task asked participants to write objectively about their schedule for the past 7 days. The following instructions were given to the participant.

> For the next 25 minutes, we would like you to write about your schedule for the past week and your schedule for the next week. In your writing, please describe what you did starting from 7 days ago until today. You can include details such as the time you woke up, what you ate, what you wore, the places you went, which buildings or objects you passed by, and who you encountered. Once you have reached today's date, begin describing your schedule for the upcoming week (in other words, the next 7 days), including as much detail as you can. Please be as objective as possible by concentrating on the facts and the details, instead of opinions, thoughts, and emotions. All of your writing will be completely confidential.

Do not worry about spelling or grammar, but try to make your text as legible as possible. After writing for 15 minutes, you will be asked to pause and complete some brief questionnaires. After completing the questionnaires, you will continue writing for another 10 minutes.

2.3 Physiological data collection

2.3.1 Apparatus

Physiological measurements were collected using an ActiPOWER, ActiCHAMP, BIP2AUX, respiratory belt, and three lead wires from BrainVision products, connected to a computer running BrainVision Pycorder 2.0. The sampling rate of physiological measurements was set to 500 Hz.

2.3.2 Equipment placement

Two ECG electrodes were placed under each rib cage and one electrode was placed under the right collarbone of each participant. The electrode under the right rib cage acted as the ground while the one under the right collarbone was the negative lead and the one under the left rib cage was the positive lead. Prior to applying the cardiac electrodes, the area of skin where the electrodes were placed were cleaned using a cotton ball dipped in rubbing alcohol. This was done to remove any skin oils and residue, which could have made it hard for the electrode to stick in place. After the electrodes were placed, ABRALYT high chloride conductive gel was placed in the wells of the electrodes with a Q-tip. Surgical tape was then applied over each of the electrodes to ensure that they remained placed and that the conductive gel would not dry out during the experiment. After ECG equipment had been set up, the respiratory belt was placed. The respiratory belt was wrapped around the participant at sternum level. Before proceeding with the experiment, signal quality of the ECG was visually inspected for clarity of R-waves. Signal quality of respiration was inspected by having the participant inhale and exhale a few times and ensuring that change in breathing was being detected by PyCorder 2.0.

2.3.3 Acquisition

Baseline physiology measurements were taken before all questionnaires and tasks were done. Baseline physiology was recorded for duration of 5-minutes. During baseline measurements, participants were asked to sit quietly with their eyes closed, try their best to relax, not move around too much, and have their feet placed flat on the ground. Participants were given 2-minutes to relax themselves prior to taking the baseline (for example, they could sit quietly with their eyes closed). This was done to allow participants to adjust to having the equipment placed on them and to acclimate to the experiment environment.

Physiological data was recorded during the 25 minutes of the writing task. Acquisition was interrupted after 15 minutes of writing to administer the VAMP. After completing the VAMP, acquisition was resumed.

2.4 Data processing

2.4.1 Processing ECG data

Kubios HRV 2.2 was used to process the acquired ECG data. Data processing followed the recommendations of the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996). All data was visually inspected for artefact and misplaced or missing R-waves. Identified artefacts were corrected. Because high levels of artefacts can significantly affect data quality, only participants with less than 20% artefact correction were included in the final data set (Berntson & Stowell, 1998). Three participants exceeded this threshold and were not processed. All other participants had required less than or equal to 5% artefact correction. HR and RSA data were collected from the software. The frequency range for RSA was set to 0.15–0.40 Hz and the Fast Fourier Transform output, in units of ms^2, from the software was used. The RSA data was then log-transformed to normality (Riniolo & Porges, 2000).

2.4.2 Adjusting RSA for RR

RSA is used as a non-invasive measure of cardiac vagal tone. In a within subject design, where RSA is measured during several phases, changes in RSA have been used to make inferences about changes in cardiac vagal tone. RSA is not a perfect measure, however. One problem is that changes in respiratory parameters can alter RSA independent of any alteration in cardiac vagal tone. For example, when RR increases, RSA decreases while cardiac vagal tone can remain unaltered (Grossman & Taylor, 2007). Therefore, when using changes in RSA as a measure of changes in cardiac vagal tone, one should either use an experimental design that does not alter RR within subjects or statistically adjust RSA values for RR values.

Preliminary analysis of the data suggested a significant within subjects change in RR between phases (see Results for details). Consequently, all RSA data was statistically adjusted for RR. A within-subjects regression method of adjusting RSA for RR levels was used (Grossman, Karemaker, Wieling, 1991; Grossman & Taylor, 2007; Ritz & Dahme, 2006). A regression was conducted for each participant using the RSA and RR for each minute of data for the participant. RSA was set as the dependent variable and RR was set as the independent variable. Unstandardized residuals were collected for the participant. This process was repeated for each participant to obtain RSA adjusted for RR for all participants.

2.4.3 Processing respiratory data

BrainAnalyzer 2.0 was used to process respiratory data. First, to remove any artefacts in the respiration data caused by non-respiratory mechanisms (Ex. pulsations from heart mechanical activity), a high and low pass filter was used. Filter edge frequencies of 0.125 Hz and 0.6 Hz were used because these are within the frequency range of human breathing rates. Second, single breaths were identified using the software's level trigger

transform function. Third, all acquired data was visually inspected to ensure that breath markers had been placed correctly and that breath markers were not erroneously placed or missed. Respiratory rate (RR; breaths/min) was then calculated by summing the number of inhalations and dividing by the duration of the acquisition period.

2.4.4 Linguistic Inquiry Word Count (LIWC) analysis of writing task

To check for participant adherence to the writing task instructions, all writing tasks were digitally transcribed and processed using a software called Linguistic Inquiry and Word Count (LIWC; Pennebaker, Chung, Ireland, Gonzales, & Booth, 2007). Of particular interest were "negative emotion words" and "cognitive processing words." The "negative emotion words" variable represents the proportion of words suggestive of negative emotions in the text. The software has 499 words in its dictionary that fall into this variable (Examples include, "sad," "worried," and "cry"). The "cognitive processing words" variable represents the proportion of words suggestive of cognitive processing in the text. The software has 730 words in its dictionary that fall into this variable (Examples include, "cause," "know," and "ought").

If participants wrote the same amount during both tasks, it would suggest that both groups were equally engaged with writing during both tasks. If the EW condition used more negative emotion and cognitive insight words compared to CW, then it would suggest that EW participants were following task instructions.

2.5 Statistical analyses

Data were analyzed using IBM SPSS Statistics for Macintosh, Version 21.0 with an alpha of 0.05. Two tailed p-values are reported in text. All data were tested for normality using Shapiro-Wilk's test. Non-parametric statistics were conducted for non-normal data.

3.0 RESULTS

3.1 Demographics

Ethnicity data showed that 39.2% of our participants identified as Caucasian, 27.5% as Asian, 15.7% as African American, 7.8% as Arab, and 9.8% as mixed ethnicity. Socioeconomic data showed that 2% of participants identified as lower class, 17.6% as lower middle class, 45.1% as middle class, 4% as upper class, and 25.5% as upper class.

3.2 Baseline measures and participant randomization

3.2.1 Baseline physiology measures

Independent samples t-tests were conducted to check if there were any between group differences in baseline physiological measures. Baseline HR for EW (M=79.11, SD=11.57) and CW (M=83.51, SD=10.61) participants, were not significantly different from each other, M_{diff} = -4.40, 95% CI [-10.65, 1.84], t(49) = -1.417, p= 0.163. Baseline

RR for EW (M=15.55, SD=2.88) and CW (M=15.10, SD=2.87) participants, were not significantly different from each other, M_{diff}=0.45, 95% CI [-1.17, 2.07], t(49) = 0.56, p= 0.58. Baseline adjusted RSA for EW (M=-0.07, SD=0.36) and CW (M=-0.15, SD=0.37) participants, were not significantly different from each other, M_{diff}=0.08, 95% CI [-0.12, 0.28], t(49) = 0.81, p= 0.42. Collectively, independent samples t-tests revealed no significant differences in baseline physiological parameters between EW and CW (see Table 1. for summary).

