HEALTH RISK FEEDBACK: THE EFFECTS OF ACE INSIGHT ON STRESS REACTIVITY

A Thesis
Submitted to the Graduate Faculty
of the
North Dakota State University
of Agriculture and Applied Science

By

Heidi Anna Rued

In Partial Fulfillment of the Requirements
for the Degree of
MASTER OF SCIENCE

Major Department:
Psychology

August 2018

Fargo, North Dakota

North Dakota State University
Graduate School

Title

Health Risk Feedback: The Effects of ACE Insight on Stress Reactivity

By

Heidi Anna Rued

The Supervisory Committee certifies that this ***disquisition*** complies with North Dakota State University's regulations and meets the accepted standards for the degree of

MASTER OF SCIENCE

SUPERVISORY COMMITTEE:

Clayton Hilmert
Chair

Kathryn Gordon

Leah Irish

Britt Heidinger

Approved:

8/3/2018
Date

Mark Nawrot
Department Chair

ABSTRACT

Exposure to adverse childhood experiences (ACEs) has lasting repercussions throughout an individual's lifetime. An adult with a history of childhood trauma is at increased risk for excessive stress reactivity, which exacerbates the development of chronic disease. It is important to investigate how this information can be used for adult trauma survivors. This study assessed the psychophysiological impacts of providing "ACE insight". Participants completed questionnaires and were given false feedback that their childhood experiences put them at increased risk for excessive stress reactivity and the development of disease. Following ACE insight, participants underwent a speech stressor task during which cardiovascular reactivity was monitored and psychological reactions were assessed. Results indicated that participants with more adverse childhoods reported feeling more worried and less happy about feedback. Further, ACE insight caused a significant increase in cardiac output for participants with a history of childhood trauma. Implications and future directions are discussed.

Keywords: Adverse childhood experiences, ACE insight, stress reactivity, blood pressure

ACKNOWLEDGEMENTS

I would like to acknowledge my mentor, Dr. Clayton Hilmert, without whom this project would have not been possible. Thank you for your expertise, guidance, and patience throughout this process. I could not have asked for a better mentor. I also want to thank my thesis committee members for their excellent feedback and direction: Dr. Kathryn Gordon, Dr. Leah Irish, and Dr. Britt Heidinger. Finally, I want to acknowledge all of the research assistants who worked diligently to help me complete this project. You all have my sincere gratitude.

DEDICATION

To my parents, Timothy and Lynn Rued. Thank you for believing in my ability to succeed.

TABLE OF CONTENTS

ABSTRACT iii

ACKNOWLEDGEMENTS iv

DEDICATION v

LIST OF TABLES viii

LIST OF FIGURES ix

INTRODUCTION 1

ACEs, Stress Reactivity, and Health 2

Trauma-Informed Care 5

Reactions to ACE Insight 6

Study Overview 8

METHOD 10

Participants 10

Physiological Recording 10

Procedure 10

Measures 13

Statistical Analyses 15

RESULTS 17

Early Childhood Scores 17

Inter-item Correlations 17

Manipulation Checks and Self-Report Measures 23

Stress Reactivity 27

DISCUSSION 30

Limitations and Future Directions 32

Implications 33

REFERENCES 35

LIST OF TABLES

Table		Page
1.	Total ACE Scores	17
2.	Inter-item Correlations Between Variables: SBP – RFQ Score	19
3.	Inter-item Correlations Between Variables: Stress Response – RFQ Score	20
4.	Inter-item Correlations Between Variables: SACL Arousal – CO Reactivity	21
5.	Inter-item Correlations Between Variables: ECC Score – PANAS	22
6.	Means for Self-report Measures by Condition	23
7.	Standardized Regressions Coefficients for Predicting Change in Self-Report Measures as a Function of Condition and ECC Score	24
8.	Standardized Regressions Coefficients for Predicting Change in Manipulation Checks as a Function of Condition and ECC Score	25
9.	Standardized Regressions Coefficients for Predicting Change in Blood Pressure Measures as a Function of Condition and ECC Score	29

LIST OF FIGURES

Figure Page

1. Interaction between condition and ECC score on reported stress response..... 26

2. Interaction between condition and ECC score on cardiac output reactivity. 27

INTRODUCTION

A growing body of empirical evidence shows that there is a remarkably strong relationship between childhood trauma and poor adulthood health outcomes (Felitti et al., 1998). Children exposed to neglect and maltreatment often experience chronic stress and elevated inflammatory and cortisol levels throughout their lifetimes (Miller, Chen, & Parker, 2011). Adults with a history of childhood trauma are at risk for a host of adverse biopsychosocial outcomes such as diabetes, cardiovascular disease, lung disease, depression, and anxiety (Felitti et al., 1998; Rich-Edwards et al., 2010). As the literature now illustrates the astonishing impact of adverse childhood experiences on adulthood health, the logical next step is to recognize how to use this information.

Clearly, the ACE studies highlight the importance of ACE prevention. Public health efforts to stop childhood maltreatment from occurring have been initiated across federal and community levels (Zimmerman & Mercy, 2010). However, it is nearly impossible to put an end to all forms of ACEs. In addition to prevention, ACE studies help us identify individuals that may benefit from a post-ACE intervention. Although efforts are being made to enhance the resilience of ACE survivors (e.g. Near@Home intervention), it is still not clear how to best intervene or what interventions are most effective for this population.

One strategy for intervention could be to inform the individual of his/her ACE status and to explain how his/her adulthood health has been and will likely be impacted. We refer to this as providing "ACE insight." However, research has not yet explored how ACE insight affects a person psychologically and physiologically. It is possible that revealing such health risk information will relieve self-blame and anxiety by simply providing an origin for adverse-health issues. On the other hand, ACE insight may promote feelings of anxiety or worry. The

knowledge that one's stress responses are a pathway from ACEs to adverse health outcomes may influence sensitivity to subsequent stress reactivity. Here, we propose a study in which participants are informed of a stress-related high risk status and examine their subsequent psychophysiological stress reactivity.

ACEs, Stress Reactivity, and Health

In the 1990's, a set of landmark studies began to closely explore the perpetuating impacts of childhood stress on adulthood health. Felitti et. al., (1998) developed the ACEs questionnaire, which now serves as a framework for understanding and exploring childhood trauma health-related outcomes. The ACEs questionnaire measures traumatic experiences on several dimensions including sexual abuse, domestic violence, childhood neglect, and unfavorable home conditions. In a recent meta-analysis, compared to those without ACEs, individuals with four or more ACEs were at two to three times greater risk for cancer, heart disease, and respiratory disease (Hughes et al., 2017). In addition, people with four or more ACEs were four to seven times more likely to engage in risky sexual behavior, use illicit drugs, and have poor mental health. Adults with high ACEs have more financial problems, higher rates of problematic alcohol use, and intimate partner violence (Felitti et al., 1998; Hughes et al., 2017).

Childhood stress is believed to cause adverse health in adulthood by influencing subsequent experiences of stress and stress reactivity (Repetti, Taylor, & Seeman, 2004). Frequent and extreme stress responses exacerbate allostatic load, leading to the development of chronic health issues (Danese & McEwen, 2012). Allostasis is the process by which biological systems respond to environmental demands (McEwen & Seeman, 1999; Danese & McEwen, 2011). Allostatic load is the physiological cost of allostatic change. For example, the increase in blood pressure after getting out of bed in the morning or release of neuroendocrines in response

to a stressor allow the body to respond appropriately to these acute challenges in our daily environments (McEwen, 2005).

These changes in our allostatic systems have consequences that include energy expenditure and wear-and-tear on the systems themselves (McEwen, 2005). Usually these consequences have minimal impact because of appropriate recovery processes. However, excessive allostatic load leads to progressive wear and tear that accumulates over time without appropriate recovery. Chronic activation of allostatic systems results in tissue damage, elevated immune responses, and cellular aging (Danese & McEwen, 2012). In turn, these cumulative impacts promote the development of chronic illnesses like cardiovascular disease (McEwen, 2005). Therefore, excessive allostatic responses to stress that begin in childhood and continue over a lifetime contribute to later chronic illness.

During early development, a child learns how to mitigate responses to stress both psychologically and physiologically. In a healthy infant, the amygdala is activated by unfamiliar stimuli as these may be signs of threat (Kagan, 1997). As a child's brain develops, the prefrontal cortex learns to differentiate sources of real threat from novel, non-threatening stimuli, ultimately mitigating unnecessary and extreme stress reactions (Taylor, Lerner, Sage, Lehman, & Seeman. 2004; Loman & Gunnar, 2010). Experiencing trauma during childhood has the potential to induce significant changes in how prefrontal mechanisms moderate amygdalar activation of allostatic systems, predisposing the individual to overreact to experiences throughout the lifetime (McEwen & Seeman, 1999; Danese & McEwen, 2012).