3.2.2 Baseline VAMP measures

Mann-Whitney U tests were conducted to check if there were any between group differences in baseline VAMP measures. Sadness scores for EW (median=1.00) were not significantly different from CW (median=1.50), U = 273, z= -1.031, p=0.303. Happiness scores for EW (median = 2.33) were not significantly from CW (median=2.00), U = 398.5, z=1.402, p=0.161. Anxiousness scores for EW (median = 2.00) were not significantly different from CW (median = 2.00), U =316, z=-0.171, p=0.865. Anger scores for EW (median=1.00) were not significantly different from CW (median =1.00), U = 262.5, z=-1.405, p=0.160. Calmness scores for EW (mean rank=30.62) were not significantly different from CW (median =21.56), U = 440.5, z=-1.373, p=0.028. Collectively, this suggests that baseline VAMP scores only differed between groups on scores of calmness; the EW group was calmer at baseline compared to the CW group (see Table 1. for summary).

3.2.3 Potential factors affecting physiology

Statistical tests were conducted to test if there were any between group differences in factors potentially affecting physiology (see Tables 1 and 2 for summary).

A Mann-Whitney U test revealed that age for EW (median=18) and CW (median= 19) participants were not significantly different from each other, U=260.5, z=-1.373, p=0.170. An independent samples t-test revealed that BMI (kg/m^2) for EW (M=22.11, SD=3.55) and CW (M=21.34, SD=2.98) participants, were not significantly different from each other, M_{diff}=0.77, 95% CI [-1.12, 2.66], t(47) = 0.82, p= 0.416. A Mann-Whitney U test revealed that there was no significant difference in time of experiment between EW (mean rank =29.04) and CW (mean rank =23.08), U=401, z=1.44, p=0.149.

Chi-square tests of association and Fisher's exact test was conducted to test whether there was an association between group and caffeine intake, large meal intake, and smoking prior to the experiment, weekly exercise frequency, and whether the participant was facing any recent or upcoming stressor in their day. There was no significant association between group and caffeine intake prior to the experiment, $\chi^2(1)$=0.545, p=0.460. There was no significant association between group and large meal intake prior to the experiment, $\chi^2(1)$=0.545, p=0.214. There was no significant association between group and having smoked prior to the experiment, Fisher's exact test: p=0.490. There was no significant association between group and exercise frequency ($\chi^2(2)$=0.024,

p=0.988). There was a trend towards a significant association between group and day stress, $\chi^2(1)=0.545$, p=0.067. The CW group had a higher frequency (19) of individuals indicating a recent or upcoming stressor in their day compared to the EW group (12).

This marginal difference in stress could also be reflected in calmness scores. The EW group shows higher levels of calmness compared to CW. To test whether an indication of stress was correlated with calmness scores, a point-biserial correlation was conducted. Calmness and stress were significantly correlated (r(51)=-0.312, p=0.026). Higher baseline stress levels are associated with lower levels of calmness.

Baseline depression scores could have also affected physiology. Consequently, PHQ-9 measures were examined for between group differences and for their correlation with baseline physiological measures. A Mann-Whitney U test revealed that there was no significant difference between EW (median=5.00) and CW (median=5.00) for baseline depression scores, U=296.5, z=-0.540, p=0.589. Pearson correlations revealed that in PHQ-9 scores were not correlated with HR (r(51)=-0.050, p=0.729), RR (r(51)=0.033, p=0.817), and adjusted RSA (r(51)=-0.119, p=0.407). This suggests that there is no relationship between baseline measures of depression and baseline physiology.

Table 1. Descriptive statistics for age, BMI, time of experiment, and baseline physiology and VAMP measures

Measure	EW (N=25)	CW (N=26)	Sig. (2-tailed)
Age†	18.44±0.71	18.81±1.13	0.170
BMI (kg/m^2)	22.11±3.55	21.34±2.98	0.416
Time of experiment†	14.10±2.69	13.12±2.65	0.149
Baseline Physiology			
HR (beats/min)	79.11±11.58	83.51±10.61	0.163
RR (breaths/min)	15.55±2.88	15.10±2.87	0.577
Adjusted RSA (ln ms^2)	-0.07±0.36	-0.15±0.37	0.420
Pre-task VAMP†			
Sadness	1.51±0.77	1.68±0.79	0.303
Happiness	2.20±0.70	1.95±0.59	0.161
Calmness	2.97±0.63	2.62±0.61	0.028*
Anxiousness	1.92±0.59	2.04±0.86	0.865
Anger	1.21±0.50	1.41±0.66	0.160
PHQ-9†	6.24±4.81	6.96±5.15	0.589

Data are mean ± standard deviation. Significant values are denoted with an asterisk (*). Significance values for measures denoted with a dagger (†) were tested with Mann-Whitney U tests (medians and mean ranks are reported in text) while all other measures were tested using independent samples t-tests.

Table 2. Descriptive statistics for frequency data.

Measure	**EW (N=25)**	**CW (N=26)**	**Chi-square Sig. (2-tailed)**	**Fisher's exact Sig. (2-tailed)**
Consumed caffeine in last four hours?	7	5	0.460	-
Consumed alcohol in last four hours?	0	0	-	-
Consumed large meal in last two hours?	4	8	0.214	-
Smoked in last two hours?	1	0	-	0.490
Recent or upcoming stressor today?	12	19	0.067	-
Frequency of weekly exercise (>30mins)			0.988	-
0 times	7	7		
0-3 times	11	12		
3 or more times	7	7		

Data are frequency data.

3.3 Participant instruction adherence

To ensure that participants were following instructions and both group were writing the same amount during the writing task, between group analyses were conducted on writing task word count, negative emotion word count, and cognitive insight word count.

An independent samples t-test revealed that word count for EW (M=585.48, SD=122.54) and CW (M=564.88, SD=117.19) participants, were not significantly different from each other, M_{diff}=20.60, 95% CI [-46.86, 88.05], t(49) = 0.614, p= 0.542. A Mann Whitney U test showed that there was a significant difference in the use of negative emotional words between EW (mean rank=38.72) and CW (mean rank=13.77), U=643, z=5.99, p<0.001. A Mann Whitney U test showed that there was a significant difference in the use of cognitive insight words between EW (mean rank=38.56) and CW (mean rank=13.92), U=639, z=5.92, p<0.001. Collectively, this suggests that the EW condition wrote the same amount of words as the CW group but used more negative emotion and cognitive insight words. The data suggest that the two conditions did adhere to the instructions that they were given and were equally engaged in the writing task.

3.4 Expressive writing and sadness

Generalized estimating equations (GEE) were conducted with time (pre, during, and post task) as a within subjects factor and group as a between subjects factor. Baseline calmness was included as a covariate. GEE revealed a significant time by group interaction for sadness (Wald χ^2 =20.30, N=51, p<0.001). Relative to pre-task sadness scores, EW participants became significantly sadder during writing (β=0.306, SE=0.073, 95% CI: 0.16, 0.45, p<0.001) and after writing (β=0.203, SE=0.066, 95% CI: 0.07, 0.33, p=0.002). Relative to pre-task sadness scores, CW participants became significantly less sad during writing (β=-0.174, SE=0.079, 95% CI: -0.33, -0.02, p=0.027) and trend towards less sad after writing (β=-0.168, SE=0.095, 95% CI: 0.35, 0.02, p=0.076). Generalized linear models (GLM) were conducted to test whether there was a between group difference in sadness during and after writing. GLM revealed that compared to CW, sadness levels were significantly higher during writing in EW (β=0.372, SE=0.114, 95% CI: 0.15, 0.60, p=0.001). Compared to CW, sadness levels were significantly higher after writing in EW (β=0.262, SE=0.114, 95% CI: 0.04, 0.49, p=0.02). See Figure 1. for line graph of sadness scores across time for both conditions. The data suggest that the expressive writing induced sadness during and after writing.