The regulation of stress responses and related allostatic systems (e.g., neuroendocrine, cardiovascular) is coordinated by a number of brain regions including the hippocampus, amygdala, and prefrontal cortex (Lee et al., 1998). Early trauma has been associated with

decreased synaptic density of the hippocampus and less hippocampal volume in adulthood (Gould & Tanapat, 1999; Vythilingam et al., 2002; Bremner et al., 1997). Individuals with childhood trauma also show abnormal amygdala development (Mehta et al., 2009; Shin, Rauch, & Pitman, 2006; Weniger et al., 2008). Further, the prefrontal cortex is particularly vulnerable during childhood and adolescence (Lupien, McEwen, Gunnar, & Heim, 2009). Research on physically abused children showed reduced volume of the orbitofrontal cortex, a region of the prefrontal cortex associated with emotional regulation and inhibition (Hanson et al., 2010). Thus, severe forms of maltreatment in childhood (e.g. neglect) adversely influence the development of the neurobiological processes involved with stress reactivity and allostatic load (Shankoff et al., 2012; Loman & Gunnar, 2010; Danese & McEwen, 2011).

A number of studies have shown that early life stress has led to extreme physiological and psychological stress reactivity later in life. For example, studies have shown that maternal separation in early life caused rat pups to have increased neuroendocrine responses (e.g. elevated corticosteroids) and anxious behavior as adults (Levine, 1967; Levine, Cheavlier, & Korchin, 1956). Studies on primates showed a similar effect of early-life stressors on dysregulated HPA activity in offspring (Lupien, McEwen, Gunnar, & Heim, 2009). In human subjects, cold, non-nurturing, conflict-ridden early family environments have been associated with higher cardiovascular reactivity (e.g. elevated blood pressure) and dysfunctional cortisol levels in college aged students (Repetti, Taylor, & Seeman, 2002; Taylor, Lerner, Sage, Lehman, & Seeman, 2004). Sustained patterns of stress reactivity like these have been repeatedly implicated in the development of chronic disease (McEwen, 2005).

Therefore, it is critical that we identify a way to short-circuit the established pattern of excessive reactivity that originated in childhood. However, once ACEs have manifested as

excessive stress responses in later life, it is not clear how to best intervene. Efforts are being made in the development of trauma-informed care interventions.

Trauma-Informed Care

Trauma-informed care is intended to integrate an understanding of the events, experiences, and effects of trauma into existing healthcare interventions (SAMHSA, 2014). It is recognized as a multifaceted approach to intervention, thus suggesting that implementation of care can be challenging.

Some aspects of trauma-informed care can be easily applied, such as trauma assessment through the use of the ACEs questionnaire. A recent study indicated that incorporating an ACE questionnaire into routine care visits was viewed as acceptable to the patients, all of whom willingly participated in ACE screenings (Glowa, Olson, & Johnson, 2016). ACE screening can provide additional patient history and may be able to be used by healthcare providers to develop a more comprehensive and effective treatment plan. However, few studies have tested methods of using patient ACEs information.

By comparison, many psychosocial and behavioral risk factors such as family history of disease, obesity, or hypertension are accompanied by clear recommendations to change behaviors in order to reduce risk of the adverse outcomes (Pellmar, Brandt, & Baird, 2002). However, it is not clear that similar interventions will positively influence the prolonged impact of ACEs that occur over a lifetime. Specifically, it is not clear how to reduce excessive stress responses or to target other mechanisms believed to link ACEs with poor adulthood health (e.g., social competence; Repetti, Taylor, & Seeman, 2002). Components of trauma-informed care suggested by the Substance Abuse and Mental Health Services Administration are ambiguous

and multifaceted constructs, including the promotion of "resilience," that are not easily defined or influenced.

It is possible that victims of ACEs do not understand the strength of the association between their early life experiences and risk of specific adverse health outcomes. Therefore, one avenue of intervention may be to inform patients of their ACE status and associated health risks. In fact, some recently developed interventions have included this course of action (e.g., NEAR@home). However, the consequences of revealing a person's risk of poor health due to ACEs, or providing ACE insight, are unclear. For instance, telling someone that their poor health is in large part due to their early life experiences may relieve self-blame, leading to positive consequences. On the other hand, this information may promote feelings of hopelessness, increasing worry and anxiety.

Reactions to ACE Insight

Health Risk Feedback. Research with patients informed of their at-risk status for disease, due to genetics or family history can provide a helpful framework for understanding responses to ACE insight. Early studies of genetic disease carriers (e.g. Huntington's disease) suggested that providing insight regarding participants' genetic predisposition may lead to significant distress immediately following feedback and at 6 months' post-feedback (Wiggins et al., 1992). For example, women who received feedback that they were carriers for a breast cancer mutation and who had no previous cancer history reported greater test-related distress and *general distress* than non-carriers (Croyle, Smith, Botkin, Baty, & Nash, 1997). This suggests that health risk feedback has the potential to initiate feelings of anxiety and distress, especially in those without pre-existing knowledge of risk.

That is, receiving health risk information may itself act as a stressor, perhaps initiating or substantiating perseverative cognition, worry, and rumination (Brosschot, Gerin, & Thayer, 2006). ACE insight for those unaware of their health risk may create a new stressor and for those already aware, it may exacerbate their existing stress. That is, ACE insight, or the knowledge that one's unalterable past is significantly influencing current and future health may lead to feeling a lack of control, more worry, and greater sensitivity to stressful experiences. Added stressors like these can produce ongoing feelings of distress that have been associated with higher physiological stress indicators, such as higher cortisol levels (Schulz, Kirschbaum, Prubner, & Hellhammer, 1998; Miller, Chen, & Zhou, 2007) and exacerbated stress reactivity (Low, Saloman, Matthews, 2009).

Despite this, most research on disease genetic risk feedback suggests no long-term psychological impacts. For instance, a systematic review of fifteen studies on predictive genetic testing showed no long-term (e.g. three years) detrimental psychological impacts of carrier test information on participants (Broadstock, Michie, & Marteau, 2000). Similarly, in a study of 167 women tested for the BRCA1 and BRCA2 breast cancer mutations, 74% reported feeling no distress regarding their test results a year later (Halbert et al., 2011). Similar research related to mental health diagnoses suggests no increase in negative emotions and attitudes following diagnosis of a mental illness (Holm-Denoma, Gordon, Donohue, Waesche, Castro, Brown … & Joiner, 2008). In fact, patients in this study experienced a significant increase in positive emotions following diagnostic feedback, presumably due to feeling hopeful for treatment. It is important to note that diagnosis of illness is separate from health risk information, as the latter precedes actual development of health issues.

Research has also shown that health risk feedback may be minimized by participants, or judged as less serious, when it is personally relevant (Jemmott, Ditto, & Croyle, 1986). This is likely due to the defensive processing of the health risk feedback where risk information is minimized in order to reduce personal distress (Croyle, & Sande, 1988; Ditto, Jemmott, & Darley, 1988; Liberman & Chaiken, 1992). In addition, poor health is often attributed to one's own behaviors, leading to the experience of self-blame. Self-blame research shows that individuals diagnosed with a serious illness (e.g., cancer, COPD) who attribute the cause of their illness to internal or self-related factors have poorer psychological adjustment (Glinder & Compas, 1999; Plaufcan, Wamboldt, & Holm, 2012). Research indicates that self-blame is correlated with higher rates of depression in patients diagnosed with cancer (Phelan et al., 2013) and is considered to jeopardize self-worth (Beck, 1967). Health risk information such as ACE insight may alleviate these negative feelings.

Thus, health risk information may relieve self-blame by providing information that risk is due to an alternative or external cause. This could promote better psychological adjustment. Consistent with this possibility, one study showed that providing obese patients with familial risk information related to weight gain led to less self-blame about eating behaviors and higher satisfaction about weight loss (Conradt et al., 2007), clear signs of improved psychological adjustment and potentially better health outcomes. In the present study, ACE insight may relieve self-blame for poor health behaviors or for one's childhood experiences, leading to lower subsequent stress reactivity.

Study Overview

In the current study, we examined the impact of ACE insight on psychological status and psychophysiological stress reactivity in the lab. ACE insight was provided in the form of false

feedback relating answers to questions about one's past to current risk of excessive stress reactivity and related disease. Participants completed the ACE questionnaire and other questionnaires concerning their childhood and past experiences. Then, they either received feedback informing them that their answers to our questionnaires indicated that they are at risk for extreme stress reactivity and the future cardiovascular health consequences of such reactivity, or they received no feedback. Following the feedback manipulation, the participants performed a standard stressor task (public speech) while cardiovascular reactivity was measured. After the stressor task, self-reported emotions and reactions to the false feedback were assessed. This is the first study to examine the impact of ACE insight on stress reactivity.