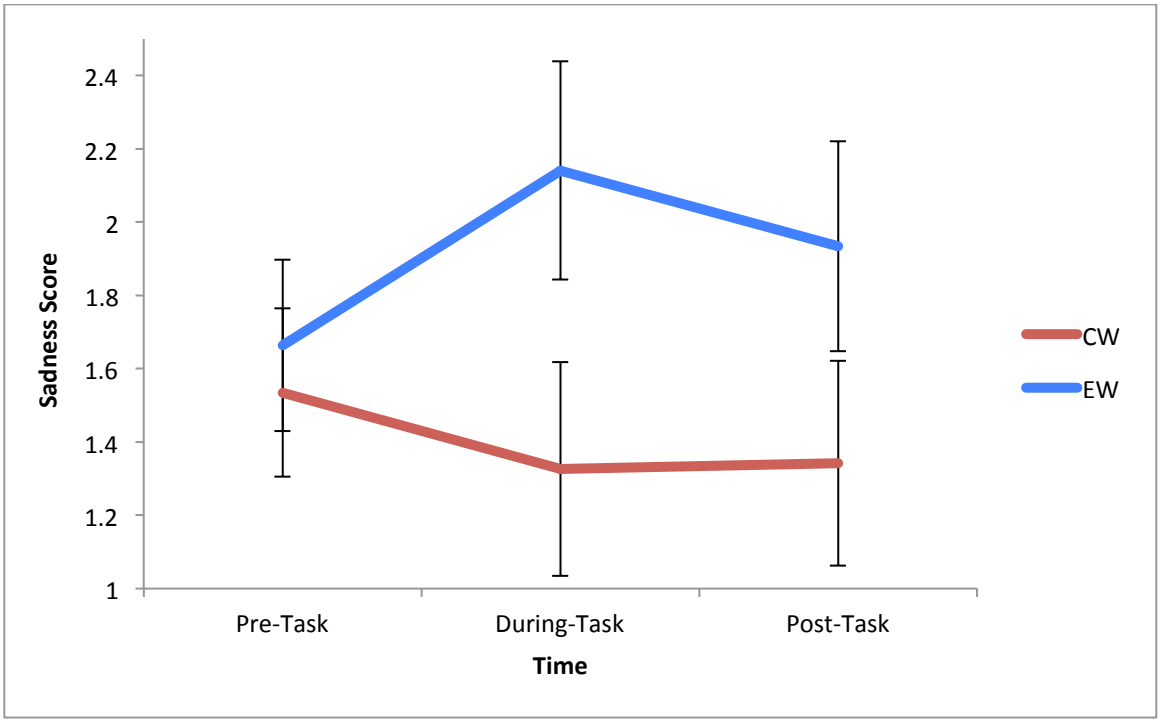

Figure 2. Means of sadness scores (baseline calmness was controlled for) across time for EW and CW. Error bars represent twice the standard error (SE).

3.5 Expressive writing and analytical rumination

GLM were conducted to test whether there was a between group difference in AR, AUR, and ASR during the writing task. One participant in the CW group provided insufficient data to construct an AR and ASR score while one participant in the EW group provided insufficient data to construct an ASR score. Consequently, these participants were not included in the respective analyses. GLM revealed that compared to CW, there was a significant increase in AR (β=0.284, SE=0.0838, 95% CI: 0.120, 0.449, p=0.001), AUR (β=0.420, SE=0.116, 95% CI: 0.193, 0.647, p<0.001), and ASR (β=0.242, SE=0.119, 95% CI: 0.008, 0.476, p=0.043), in the EW condition. This suggests that EW induced AR, AUR, and ASR. See Figure 2. for AR scores for both conditions.

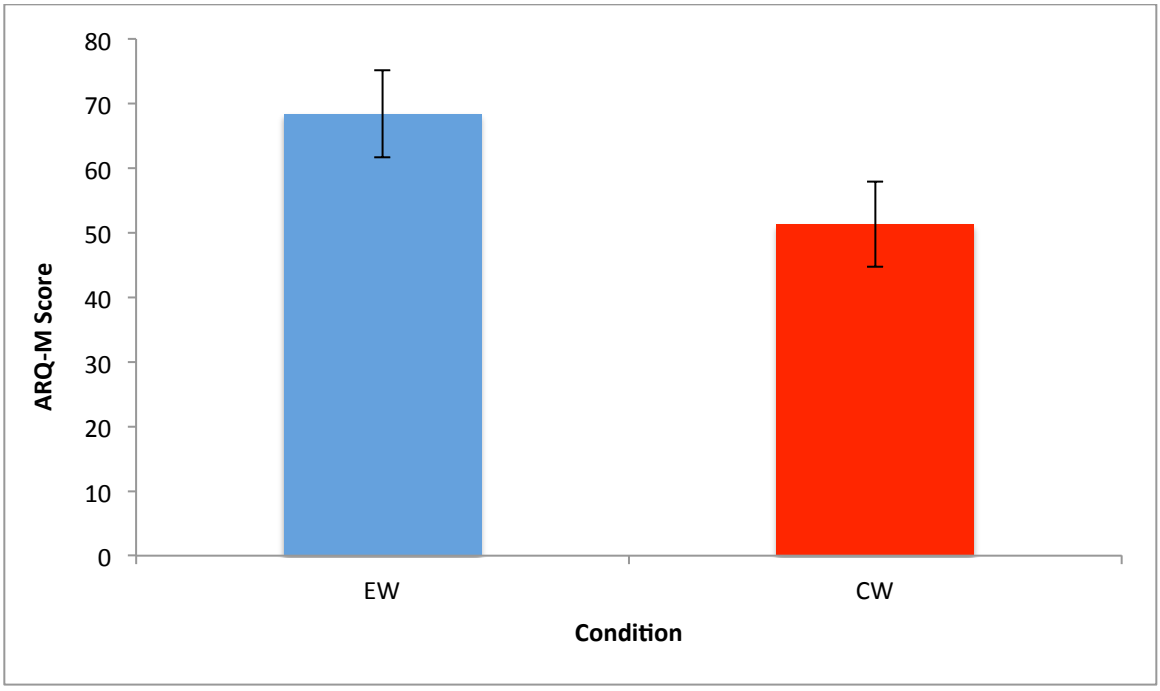

Figure 3. Means of AR for EW and CW (baseline calmness was controlled for). Error bars represent twice the SE.

3.6 Expressive writing and physiology

Repeated measures ANCOVAs (rmANCOVA) were conducted for HR, RR, and adjusted RSA. Because no specific prediction was made about differences in physiology during the first 15 minutes of writing (W15) and the last 10 minutes of writing (W10), the two phases were averaged into one variable called "Task." An rmANCOVA was run with phase (baseline and Task) as a within subject factor and group as a between subject factor. Baseline calmness was used as a covariate.

3.6.1 Heart rate (HR)

For HR, there was no significant phase*group interaction ($F(1,49)=0.473$, $p=0.495$, partial $\eta^2=0.010$) and no main effect of phase ($F(1,50)=1.191$, $p=0.280$, partial $\eta^2=0.023$). Figure 3 shows the data for phase and group. Collectively, this suggests that that HR did not increase from baseline to task for either group.