METHOD

Participants

Data from 105 participants was collected. Participants were 49.5% male and 50.5% female. Approximately 68.5% of the sample were first year undergraduate students (18%=2nd year, 7%=3rd year, 2%=4th year, 2%=fifth year). Age of participants ranged between 18-29, (M=19.28 years). Participants were asked to refrain from caffeine, intense exercise, and eating a full meal prior to the study.

Physiological Recording

Continuous blood pressure was measured throughout the experiment using a Finometer Pro (Finapres, Netherlands). The Finometer uses validated (Schutte, Huisman, Rooyen, Malan, & Schutte, 2003; Jansen, Schreuder, Mulier, Smith, Settels, & Wesseling, 2001) methods of taking beat-to-beat continuous measures of blood pressure via a finger cuff that is placed on the middle finger of the participant's non-dominant hand. All of the experimenters were trained in the proper use of this equipment. The data was downloaded onto a Dell computer and analyzed using SPSS software.

Procedure

Prior to arrival, participants were randomly assigned to one of two conditions: 1) to receive extreme stress reactor "ACE insight" or 2) to receive no feedback (control). Once the participant arrived, they were directed to sit at a table facing two chairs in a quiet room. The experimenter explained that the purpose of the study was to understand how blood pressure and heart rate are impacted before, during, and after performing a challenging task. The experimenter also showed the participant the equipment and explained that the finger cuff would take continuous measures of his/her blood pressure throughout the experiment. Also, the

experimenter explained where the electrodes would be placed on the body and that they would measure cardiovascular functions and heart rate. After consent was obtained, participants were fitted with the Finometer finger cuff. They were also fitted with a cuff on the non-dominant arm that is used to calibrate the finger cuff once.

Following the initial hook-up, the participant was instructed to sit as still as possible and to relax for a 10-minute baseline measure. The experimenter left the room for the baseline and upon return, the participant was provided with an electronic tablet to fill out the first series of questionnaires, including the Adverse Childhood Experiences (ACEs) and Risky Families Questionnaires (RFQ; below). Once the participant was finished with the questionnaires, the experimenter told the participant that they are going into the next room to analyze their answers. After 3 minutes, the experimenter returned to the room and provided ACE insight feedback or no feedback based on the condition determined prior to arrival. This manipulation is described below.

Following the feedback, the experimenter explained that the participant was going to perform a speech task in front of an audience of two experts who are trained in public speaking. The participant was also told that they would perform the speech while their blood pressure is monitored. Participants were instructed that they would have a five-minute period to prepare a speech about why they would be a perfect applicant for an open position at a company. The experimenter left the room for the five-minute preparation period.

After five minutes, the experimenter returned with two confederates of the experiment. These confederates were invited to sit in the chairs directly across from the participant and were introduced as audience members who would evaluate the speech performance of the participant. After handing clipboards to the audience members, the experimenter offered to answer any

questions before the speech began. Then the experimenter set up a video camera pointed at the participant and explained that the video would be evaluated by speech experts at a later time. This was intended to increase stress and the feeling of social evaluative threat.

Once all questions were answered and the participant and audience members confirmed they were ready to begin the participant was instructed to give the speech. Throughout the speech, the confederates maintained neutral facial expressions and performed subtle movements to increase the feeling of evaluation (e.g. look at watch, take notes, slightly shift in seat). Around the three-minute mark of the speech, the experimenter interrupted the participant with a non-supportive phrase such as "you have to keep eye contact with the audience members" or "talk about some different qualities or characteristics". Once the speech ended, the confederates were directed into the room next door to fill out evaluation forms.

Then, the participant was instructed to fill out the first set of post-task questionnaires related to emotions during the task and manipulation effectiveness (e.g. "how did you feel about the feedback provided to you"). When the participant was finished with that set of questionnaires, the experimenter asked the participant to sit and relax for a final eight-minute recovery period. Following the eight-minute recovery, the experimenter re-entered the room and unhooked the participant. Then, the participant filled out the final set of questionnaires that include health questions and demographics. The experimenter debriefed the participant and thanked them for their time.

Feedback manipulation. In the feedback condition, the experimenter returned to the room after ostensibly analyzing the participants answers to the ACE and other early environment questionnaires to read the following:

> Answers on the questionnaires you completed suggest that you could be at high risk of being an extreme stress reactor. Studies have shown that a person's early family life is strongly predictive of how they respond to stress. "Extreme stress reactor" means that you are prone to respond to stress with stronger emotions of anxiety or anger, and with larger increases in blood pressure, heart rate, and stress hormones during stress. This is important because the studies recently looking at these associations have found that a person's childhood experiences are much more strongly associated with adulthood health than we once thought. And when responses to stress are extreme they contribute to the development of heart disease, our number 1 cause of death in the US. If you have any questions about this, please let us know after the experiment is over.

In contrast, participants in the control condition, the experimenter returned to the lab and read:

> Thank you for your honest answers to our questions. If you have any questions about them, please let us know after the experiment is over. We will now move on to the next part of the study.

Measures

Throughout the experiment, the participant completed several questionnaires presented on an electronic tablet using Qualtrics software. The first set of questionnaires presented after the baseline measure contained the ACE and RFQ assessments. Following the speech task, the participant reported on their emotional responses to the task on several questionnaires including the Positive and Negative Affect Scale – Expanded Version (PANAS – X). Also, participants answered questions related to the ACE insight manipulation effectiveness. Finally, they answered a health and demographics questionnaire.

Adverse Childhood Experiences (ACE) Questionnaire. The ACE study questionnaire was originally developed by Felitti et al., (1998). The original ACE study surveyed a sample of 9,508 adults and found adequate internal consistency (Cronbach's α=.88). The questionnaire contains 10 items that are introduced with the phrase: "While you were growing up during your first 18 years of life…" Questions are related to physical, sexual, and emotional abuse, history of alcohol or drug abuse in the family, health-related behaviors, and mental health. Items require a yes or no answer. A total ACE score is the sum of yes answers.

Risky Families Questionnaire. The Risky Families Questionnaire (RFQ) was adapted from the original Adverse Childhood Experiences questionnaire (Felitti et al., 1999). This measure was designed to be more sensitive to subtler early environmental influences than the ACEs Questionnaire (Taylor, Lerner, Sage, Lehman, & Seeman, 2004). A risky family is defined as having a cold, conflict-ridden, and unaffectionate environment. In a large study of 3,248 participants, the RFQ had a Cronbach's alpha of .77 (Taylor, Lehman, Kiefe, & Seeman, 2006). The RFQ assesses the extent to which an individual experienced physical, mental, or emotional abuse or distress during childhood using a 5-item Likert scale. Items include phrases such as "how often would you say there was quarreling, arguing, or shouting between your parents?" and "would you say the household you grew up in was chaotic and disorganized?"

Positive and Negative Affect Schedule – Expanded Form. This measure was created by Watson & Clark (1994). The PANAS–X is a 60-item measure presented with instructions to indicate how a person feels. For the present study, the questionnaire will ask participants to report how they felt during the task. Answers are on a 5-item Likert scale ranging from "very slightly or not at all" to "extremely". Sub scales can be calculated to focus on several emotional dimensions: e.g., positive and negative affect, basic negative emotions (e.g. afraid, hostile,

angry), basic positive emotions (e.g. cheerful, lively, alert), and other emotions (e.g. shy, calm, amazed).

Stress and Arousal Checklist (SACL) and Manipulation Checks. After the PANAS-X, participants completed the SACL, originally developed by Mackay et al. (1978) to measure subjective feelings of stress and arousal in British University students. A study with 42 participants reported a Cronbach's alpha of .86 for the stress scale and .74 for the arousal scale (King, Burrows, & Stanley, 1983). This questionnaire includes 20 emotions (e.g., calm, passive, lively) on a 4-item scale that ranges from "definitely no" to "definitely yes".

Other Measures. The participant filled out seven questions related to thoughts about the task and five questions relating to manipulation effectiveness. These questions were designed for this particular study. Reliability analysis with the current data set indicated a Cronbach's alpha of .859. Items related to thoughts about the task were presented on a Likert scale (1-9) ranging from not at all to very much; items about the manipulation and stress effectiveness will be presented on a Likert scale (1-7) ranging from "less than average" to "more than average". Sample questions include "how engaged were you when you gave the speech?" and "how strongly did you feel each of these emotions after the experimenter gave you feedback about your answers to the early environment questionnaires?".

Statistical Analyses

We began by examining correlations among the study variables including ACE and RFQ scores, baseline blood pressure measures, and reactivity. Because it is possible that reactions to false feedback would be affected by actual ACE and RFQ scores, we considered if the effects of our manipulation were moderated by participants' actual ACE and RFQ scores. To do this, we

created an interaction variables of early childhood experiences by condition in the following analyses.

We examined the effects of the ACE insight manipulation on manipulation checks and self-reported stress and emotions using hierarchical regressions in which condition and early childhood experience score were entered simultaneously in step 1, and then the interaction in step 2. To examine the physiological outcomes of interest we tested the effect of the manipulation on the components of CVR in separate analyses.

First, to see if participants differed on baseline measures, we conducted hierarchical regressions predicting baseline physiological measures with condition and early childhood scores. Also using hierarchical regression analyses, we predicted residualized measures of systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), cardiac output, and total peripheral resistance reactivity by entering the appropriate baseline measure in the first step, followed by the dichotomized manipulation variable and early experience variables in the second step, and the interaction of these in the final step.

RESULTS

Early Childhood Scores

Participants in the sample reported a range of ACE (M = .97, SD = 1.53) and RFQ scores (M = 18.10, SD = 7.69). ACE score frequencies can be viewed in Table 1. More than half the participants reported no instances of adverse childhood experiences. However, almost 10% reported four or more ACEs, which is recognized as the "tipping point" by which ACEs contribute to substantial risk of poor adulthood health outcomes (Felitti et al., 1998). Therefore, all analyses were done with early childhood experience index score (see correlational analyses below) as a continuous measure, and with ACE scores dichotomized as zero vs some ACEs, and as zero to three vs. four or more ACEs. The results of these analyses did not differ, therefore we only report the results of analyses involving the continuous measures below.

Table 1

Total ACE Scores

Score	Frequency	Percent
0	55	52.4
1	31	29.5
2	5	4.8
3	4	3.8
4	6	5.7
5	1	1.0
6	2	1.9
8	1	1.0
Total	105	100.0

Inter-item Correlations

Correlations between the study variables are presented in Tables 2-5. As expected, reactivity measures were correlated with one another. Negative affect (e.g. sad, worried) and SACL arousal were significantly positively correlated. SACL stress and arousal were

significantly negatively correlated. In general, positive emotions about feedback (e.g. happy, relieved) were positively correlated with one another. Similarly, negative emotions about feedback (e.g. sad, worried) were positively correlated to the SACL stress scale and the PANAS negative affect scale. Total ACE score was negatively related to feeling happy about feedback, and positively related to feeling worried about feedback. Similarly, RFQ score was negatively correlated to feeling happy about feedback.

The correlation between total ACE score and total RFQ score was .753 ($p < .01$). Therefore, we created an *early childhood composite (ECC) score*, by calculating the average of the combined ACE and RFQ z-scores for each participant. ECC score was significantly negatively correlated with feeling happy about feedback. Also, ECC score was significantly positively correlated with feeling worried about feedback. ECC score was not significantly correlated with the PANAS or the SACL scales, or any reactivity measures.

Table 2

Inter-item Correlations Between Variables: Baseline SBP – RFQ Score

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Baseline SBP	1												
2. Baseline DBP	.542**	1											
3. Baseline MAP	.933**	.623**	1										
4. Baseline HR	.018	.191	.099	1									
5. Baseline SV	.056	-.155	-.105	-.267**	1								
6. Baseline CO	.071	-.119	-.083	.321**	.483**	1							
7. SBP Reactivity	-.036	.040	.026	-.080	-.114	-.081	1						
8. DBP Reactivity	-.348**	-.925**	-.378**	-.136	.068	.038	.219**	1					
9. MAP Reactivity	.076	.105	.164	-.113	-.165	-.159	.951**	.204*	1				
10. HR Reactivity	.229*	.019	.239*	-.052	-.092	.077	.197*	.135	.296**	1			
11. SV Reactivity	-.380**	-.148	-.340**	-.017	-.036	-.055	.202*	.062	.037	-.378**	1		
12. CO Reactivity	-.180	-.176	-.189	-.125	.031	-.088	.254**	.166	.162	.378**	.636**	1	
13. ACE score	-.085	-.086	-.111	-.079	-.089	-.315**	-.069	.052	-.052	-.053	-.108	.002	1
14. RFQ Score	-.044	-.069	-.060	-.099	-.105	-.258**	-.080	.059	-.045	-.081	-.165	.001	.753**

* = Significant at 0.05 level.
** = Significant at 0.01 level

Table 3

Inter-item Correlations Between Variables: Stress Response – RFQ Score

	1	2	3	4	5	6	7	8	9	10	11	12
1. Stress Response During Speech	1											
2. Emotional Response to Stress	.556**	1										
3. Response to Feedback – Happy	-.118	-.057	1									
4. Response to Feedback – Sad	.220*	.257*	-.154	1								
5. Response to Feedback – Relieved	-.004	.103	.512**	-.018	1							
6. Response to Feedback – Worried	.455**	.349**	-.257**	.631**	.052	1						
7. Response to Feedback – Hopeful	.035	.077	.652**	.006	.507**	.175	1					
8. SACL Arousal	-.065	-.168	.173	-.119	.033	-.128	.167	1				
9. SACL Stress	.484**	.360**	-.246**	.233**	.038	.403**	-.083	-.308**	1			
10. PANAS Negative Affect	.437**	.331**	-.177	.289**	-.145	.381**	-.033	-.098	.726**	1		
11. PANAS Positive Affect	.162	.167	-.353**	.098	-.058	.163	-.296**	-.385**	.542**	.368**	1	
12. ACE Score	.160	.093	-.231**	.000	-.127	.232*	-.018	.108	-.016	.071	-.022	1
13. RFQ Score	.161	.139	-.269**	-.029	-.185	.200	-.013	.099	.097	.218*	.090	.753**

* = Significant at 0.05 level.

** = Significant at 0.01 level.

Table 4

Inter-item Correlations Between Variables: SACL Arousal – CO Reactivity

	1	2	3	4	5	6	7	8	9
1. SACL Arousal	1								
2. SACL Stress	-.308**	1							
3. PANAS Negative Affect	-.098	.726**	1						
4. PANAS Positive Affect	-.385**	.542*	.368**	1					
5. SBP Reactivity	-.217*	-.005	.003	.101	1				
6. DBP Reactivity	.071	.050	.088	-.024	.219*	1			
7. HR Reactivity	-.088	.113	.113	.058	.197*	.175	1		
8. MAP Reactivity	-.182	-.003	.006	.109	.951**	-.128	.167	1	
9. SV Reactivity	.007	.032	.003	-.044	.202**	.403**	-.083	-.308**	1
10. CO Reactivity	-.005	.141	.119	-.030	.254**	.381**	-.033	-.098	.726**

* = Significant at 0.05 level.
** = Significant at 0.01 level.

Table 5

Inter-item Correlations Between Variables: ECC Score – PANAS

	1	2	3	4	5	6	7	8	9	10
1. ECC Score	1									
2. Stress Response During Speech	.169	1								
3. Response to Feedback –Happy	-.262*	-.118	1							
4. Response to Feedback – Sad	-.014	.220*	.137	1						
5. Response to Feedback – Relieved	-.162	-.004	.512**	-.018	1					
6. Response to Feedback – Worried	.228*	.455**	-.257*	.631**	.618	1				
7. Response to Feedback – Hopeful	-.016	.035	.652**	.006	.000	.175	1			
8. SACL Arousal	.110	-.065	.173	-.119	.033	-.128	.167	1		
9. SACL Stress	.038	.484**	-.246*	.233*	.038	.430**	-.083	-.308**	1	
10. PANAS Negative Affect	.146	.437**	-.177	.289**	-.145	.381**	-.033	-.083	.726**	1
11. PANAS Positive Affect	.032	.162	-.353**	.098	-.058	.163	-.296**	-.385**	-.308**	-.098

* = Significant at 0.05 level.
** = Significant at 0.01 level.

Manipulation Checks and Self-Report Measures

Participants who received feedback reported more stress, (M = 27.31, SD = 6.22) than those who did not receive feedback, (M = 24.86, SD = 7.10). Means for self-report emotion measures by condition are displayed in Table 6. That is, hierarchical regressions revealed that participants in the feedback conditions were marginally more stressed as measured by the SACL stress scale, *ß*= .179, $p = .09$. There were no main effects of condition or ECC score on the SACL arousal or PANAS scores. Results of these analyses can be found in Table 7. Also, all results for manipulation checks can be found in Table 8.

Questions that asked specifically about emotions related to feedback and one's stress response compared to the average revealed a significant main effect of ECC score on "feeling happy" about feedback, $ß = -.253$, $p < .05$, $\Delta R^2 = .076$, and "feeling worried' about feedback, $ß = .223$, $p < .05$, $\Delta R^2 = .05$. Specifically, participants with more adverse ECC scores reported being less happy and more worried about feedback than those with lower ECC scores.