3.6.2 Respiratory rate (RR)

For RR, there was no significant phase*group interaction ($F(1,49)=0.856$, $p=0.359$, partial $\eta^2=0.017$), but there was a main effect of phase ($F(1,50)=27.466$, $p<0.001$, partial $\eta^2=0.355$). RR increased from baseline to task across groups ($M_{diff}=2.355$, $SE=0.452$, $p<0.001$). Interestingly, there was also a significant phase*baseline calmness interaction ($F(1,48)=4.116$, $p=0.048$, partial $\eta^2=0.079$). To further investigate the relationship between phase and baseline calmness, multiple regressions were conducted with baseline and task RR as dependent variables and calmness and group as independent variables. There was no association between baseline calmness and baseline RR ($\beta=0.092$, $SE=0.632$, 95% CI: -1.179,1.362, $p=0.885$) but there was a marginally significant positive association between baseline calmness and task RR ($\beta=1.247$, $SE=0.655$, 95% CI: -0.069, 2.562, $p=0.063$). This relationship was not dependent on group for either baseline RR ($\beta=-0.455$, $SE=0.847$, 95% CI: -2.157, 1.248, $p=0.594$) or task RR ($\beta=0.895$, $SE=0.870$, 95% CI: -0.855, 2.644, $p=0.309$). Collectively, this suggests that baseline calmness is marginally positively associated with task respiration, RR increases from baseline to task, and there was no between group difference in RR increase.

3.6.3 Adjusted respiratory sinus arrhythmia (RSA)

Because, RR significantly increased from baseline to task, RSA measures were adjusted for RR. For adjusted RSA, there was no significant phase*group interaction, ($F(1,49)=0.329$, $p=0.569$, partial $\eta^2=0.007$). There was no main effect of phase ($F(1,50)=3.157$, $p=0.082$, partial $\eta^2=0.059$). Figure 5 shows the data for phase and group. Collectively, this suggests that adjusted RSA did not change from baseline to task for either group.

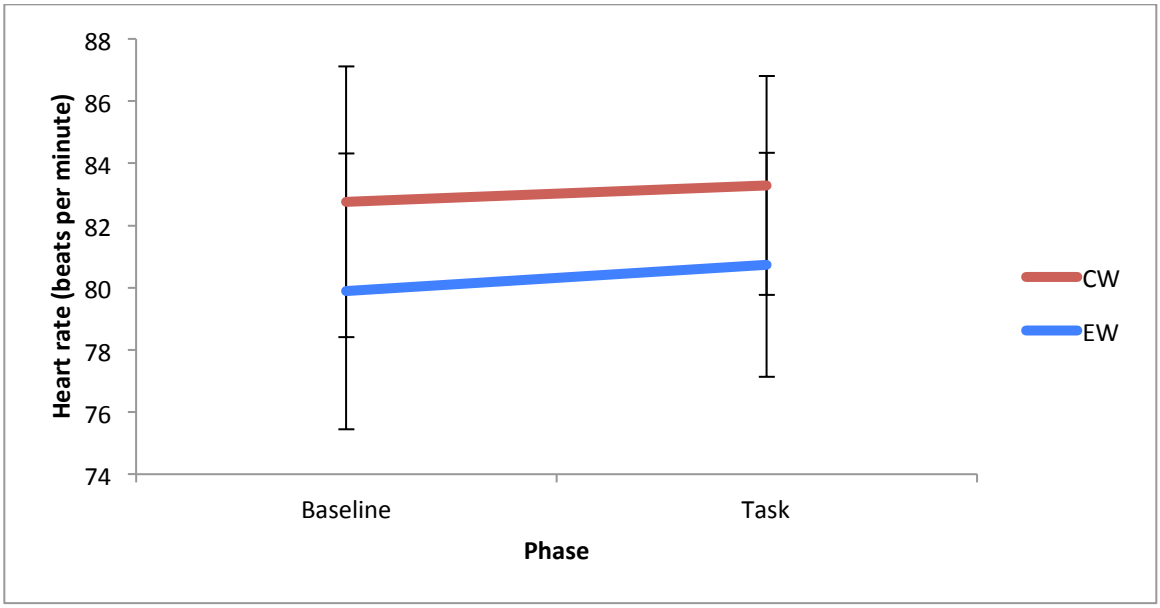

Figure 4. Means of heart rate (beats per minute) across phase and group (baseline calmness was controlled for). Error bars represent twice the SE.

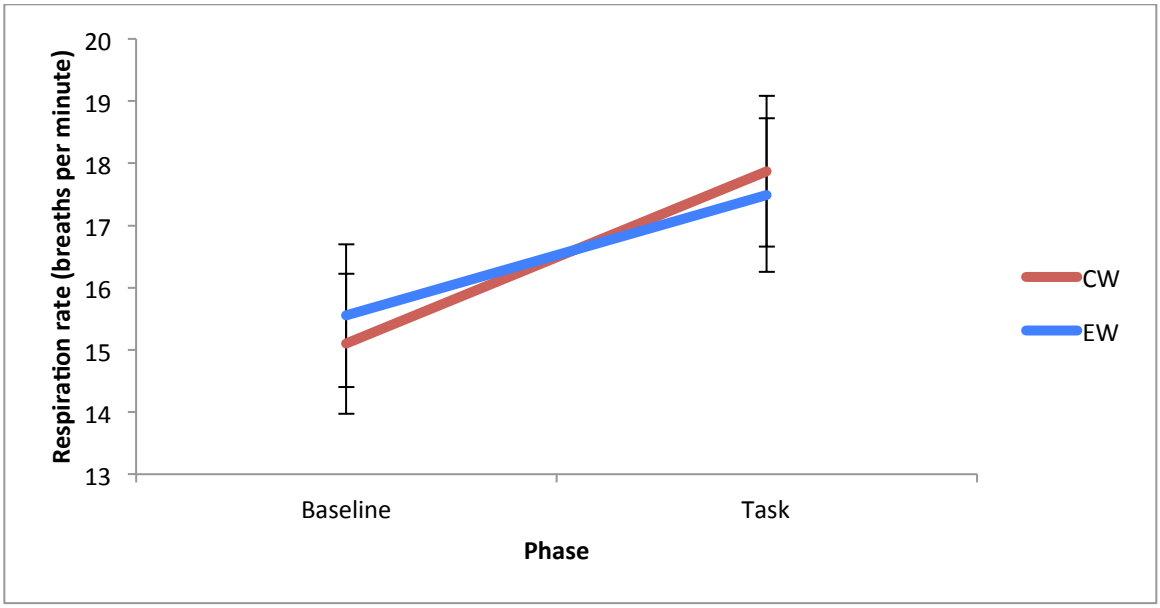

Figure 5. Means of respiration rate (breaths per minute) across phase and group (baseline calmness was controlled for). Error bars represent twice the SE.