Table 6

Means for Self-Report Measures by Condition

Condition	SACL Stress	SACL Arousal	PANAS Positive Affect	PANAS Negative Affect
1. Control	24.86	25.34	32.31	24.54
2. Feedback	27.31	23.75	33.24	25.57

Table 7

Standardized Regressions Coefficients for Predicting Change in Self-Report Measures as a Function of Condition and ECC Score

	SACL Stress		SACL Arousal		PANAS Positive Affect		PANAS Negative Affect	
Predictor	*ß*	ΔR^2	*ß*	ΔR^2	*ß*	ΔR^2	*ß*	ΔR^2
Step 1		.033		.044		.007		.031
Z-Score: Condition	.179		-.180		.078		.097	
ECC Score	.022		.127		.024		.136	
Step 2		.005		.000		.000		.005
Condition x ECC	-.072		.018		.017		.071	

ECC = Early Childhood Composite Score

Table 8

Standardized Regressions Coefficients for Predicting Change in Manipulation Checks as a Function of Condition and ECC Score

	Average Stress Response		Happy About Feedback		Sad About Feedback		Relieved About Feedback		Worried About Feedback		Hopeful About Feedback	
Predictor	ß	ΔR^2	ß	ΔR^2	ß	ΔR^2	ß	ΔR^2	ß	ΔR^2	ß	ΔR^2
Step 1		.051		.076*		.006		.027		.055		.007
Z-Score: Condition	.151		-.089		.074		.029		.055		-.084	
ECC Score	.153		-.253*		-.021		-.165		.223*		-.008	
Step 2		.034		.000		.005		.000		.000		.004
Condition x ECC	-.188		.008		.074		.002		.000		.064	

Note: * <.05, ** <.01

Finally, there was a marginally significant interaction between condition and the ECC score on reported stress response compared to the average stress response, ß = -.263, p = .07, ΔR^2 = .034. This interaction is depicted in Figure 1. ß =.358, p < .05, such that low ECC participants who received feedback reported having a greater than average stress response than those in the control condition. In contrast, when ECC was high there was no effect of feedback on reported average stress response, ß =.017, p = .894. Interestingly, simple slopes also revealed that participants with high ECC scorers in the control condition reported a greater average stress response than participants with low ECC scores in the control condition, ß =.408, p < .05. This suggests that our manipulation had its intended effect on participants with low ECC scores, and that those with high ECC scores already knew their stress responses were greater than average.

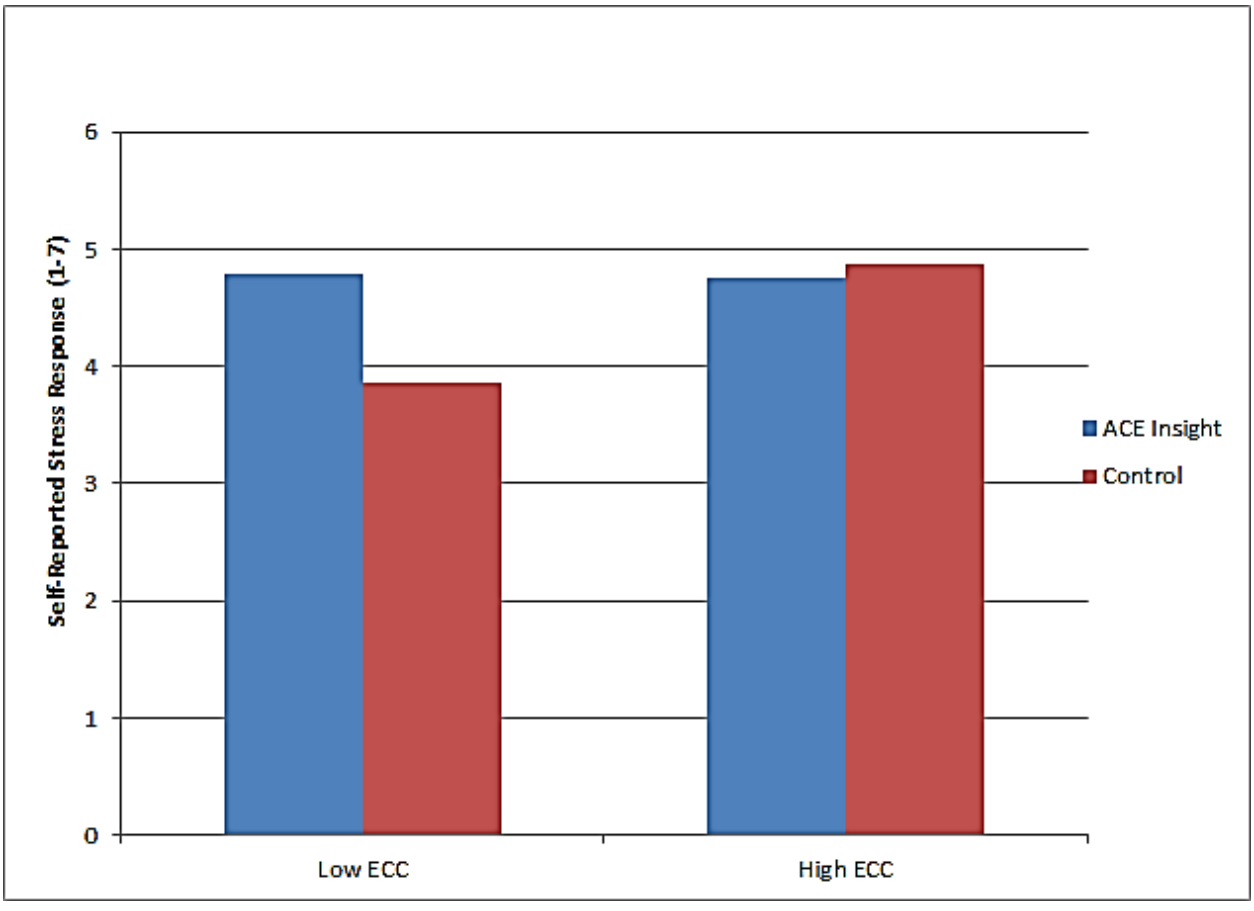

Figure 1. Interaction between condition and ECC score on reported stress response.

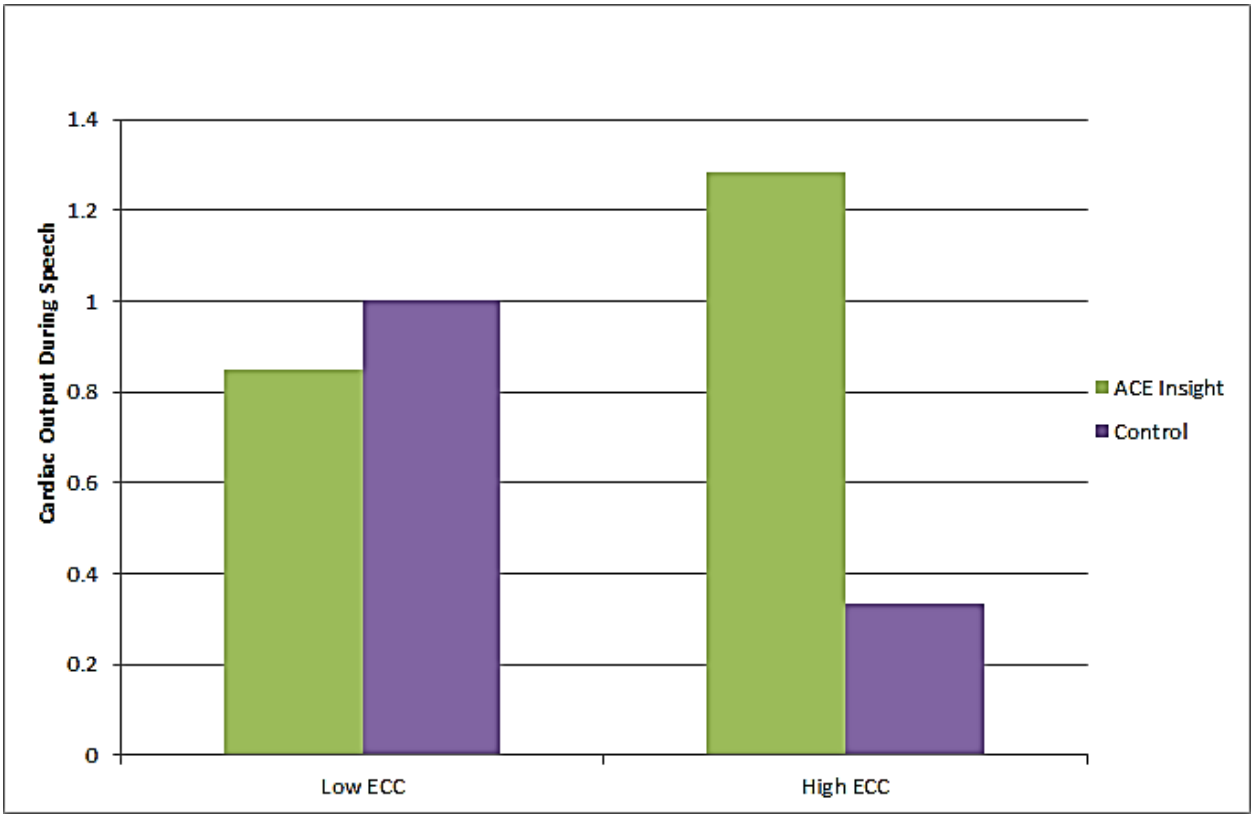

Figure 2. Interaction between condition and ECC score on cardiac output reactivity.

Stress Reactivity

Before analyzing reactivity, we first examined baseline measures in relation to condition and ECC score. These regression analyses revealed a significant association between ECC scores and baseline cardiac output, $ß = -.324$, $p < .01$. That is, participants with higher ECC scores entered the laboratory with lower cardiac output. There were no other significant associations with baseline physiological measures.

Results from separate hierarchical regressions predicting residualized SBP, DBP, HR, CO, and TPR reactivities are presented in Table 9. Analyses revealed that there was a significant interaction between condition and ECC score on cardiac output reactivity, $ß = .276$ Liters/min, $p < .05$, $\Delta R^2 = .038$. This interaction is depicted in Figure 2. Simple slopes analyses showed that when participants had high ECC scores, there was a significant effect of condition on cardiac

output, $ß = .252$, $p < .05$. However, when ECC scores were low there was no significant effect of condition on cardiac output, $ß = -.040$, $p = .677$. There was a moderately significant effect of ECC score for participants in the control condition, $B = -.374$ L/min, $p = .081$ such that those with high ECC scores had lower cardiac output than those with low ECC scores. There were no other significant effects on physiological variables.

Table 9

Standardized Regressions Coefficients for Predicting Change in Blood Pressure Measures as a Function of Condition and ECC Score

	Systolic BP (mmHg)		Diastolic BP (mmHg)		Heart Rate (BPM)		Cardiac Output (L/mn)		Total Peripheral Resistance (mmHg.min/l)	
Predictor	ß	ΔR^2	ß	ΔR^2	ß	ΔR^2	ß	ΔR^2	ß	ΔR^2
Step 1		.336		.221		.340		.513		.673
Baseline	.580		.471		.538		.716		.820	
Step 2		.005		.004		.007		.009		.000
Z-Score: Condition	.030		-.052		.084		.099		-.008	
ECC Score	-.071		-.034		-.002		-.017		-.015	
Step 3		.002		.001		.001		.020*		.008
Condition x ECC	.040		.039		-.026		.143*		-.089	

Note. * <.05

DISCUSSION

Because ACE scores and RFQ scores were highly correlated, we created the Early Childhood Composite score for our analyses. Participants with higher ECC scores reported feeling less happy and more worried about feedback, regardless of condition. This is curious because participants in the control condition received no feedback other than an expression of gratitude for completing the questionnaires. Therefore, we suspect that completing questionnaires about adverse childhood experiences was less pleasant for those with higher ECC scores across both conditions. Of course, this is only speculative as we did not have a no-questionnaire condition or a pre-questionnaire measure of mood. Nevertheless, this result is informative for interpreting many of our findings as it indicates that individuals with more childhood adversity responded differently to the experimental situations than those without a history of childhood adversity.

For instance, there was marginally significant interaction effect of ECC score and feedback condition on self-reported stress relative to the average response. Specifically, answers to the question, "Compared to the average participant, my stress response during my performance was…" revealed that receiving high-risk feedback caused participants with low ECC scores to report a greater stress response than when they did not receive this feedback (see Figure 1). However, there was no effect of feedback on participants with high ECC scores. Further analyses revealed that participants who had high ECC scores were already reporting that they were relatively high stress reactors, whereas those with low ECC scores needed feedback to report high stress responses. This suggests that our manipulation was effective in changing the perception of one's risk of extreme stress reactivity for low ECC individuals, but not for high ECC individuals because they already perceived themselves to be extreme stress reactors.

In contrast to self-reported perceptions of reactivity, psychological reactions to the speech task measured by the post-task PANAS and SACL scales did not differ across conditions or ECC scores. One exception to this was a marginally significant effect of feedback on the SACL stress sub-scale, such that participants who received ACE insight reported more stress than those in the control condition. Although we did not find compelling evidence that ACE insight affected the psychology of those with high ECC scores, physiological measures showed a different pattern.

In terms of physiological stress reactivity, it appeared that for individuals with a history of childhood trauma, ACE insight had physiological consequences. Interestingly, participants with higher ECC scores entered the laboratory with lower cardiac output. Further, when there was no feedback, cardiac output during the speech task of participants with high ECC scores did not increase as much as those with low ECC scores. It is possible that this was due to a lack of task engagement. However, the provision of ACE insight lead to a significantly higher cardiac output that matched the cardiac output of those with low ECC scores. Therefore, it is possible that feedback led to greater task engagement in those with high ECC scores. Related research has explored the relationship between task engagement and physiological responses.

Task engagement refers to a multidimensional, psychophysiological process that combines cognition, motivation, and affect to aid in effortful action and task performance (Fairclough, Ewing, & Roberts, 2009). Research concerning task engagement has shown that physiological parameters such as sympathetic nervous system activity (e.g. systolic blood pressure) are reliable of indicators of task engagement. Also, research suggests that when an individual is mentally overwhelmed, they are likely to withdraw from a task as it is viewed as too difficult to achieve. In this case the participants are more likely to exhibit patterns of physiology characterized by blunted cardiac output and increase total peripheral resistance.

For example, a study on adolescents revealed that those with childhood trauma showed significantly lower cardiac output and elevated total peripheral resistance during a social stressor task (the Trier Social Stress Task) than those without childhood trauma (McLaughlin, Sheridan, Alves, & Mendes, 2014). The researchers suggested that this pattern indicated that maltreated adolescents appraised the stressor as a threat, or considered the demands of the situation to outweigh their own capabilities. In the present study, individuals with a history of childhood adversity in the control condition were probably more likely to appraise the stressor as threatening than those without childhood adversity, resulting in less cardiac output. Additionally, this suggests that ACE insight may have influenced cardiac output and task engagement of those with higher ECC scores by influencing appraisals of the stressor task (Fairclough, Ewing, & Roberts, 2009).

This conclusion suggests that though people with childhood trauma may disengage naturally from a challenging task, ACE insight increases task engagement through appraisal and feedback mechanisms. Thus, this evidence suggests that ACE insight could be processed as a positive form of feedback for those with a history of trauma, consistent with the above research. That is, ACE insight perhaps provided an external cause (e.g., adverse childhood experiences) for one's stress responding, thereby placing blame on other factors than the individual themselves.

Limitations and Future Directions

It is possible that our ACE insight manipulation was too subtle. Thus, health risk feedback *directly* stating ACEs and RFQ answers indicated risk could lead to larger differences in physiological responses. Furthermore, this preliminary study on ACE insight provided participants with false feedback related to their early childhood experiences. Some of our effects

may have been blunted as a result of individuals not believing the risk feedback related to early childhood experiences if they had no history of childhood trauma. Additionally, our data was collected in a primarily Caucasian, middle-class community. This was reflected in our sample statistics, as rates of childhood maltreatment were primarily low. Future studies should incorporate a more diverse sample so as to further understand the relationship between childhood trauma, health risk feedback, and psychophysiological stress responses.

Implications

One important consideration when evaluating reactions to health risk information such as ACE insight is an individual's coping style. Evidence suggests that adults with a history of childhood abuse engage in maladaptive coping strategies and defense mechanisms when faced with stress or thoughts of past trauma (Futa, Nash, Hansen, & Garbin, 2003). This includes disengagement (e.g., avoidance, solitude), repression (e.g. ignoring threatening/painful information), and rationalization (e.g. providing reasons for trauma) (Ward, 1988). Methods of coping like these are associated with poor health outcomes. It is possible that ACE insight provides a person with information that helps relieve some of these maladaptive coping strategies.

Our study clearly indicates that ACE insight is perceived differently by those with and without a history of childhood trauma. Here, we have provided evidence that simply assessing ACEs can be distressing, but also that providing ACE insight may affect how people cope with subsequent stressful situations. That is, ACE insight may be affecting appraisals of stressful situations thus leading to greater task engagement, consistent with our physiological results. Despite this, our research also suggests that people who have had trauma may already be aware of their risk of high stress reactivity. Therefore, this leads to the question of whether or not ACE

insight provides any *real* insight to those with childhood adversity, at least of its impact on adulthood stress reactivity.

This suggests that we need to be cautious in making assumptions about whether or not providing ACE insight is helpful or harmful for those with a history of ACEs. Regardless, it is clear that providing ACE insight without providing resources to reduce ACE-related risks is a questionable intervention. Altogether, our findings highlight the need for additional research in this area so as to further understand the psychophysiological impacts of ACE insight on individuals with a history of adverse childhoods.