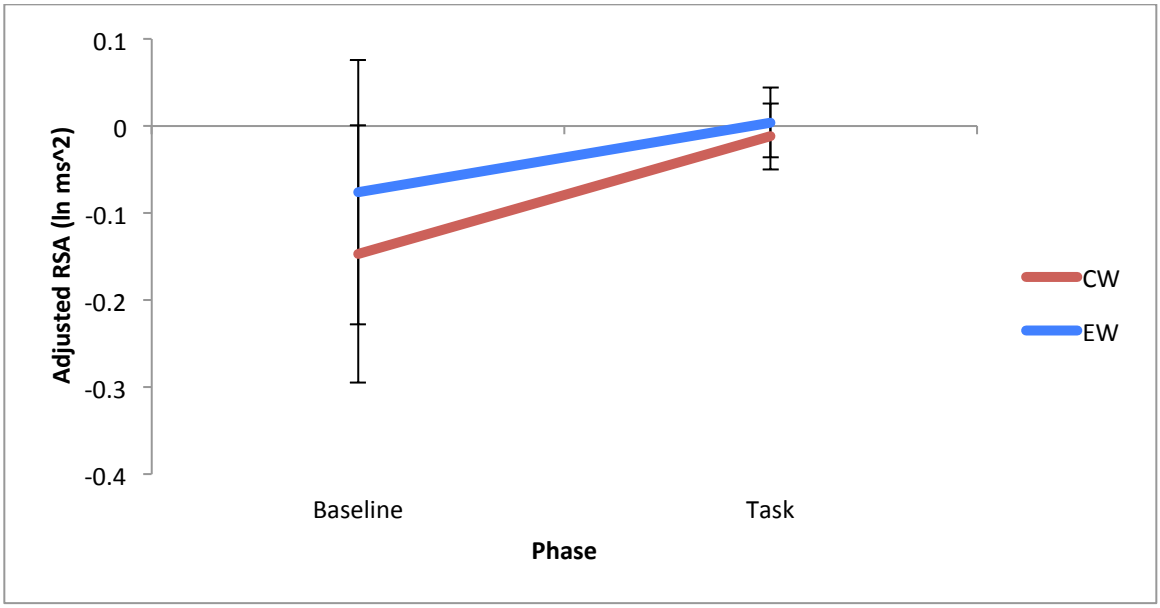

Figure 6. Means of adjusted RSA (ln ms^2) across phase and group (baseline calmness was controlled for). Error bars represent twice the SE.

3. 7 Exploratory correlations between sadness, analytical rumination, and physiological measures

A series of partial correlations were conducted between sadness, analytical rumination measures, and Task physiological measures. Baseline calmness was partialled out for all correlations. For exploratory purposes, correlations were conducted for both during task sadness and post task sadness, all three types of analytical rumination variables (AR, AUR, and ASR) were utilized. One participant in the CW group provided insufficient data to construct an AR and ASR score while one participant in the EW group provided insufficient data to construct an ASR score. Consequently, these participants were not included in the respective analyses. Fisher's r to z transformation was used to test whether there were between group differences in correlations. Uncorrected two-tailed p-values are reported. In total, 42 correlations and 21 Fisher's r to z transformations were run. To correct for multiple post hoc tests, a Bonferroni corrected $p<0.00119$ and $p<0.00238$ would be required for significant correlations and Fisher's r to z transformations, respectively.

3.7.1 During and post task sadness and analytical rumination

Partial rank correlations were conducted to test whether sadness during or post task correlated with AR, AUR, or ASR during writing. See Table 3. for summary. In EW, correlations showed no significant association between sadness during writing and AR ($r(25)=0.100$, $p=0.650$), AUR ($r(25)=0.114$, $p=0.604$), and ASR ($r(24)=0.103$, $p=0.640$). In EW, correlations showed no significant association between sadness post task and AR ($r(25)=-0.065$, $p=0.768$), AUR ($r(25)=-0.083$, $p=0.706$), and ASR ($r(24)=-0.163$, $p=0.458$). Furthermore, there were no significant between group differences in correlation for any of the measures. This suggests that increased sadness during the task was not associated with analytical rumination.

Table 3. Correlations between sadness and rumination.

Measure	**EW (N=25)**		**CW (N=26)**		**Fisher's r to z**
	Correlation (r)	Sig. (2-tailed)	Correlation (r)	Sig. (2-tailed)	Sig. (2-tailed)
During Task					
AR	0.100	0.650	0.331	0.114	0.418
AUR	0.114	0.604	0.448	0.028*	0.219
ASR	0.103	0.640	0.388	0.061	0.317
Post Task					
AR	-0.065	0.768	0.322	0.125	0.187
AUR	-0.083	0.706	0.370	0.075	0.114
ASR	-0.163	0.458	0.240	0.258	0.180

Baseline calmness scores were controlled for. The p-values reported here are not Bonferroni corrected. Significant values are denoted with an asterisk (*).

3.7.2 Partial correlations between physiological measures, sadness, and analytical rumination

Partial rank correlations between sadness and physiological parameters revealed no significant correlations between during and post-task sadness and any physiological parameter (See Table 4. for summary).

Partial correlations revealed a significant positive moderate correlation between AR and HR during Task ($r(25)=0.439$, $p=0.036$) in the EW group. Conversely, there was no significant correlation between AR and HR during Task ($r(25)=0.019$, $p=0.928$) in the CW group. However, there was no significant difference in correlation between groups (Fisher's r to z transformation: $p=0.134$). Partial correlations revealed a non significant correlation between AR and RR during Task ($r(25)=-0.179$, $p=0.414$) in EW and a trend towards a significant positive correlation ($r(26)=0.358$, $p=0.086$) in CW. Furthermore, there is a trend towards a significant difference in correlation between groups for AR and RR (Fisher's r to z transformation: $p=0.066$). Partial correlations revealed a non significant correlation between ASR and RR during Task ($r(25)=-0.027$, $p=0.904$) in EW and a significant positive correlation ($r(26)=0.504$, $p=0.012$) in CW. Furthermore, there is a trend towards a significant difference in correlation between groups for ASR and RR (Fisher's r to z transformation: $p=0.056$). Collectively, this suggests that doing EW may alter the relationship between HR and AR and RR and AR (See Table 5. for summary).

Table 4. Correlations between task physiology and during and post task sadness.

Measure	**EW (N=25)**		**CW (N=26)**		**Fisher's r to z**
	Correlation (r)	Sig. (2-tailed)	Correlation (r)	Sig. (2-tailed)	Sig. (2-tailed)
During Task					
HR	-0.158	0.462	0.081	0.699	0.418
RR	-0.019	0.930	0.216	0.299	0.424
Adjusted RSA	0.048	0.824	0.408	0.043*	0.107
Post Task					
HR	-0.170	0.427	0.075	0.723	0.407
RR	-0.132	0.540	-0.001	0.997	0.653
Adjusted RSA	0.066	0.759	0.715	<0.001*	0.005*

Baseline calmness scores were controlled for. The p-values reported here are not Bonferroni corrected. Significant values are denoted with an asterisk (*).

Table 5. Correlations between task physiology and analytical rumination.

Measure	**EW (N=25)**		**CW (N=26)**		**Fisher's r to z**
AR	Correlation (r)	Sig. (2-tailed)	Correlation (r)	Sig. (2-tailed)	Sig. (2-tailed)
HR	0.439	0.036*	0.019	0.928	0.134
RR	-0.179	0.414	0.358	0.086	0.066
Adjusted RSA	-0.108	0.625	-0.002	0.993	0.711
AUR					
HR	0.325	0.130	-0.119	0.580	0.126
RR	0.195	0.372	-0.296	0.161	0.091
Adjusted RSA	-0.001	0.996	0.095	0.660	0.749
ASR					
HR	0.324	0.132	0.188	0.380	0.632
RR	-0.027	0.904	0.504	0.012*	0.056
Adjusted RSA	-0.185	0.397	0.156	0.468	0.259

Baseline calmness scores were controlled for. The p-values reported here are not Bonferroni corrected. Significant values are denoted with an asterisk (*).

Relationships between HR and RR during Task

To explore the relationship between HR and RR during Task, a general linear model was conducted with Task RR as dependent variable and an interaction between Task HR and group as independent variables. Baseline calmness was included in the model to control for group difference. Analysis revealed that group interacts with HR to predict RR (β=0.253, SE=0.093, 95% CI: 0.065, 0.440, p=0.009). When the same model was conducted with HR and RR for W15, the effect was stronger (β=0.279, SE=0.092, 95% CI: 0.095, 0.464, p=0.004). To further investigate the relationship between RR and the group*HR interaction, a scatterplot was constructed (see Figure 7 for Task data and Figure 8 for W15 data). Baseline calmness was regressed out of both HR and RR values for each group. The resulting unstandardized residuals were then plotted and a line of best fit was added. The data show that the slope for the relationship between HR and RR is positive in CW but negative in EW.