REFERENCES

Beck, A. (1967). *Depression: Clinical, experimental, and theoretical aspects*. New York: Hoeber Medical Division, Harper & Row.

Blascovich, J., Mendes, W. B., Hunter, S. B., & Salomon, K. (1999). Social "facilitation" as challenge and threat. *Journal of Personality and Social Psychology, 77*(1), 68-77.

Bremner, J. D., Randall, P., Vermetten, E., Staib, L., Bronen, R. A., Mazure, C., & ... Charney, D. S. (1997). Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse: A preliminary report. *Biological Psychiatry, 41*(1), 23-32. doi:10.1016/S0006-3223(96)00162-X

Broadstock, M., Michie, S., & Marteau, T. (2000). Psychological consequences of predictive genetic testing: a systematic review. *European Journal of Human Genetics, 8,* 731-738.

Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research, 60*(2), 113-124. doi:10.1016/j.jpsychores.2005.06.074

Brosschot, J. F., Pieper, S., & Thayer, J. F. (2005). Expanding stress theory: Prolonged activation and perseverative cognition. *Psychoneuroendocrinology, 30*(10), 1043-1049. doi:10.1016/j.psyneuen.2005.04.008

Conradt, M., Dierk, J., Schlumberger, P., Albohn, C., Rauh, E., Hinney, A., & ... Rief, W. (2009). A consultation with genetic information about obesity decreases self-blame about eating and leads to realistic weight loss goals in obese individuals. *Journal of Psychosomatic Research, 66*(4), 287-295. doi:10.1016/j.jpsychores.2008.09.003

Croyle, R. T. & Sande, G. N. (1988). Denial and confirmatory search: Paradoxical consequences of medical diagnosis. *Journal of Applied Social Psychology, 18*(6), 473-490.

Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior, 106*(1), 29-39. doi:10.1016/j.physbeh.2011.08.019

Fairclough, S. H., Ewing, K. C., & Roberts, J. (2009). Measuring task engagement as an input to physiological computing. In *Affective Brain-Computer Interfaces (aBCI) Computers in Human Behavior (2012), workshop as part of international conference on affective computing & intelligent interaction*. Amsterdam.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., & ... Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine, 14*(4), 245-258. doi:10.1016/S0749-3797(98)00017-8

Futa, K. T., Nash, C. L., Hansen, D. J., & Garbin, C. P. (2003). Adult survivors of childhood abuse: An analysis of coping mechanisms used for stressful childhood memories and current stressors. *Journal of Family Violence, 18*(4), 227-239.

Glinder, J. G., & Compas, B. E. (1999). Self-blame attributions in women with newly diagnosed breast cancer: A prospective study of psychological adjustment. *Health Psychology, 18*(5), 475-481. doi:10.1037/0278-6133.18.5.475

Glowa, P. T., Olson, A. L., & Johnson, D. J. (2016). Screening for adverse childhood experiences in a family medicine setting: A feasibility study. Journal of the American Board of Family Medicine, 29(3), 303-307. doi: 10.3122/jabfm.2016.03.150310.

Gould, E. & Tanapat, P. (1999). Stress and hippocampal neurogenesis. *Biological Psychiatry, 46(*11), 1472-1479. doi:10.1016/S0006-3223(99)00247-4.

Halbert, C. H., Stopfer, J. E., McDonald, J., Weathers, B., Collier, A., Troxel, A. B., Domcheck, S. (2011). Long-term reactions to genetic testing for *BRCA1* and *BRCA2* mutations: Does time heal women's concerns? *Journal of Clinical Oncology, 29*(32), 4302-4306.

Heleniak, C., Jenness, J. L., Vander Stoep, A., McCauley, E., & McLaughlin, K. A. (2016). Childhood maltreatment exposure and disruptions in emotional regulation: A transdiagnostic pathway to adolescent internalizing and externalizing psychopathology. *Cognitive Therapy and Research, 40*(3), 394-415. doi:10.1007/s10608-015-9735-z

Heleniak, C., McLaughlin, K. A., Ormel, J., & Riese, H. (2016). Cardiovascular reactivity as a mechanism linking child trauma to adolescent psychopathology. *Biological Psychology, 120*, 108-119.

Hilmert, C.J., Kulik, J.A., Christenfeld, N. (2002). The varied impact of social support on cardiovascular reactivity. *Basic and Applied Social Psychology, 24*(3), 229-240. doi:10.1207/153248302760179147

Holm-Denoma, J. M., Gordon, K. H., Donohue, K. F., Waesche, M. C., Castro, Y., Brown, J. S., & ... Joiner, T. J. (2008). Patients' affective reactions to receiving diagnostic feedback. *Journal of Social and Clinical Psychology, 27*(6), 555-575. doi:10.1521/jscp.2008.27.6.555

Hughes, K., Bellis, M. A., Hardcastle, K. A., Sethi, D., Butchart, Mikton, C., & … Dunne, M. P. (2017). The effect of multiple adverse childhood experiences on health: A systematic review and meta-analysis. *The Lancet, 2*(8), e356-e366. doi:10.1016/S2468-2667(17)30118-4

Ito, Y., Teicher, M. H., Glod, C. A., Harper, D., Magnus, E., & Gelbard, H. A. (1993). Increased prevalence of electrophysiological abnormalities in children with psychological, physical, and sexual abuse. *The Journal of Neuropsychiatry and Clinical Neurosciences, 5*(4), 401-408.

Jansen, J. R. C., Schreuder, J. J., Mulier, J. P., Smith, N. T., Settels, J. J., & Wesseling, K. H. (2001). A comparison of cardiac output derived from the arterial pressure wave against thermodilution in cardiac surgery patients. *British Journal of Anesthesia, 87*(2), 212-222.

Janoff-Bulman, R. (1979). Characterological versus behavioral self-blame: Inquiries into depression and rape. *Journal of Personality and Social Psychology, 37*(10), 1798-1809.

Jemmott, J. B., Ditto, P. H., & Croyle, R. T. (1986). Judging health status: Effects of perceived prevalence and personal relevance. *Journal of Personality and Social Psychology, 50*(5), 899-905.

Kagan, J. (1997). Temperament and the reactions to unfamiliarity. *Child Development, 68*(1), 139-143. doi:10.2307/1131931

King, M.G., Burrows, G.D., & Stanley, G.V. (1983) Measurement of stress and arousal: Validation of the stress/arousal subjective checklist. *British Journal of Psychology, 74,* 473-479. doi:10.1111/j.2044-8295.1983.tb01880.x

Lazarus, R. S., & Alfert, E. (1964). Short-circuiting of threat by experimentally altering cognitive appraisal. *Journal of Abnormal and Social Psychology, 69*(2), 195-205. doi:10.1037/h0044635

Lazarus, R. S., & Folkman, S. (1984a). Coping and adaptation. In W. D. Gentry (Ed.), *The handbook of behavioral medicine* (pp. 282-325). New York: Guilford

Lee, G. P., Bechara, A., Adolphs, R., Arena, J., Meador, K. J., Loring, D. W., & Smith, J. R. (1998). Clinical and physiological effects of stereotaxic bilateral amygdalotomy for intractable aggression. *The Journal of Neuropsychiatry and Clinical Neurosciences, 10*, 413-420.

Levine, S. (1967). Maternal and environmental influences on the adrenocortical response to stress in weanling rats. *Science, 156*(3772), 258-260. doi:10.1126/science.156.3772.258

Levine, S., Chevalier, J. A., & Korchin, S. J. (1956). The effects of early shock and handling on later avoidance learning. *Journal of Personality, 24*(4), 475-493. doi:10.1111/j.1467-6494.1956.tb01283.x

Liberman, A. & Chaiken, S. (1992). Defensive processing of personally relevant health messages. *Personality and Social Psychology, 18*(6), 669-679.

Loman, M. M., & Gunnar, M. R. (2010). Early experience and the development of stress reactivity and regulation in children. *Neuroscience and Biobehavioral Reviews, 34*(6), 867-876. doi:10.1016/j.neubiorev.2009.05.007

Low, C. A., Salomon, K., Matthews, K. A. (2010). Chronic life stress, cardiovascular reactivity, and subclinical cardiovascular disease in adolescents. *Psychosomatic Medicine, 71*(9), 927-931.

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience, 10*(6), 434-445. doi:10.1038/nrn2639

MacKay, C., Cox, T., Burrows, G., & Lazzerini, T. (1978). An inventory for the measurement of self-reported stress and arousal. *British Journal of Social and Clinical Psychology, 17*, 283-284.

McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences, 840,* 33-44. doi:10.1111/j.1749-6632.1998.tb09546.x

McEwen, B. S. (2005). Stressed or stressed out: What is the difference? *Journal of Psychiatry and Neuroscience, 30*(5), 315–318.

McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. In N. E. Adler, M. Marmot, B. S. McEwen, J. Stewart, N. E. Adler, M. Marmot, ... J. Stewart (Eds.), *Socioeconomic status and health in industrial nations: Social, psychological, and biological pathways* (pp. 30-47). New York, NY, US: New York Academy of Sciences.

McLaughlin, K. A., & Lambert, H. R. (2017). Child trauma exposure and psychopathology: mechanisms of risk and resilience. *Current Opinion in Psychology, 14*, 29-34.

McLaughlin, K. A., Sheridan, M. A., Alves, S., & Mendes, W. B. (2014). Child maltreatment and autonomic nervous system reactivity: Identifying dysregulated stress reactivity patterns using the biopsychosocial model of challenge and threat. *Psychosomatic Medicine, 76*(7), 538–546.

Mehta, M. A., Golembo, N. I., Nosarti, C., Colvert, E., Mota, A., Williams, S. R., & ... Sonuga-Barke, E. S. (2009). Amygdala, hippocampal and corpus callosum size following severe early institutional deprivation: The English and Romanian Adoptees study pilot. *Journal of Child Psychology and Psychiatry, 50*(8), 943-951. doi:10.1111/j.1469-7610.2009.02084.x

Mendes, W. B., McCoy, S., Major, B., & Blascovich, J. (2008). How attributional ambiguity shapes physiological and emotional responses to social rejection and acceptance. *Journal*

of Personality and Social Psychology, *94*(2), 278–291. http://doi.org/10.1037/0022-3514.94.2.278

Miller, S. M. (1987). Monitoring and blunting: Validation of a questionnaire to assess styles of information seeking under threat. *Journal of Personality and Social Psychology, 52,* 345-353. doi:10.1037/0022-3514.52.2.345

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin, 137*(6), 959-997. doi:10.1037/a0024768

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological Bulletin, 133*(1), 25-45. doi:10.1037/0033-2909.133.1.25

Miller, S. Z., Brody, D. S., & Summerton, J. (1988). Styles of coping with threat: Implications for health. *Journal of Personality and Social Psychology, 54*(1), 142-148.

Nestler, E. J., Barrot, M., DiLeone, R. J., Eisch, A. J., Gold, S. J., & Monteggia, L. M. (2002). Neurobiology of depression. *Neuron, 34*(1), 13-25, doi:10.1016/S0896-6273(02)00653-0

O'Donnell, K., Brydon, L., Wright, C. E., & Steptoe, A. (2008). Self-esteem levels and cardiovascular and inflammatory responses to acute stress. *Brain, Behavior, And Immunity, 22*(8), 1241-1247. doi:10.1016/j.bbi.2008.06.012

Pellmar, T. C., Brandt, E. J., & Baird, M. A. (2002). Health and behavior: The interplay of biological, behavioral, and social influences: Summary of an Institute of Medicine report. *American Journal of Health Promotion, 16*(4), 206-219. doi:10.4278/0890-1171-16.4.206

Phelan, S. M., Griffin, J. M., Jackson, G. L., Zafar, S. Y., Hellerstedt, W., Stahre, M., & ... van Ryn, M. (2013). Stigma, perceived blame, self-blame, and depressive symptoms in men with colorectal cancer. *Psycho-Oncology, 22*(1), 65-73. doi:10.1002/pon.2048

Plaufcan, M. R., Wamboldt, F. S., & Holm, K. E. (2012). Behavioral and characterological self-blame in chronic obstructive pulmonary disease. *Journal of Psychosomatic Research, 72*(1), 78-83. doi:10.1016/j.jpsychores.2011.10.004

Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin, 128*(2), 330-366. doi:10.1037/0033-2909.128.2.330

Rich-Edwards, J. W., Spiegelman, D., Hibert, E. L., Jun, H., Todd, T. J., Kawachi, I., & Wright, R. J. (2010). Abuse in childhood and adolescence as a predictor of type 2 diabetes in adult women. *American Journal of Preventive Medicine, 39*(6), 529-536. doi:10.1016/j.amepre.2010.09.007

SAMHSA (2014). Trauma-informed approach and trauma-specific interventions. Retrieved June 20, 2017 from https://store.samhsa.gov/shin/content/SMA14-4884/SMA14-4884.pdf

Schulz, P., Kirschbaum, C., Prüsner, J., & Hellhammer, D. (1998). Increased free cortisol secretion after awakening in chronically stressed individuals due to work overload. *Stress Medicine, 14*(2), 91-97. doi:10.1002/(SICI)1099-1700(199804)14:2<91::AID-SMI765>3.0.CO;2-S

Schutte, A. E., Huisman, H. W., van Rooyen, J. M., Malan, N. T., & Schutte, R. (2004). Validation of the finometer device for measurement of blood pressure in black women. *Journal of Human Hypertension, 18*, 79-84. doi:10.1038/sj.jhh.1001639

Shankoff, J. P., Garner, A. S., Siegel, B. S., Dobbins, M. I., Earls, M. F., Garner, A. S., & ... Wood, D. L. (2012). The lifelong effects of early childhood adversity and toxic stress. *Pediatrics, 129*(1), e232-e246. doi:10.1542/peds.2011-2663

Shin, L. M., Rauch, S. L., & Pitman, R. K. (2006). Amygdala, Medial Prefrontal Cortex, and Hippocampal Function in PTSD. In R. Yehuda, R. Yehuda (Eds.), *Psychobiology of posttraumatic stress disorders: A decade of progress* (pp. 67-79). Malden: Blackwell Publishing.

Speisman, J. C., Lazarus, R. S., Mordkoff, A., & Davison, L. (1964). Experimental reduction of stress based on ego-defense theory. *The Journal of Abnormal and Social Psychology, 68*(4), 367-380. doi:10.1037/h0048936

Stratakis, C. A., & Chrousos, G. P. (1995). Neuroendocrinology and pathophysiology of the stress system. *Annals of The New York Academy of Sciences, 771*, 1-18. doi:10.1111/j.1749-6632.1995.tb44666.x

Taylor, S. E., Lehman, B. J., Kiefe, C. I., & Seeman, T. E. (2006). Relationship of Early Life Stress and Psychological Functioning to Adult C-Reactive Protein in the Coronary Artery Risk Development in Young Adults Study. *Biological Psychiatry, 60*(8), 819-824. doi:10.1016/j.biopsych.2006.03.016

Taylor, S. E., Lehman, B. J., Kiefe, C. I., & Seeman, T. E. (2006). Relationship of early life stress and psychological functioning to adult C-Reactive protein in the coronary artery risk development in young adults study. *Biological Psychiatry, 60*(8), 819-824. doi:10.1016/j.biopsych.2006.03.016

Taylor, S. E., Lerner, J. S., Sage, R. M., Lehman, B. J., & Seeman, T. E. (2004). Early environment, emotions, responses to stress, and health. *Journal of Personality, 72*(6), 1365-1393. doi:10.1111/j.1467-6494.2004.00300.x

Tomaka, J., & Blascovich, J. (1994). Effects of justice beliefs on cognitive appraisal of and subjective physiological, and behavioral responses to potential stress. J*ournal of Personality and Social Psychology., 67*(4), 732.

Tomaka, J., Blascovich, J., Kelsey, R. M., & Leitten, C. L. (1993). Subjective, physiological, and behavioral effects of threat and challenge appraisal. *Journal of Personality And Social Psychology, 65*(2), 248-260. doi:10.1037/0022-3514.65.2.248

Vythilingam, M., Heim, C., Newport, J., Miller, A. H., Anderson, E., Bronen, R., & ... Bremner, J. D. (2002). Childhood trauma associated with smaller hippocampal volume in women with major depression. *The American Journal of Psychiatry, 159*(12), 2072-2080. doi:10.1176/appi.ajp.159.12.2072

Ward, C. (1988). Stress, coping and adjustment in victims of sexual assault: The role of psychological defense mechanisms. *Counselling Psychology Quarterly, 1* (2-3), 165–178.

Watson, D., & Clark, L. A. (1994). *The PANAS-X: Manual for the positive and negative affect schedule-expanded form*. Iowa City: University of Iowa. Retrieved from https://www2.psychology.uiowa.edu/faculty/clark/panas-x.pdf

Weniger, G., Lange, C., Sachsse, U., & Irle, E. (2008). Amygdala and hippocampal volumes and cognition in adult survivors of childhood abuse with dissociative disorders. *Acta Psychiatrica Scandinavica, 118*(4), 281-290. doi:10.1111/j.1600-0447.2008.01246.x

Williams, L., O'Carroll, R. E., & O'Connor, R. C. (2009). Type D personality and cardiac output in response to stress, *Psychology & Health, 24*(5), 489-500, doi: 10.1080/08870440701885616

Zimmerman, F., & Mercy, J. A. (2010). A better start: Child maltreatment prevention as a public health priority. *Zero to Three, 30*(5), 4-10.