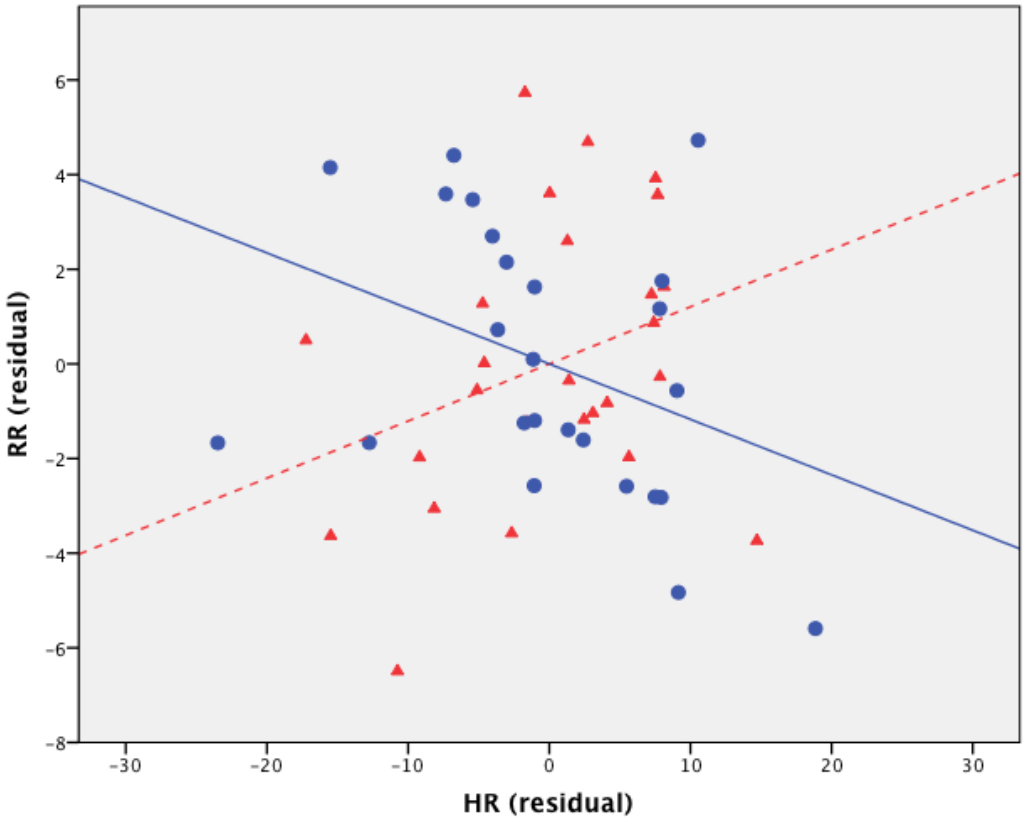

Figure 7. Relationships between HR (residual) and RR (residual) during Task. HR and RR are residuals because baseline calmness has been regressed out of each variable. The red triangles and dashed line represent data for CW. The blue circles and non-dashed line represent data for EW.

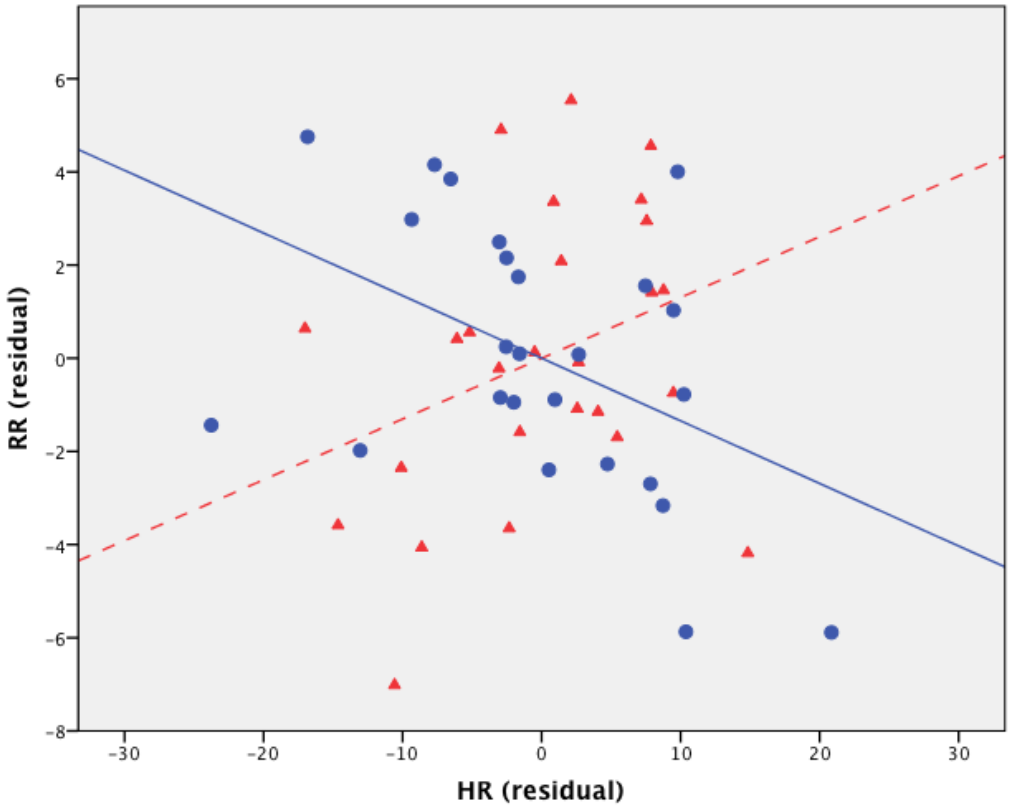

Figure 8. Relationships between HR (residual) and RR (residual) during W15. HR and RR are residuals because baseline calmness has been regressed out of each variable. The red triangles and dashed line represent data for CW. The blue circles and non-dashed line represent data for EW.

4.0 DISCUSSION

The first goal of this study was to replicate Maslej et al. (in prep) by investigating whether EW triggers exposure to depressive emotion and cognition. The second goal of this study was to extend their findings by measuring RSA and HR during EW. RSA and HR were measured to investigate whether EW increases exposure to depressive physiology.

4.1 Replicating Maslej et al. (in prep)

Participants in EW have significantly higher sadness scores during and after task, when compared to baseline measures and to CW. EW also increased exposure to AR, AUR, and ASR, relative to CW. This suggests that EW does induce exposure to depressive emotion and cognition. Research done by Hunt (1998) and Hayes et al. (2007), also support the idea that EW achieves therapeutic effect through exposure to depressive emotion and that exposure to depressive emotion is therapeutic.

Hunt (1988) exposed participants to an emotionally distressing mood induction. The mood induction involved a series of difficult cognitive tests with subsequent false, negative feedback. The mood induction induced depressed mood. After the mood induction, participants were assigned to one of three groups: emotional processing writing, disputation writing, and distraction writing. The participants in the emotional processing writing were instructed to write about their feelings about how they performed, the implications of their performance on other areas of their life, and about what could have caused them to perform the way that they did. The participants in the disputation writing were instructed to not write about how they feel or any feelings and to be as impersonal as possible, present evidence that they are brighter than the cognitive tests suggest, and to write about why cognitive tests did not have any relevance to other areas of their life. The participants in the distraction condition wrote about an episode of their favourite television show in as much detail as possible. All participants engaged in three writing sessions: after the task, during the evening, and the day after the mood induction. Emotional measures were taken after each of the writing sessions. Their results showed that there was no significant difference between groups for depressed emotion after the first writing session. However, after the second and third writing sessions, the emotional processing group showed significantly lowered levels of depressed mood compared to the other groups. Furthermore, there was no significant difference in mood between the distraction writing group and the disputation writing group. In a follow up analysis, Hunt (1998) asked whether the emotional processing group had more exposure to negative affect. The researcher analyzed the content of the writing tasks for the emotional processing group and compared it to the distraction group and found that the content of the writing in emotional processing group did indeed show significantly higher levels of negative affect. Furthermore, higher levels of depressed mood after the first writing session predicted lower levels of depressed mood after the second writing session. These results suggest that specifically engaging in and processing emotional events and exposure to depressed emotion may yield decreases in depressed mood longitudinally.

Hayes et al. (2007) integrated EW into an exposure based cognitive therapy trial for individuals with depression. The EW was designed to increase exposure and was done by patients for duration of 20 minutes weekly, between sessions with a psychotherapist. The EW instructed patients to write about "their deepest thoughts and feelings related to their depression." The trial consisted of 18 weeks of therapy. The first eight weeks were termed the *stress management phase* and they were designed to increase resilience, motivation, and to teach healthy lifestyle habits (problem solving skills, emotion regulation, and promoting healthy eating, sleeping, physical, and social activity). Weeks 9-18 were termed the *exposure-activation phase* and involved directly confronting feelings of hopelessness, negative views about oneself, and the antecedents of the depressive episode. Hayes et al. (2007) found that depressive symptoms followed a cubic pattern over the trial; depressive symptoms decreased during early weeks of the trial (this was termed *rapid early response*), increased during the *exposure-activation phase* (this was termed a *depression spike*), and then decreased after the exposure-activation phase. The researchers found that 62% of patients experienced a depressive spike and that this increase in symptoms predicted less depression after the trial was complete. Because this study involved exposure during psychotherapy sessions and EW, it's not possible to determine what contribution EW made to increase exposure. However, the EW tasks were analyzed for content suggestive of emotional processing. Analysis revealed that emotional processing indicated in EW sessions predicted post-trial depression symptom outcome. Furthermore, peak levels of emotional processing mediated the association between depression spikes and post-trial outcome.

Although the above studies suggest that EW can be used to increase exposure to depressed emotion, previous literature has had mixed results when investigating whether EW induced negative emotion. Previous research has shown that a session of EW induces negative emotion (Pennebaker & Beall, 1986; Pennebaker & Susman, 1988), however, some studies have not replicated this (Mackenzie, Wiprzycka, Hasher, & Goldstein, 2007; Smyth, Hockeymeyer & Tulloch, 2008). One explanation for the failure to replicate may include the timing of mood measurement. After a writing session is done, the mood effects of tasks may dissipate. Thus, some studies may report null results because negative emotions felt during the writing task may have dissipated after the writing task is done. Both Maslej et al. (in prep) and I have implemented a design in which we remedy this issue. We implemented mood measures before, during, and after writing the writing task was done. We both found that EW induces depressive emotion during writing.

4.2 Physiological investigation

After having shown that the data had replicated Maslej et al. (in prep), the next step was to extend her findings with an analysis of the physiological data. We have two lines of evidence suggesting that mood is not related to changes in HR and adjusted RSA. First, the correlation between baseline PHQ-9 scores and baseline physiology were examined. Results showed that baseline depression does not correlate with baseline HR, RR, or adjusted RSA. Second, data during writing was analyzed for changes and differences in physiology between EW and CW. HR and adjusted RSA were measured

during baseline and during writing Task. There was no group*phase interaction or significant effect of phase for either HR or adjusted RSA**.** The data suggest that depression may not affect these cardiorespiratory variables. This is not inconsistent with the literature. Although studies have reported that depressed individuals show increased HR and decreased RSA compared to non-depressed controls (Lehofer et al., 1999; Rottenberg, 2007; Kemp et al., 2014), not all studies replicate this finding (Moser et al., 1988; Lehofer et al., 1997; LeMoult, Yoon, Joormann, 2016). Because some studies fail to replicate such findings, it is possible that findings may be false-positives and the effects do not exist. Alternatively, the physiological variables studied may not be sensitive to the intervention used in the current study.

The relationship between depression and cardiac activity is also complicated by other factors. For instance, a meta-analysis revealed that depression severity is negatively moderately correlated with decreases in HRV ($r=-0.354$, $p<0.001$; Kemp et al., 2010). It is plausible that the current investigation did not induce severe enough depressive affect to see a distinct change in physiology. Alternatively, the physiology associated with experimentally induced acute states of depressed emotion may be different from that of chronic states of depressed emotion seen in clinical samples. Anxiety co-morbidity could, in part, explain the relationship between depression and cardiovascular physiology alterations (Kemp, Quintana, Felmingam, Matthews, & Jelinek, 2012). Kemp et al. (2012) show that depressed patients with comorbid anxiety show greater changes in HRV than depressed patients without the comorbidity. Last, it has been hypothesized that antidepressant use may be driving decreases in HRV independent of depressive symptoms (Licht et al., 2008; Kemp et al., 2010; Kemp et al., 2014; O'Regan, Kenny, Cronin, Finucane, & Kearney, 2015). For example, O'Regan et al. (2015) showed that depressed individuals not taking ADMs did not have differing HRV from non-depressed controls.

The current study showed that EW did not trigger a significant increase in HR or decrease in adjusted RSA. Because the literature is still unclear about the relationship between depression and cardiac activity, the findings of the current study should not be treated as definitive evidence that EW does not trigger depressive physiology. There are two possible explanations for why the predicted physiological changes were not found. First, it is possible that the effect does not exist or is driven by factors like co-morbid anxiety or the effects of ADMs. Second, it is possible that induced depressive states are physiologically different from those of depressed patients.

Statistical analyses revealed that there was a between group difference in baseline calmness. Specifically, EW was significantly calmer at baseline compared to CW. Consequently, baseline calmness was initially used as a covariate when running rmANCOVAs. For RR, there was a significant phase*baseline calmness interaction. Upon further investigation, analyses revealed that baseline calmness positively predicted task RR but not baseline RR. This effect was not dependent on which writing group participants were in. This is not an effect that was expected. However, a possible explanation for the data may have to do with task engagement. It is plausible that

individuals with higher baseline calmness have a greater capacity for task engagement and thus show higher RR during task. On the other hand, individuals with lower baseline calmness are less engaged by the task and consequently show lower changes in RR during task.

4.3 Exploratory correlations

To further investigate the relationship between depressive emotion, cognition, and physiology, exploratory correlations were done between during and after task sadness, analytical rumination measures, and task physiological measures. Since these are exploratory correlations, caution should be made in making interpretations. Future research needs to replicate any effects found.

Exploratory correlations revealed that AR was moderately positively correlated with HR in EW and not correlated in CW. Previous analysis showed that EW increased AR. This could suggest that depressive cognition (i.e. AR) and heart rate are positively associated. However, there was no significant between group differences in these correlations. A recent meta-analysis looked at the association between perseverative cognition and HR (Ottaviani et al., 2016). Perseverative cognition refers to repetitive and chronic thought processes that are related to a psychological stressor (Brosschot, Gerin, Thayer, 2006). Perseverative cognition encompasses anxious worry, anger rumination, and depressive rumination. The analysis by Ottaviani et al. (2016) suggested that emotional processes involving perseverative cognition are positively associated with heart rate (Hedge's g=0.20; Ottaviani et al., 2016). However, of the 10 analyzed studies for the effect, only 2 looked directly at depressive rumination; these studies reported very small effects (Gentzler, Wheat, Palmer, & Burwell, 2013; Feldman, Dunn, Stemke, Bell, & Greeson, 2014).

On the other hand, there was a trend towards a significant difference in correlation between AR and RR. In CW, the relationship between AR and RR showed a trend towards a moderate significant positive correlation. In EW, the relationship between AR and RR was a weak non-significant negative correlation. This suggests that EW may have altered the relationship between AR and RR. Interestingly, a similar pattern was also found with ASR. In any case, I am unaware of any research looking at correlations between rumination and RR. There is, however, literature looking at the effects of induced depressed affect and RR. This literature shows that depressed affect either does not affect RR or decreases it (Rehwoldt, 1911; Averill, 1969). In studies involving depressed patients, depression has been associated with increased RR (Damas-Mora et al., 1976; Damas-Mora, Souster, & Jenner, 1982). One explanation for this discordance between experimental and clinical studies may be that induced depressed emotion is physiologically different from chronic severe depression. Boiten, Frijda, and Wientjes (1994) offer an alternative explanation; they note that it is unclear whether the increased RR seen in clinical depression is driven by depressed emotion or by the anxiety that is often concurrently experience by depressed individuals. Feelings of anxiety have long

been associated with increases in RR (Christy, 1935; Finesinger, 1943; Stevenson & Ripley, 1952; Tobin et al., 1983).

General linear model analysis revealed that group interacts with HR to predict RR. To further understand the relationship between HR and RR, a scatterplot with a line of best fit was constructed. This showed that in EW, HR and RR have a negative slope. In CW, HR and RR have a positive slope. The ARH suggests that analytical rumination is a metabolically demanding process that may be energetically supported by an inefficient glycolysis pathway that metabolizes glucose without the use of oxygen (Andrews, Bharwani, Lee, Fox, & Thomson, 2015). Andrews et al. (2015) cite three lines of indirect evidence for this claim. First, glycolytic genes and metabolism have been shown to be upregulated in rodent models of depression (Uehara et al., 2006; Uehara, Sumiyoshi, Itoh, & Kurachi, 2007; Mallei et al., 2011). Second, expression of markers for oxidative energy production are decreased in rodent models of depression (Shumake, Poremba, Edwards, & Gozalez-Lima, 2000; Shumake, Edward, & Gozalez-Lima, 2001, 2002, 2003; Stone, Lehmann, Quartermain, 2007; Kanarik et al., 2011). Third, PET studies of prefrontal areas in depressed individuals show patterns suggestive of glycolysis (Drevets et al., 1992; Dunn et al., 2005). Given the evidence cited by Andrews et al. (2015), it seems plausible that the changes in brain metabolic activity may be reflected in peripheral cardiorespiratory activity. EW may be associated with a change in the relationship between HR and RR because EW increases exposure to depression. Depression places metabolic demands on prefrontal cortical areas that require high levels of glucose but relatively reduced oxygen consumption (Andrews et al., 2015).

4.4 Limitations & Future Directions

The current sample size is relatively small. There were 25 and 26 participants in EW and CW, respectively. With this sample size, the emotional and cognitive effects found by Maslej et al. (in prep) were replicated. This suggests that the current experimental design is a good candidate for further investigations of depression. The physiological effects found in this study need to be replicated with a larger sample size. Furthermore, exploratory correlations show some interesting effects, but none survive Bonferroni correction. Thus, any interpretation of data is made with caution and firm claims should only be made about exploratory effects after they are replicated.

Future research should try to replicate the findings of this study in non-clinical and clinical samples. The current study used undergraduate university students as a sample. Although this sample did include individuals experiencing low to high levels of baseline sadness, the distribution of baseline sadness data was strongly positively skewed. Most individuals included in this study were not experiencing high levels of sadness in their lives. Furthermore, sadness induced in this study was transient and not chronic. To test whether effects found in this study generalize to individuals with higher levels of chronic sadness, the experiment should be repeated with a clinical sample.

Future research should try to flesh out the physiological differences between a depressed emotional state independent of concurrent anxious emotion. Depression and anxiety are highly comorbid, share similar etiology, and are responsive to similar therapeutic interventions (Kessler et al., 1996; Barlow, 2002; Moses & Barlow, 2006). Depressive physiology and anxious physiology may differ, however. Depressed emotion induction results in a decrease or no change in RR while clinical studies of depression show increases in RR (Rehwoldt, 1911; Averill, 1969; Damas-Mora et al., 1976; Damas-Mora, Souster, & Jenner, 1982). Boiten, Frijda, and Wientjes (1994) have suggested that this discrepancy in findings may be associated with the concurrent anxiety often associated with clinical depression. Studies have suggested that the decreased HRV seen in depression may be partly driven by concurrent anxiety (Kemp et al., 2012). Clinical studies of depression should examine whether depression is related to increased RR while controlling for the presence of anxiety symptoms.

EBTs deserve more attention in being a potential therapeutic intervention for depression. Research has shown that correcting depressive cognition may yield little therapeutic effect (Gortner, Gollan, Dobson, & Jacobson, 1998; Jacobson et al., 1996; Coffman et al., 2007; Richards et al., 2016). Furthermore, research shows that exposure to and emotional processing of depressive cognition yields therapeutic effect (Hunt, 1998; Hayes et al., 2005; Hayes et al., 2007). Commonly used interventions for depression have similarly good response rates, however, they differ in their relapse rates after treatment discontinuation (Hollon, Thase, & Murkowiz, 2002; Hollon et al., 2005). Thus, the effect an intervention has on subsequent relapse rates may be a good metric for measuring how effective an intervention is. Interventions that minimize subsequent relapse should become the preferable treatment. Relapse rates after interventions with EBTs for depression are currently unknown. Hayes et al. (2007) show that an EBT follows a non-linear process of change in depressive symptoms over the course of a therapeutic intervention. The intervention is marked by spikes in depressive symptoms. Hayes et al. (2007), suggest that future studies should examine whether relapse rates for interventions with non-linear processes of change differ from therapies that show a linear process of change.

Last, future studies should further examine the relationship between analytical rumination and long-term therapeutic change. Analytical rumination is a measure of normal, adaptive, depressive cognitive processes (Andrews & Thomson, 2009; Barbic, Durisko, & Andrews, 2014). Bartoskova et al. (in prep) have shown that depression triggers a two-step analytical rumination process involving AUR and ASR. Furthermore, they show that ASR is negatively associated with depressive symptoms in a structural equation model which progresses linearly from depression to AUR to ASR. This work is cross-sectional, however. All data were collected at a single time point for all participants involved in the study. Future studies should employ a longitudinal design that examines the effects of analytical rumination on depressive symptoms over time. Future studies could use EW to increase exposure to AR and examine how depressive symptoms change over time with respect AR levels. Similarly, if future studies replicate the physiological findings reported in this study, it would be interesting to see whether exposure to

depressive physiology is associated with longitudinal changes in depressive symptoms. Physiological activation during the first session of exposure therapy for post-traumatic stress disorder (PTSD) has been shown to predict a decrease in intrusive memories across therapy (Pitman et al., 1996).

5.0 CONCLUSION

Understanding the mechanisms underlying effective therapeutic interventions is necessary to gain a better understanding of mental health conditions we treat. The RDoC emphasizes the need for a mechanistic understanding of normal and abnormal emotional and cognitive processes (Cuthbert & Insel, 2013). Specifically, this study examined the hypothesis that EW is an EBT for depression. If it is an EBT, then it should increase exposure to depressive emotion, cognition, and physiology. Malej et al. (in prep) have shown that EW increases exposure to depressive emotion and cognition. This study replicated the findings of Maslej et al. (in prep) and extended them with a physiological investigation. It was predicted that if EW is an EBT, then it should increase HR and decrease adjusted RSA. The data suggested that EW did not alter either HR or adjusted RSA. Exploratory analyses suggest the EW might induce depressive cardiorespiratory changes associated with glycolysis. Although the predicted physiological differences between groups were not found, exploratory analyses yielded results that were effective in generating new hypotheses about the cardiorespiratory changes seen in depression.

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